CATH LAB
PRACTICALS

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Indian Heart Journal

Forewords

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Dedicated to

My Father, Mr OP Mishra who has been my "Role Model" and my Mother Mrs Shakuntala Mishra who is my "Spiritual Mentor"

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Foreword

Although percutaneous coronary intervention (PCI) is considered as a safe procedure, it is not free of complications. Complications in the cardiac catheterization laboratory can be attributed to the condition of the patient upon arrival to the catheterization laboratory (cath lab) or to the technical aspects of the actual procedure. The patient-level complications can be attributed to the state of the disease, for example, patients with cardiogenic shock, acute myocardial infarction, or stent thrombosis pose a higher risk for procedural complications, such as cardiac death, bleeding, and arrhythmias, when compared to stable patients undergoing elective PCI. This complex patient subset frequently requires more intense monitoring, often an anesthesia consult and hemodynamic support with pressors or devices, such as an intra-aortic balloon pump or Impella, to secure a safe procedure.

Other measures that need to be addressed prior to the PCI to avoid complication are the optimization of the anticoagulation protocol; appropriate selection of access for the procedure, either radial or femoral; and the equipment that will be used during the PCI. Procedural PCI complications are often related to the operator technique and skills; to the nature of the lesion, calcified, torturous, or thrombotic; and to the device performance, including malfunction or misuse. These complications can lead to catastrophic events in the cath lab, such as vessel perforation, spiral dissection, distal embolization, and the no-reflow phenomenon. Device-related complications could be attributed to broken wires, deformed stents, stripping stents from the balloon, stuck balloons, rotablator burrs, etc.

Clearly planning ahead of the procedure and having the right equipment and back-up to perform the procedure are essential to minimizing complication rates. Bailout of complications in the cath lab is an art in itself, and although one complication during the procedure can be forgiven, two or more sequential complications cannot. The manual on cath lab complications focuses primarily on the procedural-related complications and is a useful guide to gain familiarity with the options and the modalities to reduce the complication rate and to treat the complication safely if it occurs. The best way to take care of complications is to avoid them, and this can be achieved with proper preparation of the procedure components—the operator, device, patient, and lesion.

Among the most common complications in the cath lab are access site complications, which result in a higher bleeding rate. But with the migration

of access from femoral to radial, the rate of vascular-related complications has been declining. Radial access, however, is not free of complications. Once the complication occurs, it is imperative to identify the complication and to treat it as soon as possible, even at the expense of differing the planned procedure, and even if the complication does not seem to be life threatening. Each device has its own potential complications, and the operator needs to be familiar not only with the use of the device but also with managing the complications that the device may cause.

One approach to minimize PCI complications is to shorten the procedure time. Staging the procedure should be considered, and it is important to know when to stop if things are not going as planned. Usually, when one strategy or device does not perform as planned, it is important to not force it on the vessel and to change the device or strategy or to abort the procedure, which is still better than experiencing the complication. Managing complications is a team effort, and therefore once a complication is encountered, it is wise to call for help and the rest of the team, including nurses, technologists, experienced operators, and an anesthesiologist. Time is of the essence when treating complications, and the more time that passes, the worse the outcome. Other noncardiac but procedural-related complications that may impair patient outcomes are induced contrast nephropathy, radiation exposure, and burns. Risk assessments of the procedure and risk adjustment are essential for planning and reporting the rate of complication per institution, especially as we move to public reporting.

Finally, we should remember that as long as we perform PCI, we will encounter complications. Therefore, learning how to bailout from these complications, and how to manage them safely is as important as knowing how to perform safe PCI. This manual is a useful educational tool to get you and your patient safely through the procedure, even if it gets complicated.

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Foreword

I fly frequently. Dividing my time between the Eternal Heart Care Centre (Jaipur) and the Mount Sinai Hospital (New York), I rely on commercial airlines. Boarding each flight, I expect to lift-off, proceed to my destination, and land successfully, with minimal discomfort and no complications. Whatever turbulence may arise in the course of travel, I expect my plane's captain and crew to have the proper training, technology and temperament to manage it.

Our patients expect no less from us. As percutaneous coronary intervention has matured, procedural success has soared toward 98–99% and complication rates have plummeted for even the most complex cases. As experience grows and equipment further evolves, ambitions are similar.

The difference between good and ideal is measured in how we handle the tough cases. In the *Cath Lab Practicals*, Professor Sundeep Mishra and his team have taken on the ambitious task of preparing interventional cardiologists for the quandaries, challenges and emergencies that can imperil success and safety in the cardiac cath lab.

With wit, savvy, clinical examples and a touch of philosophy, Professor Sundeep Mishra and his colleagues cover a broad array of potential problems in the course of coronary and structural intervention. Interventional cardiologists in practice and in training, nurses, technicians and staff would do well to digest these highly readable chapters, which detail solutions to challenges ranging from the rare (device embolization, Chapter 2) to the routine (problems with guide support, Chapter 7). By meeting percutaneous coronary intervention's most feared complications head on, the text helps demystify its most technically demanding procedures. In review of complications of rotational atherectomy (Chapter 10), for example, the authors comprehensively explore the terrifying event of burr entrapment and provide practical options for management.

"The key is not just to know," the authors write, "but to know that you know." The confidence to embark on the most complex interventional procedures grows from a comfort in one's ability to manage even the most dire complications. With such confidence, we are more likely to give the best, we have to offer to the patients who need it most. Professor Mishra and colleagues are to be congratulated on this textbook that will help all members of the interventional team feel more confident that they

can deliver the best possible interventional care, even when the going gets tough.

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Preface

Since renaissance, science has evolved as a body of empirical, theoretical, and practical knowledge about the natural world, produced by scientists who emphasize the observation, explanation, and prediction of real world phenomena. The method applied is differentiation and randomized controlled trials (RCTs) which have now become the gold standard for causal inference in medicine. However, to observe a difference, there are essentially three requirements: presence of control, sufficient number of participants to bring out a meaningful difference, and finally, a stable environment to study the difference. However, what to do when none of these conditions are satisfied? For example, cath lab complications: neither there is a control, nor sufficient numbers occurring predictably over a course of time, nor a stable environment!

The situation is somewhat akin to a "Fighter Pilot" although the essential difference here is that operator's life is not at risk (unlike the fighter pilot). Thus, to take a cue from aviation profession where procedural know-how has been over the years extensively externalized, verbalized and documented. It is verbalized not only in clearly structured instruction manuals formulated over declarative knowledge, i.e. on technical and scientific aeronautical data but also incorporated into virtual reality aviation simulators equipped with sophisticated board computers, FMS, programmed to mimic variety of real-life scenarios. Thus, throughout the training, pilots learn to master the knowledge; proceeding from simple to routine to unexpected scenarios. These established teaching processes assure objective assessments of achieved levels of professional competence by all trainees across the board independently from local circumstances and dispositions. Recently, based on cognitive research, it has been shown that acquisition of know-how may be enhanced by providing the trainees with additional contextual data embedded into concrete tasks.

However, in contrast to the field of aviation, the percutaneous coronary interventions (PCI) procedural knowledge has not been systematically verbalized, and has remained so despite over 30 years of clinical PCI practice (perhaps because of rapid changes in PCI technology). Thus, while textbooks on PCI typically beam with evidence-based data derived from numerous devices-driven trials, the fundamental cognitive processes required for the actual PCI performance are scanty; however, the "tips and tricks" which seem essential to procedural skill transfer, cover only a tiny corner in the huge PCI decision space and are by far not enough to provide for the needs. The efficacy of traditional "trainee-mentor" knowledge transfer is highly dependent on ability of trainees to perceive and mentors to explain and to demonstrate; marked heterogeneity of professional PCI competence result. With this view, we embarked upon this manual to build up the collective memory of cases, to

develop cognitive teaching programs based on retrieval of expert knowledge rather than mere mentor-guided empirical approach, to try build a mental library of cases, to try to begin to close the gap between the training modus in aviation and PCI. In other words, develop a "Cath Lab Manual" akin to aviation manual.

Sundeep Mishra

You take the red pill – you stay in Wonderland, and I show you how deep the rabbit hole goes

Acknowledgments

Guru Govind dou khade kaake lagoon paay I
Balihari guru aapne Govind diyo batay II

If both, Guru and God in form of Govind were to appear
at the door, whose feet will I worship first?
It has to be the Guru's feet first, because without him,
how would I have recognized (known) God?

First of all, I would like to thank all my teachers who brought me to a level, where I am able to write a manual. In particular Professor VK Bahl who is also my mentor and my guide as also Professors SC Manchanda, SS Kothari and of course Ron Waksman who played down my limitations, my mistakes and my weaknesses to guide me all along.

I would also like to acknowledge all the contributors to the cases and chapters.

I would like to thank the Director of AIIMS, Professor MC Misra (Center Chief), Professor Balram Airan (Head), all my colleagues and staff, All India Institute of Medical Sciences (AIIMS), New Delhi, India, Cardiological Society of India-National Interventional Council (CSI-NIC) and all my well-wishers.

I am also deeply indebted and grateful to my wife Deepti Mishra, who not only sacrificed herself so that I could complete this monumental task but also encouraged me to do it, and gave me practical advice all along. Without her help, this work would not have seen "Light of the Day." I would also like to thank my kids—Gargi Aditi and Badri Vinayak for allowing me to "sit on the computer".

Contents

Chapter 1

Slippery when Wet: Broken Guidewire, Ruptured Balloon in Cath Lab

Sundeep Mishra, Gautam Sharma

INTRODUCTION

Fractured guidewire, ruptured balloon and intra-procedure damage to other catheterization laboratory hardware are not rare complications of percutaneous coronary interventions (PCI) and can have potentially catastrophic outcomes.[1,2] With improved angioplasty hardware particularly development of more flexible and high-quality guidewire this complication might be expected to decrease. However, the risk of wire entrapment has remained same or perhaps even increased because physicians are now approaching more complex lesions. The damaged material if left *in situ* can have an innocuous outcome or it can lead to thrombosis and ischemic occlusion or it can even embolize leading to thromboembolic complications.[3] Attempts to correct the situation by force can lead to worsening of the problem, e.g. unraveling of the coiled tip of the wire, progressing to formation of knot and finally possibility of knot detachment and embolization.[4] Thus treatment has to be individualized and may vary from masterly inactivity to retrieval to cardiac surgery.[5] The key to addressing this problem like any other complications in cath lab lies in 3 "**A**s": **A**nticipation, **A**voidance and **A**melioration of the complication once it occurs.

FRACTURED GUIDEWIRE

Etiology

Fractured guidewire can be a consequence of:

- Inherent faults in design or in the manufacturing process of the guidewire or reused wire (Case 1).
- Wire entrapment in a side-branch while stenting main branch or wire wedging into a distal or winding vessel.
- Wire damage by rotational or directional atherectomy catheter.

Management

Anticipation

This problem can be anticipated while using reused material, during certain anatomies like bifurcation (when main vessel is stented while keeping wire

jailed in side-branch), calcific lesions or tortuous lesions and with certain technical procedures like rotablation[6] **(Table 1.1)**.

Avoidance

While performing main vessel stenting, the wire in side-branch should be placed deep in so that coil part of guidewire (nonmetallic tip) should not be in contact with main vessel undergoing provisional stent. Hydrophilic or polymer coated wire should not be used in side-branch and finally, jailed wire technique should not be used in some 2 stent techniques like crush technique **(Table 1.1)**.

Amelioration

The treatment of this complication once it appears is unclear and should be based on the situation. Any hardware retained in the lumen of a vessel may serve as a nidus for endothelial injury and platelet deposition, putting the vessel at risk for acute thrombosis. On the other hand, vigorous attempts

Table 1.1 Anticipation and avoidance of guidewire fracture

Structural issues
• Hydrophilic or polymer coated guidewires should not be used in side-branch
• Firm tipped guidewires should be avoided in very complex lesions
• Manufacturing defects should be looked for
• Reused material should be avoided
Clinical issues: Extra care required in
• Bifurcation lesions especially side branch being tortuous or arising at angle 90° or more
• Tortuous and calcified lesion
• Chronic total occlusion
• In-stent restenosis
• Extensive atherosclerosis especially in main vessel which can cause plaque shifting in main vessel or side branch resulting in wire trapping
• Kinky, segmented, irregularly shaped main vessel, wherein stent implantation can transmit greater radial force on the trapped wire
Technical issues
• More careful use of rotablator and directional atherectomy catheter
• Imparting sharp curve to the guidewire tip should be avoided
• Use of multiple guidewires should be avoided
• More care while performing interventions through multiple stent struts
• Extensive maneuvering with balloon and other hardware should be avoided
• Prolonged procedure time should be avoided

Table 1.2 Management strategies of guidewire fracture

- Masterly inactivity—Leaving the guidewire alone if it is in small side-branch and difficult to extract
- Guidewire retrieval
  - Use of paired wires to make a knot and subsequent withdrawal of the knot
  - Use of commercial or homemade snares
- Fixing the fractured remnant to coronary bed by a balloon or stent
- Mobilization or fixing to small side-branch
- Surgical mangement
  - Removal of guidewire
  - Removal accompanied by end-arterectomy and/or graft anastomosis

to remove the material can, by itself, cause a trauma to the vessel and prove counter-productive. Further, another device used to retrieve it can itself get trapped. Thus, the treatment options may vary ranging from masterly inactivity, percutaneous extraction of the guidewire, balloon dilatation or stent implantation over the guidewire remnants and finally surgical management **(Table 1.2)**.

Masterly Inactivity

Guidewire remnants can be left alone in a small side-branch if it is not obstructing flow and the original procedure has not damaged the vessel wall of the side-branch. These patients may be carefully followed up on systemic anticoagulation.[7] This strategy may be especially useful with hydrophilic or nonmetallic remnants (which are less likely to thrombose). However, the coronary segment which contains these fragments tends to develop progressive stenosis and therefore this strategy may not be useful in hemodynamically significant vessels.[8]

Guidewire Retrieval

Retrieval represents perhaps the most effective and popular strategy to remove entrapped material, particularly in a proximal segment.[9] However, the success rate of this strategy may be lower in a very tortuous vessel and with very mobile fragments. Guidewire retrieval may involve a simple procedure like entangling the fractured guidewire with another guidewire, making a knot together and subsequent withdrawal of the knot. However, commonly guidewire retrieval is carried out using a homemade or commercially available snare catheter using balloon as a wedge to extract damaged guidewire remnants. Details of use of snares for extraction of catheterization laboratory material are discussed in a greater detail in Chapter 2.

Crushing onto Coronary Bed

Mobilizing and fixing the guidewire fragments to coronary bed is an option when the guidewire fragment has completely detached. Hydrophilic and nonmetallic or polymer coated fragments are less thrombogenic and being slippery it is easier to mobilize.[10] Small and underinflated balloon catheters can be used to drag these fragments and lodge them to the vessel wall. Finally, the fragments can be isolated from vessel lumen by stenting over it. This strategy also takes care of damaged vessel wall likely during the index procedure. Any type of stent, bare-metal stents (BMS), drug-eluting stents (DES) or a covered stent can be used but a covered stent should generally be avoided unless there is accompanying perforation or very extensive vessel damage (Case 1).

Surgery

Surgery is the last option for these patients and should be reserved for those patients where guidewire entrapment occurs in a hemodynamically significant vessel with flow limitations and percutaneous removal of the fragments has either failed or considered too risky (entrapment into left main). Isolated removal of trapped guidewire can be attempted but often the guidewire is so firmly embedded in the vessel wall that some kind of endartectomy would be required. In those cases where the percutaneous procedure has resulted in too much damage to the vessel, graft anastomosis may be the only safe option.

CASE EXAMPLE

Case 1

Case of bifurcation lesion: Left anterior descending (LAD)-Diagonal. Provisional stenting strategy was planned. A floppy Zinger™ guidewire placed in LAD and a reused guidewire placed in diagonal branch. Following LAD stent deployment the jailed diagonal wire got entrapped between the vessel wall and LAD stent. While attempting to pull out the entrapped guidewire the guide-catheter was getting sucked in the left main repeatedly. Ultimately there was unraveling of the guidewire into microfilaments. Attempts were made to entangle the damaged guidewire filaments by 2 guidewires but they were not successful. Finally, an attempt was made to snare the microfilaments in toto into the guiding catheter but the damaged guidewire completely snapped with damaged and broken filaments floating into, LAD, left main and even proximal aorta. Patient developed slow flow which was initially managed by nitroglycerin and an extra bolus of heparin. At this point it was decided to crush the damaged guidewire and a new stent was deployed over it in left main crossing over to LAD, overlapping with the original stent. The stent was post dilated particularly at the ostium of left main

and also at the overlapped segment. Some filaments were allowed to remain in ascending aorta. Patient was discharged on dual antiplatelet therapy and low dose warfarin (INR maintained at 2). On follow-up at 4 months the stents were found patent and echocardiography demonstrated microfilaments and a few protruding stent struts out of left main into ascending aorta. On late follow-up patient remains free of angina, stress test remained negative.

Analysis of the case: There were several issues: (1) A reused guidewire was used in the side-branch (more liable to break). (2) A lot of force was applied to pull out the trapped wire as evidenced by sucking in of guide-catheter. In this case, retrieval is the best strategy which was indeed tried, first with entanglement technique and later by use of a snare. But again perhaps the force applied to pull was too aggressive which led to breaking of the wire and unraveling the microfilaments in left main. Finally, the exposed wire parts were crushed on left main which seems to be right strategy at this point. Some filaments were floating in aorta, but extra-coronary fragments, even embolization is generally benign and therefore a strategy of not intervening upon them as pursued here was the right approach. A strategy of putting the patient on triple antiplatelet therapy in this case can be debated but perhaps operators felt more safe with this approach.

NONDEFLATION OF BALLOON

Balloon deflation failure is a serious and sometimes catastrophic complication of PCI procedure. The cause could be a faulty balloon material or a reused balloon but it could also be due to inappropriate balloon preparation (use of high dye concentration vis a vis saline during preparation of diluted contrast for balloon inflation).[11]

Management

Standard deflation does not work because of development of valve mechanism inside the balloon. However, it is important not to panic and pull the inflated balloon into proximal vessel as it can lead to vessel injury, dissection or even rupture of the vessel (Case 2).

- Nondeflating balloon can be inflated at a higher pressure and ruptured but it carries a risk of vessel injury, dissection, perforation and even vessel rupture and also possibility of air embolism.
- Puncturing the balloon with a hard end of regular guidewire or using a microcatheter guided penetration with a penetrating wire like Conquest Pro may be a better solution.
- Use of laser wire for perforating the undeflated balloon is another option.
- Repeated inflations and deflations with distilled water can also sometimes lead to success in deflating the balloon.
- In some cases surgery may be the final option.

CASE EXAMPLES

Case 2

A 70-year-old lady with past history of AMI (6 months back) presented with class II angina on exertion and mild left ventricular dysfunction. Coronary angiography revealed critical stenosis in proximal LAD, distal circumflex and mid RCA with a SYNTAX Score of 19. Patient was taken up for multi-vessel PCI via right radial approach. Left coronary artery was hooked with 6 F XB 3.0 guiding catheter, lesion crossed with ACS Hi Torque™ Floppy wire and LAD directly stented with 3.5 × 21 mm Paclitaxel Stent (deployed at 14 atm.). Immediately after stent deployment patient started having chest pain with bradycardia and hypotension. Fluoroscopy revealed nondeflated proximal portion of balloon with no flow in LAD. Repeated negative suction with indeflator resulted in partial deflation of balloon but still no flow in artery was restored. In view of hypotension and bradycardia, IV Atropine was given and IV dopamine was started. A gentle tug was given to the balloon so as to pull balloon in guide-catheter and re-establish flow. However, when balloon was attempted to be pulled back only part of inflated balloon shaft came out within guide catheter while ruptured broken balloon fragment was left behind. This led to development of no flow in LAD. Since snare was not immediately available in catheterization laboratory, it was decided to inter-twine the broken balloon shaft with two guidewires. A new BMW™ wire was passed in the vessel alongside the broken ruptured balloon catheter and inter-twined with pre-existing wire and whole assembly attempted to be withdrawn in the guide-catheter. However, the above maneuver was un-successful in balloon withdrawal but on the other hand resulted in loss of first wire from balloon. No flow in LAD persisted. A second 2.5 × 10 mm balloon was introduced over the second wire in distal portion of vessel beyond ruptured balloon and inflated at 14 atm **(Fig. 1.1)**. A gentle tug was given which resulted in withdrawal of both balloons (ruptured balloon and the new balloon) into the guide-catheter. Post balloon withdrawal flow was established in LAD and patient settled hemodynamically, the whole sequence lasting 8 minutes. The procedure was completed by postdilating LAD stent with 3.5 × 15 noncompliant balloon. Examination of ruptured balloon revealed that the balloon had broken at monorail shaft junction, the usual site of balloon catheter rupture.

Analysis of the case: Probably, the saline:contrast ratio was not optimal to begin with. Secondly, more aggressive approach was used to deflate the balloon. Finally, attempt was made to retrieve the broken fragment. Initially two wire technique was tried but when that was not successful, pulling inflated balloon beyond the broken fragment and pulling back worked.

Case 3

A 50-year-old gentleman presented with non ST elevation MI, Trop T was positive. Echocardiography revealed mild basal anterolateral hypokinesia

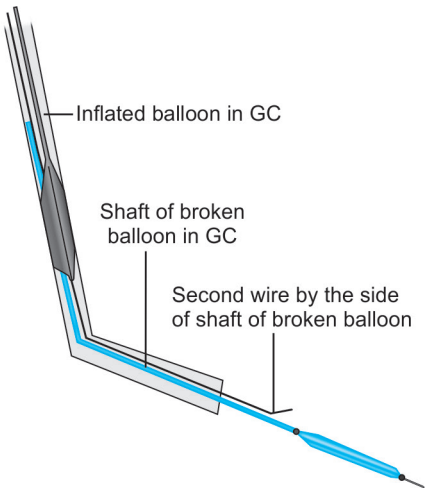

Fig. 1.1 Cartoon demonstrating proximal inflated fragment of balloon catheter in guide-catheter, ruptured broken distal fragment of balloon catheter in the vessel and a new guidewire passed alongside

Courtesy: Dr Shantanu Deshpande

with overall normal ejection fraction. Coronary angiography revealed tight LAD lesion and total occlusion of left circumflex. Patient was taken up for multivessel PCI. LAD was stented with 2.75 × 30 mm DES and the lesion postdilated with reused 3.5 noncompliant balloon. However, balloon failed to deflate and patient started getting chest pain and systolic BP dropped from 130 mm Hg to 90 mm Hg. Indeflators were changed and attempts to aspirate with 10 cc syringe failed. Saline contrast mixture was then changed to normal saline. Attempts to rupture balloon using hard end of wire were made but failed. Patient now was having severe pain and needed sedation. As a last ditch attempt balloon was inflated at very high pressure 30 atm after confirming availability of Stent Graft on shelf. Balloon got ruptured and flow was established and there was no dissection or perforation. Inspection of balloon revealed that part of the balloon catheter was missing. Attempts to entangle the damaged balloon flap with two twisted wires and remove them failed. There was brief movement of portion of balloon into left main but it migrated back to proximal LAD. Finally, the damaged balloon portion was crushed in the LAD vessel wall by a 4 × 13 mm stent. Patient did well after stent implantation with, no further pain and is doing well almost into 6 months of follow-up.

Analysis of the case: Again, probably, the saline:contrast ratio was not optimal to begin with. Trying high pressure inflation could have been dangerous because it could have ruptured the vessel (stent graft would have worked for perforation but not ruptured vessel). Maybe trying to puncture the balloon was a better strategy but perhaps there was not enough time to do it. It is very

important to carefully inspect the retrieved hardware in a case like this and indeed it revealed that the part of it was left behind. The ideal approach is to retrieve the remaining fragment but if not possible an alternate strategy of crushing it to the vessel wall may be mandatory.

BALLOON RUPTURE

Balloon rupture is a common but generally innocuous complication of PCI. However, rarely the complication can be catastrophic. The complications encountered may be of 2 types: Injury to integrity of balloon catheter and injury to adjacent vessel. Damage to vessel wall as a result of balloon rupture can be easily understood. Complications pertaining to lack of balloon integrity relate to difficulty in removing the damaged balloon catheter without injuring the vasculature and without embolization. Occasionally the catheter can get embedded in the vessel wall creating difficulties in removal.

Etiology

There are 3 types of rupture that can happen during dilatation with balloon catheter.[12]

1. *Circumferential rupture*: This occurs due to over-dilatation of the balloon, generally substantially beyond the recommended burst pressure. Typically, balloon rupture may occur with ≥50% diameter overdilation (compared to vessel size) but vessel rupture mostly occurs with only ≥100% over dilation **(Fig. 1.2)**.[13]
2. *Horizontal rupture*: This can occur when there is structural defect in the balloon catheter due to defective manufacturing process. The balloon in this situation can rupture even before rated burst pressure (RBP).

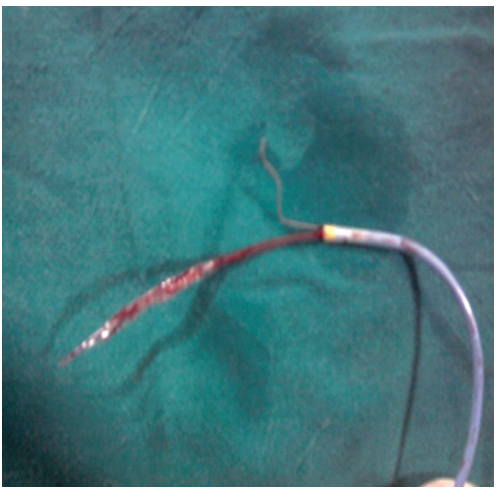

Fig. 1.2 Circumferential rupture of the balloon catheter

Courtesy: Dr Shantanu Deshpande

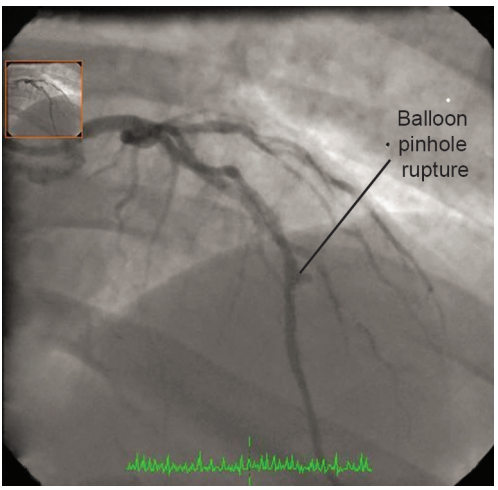

Fig. 1.3 Balloon pinhole rupture

3. *Pinhole rupture*: This is a localized rupture, which can occur in the balloon due to injury by a calcified spicule or an exposed stent strut. The balloon can rupture even before RBP **(Fig. 1.3)**.

Management

- Avoidance of balloon rupture
- Avoid using reused balloon
- Try not to go beyond rated burst pressure
- Push balloon before retrieving it
- Do not pull out the balloon if resistance is faced, rather give a gentle torque.

Trauma Inflicted Upon the Vessel Wall

This is classic "Egg first or Chicken First" debate. An elegant experimental study by Zollikofer et al. demonstrated that arterial rupture always preceded balloon rupture.[13] Thus horizontal rupture with its underlying structural defect may be completely innocuous, whereas in circumferential rupture there may be injury to all layers of the vessel caused by severe over-distension, followed by secondary rupture of the balloon because it had lost its external support. Thus, it is only balloon pin-hole rupture with subsequent jet effect wherein balloon rupture *per se* can cause damage to the vessel wall. Most of the complications due to balloon rupture can be managed conservatively. Occasionally, type 3 perforation is caused by balloon rupture may require placement of covered stents or even surgery Case 4.

Complications Associated with Retrieval of Ruptured Balloon Material

Management of ruptured balloon material has same underlying principle like other entrapped hardware including guidewire fracture. While in most cases ruptured catheter can be withdrawn without difficulty at the end of withdrawal it is very important to carefully inspect the balloon catheter to make sure that all the fragments have been recovered. Even if the balloon can be retrieved easily, intimal tears are very common and may occur in nearly 1/3rd of cases.[14] In some situations the balloon material can get fragmented and may require complex retrieval techniques mentioned earlier. In some cases, the fragments can be crushed to the side-wall by a stent Case 5. Rarely, however, even surgery may be required.

CASE EXAMPLES

Case 4

A 69-year-old gentleman presented with acute inferior MI with a window period of 6 hours with ongoing chest pain. He was hypertensive, diabetic and a chronic smoker. Coronary angiography revealed RCA lesion. Lesion was predilated and subsequently stented with 3 × 18 DES deployed at 12 atm. However, the stent balloon ruptured and post stent patient developed chest pain, hypotension and unfortunately the guidewire was also lost. Cine shoot revealed proximal dissection. The vessel was rewired and stented with 3 × 23 BMS and suitable post dilated. Finally, good result.

Case 5

A 65-year-old male presented with complaint of chest pain from last 5–6 days. ECG demonstrated ST-T changes in lateral leads. Troponin – T was negative. Echocardiography was essentially normal. Coronary angiography revealed LCX was a normal vessel but OM 1 showed tight 90% lesion. PCI of OM lesion was planned via right radial approach. Left coronary hooked with 6F EBU 3.5 catheter and OM lesion crossed with 0.014 Choice PT™ Extra support guidewire and predilated with 2.0 × 10 mm reused balloon. However, while withdrawing balloon shaft gave away with balloon in LCx. Lesion recrossed with another 0.014′ guidewire. Both guidewires were entangled with each other and pulled out simultaneously thus withdrawing the broken balloon along with the guidewires. Finally, good result.

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Chapter 2

If You Break it—You Own it: Embolization of Cath Lab Hardware

Sundeep Mishra, Ranjit K Nath

INTRODUCTION

Device loss is defined as dislodgement of a device inside the body at a location different than the intended delivery location. It is a rare but potentially fatal complication in catheterization laboratory especially if it happens within the coronary tree. Any of the interventional hardware can break and get embolized, guide-catheter, guidewire/rota wire, balloon catheter shaft, or other types of catheters and even arterial sheath but loss of a stent material is the most common. In the era of hand-crimped stents the rate of embolization was to the tune of 3–8% but contemporary studies utilizing balloon mounted stents demonstrate a much lower incidence (<0.5%) ranging from 0.27% to 0.33%.[1,2] With improvement in mounting techniques and stent design this rate may be even lower. Usually the embolizations are clinically apparent especially if they are in coronary circulation but with newer stents with lower strut thickness and lower radio-opacity the embolization may completely be missed acutely and may become apparent only later on.[3]

STENT EMBOLIZATION

Stent embolization is the most common type of embolization encountered in catheterization laboratory. The stent can be embolized as a fractured fragment or in toto, unexpanded or partially expanded. Several lesion, procedural and mechanical characters are identified which can contribute to stent embolization **(Table 2.1)**.

The clinical outcome of stent embolization depends on site of embolization. Intracoronary embolization is generally symptomatic (more than 2/3 of cases) and can be potentially fatal. It can present as myocardial ischemia, coronary thrombosis and subsequent myocardial infarction and even death.[4] In those cases where dislodged stent cannot be managed percutaneously it may require coronary artery bypass grafting (CABG).[5] Rarely stent loss in coronary vasculature can be completely asymptomatic.[3] On the other hand, extra-coronary stent loss is associated generally with minimal clinical sequelae (except limb ischemia in some cases), however, embolization in ascending aorta may sometimes cause severe acute cerebrovascular events.

Table 2.1 Factors predisposing to stent embolization

Lesion characteristics
Lesion angulation >45°
Tortuous lesions
Moderate or severe calcification
Distal location
Passage through previously deployed stent
Procedural characteristics
Direct stenting
Inadequate lesion preparation
Failure to cross a lesion with subsequent withdrawal of an undeployed stent into the guide lumen
Stenting of ostial lesions
Mechanical characteristics
Manually crimped stents
Coil stent design
Stents with low flexibility

Table 2.2 Prevention of embolizations in catheterization laboratory

Careful selection of hardware
Avoiding using reused material
Guide-catheters with increased support
Extra-support guidewires
Careful attention to technique
Predilatation before stent implantation
Proper preperation of balloon catheter
Proper preperation of vessel—Rota or cutting ballon if lesion calcific
Deploying distal stent before proximal stent

As with other complications in catheterization laboratory most of the stent embolizations are preventable. Prevention involves careful assessment of the lesion characteristics and sound application of technique starting with proper selection of guide catheter and guidewire to careful preparation of vascular bed before stent implantation can prevent embolization[6] **(Table 2.2)**. Management of stent embolization depends on whether the stent is embolized in coronary or extra-coronary circulation. If embolized into coronary vasculature it requires to be managed by redeployment, retrieval or being effectively sandwiched against the vessel wall **(Table 2.3)**. The strategy chosen depends on whether the embolized stent is still ***'riding the guidewire'*** or not. The small-balloon technique is the most common technique used

Table 2.3 Management of maldeployed/embolized stent/stent fragment within coronary circulation

If stent is riding the wire
Redeployment
• Small balloon technique
Retrieval
• Guidewire entangle technique
• Other retrieval techniques
If stent is not riding the wire
Redeployment
• Stent re-cross technique
Retrieval
• Balloon catheter technique
• Two wire technique
• Hairpin trap technique
• Homemade loop technique
• Small loop snares
Amplatz Goose Neck™ snare (ev3)
Microsnare Elite™ (vascular solutions)
En Snare™ (merit medical)
• Standard loop snare
• Multipurpose basket
• Biopsy forcep
Endomyocardial biopsy
Biliary biopsy
Crushing
CABG

when a stent is dislodged but guidewire access is maintained through the lost stent. A small balloon is advanced through the stent, inflated distal to the stent, and then withdrawn together with the lost stent. The stent is then repositioned at the intended site. If stent cannot be redeployed stent retrieval can be another option.

On the other hand if stent is lost from guidewire the most logical strategy is either some kind of retrieval maneuver or crushing strategy. In rare cases partially deployed stent can be recrossed with another guidewire (making sure that it does not cross through the stent strut) and fixed by a partially inflated balloon and redeployed at the correct site.[7] For retrieval of stent or stent fragment several strategies may be attempted, low profile balloon angioplasty catheters, double (entangling) guidewire, "homemade

loop snares", myocardial/biliary biopsy forceps, gooseneck/standard (large loop) snares or several types of retrieval baskets **(Table 2.2)**. In the ***Balloon Catheter*** technique a wire is passed by the side of dislodged stent. A balloon catheter is threaded over this new wire and passed beyond the distal part of stent. The balloon is then partially inflated and the stent may by dragged with this balloon into the guide-catheter. In ***Guidewire Entangle*** technique, another guidewire is passed beside the dislodged stent beyond its distal part and used to entangle the original guidewire on which the stent is riding. The whole assembly is then retrieved. In the ***Two-wire*** technique, two guidewires are advanced through the struts of the lost stent, and rotated several fold in order to entangle the distal end of the stent.[8,9] After ensuring a complete fixation of the stent, the wires are withdrawn and they may carry the lost device. The ***Hairpin-trap*** technique is another novel strategy that can be used for stent retrieval.[10] In this strategy, a polymer jacketed guidewire tip is bent into a hairpin loop and inserted through the Touhy-borst valve until it exits through the guiding catheter's distal end. The hairpin is then advanced through the distal end of the lost stent and then withdrawn, "hooking" the stent through the now-open hairpin. The free tip of the guidewire is then wired back into the guiding catheter and a small balloon is inflated within the guide catheter tip, adjacent to the reinserted guidewire to fix the entire system. The entire assembly is then withdrawn, removing the lost stent. In ***Homemade Loop*** technique snares can be fashioned in the catheterization laboratory by inserting an exchange-length coronary guidewire through a multipurpose catheter and reinserting the distal tip of the guidewire through the distal tip of the catheter.[11] Several custom made loop snares are also available such as the Amplatz Goose Neck™ snare (ev3), the Microsnare Elite™ (Vascular Solutions) and the En Snare™ (Merit Medical) and they may be useful for facilitating retrieval **(Fig. 2.1)**. Typically, these snares consist of a wire loop made of nitinol that is advanced through a diagnostic/guide catheter (use of micro catheter can give more control) and positioned around the lost device. It is then pulled back, trapping the device against the catheter. The catheter/loop assembly is subsequently removed from the body, along with the lost device. Microelite™ snare is 0.014′ in diameter, has loop size of 2–7 mm, is 180 cm in length and does not require a delivery catheter. The En Snare™ is also a specially designed loop snare made with 3 overlapping loops (2 mm), thus increasing the likelihood of retrieving the lost device. The stent may also be caught in a percutaneously delivered basket and retracted. Finally, it may be caught by endomyocardial or biliary biopsy forceps and retrieved.

While stent retrieval is intuitively the logical management approach to a lost stent, it is unpredictable and even potentially dangerous and may culminate in distal stent embolization or target vessel injury. Further, stent retrieval can prolong the procedure and increase radiation exposure. In this context, stent crushing in a coronary segment, that is unlikely to be significantly affected by stenting may be the most time-efficient and low-risk strategy. Further, in those situations where either redeployment or retrieval is not feasible the ***Stent Crushing*** technique is the only alternative,

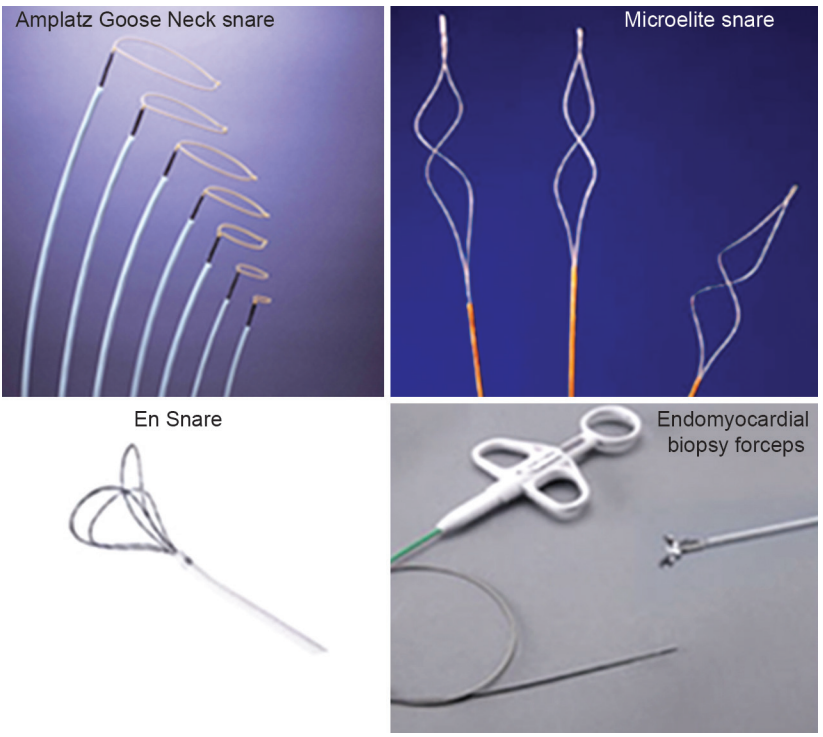

Fig. 2.1 Retrieval devices in catheterization laboratory

as a matter of fact in real world situation the stent crushing strategy is the most effective technique and is certainly most commonly employed. The technique of stent crushing involves crossing with another guidewire distal to the dislodged stent. Another stent/balloon is threaded on this wire and the catheter inflated to crush the embolized stent to the side of the vessel wall. However, it is important to make sure that the guidewire should remain clear of the embolized stent in all circumstances otherwise it may get jailed inside the crushed stent. Thus, before attempting to crush a stent, passing a balloon distal to the stent and withdrawing it in a partially inflated condition will make sure that the wire has not crossed the stent strut and thus the technique can be safely employed.

Overall, in majority of cases percutaneous management is successful in experienced hands (>85%).[5,12] Rarely however, if none of the strategies are successful patient may be offered CABG. While it is not advisable to leave stent/stent fragment in the coronary tree, occasional case reports have described an uneventful outcome. Most of the extracoronary embolizations can be left alone but for those which are symptomatic they can either be retrieved or crushed against the sidewall of the vessel.

CASE EXAMPLES

Case 1

A 72-year-old diabetic, hypertensive lady presented with acute inferior MI (4 hour of chest pain). Her echocardiography demonstrated inferior hypokinesia with overall ejection fraction of 45%. Angiography revealed a triple vessel disease with occlusion of distal RCA. RCA was hooked with 7F JR 3.5 guiding catheter; lesion crossed with All Star™ guidewire and dilated with Voyager™ balloon 2 × 12 mm at 10 atm. Subsequently, TIMI-2 flow was achieved. The proximal 70% RCA lesion was also dilated with same balloon at 14 atm. A 2.75 × 28 mm DES could not cross and so 2.5 × 12 mm was attempted to be deployed at 14 atm. However, the stent got dislodged. A second guidewire was passed and finally the dislodged stent was crushed. It was then attempted to deploy another 2.5 × 28 mm DES, but this stent also got dislodged. Another guidewire was passed and a Voyager™ balloon 2 × 12 mm passed distal to dislodged stent and the crushed stent and whole assembly withdrawn in toto. Finally two DES, 2.75 × 28 and 3 × 30 were deployed with good end result.

Analysis of the case: Probably the lesion complexity was missed and therefore despite predilatation the stent could not cross. Pulling back an undeployed stent after forcibly pushing it is the most common reason for stent dislodgement.

Case 2

A 60-year-old male, presented with angina chest pain. Coronary angiography revealed proximal RCA lesion. Stent was positioned across RCA and during the process of deployment the guide-catheter got accidently pulled before full deployment could be achieved. The stent was attempted to be retrieved by passing a balloon distal to stent and withdrawing the whole assembly, but the attempt was not successful. A 300 cm exchange coronary wire folded upon itself in the center (to make a homemade snare) and introduced via 8F JR guide catheter introduced from the opposite groin. The stent was attempted to be snared by manipulating one end of wire and thus hooking a portion of the stent on the loop of indigenously prepared snare. Once stent was entangled in this homemade snare, whole assembly was withdrawn out of the sheath. Subsequently the culprit lesion was successfully stented.

Analysis of the case: Again pulling back an undeployed stent after forcibly pushing it is the most common reason for stent dislodgement. Once stent is dislodged stent repositioning is the best strategy if stent is still riding on wire. However, in appropriate cases retrieval may be attempted.

Case 3

A patient presented with proximal LAD lesion and CTO of LCx. CTO of LCx was attempted first. The CTO was crossed with Fielder XT™ wire and dilated with semicompliant balloon. The lesion was attempted to be stented but

there was a difficulty in crossing. While attempting to withdraw the stent it got dislodged in proximal LCx and the guidewire was also lost. A new guidewire was put in Left Main–LAD and the stent was recrossed with another Fielder XT™ wire, sequentially dilated with progressively increasing size of balloon catheter and finally deployed in ostioproximal LCX (with some stent struts protruding in Left main). A cross-over stent was deployed from LAD into left Main performing the TAP procedure with LCx stent. The LCx stent was recrossed and final kissing balloon dilatation was performed. An additional; stent was put in mid-LCx lesion.

Analysis of the case: Recrossing and repositioning the embolized stent is the safest strategy but if the stent is recrossed but cannot be repositioned the stent can be even deployed in more proximal part provided stent design allows to be over-dilated.

Case 4

A 55-year-old male, known diabetic, presented with the complaint of angina on exertion NYHA class II. Overall, Echocardiographic LV ejection fraction was 45% with moderate hypokinesia of inferior and lateral wall. He was taken up for coronary angiography which revealed OM1 proximal 90% stenosis, OM2 90% diffuse disease and mid RCA 90% and ostial PLV 90% stenosis. The patient was taken up for angioplasty and stenting in RCA and LCX. After RCA being successfully stented, EBU 3.5 6F guide catheter was taken to deal with OM1 lesion. Lesion was crossed with Cougar XT™ wire and was dilated with Invatec Avion™, balloon 2 × 10 mm. Sirolimus eluting 2.5 × 32 mm stent was taken but it could not be taken across the lesion, so the lesion was once again dilated and buddy wire was taken to support the stent passage across the lesion. In an attempt to pass the stent across the lesion it got dislodged from the balloon in proximal LCX and left main with some part still in the guiding catheter **(Fig. 2.2)**. However, the wire was still across the uninflated stent. A 1.5 mm × 10 mm semicompliant balloon was taken across the stent and it was dilated and this balloon pulled back, to try withdraw the stent into the guiding catheter with the wire still across the lesion. But the balloon instead of pulling the stent into the catheter passed through and through the stent partially dilating it. Subsequently another 2.0 mm × 10 mm semicompliant balloon was taken and the process of stent extraction repeated. However, this time it was possible to pull the entire assembly (catheter, stent and balloon) little bit more towards the Guiding catheter (but not completely inside it). As a consequence, the stent now was hanging in the aorta with some part still hooked in the left main **(Fig. 2.3)**. At this point it was planned to snare out the stent. However, the available snare in the catheterization laboratory had the length of 102 cm and so the guiding catheter (which was 100 cm in length) was shortened (by cutting by 10 cm). Finally, a 6F sheath was put over the cut end and the sheaths stop cock was used as the catheters proximal end. The 10 mm snare was inserted and the stent was snared out **(Fig. 2.4)**. The check angiography was done and the lesion was rewired. The LCx lesion was finally stented with a good end-result.

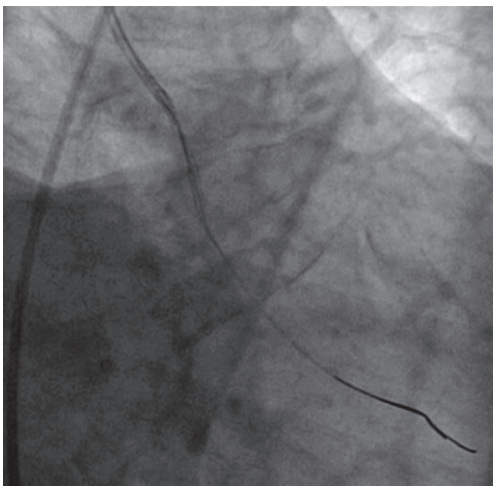

Fig. 2.2 The dislodged stent stuck in the proximal LCX, LMCA
Courtesy: Ranjit K Nath

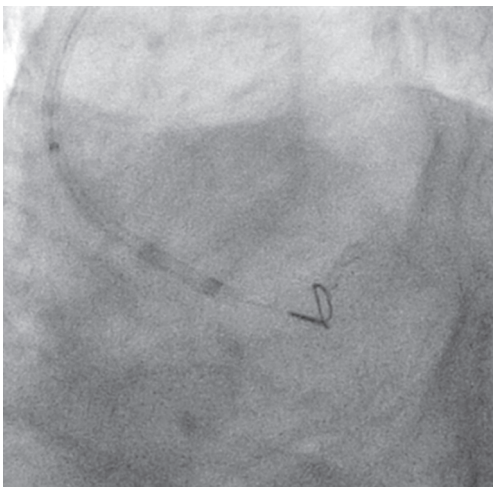

Fig. 2.3 Stent hanging in the aorta and 10 mm snare trying to catch the stent
Courtesy: Ranjit K Nath

Case 5

A 63-year-old male presented with chest pain and restlessness. Coronary angiography revealed a long calcified lesion in circumflex involving bifurcation with major obtuse marginal branch. Two wires were passed—one in circumflex, and the other one in obtuse marginal branch. Lesion pre-dilated with 2.5 × 10 mm balloon; however, it was difficult to cross through with a stent, which got entrapped in LCx. While withdrawing, the stent got

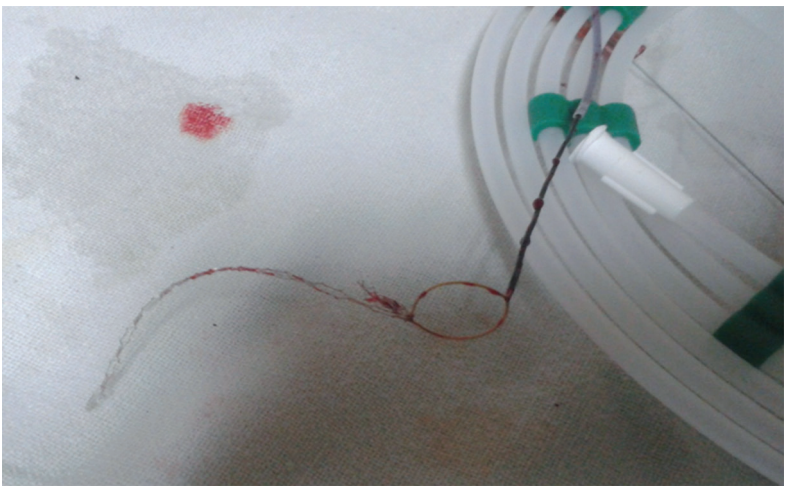

Fig. 2.4 Embolized stent extracted with a snare
Courtesy: Ranjit K Nath

dislodged in circumflex. Another wire was introduced in the circumflex artery, and balloon was then introduced over this wire. The embolized stent was crushed with this balloon at 20 atm. Further, 3 more stents were used to crush the stent completely and maintain lumen patency. Distal lesion could not be crossed and left alone. Finally, good TIMI III flow was achieved.

Analysis of the case: Trying to cross a new stent through a previously deployed stent is one of the common causes of stent embolization. Once stent gets embolized, rewiring and redeployment is the safest strategy but if it is not possible crushing the stent (especially in an hemodynamically unimportant segment) is the next best strategy.

EMBOLIZATION OF OTHER CORONARY HARDWARE

This is an extremely rare situation which may be more common with reused "*Oxford!*" material. Improper preparation of balloon catheter with contrast wherein air is not properly removed and acts as a valve preventing deflation but the whole process remaining fluoroscopically invisible. If this balloon catheter is withdrawn it faces resistance and can even rupture and embolize.[13] The clinical outcome of other embolized hardware is similar to that of a lost stent and likewise the management is also essentially similar; retrieval or crushing. However, the only difference can be that many of these devices can be bulkier than the stents and thus more clinically consequential. Even in peripheral locations they may result in compromised antegrade flow and severe ischemia. On the brighter side, because of more voluminous nature, their localization and also their retrieval is often easier. For retrieving smaller fragments, like broken rota wire fragment the usual snare techniques

described previously may be useful.[14] However, for retrieving bulky non-stent objects from the aorta (such as, retrogradely externalized guidewires), an 18–30 mm En Snare™ may be ideal because its 3-loop design facilitates object retrieval. However, it should be used with caution as the snare wires may cause vessel injury.

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Chapter 3

Interventions for Left to Right Shunts—To Plug or not to Plug: That is not the Question

Saurabh Kumar Gupta, Sivasubramanian Ramakrishnan

INTRODUCTION

Interventional closure of secundum type atrial septal defect (ASD) in cardiac catheterization laboratory has largely replaced surgical closure worldwide. Nonetheless, complication during device implantation is not uncommon and occurs with an incidence of approximately 7%.[1-3] The most common complication is device malposition and embolization. Despite growing experience, it is not uncommon for even a seasoned interventionist to cross fingers while device is being disconnected from the delivery cable. Likewise, there are complications that occur during various stages of device implantation, majority of which can be anticipated and prevented. Nevertheless, uncertainty at times is unavoidable and therefore being prepared is the key for success in cardiac catheterization laboratory.

Transcatheter closure of ASD is associated with all the risks inherent in any interventional cardiac catheterization procedure such as the risk of contrast reactions, vascular access related problems, and the introduction of infection. However, complications unique to transcatheter device closure of ASD include:

- Device malposition
- Device embolization
- Cardiac perforation
- Functional impairment of adjacent cardiac structures
- Arrhythmia and conduction blocks and
- Air or thrombus embolism.

Further, an occasional patient of ASD might have concomitant left ventricular pathology that makes them prone to develop pulmonary edema after ASD is closed.

Avoidance of complications in general is dependent on careful case selection and appropriate sizing of the defect and device to be used. In addition, avoidance of air embolism and ensuring device stability after deployment is imperative. Despite all precautions complications in cardiac catheterization laboratory still happen at a certain frequency and vigilance is required to detect complications early so that remedial actions are taken promptly.

DEVICE MALPOSITION AND EMBOLIZATION

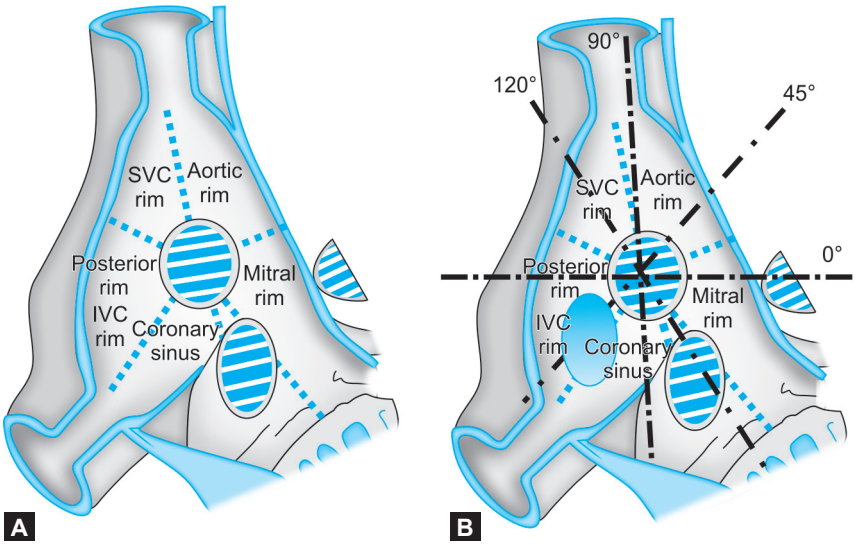
Inappropriate size or orientation of the device during deployment is central to unsatisfactory results and consequently device malposition and embolization in unfortunate few. This potentially life threatening cascade of events however, begins with inadequate imaging prior to and during the procedure. Therefore, complications during device closure of ASD can best be understood with basics of imaging in sizing the defect and choosing correct size of the device. An undersized device predisposes to technical failure as well as device embolization. On the other hand, use of an oversized device makes a patient prone to have late cardiac erosion in addition to compression of adjacent cardiac structures. Most of the complications can be avoided by appropriate case and device selection.

Case Selection

An ASD is considered ideal for device closure if the defect is circular and lies in the fossa ovalis region with good rims all around. A rim is considered good based on its high likelihood of holding the device in position and its ability to keep the device away from adjacent cardiac structures. However, such central defects are seen in only one-fourth patients. Nonetheless, with improved hardware and techniques of transcatheter closure even eccentric defects are being closed using current generation of self-centering atrial septal occluder. Atrial septal occluder (Amplatzer) is made up of Nitinol, a shape memory alloy and is comprised of left and right atrial discs and a connecting waist. The sizing of the device is based on the diameter of the waist. The left and right atrial disc diameter is 5–8 mm and 4–5 mm larger than the waist diameter respectively. Hence, an ASD even if eccentric is considered suitable for device closure if the surrounding rims measures at least 5 mm all around with an exception of retro-aortic rim. The absence of aortic rim in itself does not preclude percutaneous closure of the ASD **(Fig. 3.1A)**.[4]

Sizing of the Defect and Choosing Device Size

There are various imaging modalities that are currently being employed prior to and during transcatheter closure of ASD. Transesophageal echocardiography (TEE) is most commonly used imaging modality to assess suitability of the defect for device closure, accurate sizing of the defect and choosing correct size of the device. In addition, TEE aids in monitoring various steps of device deployment in the cardiac catheterization laboratory. Midesophageal 0° view; midesophageal 45° view at the level of aorta and bicaval view (midesophageal 90°) defines the extent of the defect with respect to adjacent cardiac structures. As is apparent from **Figure 3.1B**, measurements of a circular defect would be approximately equal irrespective of the imaging plane. On the other hand, in an oval defect, the dimension and consequently 'largest diameter' of the defect would vary depending upon the



Figs 3.1A and B (A) Anatomy of ostium secundum atrial septal defect, various rims and its relation with adjacent cardiac structures. (B) Transesophageal echocardiographic imaging planes in midesophageal 0°, 45°, 90°, 120° for assessment of atrial septal defect. Note that all planes in a central and circular defect would measure the defect equally while in an eccentric and oval defect (blue oval close to IVC rim) the measurements of same defect would vary according to the imaging plane used

Abbreviations: IVC, inferior vena cava; SVC, superior vena cava

imaging plane. Eccentric and/or asymmetric defect therefore pose substantial challenge in sizing the defect and consequently choosing appropriate device. Of all the rims in an ASD, the inferoposterior rim close to inferior vena cava is most important for transcatheter closure of ASD. The contraindication for defect margins less that 5 mm has been updated to include the inferior vena cava rim as per the revised instructions for use (4–5). It is not uncommon to have less than ideal rims in this region of atrial septum and therefore this remains 'Achilles heel' of device closure of ASD.

Device Closure of the Device

Conventionally, during device closure of ASD, device attached to delivery cable inside an appropriately sized delivery sheath is inserted in the left upper pulmonary vein. Thereafter, whole assembly is pulled with left atrial disc open and perpendicular to atrial septum. After appropriate alignment and position of the left atrial disc, the right atrial disc is deployed and as a result waist of the device is positioned across the defect. After acceptable positioning of the device across atrial septum, the position is checked fluoroscopically and on TEE. Finally, the device is released from delivery cable.

Recurrent Prolapse of the Device

In large defects, especially with deficient retro-aortic rim, it is not uncommon to have recurrent prolapse of the device necessitating repeated attempts of recapture and deployment of the device. As a result risk of trauma to adjacent cardiac structures increases that in turn increase the risk of cardiac perforation. Also, trauma to atrial septum and rims of ASD is inevitable which in rare cases may be significant enough to make ASD unsuitable for device closure.

If this happens secondary to use of an undersized device the remedy is upsizing the device. However, it is not uncommon to have recurrent prolapse of even appropriate size device despite adequate rims (other than retro-aortic rim). The problem lies in the malalignment of the device with respect to the atrial septum. During deployment of the left atrial disc, alignment perpendicular to atrial septum is the key to success.

Device Malalignment

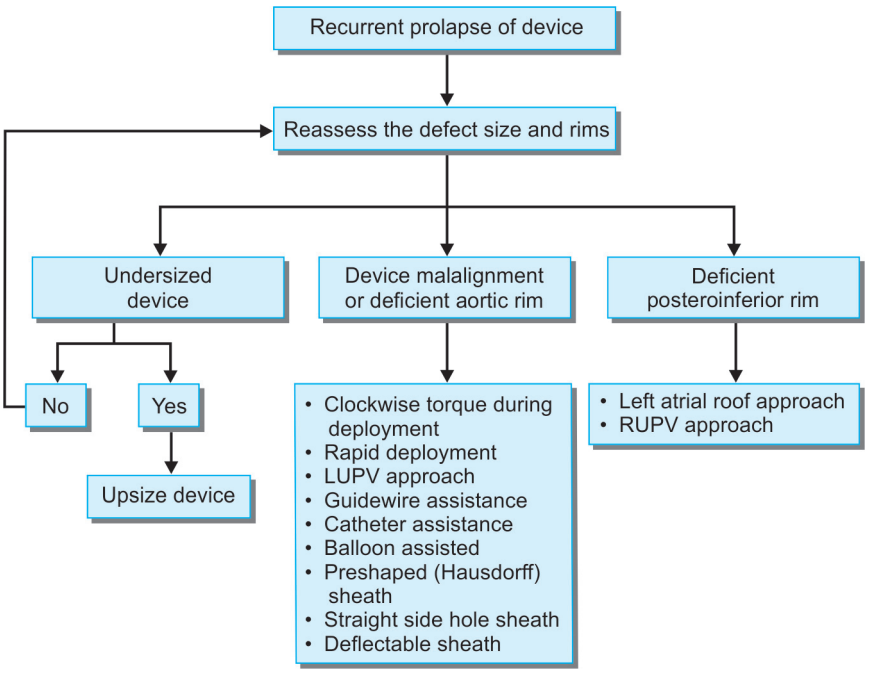
Malalignment of the device leads to prolapse of the device through the defect and/or unacceptable position across the septum and is the most common cause of failure to close the defect despite use of appropriate size device. With increasing use of TEE during the procedure identification of device malalignment is not difficult to identify. Failure to identify malalignment as the cause of recurrent prolapse may inadvertently lead to use of otherwise unnecessary larger device. On the other hand, if malalignment is identified this can be circumvented by use of various modifications **(Flow chart 3.1)**. Various modifications like rapid deployment, deployment from pulmonary vein or with clockwise torque on the sheath during deployment and use of preshaped (Hausdorff) or deflectable sheaths are being used to circumvent this problem of device malalignment. At times supporting the prolapsing portion of the device with a wire, catheter or balloon introduced from the other groin enables deployment.

CASE EXAMPLE

Case 1

Small SVC and absent retro-aortic rim: Transcatheter closure of secundum ASD in a 6-year-old girl was planned. After intubation and mechanical ventilation, TEE was performed that confirmed 17 mm defect. The defect was eccentric that was placed in the anterosuperior aspect of the atrial septum with 6 mm rim in relation to superior vena cava and absent reto-aortic rim. Despite multiple attempts including deployment from right upper pulmonary vein, 20 mm septal occluder could not be placed in satisfactory position. Review of TEE and fluoroscopic images revealed either recurrent prolapse through deficient retro-aortic rim or SVC rim not caught between the rims of the device. Thereafter, a 24 mm Amplatzer sizing balloon was

Flow chart 3.1 Approach to recurrent prolapse of atrial septal occluder



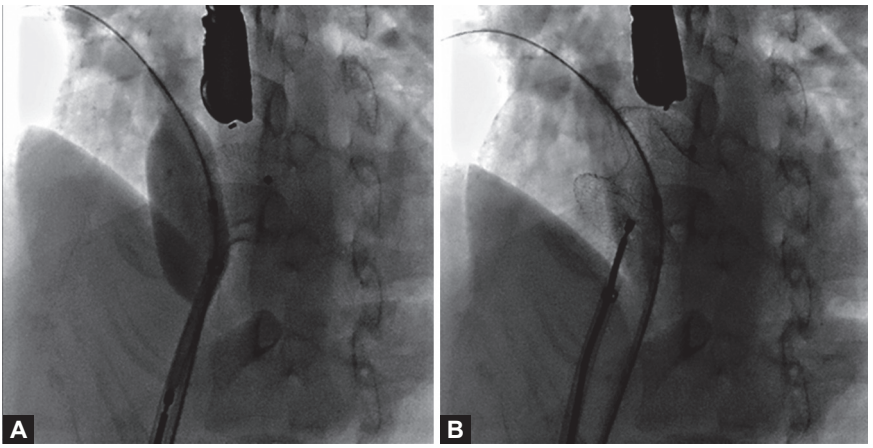
Abbreviations: LUPV, left upper pulmonary vein; RUPV, right upper pulmonary vein

placed across ASD through an additional left femoral venous access. With balloon inflated in right upper pulmonary vein, same 20 mm septal occluder was redeployed from left upper pulmonary vein **(Fig. 3.2A)**. This resulted in appropriately positioned device across the defect with all rims caught well **(Fig. 3.2B)**.

Critical analysis of the case: Though the defect and the device was appropriately sized the orientation of the device with respect to the interatrial septum was not appropriate. Therefore, during initial attempts device prolapsed through SVC-aorta region before both discs were correctly placed. The use of balloon not only prevented this uncontrolled prolapse but also allowed both discs especially the right atrial disc to get appropriately positioned and resulted in successful closure of the defect using same 20 mm occluder.

Device Malposition

Appropriate position of the device is one in which all parts of left and right atrial disc are on respective side of the atrial septum with no minimal or no residual flow without functional impairment to adjacent cardiac structures. Device malposition occurs when device is inappropriately placed across atrial septal defect without having all rims well caught. This can happen



Figs 3.2A and B Panel A shows sizing balloon in right upper pulmonary vein inflated during device deployment to prevent prolapse. Panel B shows optimal device position after deflation of the balloon

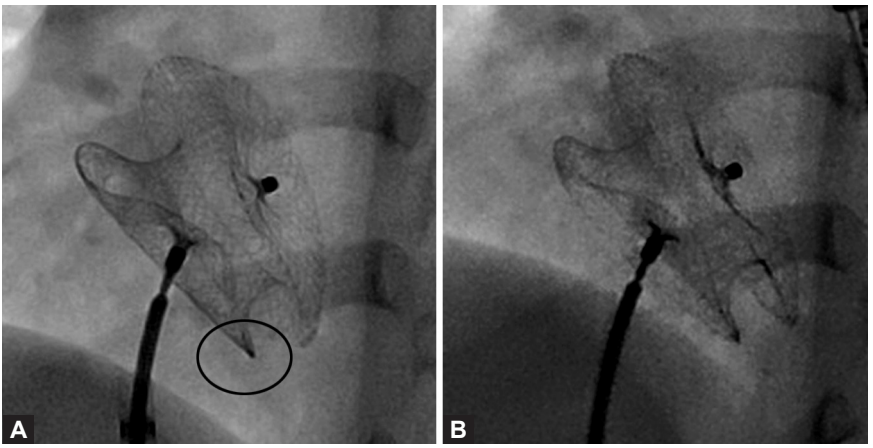
during deployment of device due to either both discs partially remaining in left atrium or coming out in right atrium. In addition, late movement of device can also lead to malposition especially with partial prolapse of right atrial disc. This late movement though can occur after release from delivery cable it is rare in a well-positioned device. Nonetheless, malposition invokes very high-risk of device embolization and should be considered part of the same spectrum. Anticipating, identifying and treating device malposition can therefore avoid many if not all of device embolization.

Device malposition can be rectified by partial or complete redeployment of device. If malposition occurs after release from delivery cable, management is different and involves attempts at reattachment of device to delivery cable using screw on the device. At times it may not be possible especially in an unstable device and would entail all strategies meant for device embolization.

CASE EXAMPLE

Case 2

A 9-year-old girl was taken in catheterization laboratory for device closure of ASD. She had single 19 mm secundum ASD with deficient (~4 mm) inferoposterior rim. A 22 mm atrial septal occluder was chosen. Device deployment from left upper pulmonary vein was relatively easy with apparently good position fluoroscopically. However, TEE evaluation raised suspicion of unsatisfactory position of occluder in inferoposterior region with both right and left atrial disc lying to the left of atrial septum. Review of fluoroscopic images showed unusually sharp lower edge of right atrial disc **(Fig. 3.3A)**. Careful review of imaging then identified device malposition



Figs 3.3A and B Fluoroscopic images in left anterior oblique view with cranial tilt after initial deployment (A) showing sharp lower edge of the right atrial disc. After partial recapture and redeployment this sharpness of the rim is no longer seen and device is better positioned across the defect (B)

secondary to lagging behind of right atrial disc during device deployment. Once this malposition was recognized remedy was easy. It only required partial recapture of right atrial disc of the device before repositioning to attain desired position across the defect **(Fig. 3.3B)**.

Analysis of case: It is very important to be sure of the position of device and multiple views and imaging modalities should be utilized till one is perfectly sure of the position. If however, it is missed during the procedure, it invokes substantial risk of early embolization. On the other hand corrective measure is easy and takes another few minutes provided it is identified in time.

Device Embolization

Device embolization is defined as migration or movement of the device outside ASD after detachment from the delivery cable. With the advancements in the understanding of imaging and interventional techniques device embolization occurs with decreasing yet finite frequency. As discussed before, inappropriate assessment of size and characteristics of the defect, inappropriate selection of device and device malposition accounts for device embolization in the majority. However, exact reason remains unknown in some patients. The device can embolize to right as well as left sided cardiac chambers and consequently might reach pulmonary or systemic circulation respectively. Understandably, embolization to left sided cardiac chambers and systemic circulation leads to greater hemodynamic instability and mandates an urgent retrieval of embolized device. Conventionally, embolized device is retrieved surgically with concomitant closure of ASD. In recent years, however, with gaining experience and improved hardware more

and more embolized devices are retrieved successfully in catheterization laboratory.

Atrial septal occluder especially when large if pulled bare can traumatize various structures like valves, cardiac chambers and vessels. Therefore, as a principle an attempt at percutaneous retrieval must include strategy to slenderize the device and a long sheath wide enough to accommodate artificially slenderized device. A sheath that is at least 2 Fr bigger than what is recommended for the embolized device is generally recommended for this purpose. The patient must be intubated and ventilated if not already done during device deployment. After obtaining appropriate vascular access, device must be initially stabilized. This can be achieved by using Bioptome or crocodile clip to hold any part of device. As an alternative a snare (gooseneck or one prepared in catheterization laboratory) can be utilized to stabilize the device. Device stabilization from two venous accesses would add to ease while pulling the device out. It is much easier to slenderize the device if screw on either side of the device can be caught as it leads to smaller device size. On the other hand, holding other areas of the device results in a larger diameter of the slenderized device. Once device is stabilized and slenderized it is not very difficult to pull the device through already placed wide bore long sheath. Percutaneous retrieval of large device requires lot of manipulations and therefore involves substantial risk of trauma to adjacent structures. Use of large sheaths and not retrieving an open device across valves especially atrioventricular valves reduces these instances to a great extent. Numerous case reports and case series of successful retrieval are reported, but for larger devices that have embolized, stabilizing the device and an emergency surgery remains better option.

CARDIAC PERFORATION AND EROSION

Recent FDA alerts have warned health care providers and patients that in very rare instances (0.1–0.3%), tissue surrounding the Amplatzer ASO can break down (erode) and result in life-threatening emergencies that require immediate surgery. Erosion is reported primarily in the roof of the atria near the aorta, which leads to aortic fistula and cardiac tamponade. Perforation during device deployment is extremely uncommon, and most of these erosions occur months to years following device placement and a few cases it followed strenuous activity. Recently warning have been modified on the product insert to include: patients with a retro-aortic rim of less than 5 mm in any echocardiographic plane or patients in whom the device impinges on the aortic root may be at increased risk of erosion.[4-6]

Following the spate of erosions reported the clinical follow-up recommendations have been updated. Specifically, follow-up with a cardiologist and echocardiograms are recommended at implant, 1 day post-implant, pre-discharge, and again at 1 week, 1 month, 6 months, and 12 months post-implant. Clinical follow-up with a cardiologist annually thereafter is also recommended. Patients are also educated to seek immediate medical

attention, if they develop signs or symptoms of hemodynamic instability such as chest pain, arrhythmia, fainting, or shortness of breath. Patients should be instructed to avoid strenuous activity for a minimum 1 month after the device implant or as directed by their physician.[5,6]

IMPINGEMENT OF ADJACENT CARDIAC STRUCTURES

Avoiding impingement of adjacent cardiac structures is imperative to achieve during device closure of atrial septal defect. In a young child device reaching close to various cardiac structures is not uncommon. In absence of any hemodynamic compromise these devices may be left *in situ* as with growth of various cardiac structures in these children device finally becomes away from these structures. However, the device should not be released from the delivery cable if the device interferes with any adjacent cardiac structure, such as superior vena cava, pulmonary veins, mitral valve, coronary sinus or aorta and cause hemodynamic compromise. The device should be recaptured and redeployed. If still unsatisfactory, recapture the device and replace with a better sized device.

ARRHYTHMIA AND CONDUCTION BLOCKS

A few atrial ectopics are not uncommon with closure of large ASDs, but they disappear immediately. The rhythm should be carefully looked at prior to release from the cable. If blocks or significant atrial tachycardias persist, the device sizing and placement have to be reassessed. Incessant atrial and/or ventricular ectopics if persists may be a pointer towards device embolization and must be taken seriously.

AIR EMBOLISM AND THROMBOEMBOLISM

Introduction of a larger sheath into the low pressure left atrium predisposed to air embolism. Air embolism is avoided by pre-flushing the sheath, flushing the sheath while advancement, use of a under-water seal while the dilator is removed or removal of the dilator in IVC-RA junction, allow for back bleeding and then advance the sheath. In the supine position symptomatic air embolism (mostly to right coronary artery) results in ST segment elevation or in rare cases neurological complications. Thromboembolism is another serious complication that should be kept in mind during interventional closure of ASD. Adequate heparinization must be ensured and frequent flushing of sheaths and catheters must be practiced during entire procedure.

CONCLUSION

Transcatheter closure of ASD using self-centering atrial septal occluder is an established procedure. Even with growing experience complications

occur with certain frequency. These complications are best prevented and anticipated early so that they can be promptly managed.

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Chapter 4

Delicate Sound of Thunder: Coronary Perforations

Sundeep Mishra, Neeraj Parakh

Perfect is the Enemy of Good

INTRODUCTION

Coronary perforations, albeit rare, are one of the most dreaded complications occurring in cath lab, perhaps because of high morbidity and mortality associated with it. The incidence varies from 0.2% to 0.6% but is higher with plain balloon angioplasty (0.61%) or with a debulking procedure (1%) like rotablation, cutting balloon or directional atherectomy.[1-5] The incidence is reduced with the use of stents (0.16%) but is unaffected by the use of IIb/IIIa antagonists.[5]

TYPES OF PERFORATIONS

Coronary perforations are graded according to anatomic classification proposed by Ellis and co-workers.[3] **Type I** = crater extending outside the lumen only in the absence of linear staining angiographically suggestive of a dissection; **Type II** = pericardial or myocardial blush without an exit hole ≥1 mm; **Type III** = frank streaming of contrast through an exit hole ≥1 mm; and **Type III Cavity Spilling** = perforation into an anatomic cavity chamber such as the coronary sinus, the right ventricle, left ventricle, etc. In addition there is another type of **Localized Perforation** which occurs postcardiac surgical procedures like coronary artery bypass grafting (CABG). The importance of this classification lies in the fact that it is co-relative of major clinical outcomes and therefore also the treatment strategy to be used.

FACTORS PREDISPOSING TO PERFORATION

Anticipation of patient and lesion characteristics is the most important step in the management of this complication and therefore a careful assessment of predisposing factors must be made in each and every case. The predisposing factors can be clinical, angiographic or technical **(Table 4.1)**. These factors may predict not only occurrence of perforation but also severity and may even influence the treatment decisions. Complex coronary anatomies are perhaps most important predictors of perforation and some co-relation exists between ACC/AHA lesion Class B2/C.[6] Among technical factors, the

Table 4.1 Predisposing factors of perforation

Clinical
• Advanced age
• Female gender
• Previous coronary artery bypass grafting (CABG)
Lesion morphology
• Calcific lesions
• Chronic total occlusion (CTO)
• Tortuous lesions
• Long lesions
• Eccentric lesions
• Ostial lesions
• Right coronary artery (RCA) lesions
• Fragile venous grafts
Technical factors
• Use of ablative devices
• Non use of stents
• Less use of intravascular ultrasound (IVUS)
• Use of hydrophilic and chronic total occlusion wires
• Use of extraction devices
• Device oversizing
• Use of high inflation pressures

use of atheroablative techniques resulted in a higher incidence of perforation (odds ratio of 16.3) and an increased severity of perforation type (odds ratio 28.9 for development of Type III perforation in atheroablative versus non-atheroablative techniques).[6,7] Attempts to over do the procedure like use of high inflation pressures and oversized balloon are also co-relative of perforation, amply justifying the idiom that '*perfect is the enemy of good.*' Surprisingly, no association existed between abciximab use with either the incidence or the angiographic classification of coronary perforation.

CLINICAL OUTCOMES OF PERFORATION

The clinical outcomes after perforation vary from innocuous to pericardial tamponade to MI, cardiogenic shock and need for CABG to cardiac arrest and death (up to 20% in Type III perforations). However, it should be remembered that most cases of perforations are not accompanied by any clinical sequel. The outcomes are determined to a large extent by the severity of perforation as defined by its classification, use of IIb/IIIa antagonists and occurrence

Table 4.2 Clinical outcomes after perforation

Classification	*Pericardial tamponade*	*MI*	*CABG*	*Death*
Type I	8%	0%	15%	0%
Type II	13%	14%	10%	0%
Type III	63%	50%	56%	19%
Cavity spilling	0%	0%	0%	0%
Contained perforations	6%	0%	24%	6%

of guidewire perforation **(Table 4.2)**. Ajluni and co-workers also found that overall clinical outcomes were worse for patients with free perforations (tamponade 20%, CABG 60%, death 20%) than with contained perforations (tamponade 6%, CABG 24%, death 6%).[6]

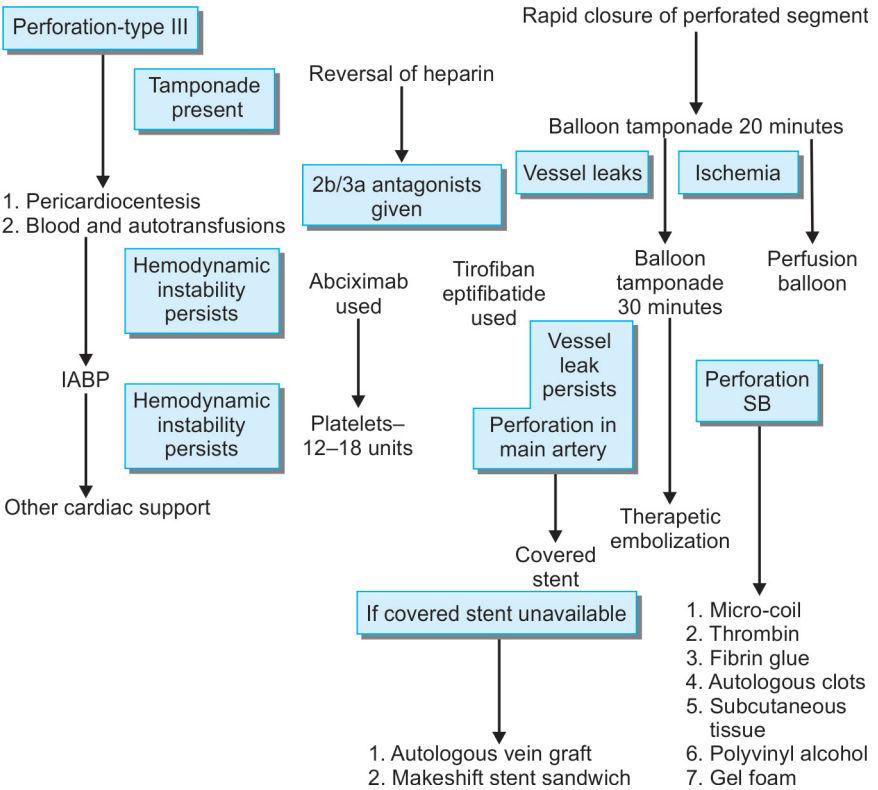
MANAGEMENT OF PERFORATION

Like in so many other complications, anticipation is the key to management of perforation. Thus patients with complex coronary anatomies or in those patients where use of adjunctive devices like rotablation is contemplated this problem should be anticipated and actively prevented. The best way of prevention of perforation is adequate lesion preparation. However once perforation happens it should be treated according to classification of perforation.

While Type I and Cavity Spilling perforations require no special treatment other than close observation because of their innocuous nature. Type II perforation also does not require much more than prompt recognition, discontinuation of glycoprotein IIb/IIIa antagonist and serial echocardiograms on follow-up. If however, cardiac tamponade develops (rare), immediate pericardiocentesis, prolonged balloon tamponade and blood transfusions and autotransfusions may be required. If patient develops myocardial infarction or intractable ischemia CABG may be required as a last resort.

The management of Type III perforation could however be more complex **(Flow chart 4.1)**. Conceptually, 2 treatment streams are simultaneously pursued. Attempts are typically made towards (a) hemodynamically stabilizing the patients (pericardiocentesis, blood and autotransfusions, intra-aortic balloon pump (IABP) and other cardiac support) and (b) sealing the leaking artery (prolonged balloon tamponade, tamponade with perfusion balloon, discontinuation of 2b/3a antagonists, reversal of heparin, intravenous platelet transfusions, or use of interventional sealing device like covered stent, autologous vein graft, makeshift stent sandwich, micro-coil embolization, injection of thrombin, fibrin glue, autologous clots, subcutaneous tissue, polyvinyl alcohol or gel foam. Open repair of the leaking artery along with CABG may be the last option.

Flow chart 4.1 Treatment algorithm of type III perforation



Sudden accumulation of blood in the pericardial space may lead to pericardial tamponade and even death. Therefore, prompt recognition, pericardial aspiration and rapid closure of perforated segment is crucial to reverse the fatal cascade. This generally involves immediate balloon tamponade of leaking vessel, rapid reversal of anti-coagulation and/or anti-platelet therapy, addressing hemodynamic instability, involvement of surgeons if appropriate, and specific treatment of leaking vessel with a bail-out device such as a covered stent or embolization coils. For addressing the leaking vessel generally balloon tamponade is the first treatment option and may be the only maneuver required in majority of cases. A balloon (with a balloon to artery ratio ~1.0) should initially be positioned over the site of perforation and inflated for at least 20 minutes. If the patient is unable to tolerate ischemia during balloon inflation, a perfusion balloon should be used. Perfusion balloons allow distal vessel perfusion, thereby reducing ischemia during prolonged inflations. Subsequently immediate attention should be directed towards reversal of heparin with the aim to achieve a PTT < 60 sec or ACT < 160. Usually, IV protamine sulfate is required to achieve this.[8] If abciximab is used platelet transfusion may be useful but it

does not work if small molecules (eptifibatide or tirofiban) are used for IIb/IIIa antagonism. In minority of cases if the vessel still continues to leak, definitive percutaneous therapy may be required. The major advance in this context is the development of a covered stent. Polytetrafluoroethylene (PTFE) covered stents can successfully seal the site of coronary perforation and consequently can lead to a decrease in mortality and the need for emergency surgery. However, the utilization of these devices may be limited by availability of covered stents in labs and limitation in available sizes contributing to marked under-utilization of this strategy.[6,9] In case of non-availability of this device other strategies to seal the leaking vessel like autologous vein graft and makeshift stent sandwich can also be attempted.[10,11] If the perforation is not in the main vessel but involves small, clinically insignificant side-branch, therapeutic embolization may be attempted. While there may be several ways by which it can be technically achieved (thrombin, fibrin glue, autologous clots, subcutaneous tissue, polyvinyl alcohol or gel foam), micro-coil embolization remains the most popular of all.[12] After achieving stabilization patient is further monitored in CCU and serial echocardiograms performed for at least 48 hours.

CASE EXAMPLES

Case 1

A 62-year-old male patient presented with recent worsening of angina since last 3 months. Echocardiogram revealed regional wall motion abnormalities (hypokinesia) in inferior territory. Coronary angiogram revealed a CTO of RCA with collaterals from LAD. Patient was taken up for angioplasty of RCA. Lesion cannulated with JL guiding catheter, lesion attempted to be crossed with 014 penetrating tip guidewire. However, despite repeated attempts wire could not cross and patient developed Type 1 dissection. In view of the dissection the procedure was abandoned and patient remained stable on follow-up and is scheduled for another PCI procedure for RCA.

Analysis of the case: Patient had CTO of RCA and a penetrating wire was chosen. CTO lesion morphology is more prone to developing perforations and so is the use of penetrating wire. Type I dissections are generally innocuous and by themselves may not require any treatment.

Case 2

Hypertensive, diabetic, 58-year-old post CABG patient done 12 years earlier presented with rest angina. Coronary angiogram revealed Native LAD disease with tight lesion in mid LAD, diffuse LCx disease 40–50% and diffuse RCA disease. LIMA to LAD was occluded but SVG to OM and PD were patent but 60% disease in ostial SVG to RCA graft. Patient was planned for PCI to native LAD and SVG to RCA. LAD was hooked with XB catheter, lesion crossed with BMW™ wire and dilated with 2.5 × 15 semi-compliant balloon.

However, waist was persisting in mid-LAD, therefore the lesion was dilated with 2.75 × 12 semi-compliant balloon. On check angiogram there was a cavity spilling perforation. The patient remained hemodynamically stable and the procedure was abandoned and patient carefully monitored in CCU. Patient was discharged in a stable condition.

Analysis of the case: Patient probably had a fibrotic lesion in LAD which was more resistant to opening. At this stage, may be cutting balloon or even Rota may have helped. However, use of over-sized balloon (for the artery) at high pressure led to the perforation. Luckily, it was Type III Cavity Spilling Perforation which really only causes coronary-ventricular fistula and therefore, by itself generally does not require any treatment.

Case 3

Hypertensive, 58-year-old male presented with recent onset, crescendo angina. Coronary angiogram revealed left coronary system normal but total occlusion from mid-RCA. RCA was cannulated with JL 3.5 guiding catheter, lesion attempted cross with 014 hydrophillic guidewire, but the guidewire could not cross. A rapid exchange balloon was taken to improve the guidewire support, but still the guidewire could not cross. A check angiogram was taken and revealed a ghastly intramyocardial perforation. Bed-side 2D-echocardiography revealed no fluid in the pericardium. Patient remained hemodynamically stable, therefore masterly inactivity was planned and patient observed in CCU for 48 hours and ultimately discharged after 5 days in a stable condition.

Analysis of the case: Again an occlusive lesion of RCA which can predispose to perforation. In this case wire was not crossing (was most likely lodged in the false lumen), but inability to cross was construed as a tough lesion and was forced to cross by taking an additional support by an OTW balloon. This forcing in the false lumen led to extrusion of wire tip out of vessel wall but fortunately not in pericardium but in myocardium leading to development of intramyocardial perforation. By itself intramyocardial perforation if it remains stable may require no treatment other than reversing the effect of heparin.

Case 4

A 62-year-old post CABG patient (done 15 years earlier), diabetic presented with NYHA Class angina of 6 months duration. Coronary angiogram revealed native triple vessel disease, mild left main, LAD calcific and diffusely diseased, diffuse disease in LCx-OM territory, RCA occluded, anastomotic tight lesion in LIMA to LAD, SVG to OM1 and OM2 patient and SVG to RCA occluded. Patient was planned for PCI to native occluded RCA followed by PCI to LIMA-LAD stenosis. RCA was cannulated with JL guiding catheter, but adequate support could not be obtained. The lesion was crossed with 014 hydrophilic guidewire. The presence of distal guidewire tip in true lumen was confirmed

by filling the distal RCA via injection through microcatheter. The occluded portion of RCA was sequentially dilated with 1.5, 2, 2.5 and 2.75 semi-compliant balloons at 15–20 atm. However, the waist in mid-RCA was still persisting. Finally, the lesion was dilated with 3 × 15 semi-compliant balloon at 22 atm but the waist was still persisting. Meanwhile, patient complained of severe chest pain and started dropping pressures. Check angiogram revealed a Type III perforation. Immediately heparin was reversed and balloon tamponade of proximal RCA (keeping from true to true lumen) was instituted and continued for 20 minutes. The patient stabilized. Echocardiography revealed mild pericardial effusion. Subsequent to tamponade angiogram revealed the leak had stopped but another angiogram few minutes later revealed re-leak. Another balloon tamponade was instituted for 30 minutes and subsequently a Jomed Jo™ Stent Graft 3 × 26 mm at 16 atm taking great care that the stent is deployed from "true lumen to true lumen." Subsequent to the stent graft deployment patient stabilized but there was no flow in RCA and the procedure was abandoned. Patient was carefully monitored in CCU for 48 hours and discharged in a stable condition.

Analysis of the case: Again RCA occlusion was attempted to be crossed. Most likely hydrophilic guidewire initially crossed into false lumen and then subsequently re-crossed into true lumen (as evidenced by filling of the distal RCA after injection was made through the microcatheter. So, when balloon was dilated, part of it was dilated in the false lumen. However, the false lumen could not accommodate the final balloon dilatation with the larger balloon and that probably lead to perforation. Once patient developed perforation he was managed by reversal of heparin and balloon tamponade. This technique did stabilize the patient and even sealed the perforation for some time but there was a tendency for re-leaking. As such a covered stent was deployed making sure that it extended from true lumen to true lumen.

Case 5

A 71-year-old female, PO CABG '91, hypertension, hyperlipidemia, DJD, hypothyroidism, post-tubal ligation. Stress Thallium revealed reversible ischemia in lateral territory. Coronary angiogram revealed native triple vessel disease, LAD occluded; significant calcific lesion in OM and RCA was occluded. LIMA to LAD was patent but SVG to PD and OM were occluded. Patient was planned PCI of OM. LCA cannulated with 7F 3.5 CLS guide catheter and the OM lesion crossed with PTGI™ guidewire. The guidewire was exchanged over OTW balloon with a Rota Floppy™ guidewire. 1.5 mm Rota burr was chosen and a total of 8 runs made (total time 115 sec). Angiogram after rotablation revealed a type II perforation. Patient complained of chest pain but remained hemodynamically stable. Urgent echocardiogram revealed a mild pericardial effusion with no evidence of tamponade. Heparin was reversed and LCx rewired with PTGI wire and rota wire removed. Subsequently, balloon tamponade was carried out with 2 × 20 balloon at 6 atm for 285 sec. However, as the perforation was persisting, another attempt at balloon tamponade was made for another 255 sec. Check angiogram revealed persisting perforation.

In view of persisting perforation a covered stent (Jomed Jo™ Stent) 2.75 × 26 was deployed at 16 atm. However, subsequent angiogram revealed worsening in the grade of perforation from Type II to Type III. Chest pain increased and patient became hemodynamically unstable. Echocardiogram revealed increasing effusion. LCx was rewired with BMW™ wire and PTGI™ wire removed. Another attempt at balloon tamponade with 2 × 20 balloon made followed by deployment of another Jomed Jo™ Stent Graft 2.75 × 18. These procedures lead to partial sealing of perforation but it was still persisting. Patient got stabilized and relived of chest pain. However, in view of persisting perforation patient was sent for CABG.

Analysis of the case: Calcific lesions and use of rotablation have a higher chance of developing perforation. Perforations with rotablation are generally caused when rota burr fails to advance and gets stuck in the same position (with loss of speed). Here the perforation can occur because of maldirection of the burr consequent to wire bias. Perforation less than Type III generally require no treatment unless accompanied by hemodynamic compromise or pericardial tamponade. In this case implantation of covered stent was executed in type II perforation with disastrous consequence because probably it was deployed partly in false lumen. Subsequently the lesion was rewired and another covered stent deployed with only partial sealing of the defect. In patients where interventional treatment fails or only partially successful one should not shy away from sending the patient to bypass surgery.

GUIDEWIRE PERFORATION

Guidewire perforations are the 2nd most common mechanism of perforation (only less common than balloon induced perforations: compliant or noncompliant balloons and the stent-balloon delivery system) occurring in nearly half the cases in some series.[5,13] Typically, these perforations do not manifest early but in as many as half the cases the pericardial effusion or tamponade may become clinically manifest only after several hours following the coronary procedure.[6] While in many cases minor perforation is missed during the procedure, in some cases it may not be angiographically obvious at the end of the procedure. The mechanism of this kind of perforation is a leak from a minor vessel, slowly filling up the pericardium and manifesting later. While most such cases occur in context of distal branch puncture with the guidewire tip such as a hydrophilic wire and the administration of glycoprotein IIb/IIIa antagonist, it may also be associated with use of debulking devices like cutting balloon and rotablator. **Table 4.3** enumerates the mechanisms of guidewire perforations.[5] Most guidewire perforations are of the type I and II perforations predominantly caused by hydrophilic and stiff wires, and do not require pericardial drainage or surgical intervention. However, many guidewire perforations can be type III as well and because they are usually "relatively small leaks" in the distal coronary bed or a side branch, they may be successfully treated by coiling or particle embolization technology.

Table 4.3 Mechanism of guidewire perforations

Mechanism	*Incidence*
Procedural characteristic	
Crossing lesion	51%
Distal wire penetration	30%
Wire fracture	10%
Hardware characteristic	
Hydrophllic wire	50%
Standard and intermediate wires	14%
Floppy tip wires	29%
Rotawire	11%

CASE EXAMPLE

Case 6

A 46-year-old lady presented with NYHA Class III angina. Coronary angiogram revealed long tight mid-RCA lesion. RCA engaged with JR 3.5 guiding catheter and lesion crossed with 014 guidewire. It was stented with 3.5 × 32 drug eluting stent (Biomime™) and post-dilated with 3.5 × 12 Sprinter NC™ balloon at 18 atm for 30 sec. Angiogram after the procedure showed a good end result. Five hours after the procedure patient developed severe chest pain along with hypotension. Echocardiography revealed large pericardial effusion with tamponade. Tamponade was immediately drained and patient brought to cath lab for a check shoot. Angiogram revealed Type III perforation in the proximal part of the stent. RCA engaged with the guiding catheter, wired and a covered stent 3.5 × 19 deployed at 14 atm for 30 sec. Finally there was a good end result.

Analysis of the case: Generally delayed perforations are guidewire related (being associated with hydrophilic or penetrating guidewires); however, occasionally they could be related to other cause. In this particular case the perforation arose as a result of proximal edge dissection which manifested late probably due to straining by the patient during the course of observation in CCU. In many cases guidewire perforations are innocuous leaks at the most requiring a pericardial tap. The only point of interest being awareness of the CCU physician of this phenomenon post-procedurally. Once the condition is suspected it warrants an immediate performance of echocardiogram to diagnose and a pericardial tap. Occasionally, however, it can be more malignant requiring definitive treatment of the perforation and in some cases if the treatment is delayed even mortality may ensue. In this particular case, the gravity of situation was recognized and corrected by use of a covered stent.

CONCLUSION

Perforations in cath lab are rare but dreaded complication because of serious morbidity and even associated mortality. As always "prevention is better than cure", the predisposing factors like CTO, use of hydrophilic wires or athero-ablative procedures should be carefully accounted. Once perforation happens recognizing the type of perforation is very important because some varieties (Type I, intramyocardial or cavity spilling perforations) may require no treatment except monitoring and masterly inactivity. On the other hand for more serious perforations a careful well defined approach (treatment algorithm) should be pursued.

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If I were a swan, I'd be gone.
If I were a train, I'd be late again.
If I were a good man, I'd talk with you
More often than I do.

Chapter 5

Bark at the Moon: Technical Difficulties in Cath Lab

Sundeep Mishra, Rakesh Yadav

INTRODUCTION

With the increasing age of the PCI population and the increasing complexity of cases being undertaken in many centers, technical failure is now encountered more frequently, particularly in high-volume centers dealing with elderly patients. These difficulties may range from balloon failure (wire crossing but balloon not crossing, balloon not expanding) or stent failure (balloon crossing but stent not crossing, inability to expand the stent completely) to challenging situations like cork-screw effect after wire passage in a tortuous artery to serious ones like balloon not deflating or stent not properly deploying. However, there may be no simple answer to these problems. Many a times multiple solutions exist and which is the best solution one learns either by experience or by mentor guided empirical approach. Unfortunately, unlike aviation science, PCI procedural knowledge have not been systematically verbalized or available in textbooks and has remained so despite over thirty years. That is why "Tips and Tricks" session is so popular. Herein we deal some of these technical problems and attempt to build up your library of cases by suggesting different alternatives by way of case examples.

WIRE NOT CROSSING

Wire not crossing is really a domain of CTO intervention and best dealt there. Suffice to suggest some tricks; getting support by a balloon or microcatheter is the simplest of them.

Acute Total Occlusions

If not crossing with standard wires, use an hydrophilic wire with low tip load (Crosswire™ or Fielder™ wires which have low friction and can easily pick up micro-channels).

Recent Total Occlusions

A drilling wire with higher tip load (>2) such as Miracle Bros™ or Conquest™ Wire (intermediate tip load) may be a good option.[1]

Older Chronic Occlusion

They generally require a penetration tip wire with higher tip load, a Conquest Pro™ or Progress HT™ for example.

Lesions in Tortuous Arteries

Require initial use of a light support wire (lateral support ≤10 Units) such as Pilot 50™ or Whisper LS™ followed by its exchange for a heavier duty wire.[2]

Negotiating Acute Bends/Going through Stent Struts

Give an appropriate curve to the tip, sometimes a gradual broad curve or even use a withdrawal technique (make a big curve on the guidewire, go beyond the stent struts and then gently withdraw the wire to engage the strut of the stent. Avoid unicore wires and use a dual core wire with a shaping ribbon such as BMW™ wire for these situations. Wiggle wire™ is a good wire specially designed for crossing stent struts.

Calcific Tight Lesions

Use wires with higher tip load and higher lateral support **(Table 5.1)**.

WIRE CROSSING BUT BALLOON NOT CROSSING

Typical lesions encountered in balloon failure may be calcified stenoses, CTOs or noncompliant plaques especially in context of tortuous lesions. In most cases simple techniques which increase balloon trackibility can help but sometimes more drastic measures contributing to adequate lesion debulking (like use of laser, cutting balloon or even rotablation) may be considered mandatory. Generally maneuvers that can help are:

- Dilating the proximal even mild lesion.
- Increasing guidewire support. Using heavier duty wires. The parallel or the anchor wire or balloon technique may also assist passage of balloon or microcatheter across the lesion, but success is variable and often dependent on operator experience[3] **(Table 5.2)**.
- Increasing the guide catheter support.[4] Techniques that are most frequently used are upsizing the guide catheter, deep-throating, and the use of the GuideLiner™ **(Table 5.2)**.
- Use a low profile balloon or short balloon **(Table 5.3)**.
- Rota-Ablation is considered the adjunctive treatment of choice in this situation but the use of this technique is requires special hardware (dedicated Rota™ Wire) and advanced skills.[5]
- Creating channels through the lesion to permit subsequent passage of balloon catheter or a microcatheter. Tornus™ penetration catheter and Corsair™ Microcatheter. Tornus™ catheter has been compared with rotablation in a retrospective study of CTO cases but there are no direct prospective comparative trials of these devices against each other.[6]

Table 5.1 Heavy duty wires

Wire	*Core*	*Tip*	*Coat*
Cordis stabilizer plus	Stainless steel	Platinum Nickel Coils, Soft Tip	Hydrophobic
Cordis stabilizer XS	Stainless steel	Platinum Nickel Coils, Soft Tip	Hydrophobic
All star	Stainless steel		Hydrophobic
Balance heavy weight	Elastinite nitinol	Tungsten+ stainless steel coil, shaping ribbon, Intermediate Polymer Cover	Hydrophilic
HT extra S'port	Stainless steel	Core to Tip, No Polymer Cover, Radio-opaque Coil	Hydrophobic
Asahi grand slam	Stainless steel	Core – Tip, Joint less, Fused Stainless Steel+ Platinum Coil (40 mm), No Polymer	Hybrid
Asahi intermediate	Stainless steel	Core – Tip, Joint less, Fused Stainless Steel+ Platinum Coil (200 mm), No Polymer	Hybrid (Tip hydrophobic, working coils hydrophilic)
Asahi standard	Stainless steel	Core – Tip, Joint less, Fused Stainless Steel+ Platinum Coil (200 mm), No Polymer	Hybrid (Tip hydrophobic, working coils hydrophilic)
HT iron man	Stainless steel	Core to tip, with radio-opaque coils, No polymer cover	Hydrophobic
ChoICE PT ES	Unibody stainless steel	Polymer Tip	Hydrophillic ICE Coating
ChoICE ES	Unibody stainless steel	Floppy/spring coil	Hydrophilic; distal 3 cm spring coils uncoated
Forte MS/ES	Stainless steel	Core-Tip, No Polymer Sleeve	Hydrophobic
Bsc mailman	Unibody stainless steel	Floppy/spring coil, No Polymer Sleeve	Hydrophilic; distal 3 cm spring coils uncoated
BSC platinum plus	Unibody stainless steel	Intermediate/Spring Coil	Hydrophobic
Medtronic zinger support	Stainless steel	Floppy/spring	Hydrophilic or Hydrophobic
Medtronic thunder	Stainless steel	Core-Tip	Hydrophobic
Whisper ES	High tensile steel (Durasteel)	Stainless steel coil in Polymer Coat	Hydrophilic
ABV wiggle	Stainless steel	Shaping Ribbon	Hydrophobic

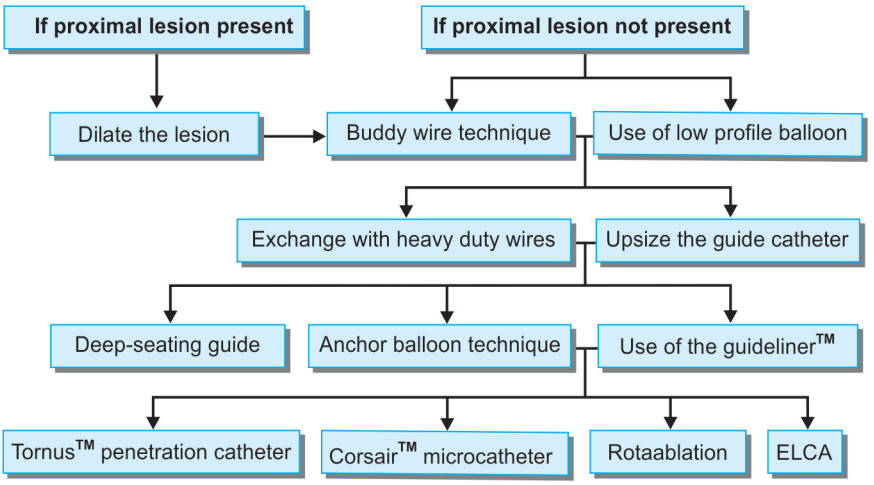
Table 5.2 How to increase guide support

- Use co-axial guides: choose appropriate guide according to ostial orientation
- Use support from opposite wall of aorta: XB; EBU guides
- Use support of aortic root: Amplatz guides: AL; AR
- Upsizing guiding catheter
- Deep seating of guide
- Buddy wire/Triple wire technique
- Anchor Balloon Technique
- Child in Mother Guide: Guideliner™

Table 5.3 Low profile balloons

Manufacturer	*Access*	*Min Balloon Dia*	*Shaft Dia (F)*	*Tip Profile*	*Crossing Profile*
Terumo					
Ryujin™		1.25	2.5	0.017	0022
Ryujin Plus™		1.5	2.5	0018	0023
Invatec					
Avion™		1.25	2.2	0016	0022
Falcon™	5 F	1	2.2	0016	0021
Abbott Vascular					
Voyager™	6F	1.5	2.5	0017	0024
Mini Trek™		1.2		0017	0021
Boston Scientific					
Maverick 2™				0017	0028
Apex™		1.5	2.3	0017	0023
Medtronic					
Sprinter™	5/6F	1.5	2.5	1.25 (0016), Rest (0019)	1.25 (0021), Rest (0024)
Cordis					
Ninja™		1.5	2.7	0018	0025
Firestar™	6F	1.5			0028
Clear Stream					
Eze CTO™		1.25		0017	0022
Blue Medical					
XTRM™	5F	1.1		0016	
Vascular Perspectives					
NIC Nano 0.85™	5F	1		0017	00195
Beo NC™	5F	1.5			

Flow chart 5.1 Algorithm to approach the problem of wire crossing but balloon not crossing



7. Excimer laser coronary atherectomy (ELCA) has an established role in this situation especially in context of mild-moderate calcification.[7-11] The technique is relatively simple to master and more easily adoptable (does not require additional hardware and technique) unlike rotational atherectomy.[12]
   Flow chart 5.1 suggests an algorithm to approach this problem.

CASE EXAMPLES

Case 1

A 60-year-old male presented with chronic stable angina. His coronary angiogram revealed Mid LAD 80% stenosis with some extraluminal calcification. The lesion was crossed with BMW™ wire but due to tortuosity and calcific nature, balloon or stent could not cross. At this point buddy wire was placed to straighten the tortuosity and 2 × 10 mm PTCA balloon was successfully tracked over the BMW™ wire and the lesion was pre-dilated. A 3.0 × 15 mm stent deployed at 12 atm. and stent post dilated with 3.5 × 15 mm PTCA balloon. Subsequently an edge dissection was detected at distal edge of deployed stent which progressed to total occlusion of LAD. On table patient developed hypotension and cardio-respiratory arrest, followed by VT which was DC Verted, CPR given and also patient intubated. Further, on the original BMW™ wire no balloon was tracking. Patient continued to drop pressures. At this time Extra-Support Whisper™ wire was taken to cross the lesion which was successful. Later stent was deployed at the edge of previous stent sealing of the dissection and restoring normal flow. Patient recovered and was shifted to ICCU where later ECG showed no new changes; the ST changes settled down and patient remained asymptomatic and was subsequently discharged in a stable condition.

Analysis of the case: The lesion involved was calcific and somewhat tortuous. Early on the difficulty in passing the balloon was overcome by using a buddy wire technique. The edge dissection following post dilatation of the stent was probably in the context of dilating with an over-sized balloon in a calcific milieu. With a similar pre-existing substrate (calcific and tortuous) along with a fresh tight stenosis (as a result of edge dissection), there was even a greater difficulty in passing a balloon. This difficulty was successfully overcome by using an Extra-support wire and the edge dissection treated by placing another stent.

Case 2

A 58-year-old male presented with ACS. Coronary angiogram revealed a near-total occlusion of mid-RCA. RCA hooked with JR guide catheter and lesion crossed with a standard floppy guidewire. However, 1.5 × 12 mm, balloon catheter could not cross the lesion. Guide support was improved by making it co-axial with the RCA, but the balloon still would not cross. A smaller balloon 1.25 × 10 mm chosen but still could not cross the lesion. Another guide-wire (employing the buddy wire technique) used but still unable to cross the lesion. Finally, decided to deep-throat the JR guide, deep into mid-RCA. Subsequently able to cross with the balloon and stent. Finally, good result.

Analysis of the case: Again it was a very tight stenosis and Judkins Right catheter did not provide enough support. Techniques for improving the guide support like making guide more co-axial, buddy wire techniques were tried but lesion could not be crossed with the balloon. A smaller sized balloon was also used but to no avail. Finally, sufficient guide support was obtained by deep-throating the RCA guide which allowed the lesion to be dilated and stented.

Case 3

A 65-year-old male, hypertensive for last 25 years, suffered anterior MI and underwent primary PCI to LAD with DES (window period 1 hour). An attempt to dilate CTO of RCA after an interval of 6 weeks failed. Patient had normal overall ejection fraction but SPECT imaging revealed ischemia in apex and inferoposterior lateral wall, area of ischemia being approximately 25%. Initially RCA hooked with a JR guiding catheter and the lesion crossed with hydrophilic guidewire. However, 1.5 × 12 mm, balloon catheter could not cross the lesion. JR guide removed and RCA hooked with Guideliner ™ catheter and crossed with previous wire. This time balloon crossed and multiple dilatations made with increasing profile of balloon catheters. Then Guideliner™ was removed and RCA hooked again with JR guide catheter and multiple stents deployed. Finally, good end result.

Analysis of case: A CTO of RCA required somewhat more support than provided by a regular JR guide catheter. This support was obtained by the use of Guideliner catheter.

Case 4

Patient was a 59-year-old male, diabetic, hypertensive, chronic smoker and COPD and benign prostatic hypertrophy, had past history of inferior wall MI and failed PCI to left circumflex vessel. He presented with NYHA class III angina. Echocardiography revealed aneurysm in posterior and inferior walls and akinetic basal septal and lateral walls. Overall ejection fraction was 30–35% with diastolic dysfunction as well. Coronary angiography revealed triple vessel disease with normal left main. Distal LAD had 60% lesion, circumflex showed a CTO lesion with retrograde filling via LAD and RCA was also a long segment CTO with retrograde filling via LAD. Left coronary was hooked with 7F XB 3.5 guiding catheter, lesion tried to be crossed with hydrophilic Whisper™ wire which could not cross. Ultimately crossed with Cross-it™ 200 wire. Another attempt was made to cross with ACROSS CTO™ 1.5 × 10 mm balloon catheter but again failed. Used buddy wire technique to cross the lesion but still could not cross with the balloon. Subsequently decided to use a lower profile ACROSS CTO™ 1.1 × 10 mm balloon but without success. Finally crossed the lesion with 1.25 × 6 mm Sprinter™ balloon and dilated the lesion at 12 atm pressure. Further dilated the lesion with 2.5 × 12 mm Sprinter™ balloon at 16 atm. Finally, the lesion was stented with 2.5 × 18 mm DES at 12 atm and post-dilated with 2.5 × 12 mm Sprinter NC™ balloon at 14 atm. There was a good final result.

Analysis of the case: It was a CTO lesion, therefore a larger guide which provides good support (7F XB) was chosen to begin with. However, even this was inadequate to provide enough support to enable the balloon to cross. Other maneuvers to increase guide support such as use of a buddy wire or a lower profile dedicated balloon were also unsuccessful. Finally, the lesion was crossed with a shorter balloon (1.25 × 6 mm Sprinter).

FAILURE OF THE BALLOON TO EXPAND

This is another well recognized though rare complication encountered in cath lab particularly in context of fibrotic or calcific lesions. The management of this problem lies in adequate plaque modification or in simple words vessel preparation. Techniques which aid in passage of balloon like use of Guideliner™ or parallel wire and anchor balloon technique or even use of Tornus™ device do nothing in this situation.[6,13] Again rota-ablation is the gold standard for adequate lesion debulking, especially in calcific lesions, and contributing to even improved outcomes.[14,15] Other techniques such as cutting balloons or double coated balloon may be simpler but may not equal the results of rotablation.[15-17] Use of ELCA in this situation is another good option.

Balloon Crossing Stent not Crossing

Undeployed stent-balloon assembly has a higher profile (compared with balloon catheters) and therefore requires a higher tackibility and support to cross the lesions. Typically all the maneuvers that increase the crossibilty

of balloon catheters like increase guide catheter and guidewire support can aid in delivery of the stent. In rare cases where despite all these maneuvers the stent cannot be deployed (and also there is good balloon result), the procedure may be ended using a drug eluting balloon.

CASE EXAMPLES

Case 5

A 55-year-old male diabetic and hypertensive presented with history of recent inferior wall MI and post infarction angina. Coronary angiography revealed acute total occlusion of mid-RCA. The lesion was crossed with hydrophilic wire and dilated with balloon catheter but a long stent could not be delivered across the lesion. A buddy wire was positioned in distal RCA across the lesion and this enabled the long stent to be delivered across the lesion. Once the stent was delivered the buddy wire was withdrawn and the RCA lesion stented with a good result.

Analysis of the case: A buddy wire technique was used to increase support to deliver the long stent.

Case 6

A 53-year-old male presented with unstable angina. Coronary angiography revealed LAD long segment lesion, LCX was large, super dominant, and RCA was small but normal. Left coronary was hooked with XB catheter and lesion crossed with BMW™ wire. The lesion was predilated but stent could not cross the lesion. Proximal mild lesion was also dilated and a buddy wire technique used to increase support. Subsequently, 3 × 28 DES was deployed distally. Proximal lesion was predilated with stent balloon but it resulted in mild proximal dissection. Proximal stent placement was tried but in the attempt there was a left main dissection which further progressed to left main hematoma extending to LCX as well. The left main hematoma was managed with a short stent. Some hematoma persisted and there was a distal perforation as well, but TIMI III flow was achieved and the patient remained stable so heparin was reversed and he was conservatively managed.

Analysis of the case: Proximal edge dissection after balloon angioplasty is a known phenomenon which occurs if oversized balloon is used in context of high pressure balloon dilatation or with a calcific spicule in the lesion. If wire is across the lesion it can be easily managed by tacking the dissection with a stent. In this case however, there was a difficulty in delivering the stent probably related to a calcific spicule (as evidenced by difficulty in passing the distal stent even in the beginning which had to be overcome by use of a buddy wire). Buddy wire technique could have been used again to try to cross the stent again, however, patient had developed not only dissection but also intramural hematoma and therefore it would have been difficult to pass another wire. Further an unsuccessful attempt would also have entailed loss

of some time. Thus a short stent was used successfully to tack the dissection and bail out the procedure.

DIFFICULTY IN STENT POSITIONING

Correct positioning of the stent during PCI is imperative. Incorrect positioning of the stent may have serious consequences especially in case of ostial or bifurcation lesions. As a minor problem, poor positioning may translate into inability to cover the lesion, entailing use of additional stent which not only increases cost but can also lead to stent thrombosis or restenosis (total stent length is co-relative of long term complications). On the other hand, poor positioning can also lead to stent struts into main branch which may compromise a side-branch with more devastating consequences. Accurate positioning of the stent can be thwarted by the pulsatile movement of the stent caused by vigorous contraction and relaxation of the ventricle, by the flow of blood in the vessel, even by respiratory efforts. Most commonly ventricular contraction and relaxation is transmitted to epicardial coronary arteries so the stent undergoes a longitudinal antegrade and retrograde **"hum and buzz effect"** or splay. A number of techniques have been described to stabilize the splay of stent. These techniques include simple maneuvers like deep seating the guiding catheter, to use of longer stents, to partial inflation of stent while positioning or even positioning the buddy wire in coronary sinus to stabilize the guide. Other techniques include either induction of temporary asystole using adenosine or rapid ventricular pacing (trans-ventricular or even trans-coronary).[18] The major limitation of using adenosine is that duration of asystole induced by it is short and unpredictable and it must be used with caution in patients with asthma. Transvenous RV pacing requires additional catheters and may be associated with potentially serious complications in 1–20% of cases, RV perforation with tamponade, femoral AV fistula formation, DVT and pulmonary embolism, and bleeding at the femoral access site (by inadvertent arterial laceration), to name a few.[19,20] Transcoronary pacing is associated with none of these complications. However, minor side effects include coronary spasm, diaphragmatic stimulation (ameliorated by changing the wire position) and a stinging sensation at the site of the anode (prevented by using a larger electrode or the anesthetized groin for the anode contact).

Another option is using the Szabo technique which requires 2 guidewires, the Primary wire and the Anchor wire. Briefly the technique is as follows: Outside the guiding catheter the stent is first partially inflated and removed from the balloon catheter. The proximal end of the anchor wire is carefully threaded through the last strut of the index stent, the stent is then crimped on a balloon catheter and loaded onto the primary wire (one which wires the lesion). The stent is then advanced into the guiding catheter over both the primary wire and the anchor wire which remains outside the ostium of the index artery. The stent is then advanced over both the wires into the ostium of the diseased artery, its advancement stopped by the anchor wire when the last strut is reached. The stent is then deployed at low pressure

(with one stent strut hanging out of the ostium into the main branch/aorta). The anchor wire is then removed and the stent deployed at high pressure. This technique ensures accurate positioning; however there have been several reports of excessive resistance when attempting to remove the tail anchoring wire which may lead to telescoping of the guide into the stented vessel. Thus there is a risk of vessel dissection and wire fracture.[22] Further, the insertion of the tail anchor wire under the last stent cell can potentially damage the stent balloon and distort the architecture of the stent.[21,22]

CASE EXAMPLE

Case 7

A 45-year-old male, hypertensive, smoker presented with ACS with persisting NYHA class III angina. Coronary angiogram revealed plaque in proximal LAD and a tight stenosis in ostial Ramus Intermedius. The left coronary artery was hooked with 6FL JL 3.5 guiding catheter and the lesion crossed with Zinger™ guidewire. Ostial lesion was predilated with 2.5 balloon and then taken up for stenting. A 2.75 × 18 mm Everolimus eluting stent was positioned across the lesion but the stent kept moving to and fro with each contraction. The guide was deeply intubated which stabilized the movement of the stent and it was successfully deployed with good results.

STENT NOT EXPANDING

Calcific or fibrotic lesions often require use of rota-ablation or cutting balloon for preparation of bed before stent can be implanted. Subsequently the lesion should be predilated with appropriately sized to confirm and enable the deployment of stent. Sometimes when some of these steps are omitted or even despite them, stents implanted into hard, calcified lesions do not always fully expand, even though they are inflated at very high pressure. Once the device has been released these stents being under-expanded are prone to acute abrupt closure. Even if acute closure does not occur the chances of late complications like stent thrombosis or restenosis are more likely. One of the options is to use an ultra-higher pressure noncomplaint balloon which can generate a pressure up to 40 atm or more. OPN NC™ High Pressure Balloon (RBP 35 atm) is one such option. It has a unique twin-layer balloon technology with ultra-low compliance and can generate a pressure >40 atm. It comes in sizes 2, 2.5, 3, 3.5, 4 mm and lengths 10, 15 and 20 mm and comes with 40 atm. High pressure inflation device. In some cases however even this pressure may not be enough to properly expand the stent or it may cause stent fracture (with current generation stents with low strut thickness). In any case these balloons are not universally available. In those cases rota-ablation may be a reasonable alternative. Although there is certainly a risk of certain potential complications, such as the distal embolization of metal particles or late thrombosis, many case series have demonstrated that it can be successfully performed. The success may be attributed to the ablation of

the stent rings and the calcium that protruded through them, resulting in the thinning of the wall, facilitating balloon expansion and subsequent stent deployment.[23]

CONCERTINA EFFECT AFTER GUIDEWIRE INSERTION

A mechanical alteration during maneuvering of guidewires in coronary arteries occasionally induces straightening effect and vessel wall shortening which produces coronary pseudostenosis, referred as accordion phenomenon. This phenomenon is especially common when highly tortuous arterial vessels are linearized with a stiff guidewire or balloon catheter.[24-27] Further, it can be simply be reversed by the withdrawal of the mechanical device causing the artery deformation. The RCA is especially prone to this phenomenon because the artery is entrenched in the epicardial fat tissue and courses rather freely in the atrioventricular groove but it has been described with LIMA, left main, iliac artery and during carotid stenting.[24-26,28,29] In many cases the outcome is innocuous but the coronary artery elongation however may induce angiographic defects-"web-like" eccentric constrictions-attributed to accordion phenomenon which can be inappropriately identified as coronary spasm, dissection or thrombus development, which may falsely lead to unnecessary stenting at the pseudo-narrowing lesion, turning a totally reversible event into a true iatrogenic complication.[30,31] Sometimes however because of straightening of vessel and consequent production of pseudo-lesions, it can produce even ischemia. The commonest differential is vasospasm which is generally responsive to intracoronary vasodilators like nitroglycerin (100–200 μg) or calcium channel blockers. On the other hand vasodilators are especially ineffective in relieving pseudo-lesions and the only therapeutic management is to remove the angioplasty guide wire.[32] Intravascular ultrasound imaging may be helpful in this occasion to rule out dissection or thrombus extension prior to guide-wire removal.[33] In many cases it is very difficult to perform PCI because it may be difficult to separate true lesions from pseudo-lesions. The only way to overcome this problem is to use a soft microcatheter which takes the curve of the artery allowing clear differentiation.

CASE EXAMPLES

Case 8

Patient was 71-year-old male, diabetic, hypertensive, known case of CAD, post CABG with LIMA to LAD and SVG to OM. Presented with ACS, rest angina. Coronary angiogram revealed LAD occluded proximally and 90% stenosis in mid LAD after the insertion of LIMA graft. SVG to OM was also a degenerated graft with 90% stenosis in the prozimal part of the graft. First PCI with stenting of SVG to OM done, followed by PTCA to distal LAD via the LIMA graft. While passing the guidewire through the LIMA graft it showed a classical cork-screw effect. However, there was no resultant ischemia as a

result of these pseudolesions. Further, the site of interst was distal to LIMA in main LAD and therefore it caused no mis-interpretations while doing PCI. As such PCI to LAD was done in routine fashion ignoring the false lesions.

Case 9

A 68-year-old female, non-diabetic and hypertensive presented with chest pain of four day duration. Troponin T was elevated but otherwise routine biochemistry was normal. Echocardiogram revealed a normal left ventricular function with overall ejection fraction of 60% with no evidence of valve regurgitation. Coronary angiography revealed that left main coronary was normal, LAD showed mid discreet 95% lesion, LCx was normal and RCA showed a distal subtotal occlusion with tortuosity. A right radial approach was chosen. Left coronary was hooked with EBU left guiding 3.5 guiding catheter. The lesion crossed with BMW™ wire and predilated with 2.5 × 10 mm balloon followed by deployment of 4 × 20 mm DES with good end result. RCA hooked with JR 3.5 guiding catheter, the lesion crossed with High torque™ floppy wire which revealed a concertina effect in RCA. Nevertheless based on previous angiogram the true lesion was predilated with 1.5 × 10 mm balloon and stented with 2.5 × 24 DES. However, during manipulations, the soft end of the wire at concertina site, produced a wire induce dissection. Further as the proximal concertina effect persisted the wire was withdrawn to get a clear idea. However, immediately after removal of wire the vessel went into an abrupt closure. The lesion was re-crossed with BMW™ wire and stented with 4 × 28 DES based on previous landmarks.

Analysis of the case: The pseudolesions produced as a result of accordion effect are not always innocuous; they can not only cause ischemia, they can complicate the course of subsequent PCI by increasing misinterpretations because of angiographic artifacts. Further, they can even induce guidewire traumas to the site of these false lesions. However, once the complications are suspected it is very important to keep the wire in place and to exchange it with microcatheter if required for better evaluation. In this case wire was removed hoping for better visualization and correct diagnosis but this led to abrupt closure. Fortunately true lumen could still be negotiated using a nonhydrophilic wire and the site of dissection appropriately tacked by another stent.

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You know the skill destroys the knowledge
Knowledge divides the skill
Tried to run
Tried to hide
Break on through to the other side

Chapter 6

Honey I Shrunk the Stent: Issues in Stent Implantation

Sundeep Mishra, Ranjit K Nath

HISTORY OF STENT TECHNOLOGY

During past several years stenting has emerged as the predominant accompaniment of PCI. The favorable short and long-term outcomes of stenting pertain to the ability of these metallic endoprosthesis to seal dissection planes and resist elastic recoil of the diseased vessel translating into reduction in thrombosis and restenosis. Initially two types of stent designs were available. The slotted tube type (Palmatz Schatz™ stent) and coil type (Wiktor™ stent). While slotted tube design provided more radial support (and therefore more useful in ostial and proximal lesions), the coil design was more deliverable. Over the course of time it was realized that coil stents when employed in ostial and complex lesions suffered a special problem: they had a tendency to unravel and uncoil and this deformation contributed to an unacceptable restenosis rate. Further, this type of stent was also more prone to stent recoil. Consequently, design trends swung away from coiled wire stents to slotted tube (designs that were laser cut from stainless steel tubes) that had higher radial strength, and greater longitudinal integrity but lower flexibility.

In the PCI procedures physicians desire several qualities in their stents: deliverability (tracking and flexibility), conformability, radiopacity, radial strength and cells accommodating side-branch access are some of them. However, on long-term basis antirestenotic and antithrombotic properties are most important. Reducing strut thickness and a hybrid design (and more open cell design) contributed to reduced restenosis rate and even more deliverability, but ultimately led to less radial strength and consequently more stent fractures. This led to research on new stent material with more radial strength (cobalt chromium and platinum chromium) and intelligent stent design (reduced number of connectors between the hoops). However, these modifications led to a new set of problems like reduced radiopacity and longitudinal stent distortion. Meanwhile, evolution has occurred in other areas as well; development of potent antirestenotic drug (DES) and bioresorbable polymer to completely bioabsorbable stent. However, it is important to realize that while any new technology solves one problem, it brings forth a completely new problem. Thus need of the hour for the stent scientists is to achieve a balance between the various properties to achieve an optimum solution and for the physicians to understand the individual properties of currently available stents and judiciously choose them appropriate to the lesion and the patient characteristics.

DEFINITIONS

Stent fracture: Fracture of one or multiple stent struts with or without gap within the stent structure **(Fig. 6.1)**.

Stent longitudinal compression: Percentage by which the length of deployed stent decreases due to force on its ends.

Stent fore-shortening: Percentage by which the length of a stent decreases from its crimped state to its deployed state.

Stent recoil: Percentage by which the diameter of stent decreases from its expanded diameter to the diameter after its deployment in a vessel.

Stent Fracture

Introduction

With the evolution of DES there is improvement in stent platform; lower strut thickness, better composition of stent (from stainless steel to cobalt chromium to cobalt platinum), improvement in cell design (designing of cell connectors, intelligent cell design with placement of closed cells at the edges and open cell design in the center, etc) improvement in polymer (from stable to biocompatible to bioabsorbable to lower thickness of polymer coat to even no polymer) to improvement in antirestenotic drug (type of drug and lower load of drug). This has led to improved third generation DES which may, at one hand cause much less-long-term inflammation and vascular reactivity and be more deliverable but also be more prone to stent damage on the other. However, despite several case reports, stent damage (stent fracture, longitudinal stent compression or even stent rupture) is not a

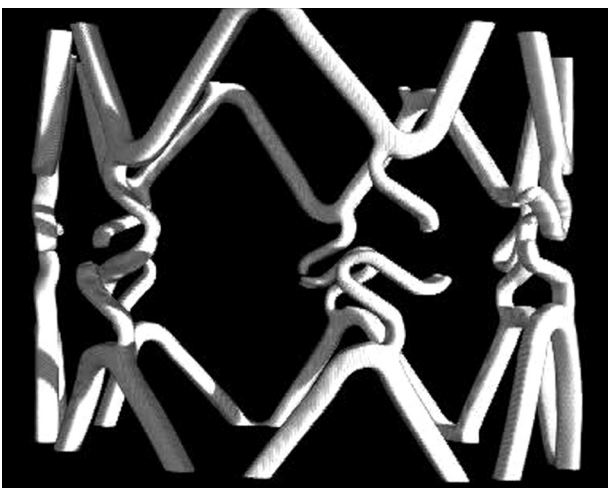

Fig. 6.1 Strut fracture

well-recognized entity and incidence rates are likely to be underestimated. One of the reasons why stent damage is missed is that it can be difficult to pick it up on conventional fluoroscopy because of poor stent radiopacity of most 3rd generation DES due to their lesser metal load (low strut thickness) and if definitive imaging using IVUS or OCT is not done it may not be picked. However, though difficult to diagnose the consequences of stent damage may not be that innocuous **(Table 6.1)**. Stent fracture (SF) in particular is a potential cause of stent restenosis and thrombosis, which can lead to adverse clinical outcomes such as recurrent angina, myocardial infarction (MI), and even sudden death.[1]

Incidence

The reported incidence of SF varies widely between different studies but occurs in 1% to 8% of patients who are given DES.[2,3] These variations are related to many different factors including definition of SF, the methods used to detect SF, type of stent used, the population studied and also on the percentage of patients who undergo follow-up with the available imaging modalities with variable sensitivity of detection. In an autopsy study, the incidence of SF was 29%, which is much higher than clinically reported.

Classification of Stent Fracture

The definition of SF varies from study to study and various morphologic classification schemes have been proposed but perhaps the best is one by Nakazawa and co-workers **(Table 6.2)**, based on number and degree of strut fracture, because this classification seems to co-relate with clinical outcomes as well. A high rate of adverse pathologic findings was observed in lesions with grade V SF, whereas fracture with grade I-IV did not have a significant impact on the pathological outcome.[4] The points of SFs are usually located at hinges subjected to either medial or shear forces created by nonuniform vessel anatomy.[5] In a study by Park et al., 50% of SFs were of type 1, and overall, types 1, 2, and 3 represented 96% of all cases, while type 4 represented only 4% and there were no type 5 fractures.[6]

Table 6.1 Clinical consequence of stent fracture

Study	*Incidence*	*Complication (%)*
Bilen and co-workers	1.9%	60% restenosis, 10% stent thrombosis
Lee and co-workers	1.5%	53% restenosis
Ino and co-workers	4.9%	33% restenosis
Chung and co-workers	0.8%	65% restenosis
Aoki and co-workers	3.1%	38% restenosis
Umeda and co-workers	7.7%	155 restenosis

Table 6.2 Nakazawa classification of stent fracture

Type	*Description*
Grade I	Single strut fracture
Grade II	≥2 strut fractures without deformation
Grade III	≥2 strut fractures with deformation
Grade IV	Multiple strut fractures with acquired transection but without gap
Grade V	Multiple strut fractures with acquired transection but with gap in the stent body

Table 6.3 Factors predisposing to stent fracture

Anatomic features
• Location of lesion
• RCA
• SVG
• Abnormal course of coronary between sternum and dilated aortic root
• Tortuous and highly angulated vessel
• Long lesions
• CTO lesions
• Instent restenosis
• Other complex anatomy
• Myocardial bridge
• Clinical context
• Chronic kidney disease
Technical factors
• Strut thickness
• Link connectors
• Cell design: Closed cell
• Stent conformability
Procedural characteristics
• Balloon or stent overexpansion
• Full metal jacket stenting
• Inappropriate stent handling: Crush technique
• Biventricular pacing

Predisposing Factors

The predisposing factors can be anatomic, technical or procedural but technical factors (stent type and conformability) are the most important factors which correlate most with stent fracture **(Table 6.3)**.

Technical factors
Cell design: Stents with closed-cell structure that result in greater straightening of the vessel, which subjects the stent to greater forces during the cardiac cycle may be predisposed to strut fracture.[7]

Number of link connectors: Although most SFs may occur in all types of stents (DES or BMS), they are common with stents that have more link connectors between the stent hoops **(Fig. 6.1)**. Thus while it can occur in all types of stents it was most common with Cypher™ stent having 6 link connector between the hoops, intermediate with Xience V™ and minimum with those with 2 link connectors, Omega/Element™, Driver/Endeavor™ or Nobori stent.[8-19] In fact in a series of 530 patients (out of a total 2728 patients treated with DES) followed up with repeat angiography, SF was identified in 10 patients (a prevalence of 1.9%) and all occurred with sirolimus-eluting stents.[2]

Stent conformability: It is defined as the degree to which a stent can bend around its longitudinal axis after deployment. Decreased stent conformability can lead to straightening of the vessel wall subjecting the stent to constant shear stress and strain leading to SF.[20]

Anatomic and pathologic factors
These include—tortuous and highly angulated vessel, long lesions, chronic total occlusion (CTO) lesions and other complex anatomy.[21-23]

RCA and SVG locations: These locations undergo dynamic movement during cardiac contractions and stents in these locations may be subjected to repetitive distorting forces (as some segments of these vessels have more flexion points during the cardiac cycle). Repetitive cardiac contraction exposes the stent to compression, torsion, kinking, elongation, bending, and shear stress contributing to mechanical fatigue, culminating in SF. In a meta-analysis of eight studies with 108 SFs in 5321 patients, the incidence of SF in right coronary artery (RCA) was the highest while left main (LM) stents were least likely to fracture.[7,24-26]

Procedural characteristics
Balloon or stent overexpansion.[27]

Full metal Jacket stenting: Stent overlap results in localized rigidity creating hinge points that deform the stent leading to SF.[28,29]

Inappropriate handling of stent: For example crush technique in bifurcation stenting.[30]

Clinical Course

Some patients with SF might be asymptomatic, particularly in case of mild forms of SF (isolated strut fractures). On the other hand more severe grades of SF are usually associated with a clinical sequelae which may range from recurrent angina, MI, stent thrombosis and even sudden death.[31-33] It may be

interesting to note that STEMI occurred less often with BMS perhaps because thicker neointimal layer that develops limits the contact of stent material with the arterial lumen inhibiting thrombus formation.[6] Chakravarty and co-workers reported a mean incidence of stent fracture in 4% and an adverse clinical outcome in 38%.[34] However restenosis is the most frequent clinical outcome with SF perhaps related to poor distribution or interruption of drug delivery consequent to SF.[6] Again higher grades of SF are associated with more TLRs.[2,5]

The temporal course of occurrence of SF, i.e. the time between SF and the development of ISR or ST, and the occurrence of symptoms are still not well recognized. Some patients present as early as 3 days after stent implantation (Xience) while others may present even after several years.[6,35,36] Similarly SF may also not be clinically apparent immediately, the median time interval from stent implantation to detection of fracture at repeat angiography was 226 days (ranging from 7 to 620 days) in a study by Lee et al.[2]

Stent Fracture and Coronary Aneurysm

The SF have been associated with coronary aneurysms.[24,37-39] High pressure balloon dilatation with oversized balloon culminating in tearing of intima-media may be one cause. A DES drug and/or polymer hypersentivity reaction leading to advential inflammation and causing weakening of the media, excessive dilatation may be another factor. Finally, the coating drug of DES might inhibit the process of healing, leading to aneurysmal dilatation. There is also an association between SF, pseudoaneurysm, and infection.[40] On the other hand aneurysm and malapposition of stent might lead to excessive motion of the stent, leading to SF. In fact whether cause or effect nearly all post stent coronary aneurysms are associated with SF.[41]

Diagnostic Modalities

Conventional fluoroscopy

Stent visibility is limited on conventional fluoroscopy. There are several factors that contribute to stent visibility, including the patient's build, a stent platform, and stent thickness and stent material. Stainless steel stents are more visible than cobalt chromium which is also less visible than platinum chromium. Third generation DES which have low strut thickness like Xience V (81 μ) are much less visible.[42]

Stent boost

This technology has improved the visibility of stent struts and involves the automated detection of proximal and distal markers of balloon catheters in each cine frame (through the identification of blob-like structures). It does not add to cost but is limited by the fact that a balloon catheter needs to be placed in the vicinity of a stent or stented segment in order to acquire images.[42,43]

Intravascular ultrasound
Intravascular ultrasound (IVUS) has a greater sensitivity to pick up SF compared to conventional angiography. In addition it can also identify mechanisms of stent failure by providing information regarding neointima formation, vessel remodeling, perivascular tissue, stent expansion, stent strut distribution, and malapposition.[41,44] A major limitation of IVUS is, however, its difficulty of passage through damaged fractured stent struts and that they frequently cause artifacts.

Multidetector computed tomography (MDCT)
Under ideal *in vitro* conditions, CT has a high accuracy when used to evaluate coronary SFs which may be better than conventional angiography. Further, stents gap detected by MDCT have also correlated with in-stent restenosis (ISR), one study finding (SF) in 46% of stent gaps.[45,46]

Optical coherence tomography (OCT)
This imaging modality may be ideal to detect SF (10 times better resolution than IVUS and lesser artifacts).[47] The absence of stent strut was the most common morphological feature of SF in OCT but it can also pick up localized neointimal proliferation which can be highest at the fractured stent site.[41]

Management
The management of SF depends on the presence of clinical features and the grade of fracture. If it is a minor SF and the patient is asymptomatic, it may require no treatment. In symptomatic patients there is no consensus about the ideal management of SFs. The decision should depend on the type of fracture, presence of ischemia, and the presence of factors that predict possible recurrence. If the reason of SF was stent overexpansion, then restenting the lesion again is possible with avoidance of stent overexpansion. On the other hand, when SF is caused by a nonmodifiable factor like excessive vessel tortuosity, then referring the patient for coronary artery bypass graft (CABG) is more reasonable when there is a clear need for revascularization. A useful tip in the management of SF by restenting, when there is difficulty in passing the wire or balloon across the lesion, stent boost guidance may be utilized to manipulate the wire and balloon across the lesion.[29]

CASE EXAMPLES

Case 1

Patient was a 55-year-old male, known case of hypertension and diabetes since 5 years, admitted with history of unstable angina. LV EF was 60%. PTCA stenting to RCA was done using 2 DES (Taxus™ stent); 3 × 16 mm for proximal RCA and 3 × 32 mm for mid RCA. Check CAG done 6 months later showed well flowing stent with 30% in-stent restenosis in mid RCA. Patient was left on medical management. Patient presented with acute IWMI nearly 4 months later. Check angiogram showed proximal RCA in-stent total occlusion with

a heavy load of thrombus. The lesion was crossed with Pilot 2™ 0.014 wire. Repeated thrombosuccession was done using 6F Diverse E™ catheter. Stent boost was used to diagnose stent fracture of proximal RCA Taxus™ 3 × 16 mm stent. Lesion was predialted with Pantera™ 1.5 × 6 mm balloon at 10 atm. Followed by 2 and 2.5 mm balloons at 12 atm. Lesion was stented with Endeavor™ (3 × 24) at 14 atm. Post dilatation was done using Voyager NC™ 3.0 × 20 mm at 18 atm.

Analysis of the case: Taxus™ is a stainless steel stent which lies intermediate in the risk of stent fracture (less than Cypher™ but more than 3rd generation cobalt chromium stents). Many a times minor stent fractures do not manifest early but become apparent only on follow-up. Endeavor Sprint™ stent is a cobalt chromium stent with only 2 connectors and therefore is among the least likely to fracture.

Case 2

A 52-year-old lady presented with post ACS, Class III angina. An angiogram revealed an occluded RCA, the remaining arteries having no significant disease. She was taken for PCI of RCA. The RCA was hooked with 6F 3.5 JL guiding catheter and the lesion crossed with BMW™ guidewire. The lesion was dilated with 2 × 10 and 2.5 × 15 semicompliant balloon and then stented with 3 × 18 Vison™ stent. Check shoot revealed that stent was under-deployed. The stent was post dilated with 3 × 15 noncompliant balloon but angiogram revealed that stent was still under-deployed. Therefore stent was again post-dilated with 3.5 × 15 semi-compliant balloon with a seemingly good result. However, within 10 minutes post-procedure she developed severe chest pain. Angiogram done immediately revealed a filling defect in RCA stent suggestive of acute stent thrombosis (ST). The cause of ST was debated. It was a thrombogenic PCI milieu, therefore ST was possible, but 7000 U of unfractionated heparin had already been administered at the beginning of PCI, and ACT was 270, moreover Tirofiban was already on-board. Therefore possibility of stent strut fracture seemed more likely. An IVUS was planned but strut fracture was anyway confirmed because there was a great difficulty in crossing the filling defect with a BMW™ wire and the lesion was crossed with a great difficulty with another wire using buddy-wire technique. First it was attempted to dilate the lesion with 2 × 10 semi-compliant balloon, but it couldnot cross initially. Subsequently the lesion was crossed and dilated with 1.5 × 10 semi-compliant balloon. Then it was progressively dilated with 2 × 15, 2.5 × 15 and 3 × 15 balloons. Finally the lesion was stented with 3.5 × 15 Vision™ stent. Check angiogram revealed a good end result. Patient remains asymptomatic more than 5 years after follow-up.

Analysis of the case: Most probably the initial stent was undersized for proximal part of the artery. Attempts to correct it using larger compliant balloon with high pressure dilatation caused high grade stent fracture/collapse of the stent warranting progressive dilatation of the damaged stent

with compliant balloons 1.5 × 15, 2 × 15, 2.5 × 15 and 3 × 15 and finally, damaged stent crushed against wall by another 3.5 × 15 stent.

Stent Longitudinal Deformation

Introduction

Any change in length of deployed stent, strut overlap, strut separation which may or may not culminate in malapposition and/or luminal obstruction is known as longitudinal stent distortion (LSD). Radial strength of the stent depends on material of the stent, strut thickness and number and geometry of connectors between the hoops. On the other hand deliverability of stent depends on strut thickness, total metal to artery ratio and stent design particularly the number of connectors between the hoops. Mechanical engineering of stents is a science of compromise. While reducing the strut thickness, metal artery ratio, number of fixed connectors between cells and altering the geometry of these connectors (and their longitudinal distribution), enhance flexibility/deliverability and conformability but at the expense of radial and longitudinal strength of the stent structure.[48]

Effect of Stent Material and Design

Older generation stents like Cypher™, which had higher strut thickness and 6 connectors between hoops culminating in a higher radial strength, but they were much less deliverable and prone to strut fractures. On the other hand newest generation Element™ Stent composed of platinum chromium and having much lower strut thickness, only two connectors with peak offset design has much higher flexibility, deliverability and conformability. Due to its platinum chromium composition, it maintains its radial strength despite much lower strut thickness. However, because of its lower strut thickness, only 2 connectors and peak offset design of connectors it is most prone to LSD. Driver™ stent despite having two connector design has higher strut thickness and also different connector geometry and has consequently slightly greater longitudinal strength. Other 3rd generation stents like Xience™ series, Biomatrix™, etc. lie in between in flexibility as well as longitudinal strength. An interesting development in this area is Integrity™ stent platform which is actually a variety of coil stent, employing continuous sinusoidal technology permitting superior deliverability without compromising on longitudinal and radial strength as well. This is achieved by a unique manufacturing process and aligned-crown design. In addition the helical single wire winding from one end of the stent to the other behaves like a third connector.[48]

Incidence

The overall reported incidence of LSD is around 0.2% over 4-year time period but it may be slightly higher with Element™ platform (0.6–0.8%). One of the

reasons for this phenomenon may be that while LSD may be under-reported with other 3rd generation stents because of low radiopacity, on the other hand because of higher radiopacity (platinum chromium base) this complication is easily recognized in this platform.[49]

Predisposing Factors

Several factors can contribute to development of LSD. They could be; lesion characteristics particularly complex subsets like ostial or proximal, bifurcation lesions, calcific and tortuous lesions; technical characteristics like malapposition, wire bias or rough handling during the procedure. However, the stent characteristics particularly less number of connectors between hoops, orientation of the connectors and other stent design features are perhaps the most important factors[48,49] **(Table 6.4)**.

Mechanism of Longitudinal Stent Distortion

The etiology of LSD can be heterogeneous. While it can be induced by a variety of devices passed into the stent (slightly malapposed) such as post dilation balloon, IVUS/OCT catheter, thrombectomy catheter, filterwire etc, even withdrawal of jailed buddy wire but in 2/3rd of cases it is caused by deep engagement of guiding catheter or guide-catheter extension like Guideliner™ **(Table 6.5)**.

Table 6.4 Factors predisposing to longitudinal stent deformity

Lesion characteristics
Complex lesion subsets
• Ostial/proximal
• Bifurcation: Provisional stenting technique
• Long lesions
• Calcific lesions
• Tortuous lesions
Technical characteristics
• Malapposition–undersized stent, direct stenting procedure
• Wire bias after stenting (prior to postdilatation)
• Use of excessive force to pass various devices through a recently deployed stent
• Rough handling of the PCI procedure
Stent characteristics
• Strut thickness
• Less connectors between the hoops
• Stent design alignment of connectors with the long axis of the stent—connector link the offset, in-phase hoop peaks

Table 6.5 Etiology of longitudinal stent distortion

Proximal edge
• Guide catheter trauma
– Deep intubation of guide catheter
– Guide catheter extension—GuideLiner
• Passage of devices after stent deployment
– Postdilatation balloon IVUS/OCT catheter
– Thrombectomy catheter
– Proximal embolic protection device
Distal edge
• Withdrawal of devices after stent deployment
– Thrombectomy catheter
– Distal protection devices
– Devices involved in PCI distal to deployed stent
– IVUS/OCT withdrawal
• Withdrawal of jailed buddy wire

Clinical Features

Most of the times effect of LSD is clinically innocuous, appearing as a radiopaque line at the edge of the stent (as a result of separation and bunching or folding of stent struts) which in extreme cases may manifest as "concertina" or "accordion" effect. It may initially be recognized as a difficulty in the passage of a device such as postdilation balloon. In more severe cases, it can produce acute obstruction to flow of blood contributing to acute stent thrombosis. In addition because of suboptimal result, additional stent may be deemed necessary. In rare cases even emergent CABG may be required. On long-term basis because of essential malapposition of stent struts it may lead to sub-acute or late stent thrombosis or restenosis as well.[48,49]

Management

In general prevention is better than cure. An anticipation of problem and a careful application of technique is generally sufficient. Several tips and tricks can be employed to prevent this situation **(Table 6.6)**. Once LSD develops management depends on the severity of the problem. In minor cases (when incidently recognized) high pressure postdilatation with a NC balloon is generally sufficient (which will take care of underlying under-expansion and malapposition). If there is difficulty in passing the NC balloon a smaller compliant balloon should be first used and the procedure completed with an adequately sized NC balloon. In some cases when the defect cannot be corrected by mere postdilatation or significant part of lesion gets uncovered as a result of LSD, additional stent will be required and rarely the patient may have to be sent for CABG.[49]

Table 6.6 Tips and tricks

- Better plaque modification by careful predilatation of the lesion (avoid direct stenting) or even rotablation in calcific lesions
- Prolonged balloon inflation during stent delivery
- Proximal optimization technique mandatory when doing provisional stent technique for bifurcation lesions
- Keeping guide catheter co-axial at all times; particularly re-engagement of the guide co-axially over the deflated stent balloon during withdrawal or withdrawal of IVUS catheter
- Deep intubations with guide catheters or guide extensions (Guideliner) should be performed with extreme care
- If the proximal part of the stent lies in a tortuous segment high possibility of wire bias while attempting postdilatation should be anticipated
- Specific maneuvers may be considered to avoid guide catheters moving in and out of the proximal coronary segment with respiratory or cardiac motion such as leaving an additional guide wire in sinus of valsalva to stabilize the guide catheter
- Proper handling and a resistance free passage of various devices across the stented segment
- After stent deployment if there is a resistance to delivery of postdilatation balloon or imaging catheter suspicion of LSD should arise and careful radiographic assessment be performed to diagnose LSD and also to determine the extent of lesion uncovered by the problem
- Choose stents carefully when problem anticipated
- In case of long stents there is a relatively high chance that proximal part of stent is under-deployed in the large diameter proximal segment and therefore this part should be carefully high-pressure dilated with adequately sized NC balloon
- Avoid use of IVUS or OCT to diagnose the problem; because though they may provide useful diagnostic information there is a risk hat they may cause further deformation by interacting with deformed struts.

CASE EXAMPLE

Case 3

A 63 year, hypertensive male patient presented with history of exertional angina class III since 6 months. Echocardiography was essentially normal. Coronary angiogram revealed lesion in proximal LAD. Left coronary hooked with XB 3.0 – 7F catheter and lesion crossed with ALL STAR™ wire and another wire kept in diagonal vessel (provisional stenting). Lesion predilated with 2.5 × 12 balloon at 12 atm and stented with Xience V™, 2.75 × 18 @ 12 atm and stent post-dilated with NC balloon 2.75 × 13 @ 14 atm. There was some difficulty in removing the buddy wire and later cine revealed crumpling at the proximal edge of the stent. The proximal edge was subsequently postdilated with NC balloon 3 × 10 @ 16 atm. Finally, there was a good result.

Analysis of the case: Provisional stenting technique for bifurcation lesion is a common precursor for LSD. Very commonly, during withdrawal of jailed wire, guide catheter may get sucked in causing longitudinal compression of proximal edge of the stent, which was the likely cause here.

Radial Stent Recoil

Acute Stent Recoil

Historically, one of the reasons for widespread acceptance of stents over POBA was high prevalence of recoil of ballooned artery post–procedure. It was found that stents (slotted tube) prevented this recoil following POBA. However, in 1990 IVUS studies demonstrated that most stents themselves remain under-expanded despite an excellent angiographic appearance. The reason for this phenomenon was found to be postdeflation stent recoil. At that time it was realized that stent recoil was more with coil stents (18%) vs slotted tube design (7–10%). With the evolution in design and material of stent the recoil is much lower with current 3rd generation DES.[50]

Late Stent Recoil

Stent recoil which occurs over a 4 month period is known as late stent recoil. Classically, this is not an issue with self-expanding stents but occurs only in balloon expandable ones. It is not much of an issue with metallic stents: Palmaz Schatz™ stents exhibited only a little recoil (0.1 mm^2 and 0.6% at 4 months). It is even lower with current generation DES: Xience V™ (0.02 mm^2 and 0.35 at 6 months). However, this could certainly be an issue with recent bioabsorbable plastic stents: Absorb™ (0.65 mm^2 and 7.6%). This problem could be overcome to a greater extent by a self-expanding design for example earlier IgakiTamai™ stent actually underwent an expansion (0.71 mm^2) rather than recoil.[51]

Factors Contributing to Stent Recoil

As discussed recoil is dependent on the material of stent (platinum chromium < cobalt chromium < stainless steel < PLLA) but also on design (self expanding < balloon expandable) but it also depends on lesion characteristics (calcific lesions < fibro-fatty plaques).

Clinical Outcome

Small amount of stent recoil is mostly in-consequential but it may contribute to underexpanded/malapposed stent struts, which could be a recipe of stent thrombosis or restenosis.

Management of Stent Recoil

As always prevention is better than cure and proper application of technique is the best way. Optimal technique involves use of compliant slightly oversized balloon (1.1 : 1, balloon: artery ratio at least with older generation DES) followed by high pressure postdilatation with noncompliant balloon (to increase stent cross-sectional area (CSA) and improve stent geometry as well). The aim is to achieve an optimal stent expansion defined as ratio of stent to reference lumen CSA of 0.9-1:1.

CASE EXAMPLE

Case 4

A 50-year-old male presented with a complaint of angina on exertion (since 8 months). He was hypertensive and diabetic. Overall LVEF was normal. Coronary angiogram revealed proximal LAD and OM1 lesion. LAD lesion crossed with BMW™ guidewire and predilated with 3.5 × 10 mm balloon @ 8 atm. Angiogram revealed dissection after predilatation and lesion stented with long Absorb™ 3.5 × 28 mm. Stent boost guided postdilation was carried out with NC balloon 3.5 × 10 mm @ 20 atm revealing good end result in LAD. BMW™ crossed OM1 and predilated with balloon 2.5 × 10 mm @ 14 atm. Subsequently, Absorb™ 2.5 × 18 mm was placed across the lesion and postdilated with NC balloon 2.75 × 12 @ 16 atm. Finally, there was a good result. Patient was instituted on routine post-PCI treatment (including anti-platelet Rx). The following day patient suddenly felt chest discomfort, acute breathing trouble with sweating. He was immediately transferred to Cathlab for Emergency PCI. Angiogram revealed stent thrombosis of both the stents. Within minutes patients developed cardiorespiratory arrest and could not be revived.

Analysis of the case: Patient developed early SAT despite following all the recommended steps. The stent being a plastic stent, possibility of stent recoil should be kept high.

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Chapter 7

Cannulation Blues: Issues with Guide Catheter

Sundeep Mishra, Sunil Verma

GUIDING CATHETERS

Guiding catheters remain one of the crucial components in angioplasty hardware. The initial crude Teflon catheters had small lumens, minimal torque control, and sharp edges. However, with evolution in design while they are nearly as safe as the diagnostic catheter, they have a lumen diameter at least twice that of a typical diagnostic catheter (typically 0.076-inch or more). The design involves reducing wall thickness to less than 0.010-inch, but at the same time still incorporating a Teflon liner to reduce friction, metal or plastic braid to transmit torque and provide sufficient stiffness to offer "backup" support during device advancement, and a smooth outer coating to resist thrombus formation. This extraordinary feat is achieved by use of special materials whose properties are typically varied along the length of the catheter to optimize the balance between support and flexibility at each point. Currently, most guiding catheters include a very soft material in the most distal 2 mm of the catheter to reduce the chance of vessel trauma during engagement of the non-tapered tip. With issues in vascular access there is a tendency to reduce the size of guiding catheter so that at present 6F or even 5F guiding catheters are pretty much standard for the common "garden variety" of situations. However, for complex procedures where double balloon/stents or devices like rota-ablators are likely to be used, larger bore guiding catheters may be required. The standard guide catheter length is 100 cm, but shorter (90 cm) guides are available to allow more distal passage of devices with limited working lengths during procedures on distal lesions in saphenous vein graft (SVG) or left internal mammary artery (LIMA) interventions.

Depending on the site, take-off of the ostium and the amount of support required, the guiding catheters are available in shapes similar to conventional Judkins and Amplatz curves, as well as a wide range of custom shapes, such as hockey-stick, multipurpose, and Voda, that are designed to provide support and co-axial engagement. While Judkins is very useful as a diagnostic catheter because its primary angle is fixed and it can intubate only a relatively small area of the ostium, however, it has several limitations when used as a guiding catheter **(Fig. 7.1)**. Firstly, the primary curve is fixed so in many cases it is impossible to intubate co-axially, also because of the fixed primary curve it may form a 90° angle (180° for the circumflex artery) with the ostium making it extremely difficult to pass any device. Finally, there is a minimal or even no point of contact with either the root of aorta or the

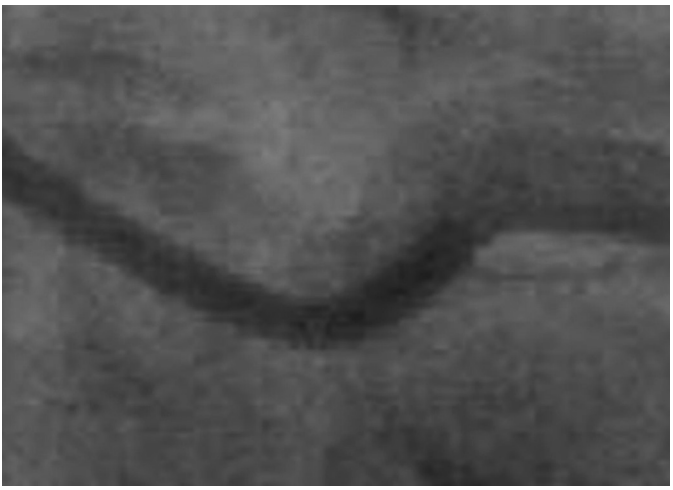

Fig. 7.1 Limitations of Judkins catheters as a guide catheter

- 1° curve fixed: may not be co-axial
- 1° curve makes >90°: difficult to pass balloons, etc.
- 180° for LCX
- 2° curve long and straight and makes another <90°
- JL—Point of contact on ascending aorta very high (chance of prolapse) and narrow (chance of dislodgement)
- JR—No point of contact on ascending aorta

opposite wall culminating in very poor support for passage of the devices. Amplatz guiding catheter on the other hand is generally co-axial and gets good support from not only opposite wall but also the root of the aorta (AL catheter) **(Fig. 7.2)**. Unfortunately, because primary curve is fixed in Amplatz catheter as well it is most liable to cause catheter trauma to the lumen. In this context, a good balance between safety and efficacy is provided by extra back-up catheters (XB or EBU). First of all their primary curve is not fixed and can change according to requirement and the resistance faced (as such these catheters remain co-axial in all circumstances). Further, they may provide support not only from the opposite wall but also by deeply intubating (since primary curve can vary).

The type of guiding catheter chosen also depends on the position of index artery. The aim is to achieve co-axial alignment along with support. Generally in cases of anomalous origin of native arteries or their superior take-offs Left Amplatz (AL 0.75,1 or 2) is the best catheter. In case of inferior take-offs, however, Multipurpose may be the ideal catheter. For SVGs located in different regions in ascending aorta and even LIMA interventions Right Judkins may be the right catheter. However, despite this general approach, difficulty may be encountered in cannulating the index arteries especially abnormally located arteries and providing enough support.

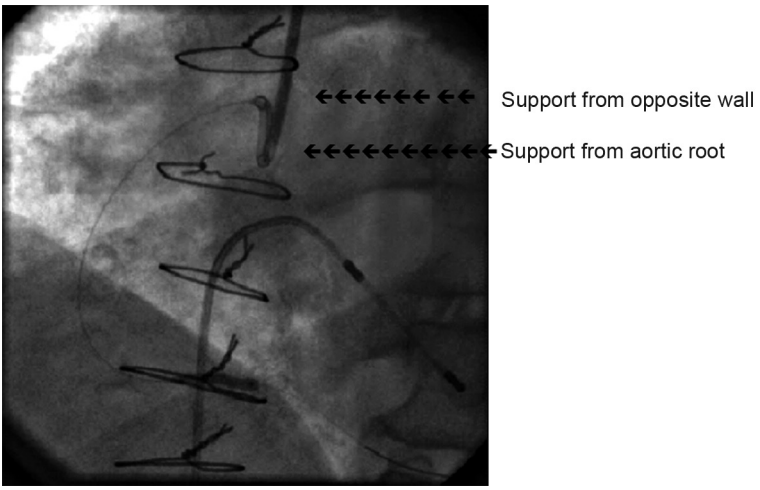

Fig. 7.2 Support provided by Amplatz guiding catheter

NATIVE CORONARY ARTERIES

Generally no difficulty is encountered in cannulating native arteries unless aortic root is abnormal, the origin of arteries is abnormally located or oriented (Inferior or Superior Take off)[1] **(Table 7.1)**. As a general rule for interventions in LAD a smaller curve of guiding catheter (JL3.5) and a clockwise rotation may suffice. Some specialized catheters like XB LAD also are available. On the other hand for left circumflex, an anti-clockwise rotation or use of specialized catheters like Voda may help. For right coronary artery (RCA) generally a JR or Hockey Stick catheter suffices but if the RCA origin is inferiorly oriented, a multipurpose catheter may be useful. On the other hand, if RCA origin is superiorly oriented AL catheter (0.75, 1 or 2) may be required.

Shepherd's Crook RCA

This is a special situation where RCA origin is not only superiorly oriented but RCA courses superiorly to some extent before making a U turn. The technical requirements of a guiding catheter are thus very stringent; not only the catheter be co-axial but it should provide enough support to guide wire and other devices which have to make a 180° turn-around. The regular JR catheter is particularly unsuited for this complex anatomy and if interventionist persists with this catheter despite problems it can lead not only to a failed procedure but also carries a high risk of aorto-ostial dissection. In extreme anatomy, AL catheter (0.75, 1 or 2) is the only solution. However, if there is only a mild Shepherd's Crook, 3DRC catheter provides a very nice balance between safety and success.[1]

Table 7.1 Guide catheters for native coronary arteries

Left guide catheters
• *Normal aorta*: JL4, AL2, XB 3.5, EBU, VODA
• *Dilated aorta*: JL5, AL3, XB4
• *Dilated ectatic aorta*: JL6, AL3, XB4.5
• *More back-up*: EBU, XB3.5, VODA, XB-LAD
• *Superior take-off*: AL2 ST
• *Inferior take-off*: MPA, JL 4 MOD
• *High take-off*: AL3, AL4, JL3.5
• *Low take-off*: AL1.5
• *Posterior take-off*: AL2
• *Anamolous CX*: MP
Right guide catheters
• *Normal aorta*: HS, JR4, AR2
• *Dilated aorta*: JR5
• *Superior take-off*: AL, AR 2, HS, DA 75
• *Inferior take-off*: MPA, HS, AL, AR, JR 4 ST, JR 4 MOD
• *High take-off*: AR2, JR 3.5 (0.5 smaller size), RCB, MP
• *Anomalous origin*: JL, HS, 3DRC, AL1, MP, AL2, LCB, JR
• *Anterior origin*: AL, MP
• *Posterior origin*: AL1, AR 2
• *Shepard crook RCA*: AL, LIMA, HS, MP

SAPHENOUS VEIN GRAFTS

For patients who have undergone coronary artery bypass surgery, knowledge of graft anatomy is important for planning the PCI. Different guide catheters may be chosen according to location of the graft **(Table 7.2)**. However, generally JR catheter is most useful in these situations but in some cases other guiding catheters may be required like LCB catheter, AL (for high take-off) or MP (for low take-off) **(Table 7.2)**.

INTERNAL MAMMARY ARTERY

The main challenge in IMA intervention is that it is tortuous and redundant. Thus guide should be able to provide enough length to other hardware like guidewires and balloons/stents and therefore short length guiding catheter or even radial (generally left) approach may be useful. The second challenge is need to provide enough support. The choice of guiding catheter depends on the anatomy of the artery requiring intervention. The left IMA (LIMA) originates anteriorly from the caudal wall of the subclavian artery and is distal to the vertebral artery origin. The JR catheter usually

Table 7.2 Guide catheters for bypass grafts

Saphenous vein grafts
• *RCA graft usual location*: Primary—MP; Alternate—JR, AL, RCB, HS, EGB
• *RCA graft anterior location*: Primary—AL; Alternate—JR, MP, HS
• *LCA graft*: Primary—JR, HS; Alternate—AL, LCB, MP, EGB (El Gamal)
• LCA *graft ant location*: Primary—AL, HS; Alternate—JR, LCB, MP
LIMA grafts
• LIMA
• IMC

is good for cannulating the artery but may not provide enough support. LIMA or left coronary bypass (LCB) catheter may provide more support but carry a risk of ostial dissection **(Table 7.2)**.

ANOMALOUS ORIGIN OF ARTERY

Anomalous origin of one or more coronary arteries is an infrequent occurrence but more likely in presence of certain congenital heart diseases. Its prevalence varies from 0.6% to 1.2%.[2] Abnormalities of conal artery are the most common anomaly followed by anomalous origin of right coronary artery. Some of these anomalous vessels can be diseased requiring coronary interventions.

Anomalous origin of RCA from left sinus of valsalva (ARCA) is the 2nd most common coronary anomaly. It was first described in 1948 by White and Edwards and its prevalence seems to be higher in oriental populations (0.25%). Typically RCA arises from the left coronary sinus and its origin is oriented superiorly and anteriorly to that of left coronary. After its origin the proximal segment of ARCA takes an abrupt rightward and downward course between the aorta and pulmonary artery before it pursues its course posteriorly in the right AV groove. Since it arises at an acute angle from the aorta the luminal orifice at aorta may not be round but is rather slit-like or chink like. Thus, it is easy to appreciate that this unique location, its abnormal take-off and chink like anatomy of the ostium makes it singularly difficult for the operator to locate and cannulate the ostium and even more difficult to provide adequate support. One special tip while doing PCI is to obtain a RAO view which clearly defines position of right coronary artery (RCA) ostium and its initial course. Guide-catheter intubation and guide-wire passage should be made under this view. Several strategies have been tried to overcome this problem.[3] With JL catheter it is practically impossible to cannulate this anatomy and AL catheters should be the first choice. Sometimes, simple clockwise rotation of the catheter beyond the LCA (which orients the catheter superiorly) may be enough. However, use of specialized catheters like left coronary bypass may help. A specially designed Leya catheter can not only cannulate but also provide enough support in majority of cases.

Leya catheter is a modified AL catheter wherein the primary curve is fashioned 90° rightward and anteriorly, thus conforming to the unique anatomic features of this anomaly.[3] Another catheter, LCB has been found useful in some cases.[4] Some maneuvers have also been found useful if this specialized catheter is not available. One of them is positioning the catheter as close to the ostium as possible and then crossing into the anomalous artery by a guidewire and then threading the guide catheter over this guidewire.[5] Another technique is to wire the LCA first to provide support to the assembly and then carefully withdrawing the guide-catheter and trying to intubate the anomalous RCA (with the added support).[6]

Rarely, anomalous RCA may have a posterior origin from left coronary sinus. In that case several catheters can be tried. Use of AR or even XB catheters may sometimes help.

Anomalous origin of RCA from right sinus of valsalva or even RCA (forming right main artery) is another common anomaly. In general it requires no special technique or hardware (other than advanced skill of the operator) and recognition and proper evaluation of the origin of LCx. However, there could be serious problems during PCI if this essential evaluation is not properly performed. The first case series of PCI performed on such aberrant vessels was described in 1982.

CASE EXAMPLES

Case 1

A 56-year-old, diabetic male presented with class III angina, uncontrolled on medication. Coronary angiography revealed a single vessel disease: dilated tortuous RCA with multiple tight tandem lesions in mid-distal RCA. Initially conventional Hockey Stick guide-catheter was used which unfortunately was not co-axial and did not allow enough support to wire this dilated tortuous artery. Finally, AL1 guiding was used which provided enough support.

Case 2

Case of inferior take-off of RCA in post bypass case. Co-axial cannulation and proper support obtained by multipurpose catheter.

Case 3

Shown a case where AL 0.75 was not co-axial but AL 1 was appropriate. In a case of Shepherd's Crook RCA. JR and HS catheter is generally not co-axial. If persistent attempts are made with a non co-axial catheter it can cause ostial dissection (IM catheter). Even AL catheter curve should be carefully chosen.

Case 4

Case of SVG grafts. Generally, it is adequately dealt with JR or HS catheter. However, in some cases LCB or even AL catheter may be required.

Case 5

For LIMA interventions JR can be used but generally does not co-axial and may not provide enough support, so that once a complication happens difficult to quickly pass devices to handle it. LIMA or IMC catheters may provide enough support but should not deeply intubated, otherwise there could be a risk of dissection.

Case 6

A 62-year-old diabetic hypertensive male with past history of PCI to LAD 2 years ago with DES and 3 months ago with DES to anomalous RCA arising from left coronary artery, presented with rest angina. Coronary angiogram revealed instent restenosis of anomalous RCA. Initially RCA cannulation was carefully attempted with JR but could not hook it. Then AL 1 catheter was tried but could not intubate the ostium. Finally AL2 catheter was used which on clockwise rotation came very close to the ostium but was still semi-selective. The ostium was attempted to be wired but wire could not cross into the lumen of RCA. On RAO view it was realized that the catheter was still some distance away from the ostium. The RAO view was used as guide to wire the ostium which was now possible and finally the AL2 catheter was threaded over the guidewire to nicely cannulate the ostium of anomalous RCA. Subsequently the procedure was routine.

Analysis of the case: Use of JR catheter in this type of anatomy, though safe is completely useless. AL catheter of appropriate curve is generally the catheter of choice. If despite several attempts the catheter is close to intubating but still not selectively intubating wire should be used to thread the index artery. If still not successful several views (particularly RAO) should be obtained to assess the distance and direction to be traversed by the wire.

Case 7

A 70-year-old hypertensive elderly lady had inferior wall MI and was thrombolysed outside in local hospital, presented with post-MI angina. ECHO showed fair LV function with inferior wall hypokinesia. Coronary angiogram revealed anomalous RCA arising from left sinus posteriorely (Type D according to Sarkar et al. classification of anomalous RCA). The anomalous RCA had total occlusion mid RCA. The culprit RCA was attempted to cannulate with JR 4.0, AR-1, AL-1, AL-2 but not successful. Finally, XB RCA 3.5 guiding catheter was successful in engaging the anomalous RCA. Whisper™ wire was used to cross the total occlusion. After dottering with 1.2 × 10 mm balloon, the lesion was predilated with 2.5 × 10 balloon. Initially 3.0 × 28 mm Xience V™ stent was chosen but found that it may not cover the entire lesion. During the course, check shoot revealed that the vessel was closing again because of thrombus. When trying to take longer stent the whole guide system and wire was disengaged from RCA. The anomalous RCA had to be reintubated and re-crossed with same Whisper™ wire. The long segment lesion was stented with 3.0 * 38 mm Xience Prime™ stent. TIMI III result achieved.

Analysis of the case: In posteriorly oriented ARCA even AL may not be helpful. In this case several different types of catheter may be tried JR, HS, AR or XB catheter may be tried. This case was finally successfully done using XB catheter. However, XB catheter may not give as good support as AL catheter.

Case 8

Old inferor MI presents with Troponin positive ACS. Coronary angiogram reveals anomalous Cx arising from RCA. Started with diagnostic JR, crossed the lesion with exchange guidewire, then exchanged with guide catheter, dilated and stented.

Case 9

A 46-year-old male, HTN, obese (80 kg), presented with inferior MI. Echo revealed inferoseptal, inferior and posterior wall hypokinesia with overall LVEF ~40%. Patient was preloaded with aspirin and prasugrel and shifted to cath lab for PCI in view of persistent angina. Coronary angiogram revealed LCx was not visualized from left main but retro filling OM and LCx (probably anomalous origin of LCx which was totally occluded with nonperfused myocardium-one of the angiographic sign of anomalous coronary artery). RCA cannulation was attempted with JR 3.5. However, JR was selectively cannulating conal branch but stumps of occluded RCA and LCx were visible with inferior orientation. MPA1 diagnostic catheter was first used to clearly visualize the anatomy (RCA totally occluded, LCx arising from RCA but also totally occluded). Patient was taken up for primary angioplasty of RCA followed by PTCA to LCx as a staged procedure. Using 7F multipurpose guiding, primary PTCA of RCA was carried out. Export™ thrombus extractor was used to extract large thrombus burden from RCA. Xience Prime™ 3.5 × 38 was deployed in the distal lesion and 3.5 × 18 used in the proximal lesion. PCI to anomalous LCx attempted after a week. Multipurpose guide catheter was positioned just at the ostium of RCA and LCx (No other guiding was engaging the LCx). Simultaneous LCA (JL 3.5) and RCA injections done to visualize a channel with antegrade flow in the direction of retro filling LCx). Fielder FC™ wire was repeatedly buckling and guide catheter disengaging on any attempt to manipulate the wire further. Repeated attempts with 1.25 × 6 mm balloon support also failed. Wire was repeatedly moving into the twig. Since quantity of contrast and time exceeded during repeated manipulation of guide catheter and wire, it was decided to attempt once again next time. Anomalous LCx was reattempted after a week, using microcatheter. This time wire moved further but in a branch. Angiogram through microcatheter in true lumen revealed a dissection but identifying true lumen provided direction and ultimately managed to cross the wire into, true lumen (confirmed with free movement of microcatheter over the wire). RAO caudal view was used to confirm LCx position. Multiple inflations were delivered using 1.25 × 6, 1.5 × 10, 2 × 20 mm balloon catheters. Integrity™ stent 2.25 × 33 mm and 2.25 × 30 mm used to cover the entire lesion. Finally good result was achieved.

EXCHANGE OF GUIDE CATHETER

Sometimes the guide catheter is co-axial and allows the passage of guidewire but does not provide enough support to pass devices like balloon and stent. In that case the guide catheter needs to be exchanged without losing the guidewire position. The technique involves passage of microcatheter or over the wire balloon and exchange of the guidewire with a heavy duty guidewire. Subsequently the guide catheter can be withdrawn and the requisite guide catheter carefully threaded over the heavy duty guidewire to achieve proper intubation of coronary ostium. Subsequently the procedure can be carried out as per plan.

TIPS AND TRICKS

- Catheter size and shape are selected as per case. It should be minimally traumatic while providing optimal backup support
- Catheter advancement and rotation should always be gentle and gradual
- If difficulty is encountered in cannulating, a different size or shape should be tried early instead of persisting with a catheter
- Deep cannulation of the vessel should be avoided
- If aorto-ostial disease is present the catheter chosen should be relatively fixed (not freely moving) and not deeply intubated. Generally, Judkins catheters are a good choice. Catheter with side-holes should be considered in this situation to allow some flow into artery intra-procedurally.

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Chapter 8

Valvular Interventions: Keep Your Eyes on the Door and Your Mind on the Procedure

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INTRODUCTION

Valvular interventions are one of the most rewarding procedures in interventional cardiology. Balloon dilatation of stenotic valves offers simple and long-lasting results in most of the cases. Surgical therapy in the form of valve replacements requires lifelong care in the terms of anticoagulation and its associated side effects. Balloon mitral valvotomy (BMV) by far remains the most common valvular intervention in the developing world due to high prevalence of rheumatic heart disease. On the other hand, aortic valve balloon dilatation (AVBD) and pulmonary valve balloon dilatation (PVBD) are still performed all over the world as these are mainly of congenital etiology. They are performed more commonly in the younger age group.

BALLOON MITRAL VALVOTOMY

Inoue's novel innovation has revolutionized the treatment for mitral stenosis.[1] Although Inoue balloon has simplified the BMV to a large extent, difficulties may be encountered at various anatomical levels and in certain subsets of patients. BMV has a relatively steeper learning curve and knowledge of certain tips and tricks may be useful at the time of need **(Table 8.1)**.

At Groin

Too slim or too obese patients may rarely pose difficulty in entering the femoral vein at groin. Changing the direction of balloon and taking a more vertical direction usually succeeds. If still not successful, then 14 F sheath may be used to give easy passage to the Inoue balloon. In very rare cases of IVC interruption, BMV can be performed via internal jugular route.

At Septum

Trans-septal puncture is the most critical step during BMV. Fluoroscopic technique described by Hung is usually followed.[2] In case of difficult anatomy, echocardiographic assistance may be of great help. Septal angiogram performed by pulling the Mullin's sheath while injecting dye defines the septal

Table 8.1 Anatomical levels of difficulties during BMV

At groin:

- Difficult entry of balloon (too thin or too obese patient)

At septum:

- Bulging interatrial septum
- LSVC to coronary sinus
- Large right atrium
- Thick septum
- Abnormal septal profile
- Cardiac malposition
- IVC anomaly

At mitral valve:

- Very tight mitral stenosis
- Large left atrium
- Small left atrium
- Abnormal septal puncture
- Subvalvular deformity
- Unusual orientation of mitral valve

anatomy and aids in the septal puncture. Sometimes, staining the septum by injecting dye into the septum helps in identifying the correct site of septal puncture. In case of bulging septum, Brockenbrough needle may be bent a bit more to prevent it from slipping on septum. Sometimes, a thick septum prevents crossing of balloon at septum. Some clockwise rotation of balloon in a screwdriver manner is successful in most cases. If still not successful, the interatrial septum (IAS) should be re-dilated with a 14 F dilator.

At Mitral Valve

When left atrium (LA) is very large or a subvalvular deformity is present, the usual method of crossing the mitral valve may fail. In these situations, modification of the curve of J stylet may help in left ventricle (LV) entry; a larger curve for large LA and smaller curve for small LA. If still unable to cross a reverse loop or semireverse loop can be attempted to try cross the mitral valve. In brief, this technique requires clockwise rotation of the J-shaped stylet. In case of very tight mitral stenosis, if Inoue balloon cannot be negotiated through the mitral valve, then an exchange length 0.032″ wire on a Swan Ganz catheter can be used to cross the mitral valve and over this wire Inoue balloon can be tracked.[3,4] If it still fails, then a smaller peripheral balloon can be used to make way for Inoue balloon **(Table 8.2)**.

Table 8.2 Left ventricle entry in difficult cases[5]

- Reverse loop
- Semireverse loop
- Changing stylet curve
- Floating balloon by partially inflating balloon
- Over the wire
- Predilatation with smaller peripheral balloon
- Push-pull technique
- Modified over-the-wire (OTW) technique using Mullin's sheath

TACKLING COMPLICATIONS

Cardiac tamponade is a frequent (may occur up to 1%) and most dreaded complication of BMV procedures. Most of the cases are simply managed by pericardiocentesis and reversal of heparin. Occasionally, surgical repair is required. Some operators have used application of thrombin glue to seal the perforation site. Mitral regurgitation (MR) is another feared complication of BMV. Increase in the grade of MR is relatively frequent, occurring in more than 5% of cases but severe MR may be rare. However, with more and more challenging cases being taken up for BMV, MR has become a real challenge. Proper patient selection is the key to prevent this complication. Urgent surgery is required in most of the cases. Balloon dilatation of interatrial septum helps in decompressing the LA, and may decrease the pulmonary congestion but at the cost of lower cardiac output. Cerebrovascular accident during BMV is another frequent complication associated with BMV (seen in less than 1% of cases). The source of embolus may be clot in left atrial appendage or LA, generally in context of AF. However, it could be calcium or even tissue material from the thickened fibrotic valve. Urgent thrombolysis with or without percutaneous catheter retrieval of thrombus material is the treatment of choice. Many special catheters are available to retrieve the embolic material and are very useful, if the embolic material is organized thrombus or calcific spec from the valve as these will not get dissolved by thrombolysis.

Aortic Valve Balloon Dilatation

Aortic valve balloon dilatation (AVBD) is another non-coronary intervention frequently performed in a busy cath lab. The most common problem associated with this procedure is inability to cross the aortic valve. Using a straight tip, 0.038″ wire is quite useful. Judkin's right, pigtail and multipurpose catheters are commonly used. Amplatz left 2 is very useful in difficult cases as it directly looks towards the aortic orifice and has a wider reach. An occasional coronary dissection has been reported and should be dealt as with coronary angioplasty.

Pulmonary Valve Balloon Dilatation

It is another common non-coronary intervention. In adults, Inoue balloon or double balloon techniques are used. Crossing of pulmonary valve with Inoue balloon is at times tricky. There should be no loop or step in the balloon length during crossing the valve. During valve deflation, it is better to push the balloon into pulmonary artery to restore the blood flow early, and then it can be pulled out. An important precaution in this procedure is to monitor both blood pressure and arterial saturation by pulse oximetry (as in patients with patent foramen ovale (PFO) or atrial septal defect (ASD), the blood pressure will be maintained due to right to left shunt but saturation will fall dramatically and may result in neurological injury, if it persists for longer period).[6]

CASE EXAMPLES

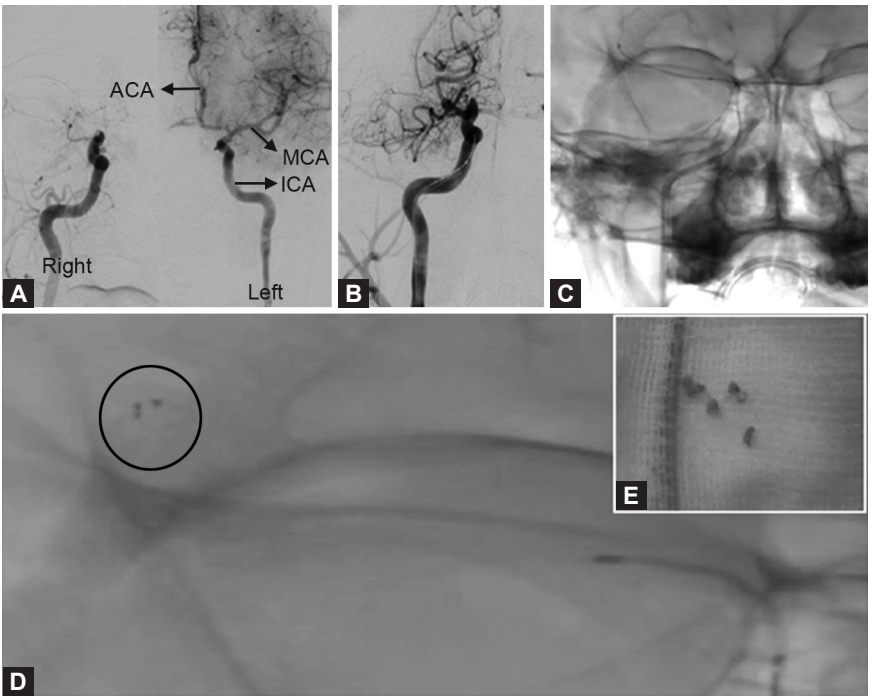
Case 1

A 24-year-old male underwent BMV for severe mitral stenosis. Procedure was smooth and without any difficulty. However, immediately post-procedure he developed left hemiparesis. There was no LA or LA appendage clot in pre-procedural echocardiography. Noncontrast computed tomography (NCCT) head shows no evidence of intracranial bleed. Digital subtraction angiography of cranial vessels revealed occlusion of right middle cerebral artery. Using 0.021 PROWLER SELECT Plus™ (Cordis Neurovascular, Miami Lakes, Fla) microcatheter, the lesion was crossed successfully with 0.014 Transcend™ guidewire (Boston Scientific, Natick, Mass, USA). Solitaire FR™ clot retrieval device (ev3, Irvine, California) was used to retrieve extremely hard fibrotic material **(Fig. 8.1E)** from the lesion. Some flow was restored and there was some improvement in the neurological condition of the patient.[7]

Analysis of the case: Embolization of thrombotic material is possible even in patients where no clot is visible on transthoracic or even transesophageal ecocardiography (TEE) echo. On the other hand, embolized material can be calcium or tissue from the valve. Clot-retrieval devices are useful in cases where embolism could be due to calcium or tissue tags. Timely, intra-arterial or even intravenous thrombolysis is a good solution, if retrieval is not possible **(Fig. 8.1)**.

Case 2

A 38-year-old man developed acute pulmonary edema and hypotension while waiting for mitral valve replacement surgery for severe calcific mitral stenosis. He was put on mechanical ventilation and was planned for emergency BMV. Calcific valve prevented LV entry of Inoue balloon. LV was crossed with the help of Swan-Ganz catheter which was tracked to aorta. A 0.032″ exchange length wire was tracked through the Swan-Ganz catheter. Inoue balloon was now tracked to LV over this exchange length wire and BMV was performed successfully **(Fig. 8.2)**.



Figs 8.1A to E (A) Intra-arterial digital subtraction angiography (IADSA) showing normal left ICA, MCA and ACA, while the right ICA is completely blocked before its bifurcation into right MCA and ACA; (B) Attempting to cross the lesion using 0.021 PROWLER SELECT Plus microcatheter; (C) Lesion successfully crossed by 0.014 transcends guidewire; (D) Solitaire FR clot retrieval device *in situ* (circled); (E) Retrieved hard and fibrotic embolised material

Abbreviations: ICA, internal carotid artery, MCA, middle cerebral artery; ACA, anterior cerebral artery.

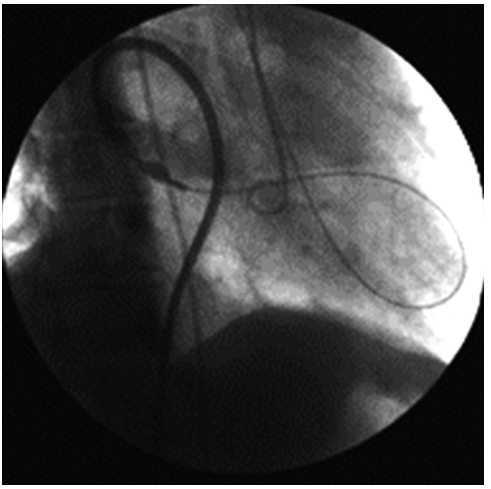


Fig. 8.2 Tracking of Inoue balloon over 0.032″ exchange length wire

Analysis of the case: In case of difficult LV entry due to tight, non-compliant mitral valve LV entry can be achieved by use of over the wire Swan Ganz. Sometimes despite Swan Ganz crossing, because of very small mitral valve Inoue balloon may still not cross. In this case, dilatation of the mitral valve with a smaller Inoue balloon or even peripheral balloon can be performed (which will permit crossing of larger Inoue balloon).

Case 3

A 21-year-old lady with severe mitral stenosis was taken up for BMV. Septal puncture appeared difficult as needle was repeatedly slipping into the coronary sinus. An angiogram of coronary sinus with the help of Mullin's sheath revealed hugely dilated coronary sinus due to LSVC draining into coronary sinus. A successful septal puncture was performed with the help of echocardiography. Septal tenting by the needle tip was confirmed on echocardiography before puncturing the septum. Subsequently, mitral valve was dilated successfully.

Analysis of the case: Septal puncture is often difficult in BMV when LA is large, particularly when septum is bulging. In this case, usual landmarks may not help. Here, better visualization of septum can be achieved with septal staining but in most difficult cases, transthoracic echo or even TEE can help identifying septum and accurately positioning the needle.

Case 4

A 30-year-old female with very tight mitral stenosis. 26 size Inoue balloon failed to cross the valve. Tried with 20 size balloon but could not succeed. An angiogram through balloon lumen showed that balloon was placed at the right position but was not able to cross due to very narrow lumen. Coiled wire was tracked to LV through balloon lumen, and then it was tried to track into LV over the wire without any success. Subsequently, coiled wire was exchanged with 0.032″ exchange length wire and mitral valve was dilated with 6 × 40 mm peripheral balloon. This facilitated tracking of Inoue balloon and successful dilatation of mitral valve **(Figs 8.3 and 8.4)**.

Analysis of the case: In case of difficult LV entry due to tight, non-compliant mitral valve, LV entry can be achieved by use of over the wire Swan Ganz. Sometimes despite Swan Ganz crossing, because of very small mitral valve orifice, Inoue balloon may still not cross. In this case, dilatation of the mitral valve with a smaller Inoue balloon or even peripheral balloon can be performed (which will permit crossing of larger Inoue balloon).

Case 5

After successful dilatation of mitral valve in a young lady, the balloon failed to deflate. Anticipating delay in the procedure, extra dose of heparin was given. Balloon was attempted to be deflated by aspirating from the other vent but very little fluid could be aspirated. Removing the wire stylet from

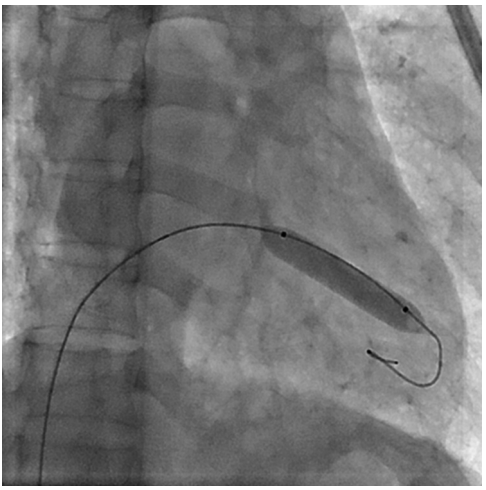


Fig. 8.3 Dilatation of very tight mitral stenosis by 6 mm × 40 mm peripheral balloon over 0.032″ exchange length wire

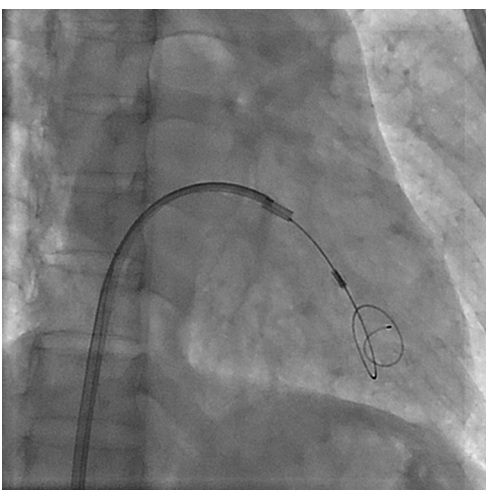


Fig. 8.4 Attempted crossing of mitral valve over the coiled wire placed in left ventricle

the other vent by unscrewing the stopcock from 'W' connector resulted in drainage of contrast and deflation of balloon.

Analysis of the case: In failed case, just waiting for a while results in deflation of balloon as 2 tiny holes are provided in the Inoue balloon may allow only slow seepage of contrast from the balloon. However, other Inoue type balloons like Accura, Blue arrow, etc. where this safety mechanism does not exist, other techniques will have to be applied. Generally, non-deflation is

related to higher contrast content in the dilatation fluid which results in the balloon acting like a one-way valve. Thus, a simple solution to the problem is introducing some more saline in the balloon (which will reduce the contrast: saline ratio) allowing a subsequent deflation of the balloon. However, in some cases puncturing by using septal puncture needle may be the only option.

CONCLUSION

Percutaneous dilatation of stenotic valves is a challenging field in cardiology due to inherent unpredictability in the outcome and steeper learning curve especially for BMV. With interventionalist taking up much more challenging cases for intervention the anticipated difficulties are also increasing. Knowledge of various simple maneuvers and techniques as well as troubleshooting and management plans can help in cases of emergency.[8]

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Chapter 9

Management of Resistant Thrombus: Catastrophe and the Cure

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INTRODUCTION

Thrombus containing lesion is a common substrate in patients with ACS particularly those presenting with acute MI. This is one of the reasons that the outcomes in primary PCI are worse than a regular PCI. Several studies have shown that primary PCI is associated with poorer acute outcomes (MACE rates between 2%–14%) versus standard PCI (<2%). Primary PCI is also associated with poorer long-term outcomes compared with standard PCI: Mortality (2–4.5% vs <1%. The reason for poor outcomes in primary PCI is persistent thrombus (sub-optimal result) or embolizations of formed thrombus (slow flow and no reflow phenomenon) and inappropriate vessel sizing which can lead to malapposed stent (acute thrombosis or SAT).It is estimated that BMS malapposition in AMI occurs in more than 1/3rd of patients post-procedure and can be even higher with the use of DES. On the other hand poor long-term outcomes can be related to inappropriate vessel sizing which can lead to malapposed stent (recipe for SAT) or deployment of stent over the site of plaque rupture. Nearly 1/4th of the patients undergoing primary PCI develop late stent malapposition over the period of 1 year. Persistent thrombus is one of the main culprits in this context. It not only directly leads to suboptimal result and development of slow flow and no-reflow phenomenon but may also contribute to inappropriate vessel sizing. Thus management of thrombus is one of the main concerns in patients with persistent/resistant thrombus.

HUGE THROMBUS BURDEN

While presence of some thrombus is very common in many types of lesion (practically invariable depending on how it is visualized), huge thrombus loads can be present in following situations:

- Acute STEMI (practically all)
- NSTEMI (majority)
- Aged degenerated vein grafts
- Stent thrombosis.

PATHOPHYSIOLOGY OF THROMBUS CONTAINING LESIONS

In a given case two factors are important in context with thrombus: overall thrombus load and organization of thrombus (whether fresh or organized thrombus).

Overall thrombus load: It has been demonstrated that patients with a higher thrombus burden, treated with DES, have a much higher acute failure (8.2%) and much higher MACE rate in STEMI.[1] Even in SVG intervention a huge thrombus burden dramatically increases the MACE rates and likelihood of death (10%).[2]

Organized thrombus: In context with organization of thrombus; treatment-wise, organized thrombus is much more difficult to manage. Thus while thrombolysis is very effective early (The Golden Hour) its efficacy goes on decreasing as the time between the onset of chest pain in AMI increases so that it is effective much less after a delay of 12 hours and if it is given after 24 hours its risks actually out-weigh benefit. Primary PCI has a much longer time window but after 3 days (OAT Trial), even here risks outweigh benefit.

EVALUATION OF THROMBUS CONTAINING LESIONS

- *Angiography*: One of the challenges of conventional angiography is that angiography is a poor tool for thrombus evaluation by itself. Even in patients with NSTEMI thrombus may be angiographically visible in less than 1/5th of the cases.[3] It being such a poor discriminator, presence or absence of thrombus should depend on context (ACS, SAT etc) rather than actual cine visualization.
- *IVUS*: It is another poor tool for evaluation of thrombus because it cannot differentiate between blood and thrombus.
- *OCT*: OCT is a better tool for evaluation of thrombus.
- *Angioscopy*: Angioscopy remains "Gold Standard" for evaluation of thrombus containing lesions. In patients with NSTEMI thrombus was demonstrated in 86% of patients (vs. 18% by plain angiography).[3]

Outcome of Thrombus Containing Lesions

The clinical sequelae of thrombus formation depend on thrombus itself or distal embolization (as a consequence of mechanical interference with the thrombus or plaque by various devices during PCI or even spontaneous embolization of thrombus or ruptured plaque). The consequences range in severity from small MIs to cardiogenic shock or even death. The outcomes depend on:

- Extent of embolization
- Size of the vascular bed involved
- Hemodynamic status
- Associated co-morbidities.

The outcomes can be objectively quantified in the following manner:

- *Angiographic outcome*: Presence of thrombus may be related to sub-optimal outcome related to persisting thrombus.
- *Epicardial blood flow*: The primary goal of primary PCI is restoration of epicardial blood flow to provide perfusion into the infarcted part of the myocardium. The rate of restoration of epicardial blood flow after PCI can be assessed by the angiographic reperfusion in myocardial infarction (TIMI) flow grade **(Table 9.1)**. Achieved TIMI flow rates have a prognostic impact of long-term outcomes after PCI.[4] Reduced perfusion is associated with significantly high 1-year mortality rate: 4.5% for absent perfusion, 3.9% for a reduced perfusion versus 1.4% for normal perfusion.[5] TIMI 3 flow rates are more difficult to achieve in thrombus containing lesions. Slow flow or no reflow phenomenon is much more common in thrombus containing lesions.
- *Myocardial blood flow*: Epicardial blood flow may not be synonymous with myocardial blood delivery. Despite restored epicardial blood flow, a substantial percentage of patients have signs of impaired myocardial reperfusion and therefore have an impaired prognosis.[6,7] On the other hand myocardial blood flow is the one which correlates more closely with clinical outcomes including infarct size and has even been correlative of 1-year all cause mortality in patients with STEMI in routine clinical practice. Myocardial blood flow is determined by myocardial blush grade (MBG) score **(Table 9.2)**.

Table 9.1 TIMI grade flow

'TIMI grade flow' is a scoring system from 0–3 referring to levels of coronary blood flow assessed during PCI:

- TIMI 0 flow (no perfusion) refers to the absence of any antegrade flow beyond a coronary occlusion.
- TIMI 1 flow (penetration without perfusion) is faint antegrade coronary flow beyond the occlusion, with incomplete filling of the distal coronary bed.
- TIMI 2 flow (partial reperfusion) is delayed or sluggish antegrade flow with complete filling of the distal territory.
- TIMI 3 is normal flow which fills the distal coronary bed completely.

Table 9.2 Myocardial blush grade

MBG of the myocardial infarct region was classified as:

0: No myocardial blush (persisting myocardial blush was also graded as 0 because this is considered to be extravasation of angiographic contrast medium associated with hemorrhage)

1: Minimal myocardial blush

2: Moderate myocardial blush

3: Normal myocardial blush

All compared with the MBG of myocardial regions of noninfarct-related arteries.

- *ST resolution*: This ECG sign is the classical sign for success of thrombolysis in absence of other sophisticated signs like angiography.
- *Infarct size*: The reason for attempting to reperfuse myocardium is to reduce infarct size as much as possible. Infarct size is measured enzymatically.
- *Preservation of LV function*: This is one of the clinical markers of successful reperfusion therapy in AMI.

However, besides thrombus embolizations distally, there is also small but finite possibility of thrombus migrating proximally (especially while performing the PCI procedure), which may be especially dangerous in case of occlusion of ostial left anterior descending artery (LAD) or left circumflex artery (LCx).

CASE EXAMPLES

Case 1

A 46-year-old male presented with acute onset, prolonged rest angina (window period 24 hours). Risk factors were diabetes, hypertension and dyslipidemia. ECG demonstrated anterior wall MI and low blood pressure and cold extremities suggesting cardiogenic shock. Echo revealed regional WMA in LAD with overall EF of 32% with no mechanical complications. ABG also suggested desaturation. IABP was inserted and patient intubated. Radial approach was obtained. Angiography revealed osteoproximal LAD occlusion. LCA was cannulated and LCx wired. LAD lesion attempted to be crossed with non-hydrophillic guidewire, but could not cross. Finally, lesion was crossed with hydrophilic wire. The lesion was dottered with 1.5 mm balloon and subsequently thrombosuction performed but there was no antegrade flow. Another attempt at thrombosuction performed but resulted in only slight antegrade flow. It was decided to resolve the problem by crushing the thrombus with low pressure dilatations with a balloon catheter. As such 2 × 15 mm balloon used but resulted in no improvement rather the flow diminished. Therefore decided to dilate with a larger balloon; 2.5 × 15 mm balloon. With this approach antegrade flow in the LAD was established but on the other hand thrombus migrated into LCx. Meanwhile patient developed severe chest pain, and VF, which was cardioverted. However, patient remained hemodynamically unstable. TPI was inserted. LCx was crossed with guide wire and thrombosuction performed followed by low pressure balloon dilations to crush the thrombus in LCx. Subsequently, intracoronary thrombolysis was delivered to deal with resistant thrombus. Finally, LAD lesion was stented with 3 × 32 mm DES but there was a TIMI 1 flow post-procedure. Stent boost revealed that middle part of the stent was probably under-deployed and so dilated with 4 mm NC balloon at nominal pressure. Finally, good end result. The nonculprit lesion was left alone for the moment.

Analysis of the case: This case reveals a particularly resistant thrombus, which while being dealt aggressively with PCI (repeated thrombosuction and

multiple balloon dilatations) culminated in migration of thrombus proximally into left main and LCx. The point where balloon catheter is withdrawn after an unsuccessful attempt at crushing the thrombus is especially susceptible for pulling thrombus back proximally. Some of the techniques for preventing thrombus migration include flushing the sheath before pulling out the balloon, keeping the guide deeply intubated to provide enough support for smooth balloon removal. In this case, despite these precautions the thrombus migrated proximally and was retrieved with thrombus extraction from LCx. The resistant thrombus was finally managed with intracoronary thrombolysis.

Case 2

A 56-year-old male smoker, presented with unstable angina. ECG demonstrated ST ↓, T wave inversion in precordial leads [LAD T wave inversion pattern]. Echo revealed anterior hypokinesia and overall EF – 45%. Angiogram revealed LM normal, mid LAD – 99% stenosis; discrete; with TIMI 2 flow distally, LCx – nondominant; normal. RCA – dominant; no significant stenosis and anterior wall hypokinetic. Patient was planned for PCI and stenting to mid LAD with DES. LCA was cannulated and the lesion crossed with guide-wire. Subsequently lesion dilated with balloon catheter. However, after balloon dilatation, there was slow flow in the LAD and the patient had bradycardia followed by cardiac arrest. Despite all attempts, patient could not be resuscitated and was lost.

Analysis of the case: Taking up a case with thrombus containing lesion for routine angioplasty with balloon catheters could be a big mistake. Such patients often require mechanical thrombectomy along with IV or IC GpIIb/IIIa antagonists. Once no-reflow occurs in a major artery it could be fatal.

MANAGEMENT OF RESISTANT THROMBUS/DISTAL EMBOLIZATION: CURRENT STRATEGIES

Currently the management of resistant thrombus and prevention of distal embolizations focuses more on mechanical approaches. These are especially more useful in cases of relatively organized clots. The reason is inability of antithrombotic/thrombolytic agents to penetrate heavy thrombus burden core.

Multiple Low Pressure Balloon Dilatations

Often called a "***poor man's thrombectomy***" is one way to handle resistant thrombus. However, it may not be particularly effective and therefore should be employed only in cases of huge thrombus when thrombectomy device is not available or in conjunction with it. Sometimes after achieving TIMI III flow post balloon dilatation the lesion may be left alone (because of thrombogenic milieu and general arterial spasm) and stented at a later date.

Distal Protection Devices

Technically filter devices can trap the embolic material in the mesh of the filter and balloon devices and prevent embolization by occluding the distal lumen (permitting aspiration of the debris). However, these devices by themselves can contribute to trauma to the vessel and consequent embolization of thrombotic debris. Thus, in the setting of AMI they may fail to show any benefit. Currently, there use may be restricted to AMI in vein grafts.[8]

Proximal Protection Devices

Their use is based on balloon occlusion proximal to the lesion and aspiration of released debris during the procedure.[9] However, their benefit in real world situation has not been demonstrated yet.

Direct Extraction of Thrombus via Guide-catheter

This technique may sometimes work when thrombectomy catheters are not available, especially when the thrombus is proximal and catheter wide bore. However, this technique carries a risk of proximal migration of thrombus.

Thrombectomy Devices

These devices directly extract the thrombus reducing the thrombus load. However, by themselves they may cause trauma to the vessel wall contributing to embolization per se: the more invasive the device, the poorer the outcomes. Thus, Posis Angiojet™ while it causes aggressive de-thrombogenization has not shown any benefit (perhaps related to mechanical trauma caused by it). There is some controversy as to the use of other less invasive devices but in day-to-day clinical practice, even they might not be useful in all the cases. Currently, their use may be warranted in lesions with visible thrombus/high thrombus load.

Plaque Trapping Devices

Technically, they are ideal because they themselves cause no mechanical trauma but at the same time they limit the embolization by trapping the thrombus. Sometimes even a regular stent can do the job but currently, some specialized devices are available.

Covered Stents

While they may be very effective thrombus trapping devices, but by virtue of their construction, they may contribute to more stent thrombosis (therefore no reduction in MACE) and higher restenosis rate.

Embolic Protection Stents – MGuard

The MGuard stent is an ultra-thin, micron level, flexible mesh net fabricated by circular knitting. The technology combines the clinical benefits of stent efficacy with embolic protection at the target lesion site. The way it works is that during the stent deployment, the net stretches and slides over the expanding stent struts, creating custom–designed pores parallel to the arterial wall. Thus embolic debris is captured between the fiber net and the arterial wall and isolates the pro-thrombotic intimal components from the blood stream. The MASTER trial enrolled a total of 433 patients with STEMI presenting within 12 hours of symptom onset undergoing PCI who were randomized at 50 sites in 9 countries to the MGuard EPS (n = 217) or commercially available bare metal or drug-eluting stents (n = 216). Significantly more patients developed ST segment resolution (57.8% vs 44.7%, P = 0.008) 60–90 minutes post-procedure, the primary endpoint. ST-segment resolution is historically known to be a strong predictor of mortality. Secondary endpoint clinical outcomes continued to show a lower mortality rate with the MGuard EPS compared to the control (1.0% vs 3.3%, p = 0.092) at 12 months as it was at 6-months.[10] However, being a bare-metal stent the TLR rates were expectedly higher.

Stentys Stent

Stentys is a nitinol based self expanding stent which can trap the thrombus. The APPOSITION III study was a large, real-world, 1000-patient registry designed to measure the long-term safety and performance of the STENTYS Stent. The primary endpoint was MACE at 12 months, with subsequent follow-up at two years. Safety was demonstrated with only 1.2% mortality and low MACE rate at 30 days. Final one-year data presented at ACC. 13 by G Montalescot et al. showed a low cardiac death rate (2% with Stentys vs 3.9% with other stents).

Systemic administration of antithrombotics and even thrombolytics are useful as a supplement to mechanical approaches. The reason is that while mechanical approaches target in-situ thrombus, they fail to address thrombus that may form in the myocardial bed or that which was previously embolized. Thus a combination of both, mechanical and pharmacological approach may be ideal in most cases.

Local Thrombolytic Therapy

Initially it was the preferred therapy (before mechanical approaches were available) but high bleeding risk with this approach was the major limitation, so that now its use is restricted to very few selected cases.

Systemic (IV) Antithrombotic Therapy

IV GpIIb/IIIa antagonists have shown good efficacy but are limited by limited penetration into the organized thrombus.

Local Intracoronary Delivery

Local delivery via guide catheter or end-hole catheters may be more effective than IV delivery and also safer because they may utilize only bolus dose and may not require a maintenance dose.[11,12] However, they may still be limited by incomplete penetration into the thrombus and the possibility of wire loss or introduction of air embolus. Further, in case of bifurcation lesion it will result in significant drug loss into non-infarct related artery.

Local Therapy with Clear Way Rx Catheter

The Clear Way Rx is a monorail, highly trackable, low-profile device that is easy to use without the loss of wire. In an occluded thrombotic vessel, positioning of this catheter inside the thrombus increases the contact surface within the thrombus addressing residual clot within the lumen but also the clot embolized beyond the occluded artery.[13,14]

Laser

In case of resistant thrombus laser is another option which can be used if the facilities exist in cath lab.

Persistent Thrombus

Sometimes despite all efforts thrombus may remain and it may be best to leave them alone in those cases.

CASE EXAMPLES

Case 3

Patient was a 59-year-old female, hypertensive smoker, admitted with chest pain; onset of symptoms 24 hours before. She was having angina on exertion for past two months. EKG demonstrated ST elevation + Q waves in the inferior leads. Echocardiogram revealed LVEF 30%; posteroinferior akinesia. IV heparin, Ticagrelor 180 mg PO, ASA 500 mg IV were administered in the ER and patient sent immediately to the catheterization laboratory. Right radial artery approach was chosen and angiogram revealed a significant stenosis of proximal LAD, however, a thrombotic occlusion on aneurysm of proximal RCA was the culprit lesion. RCA was cannulated with JR4 6F guiding catheter and crossed with BMW™ guidewire. Repeated thromboaspiration with an Eliminate™ catheter 6F (Terumo) was carried out and intracoronary bolus of abciximab followed by continuous IV infusion given **(Fig. 9.1)**. Multiple balloon dilatation, with increasing diameter (2.5/15 mm; 3.0/20 mm; 4.0/15 mm @ 12 atm) were also carried out. However, repeated angiograms over a period of 40 minutes still revealed slow flow. Therefore, thromboaspiration was still carried out. Finally, an acceptable flow (TIMI 2-3) was obtained;

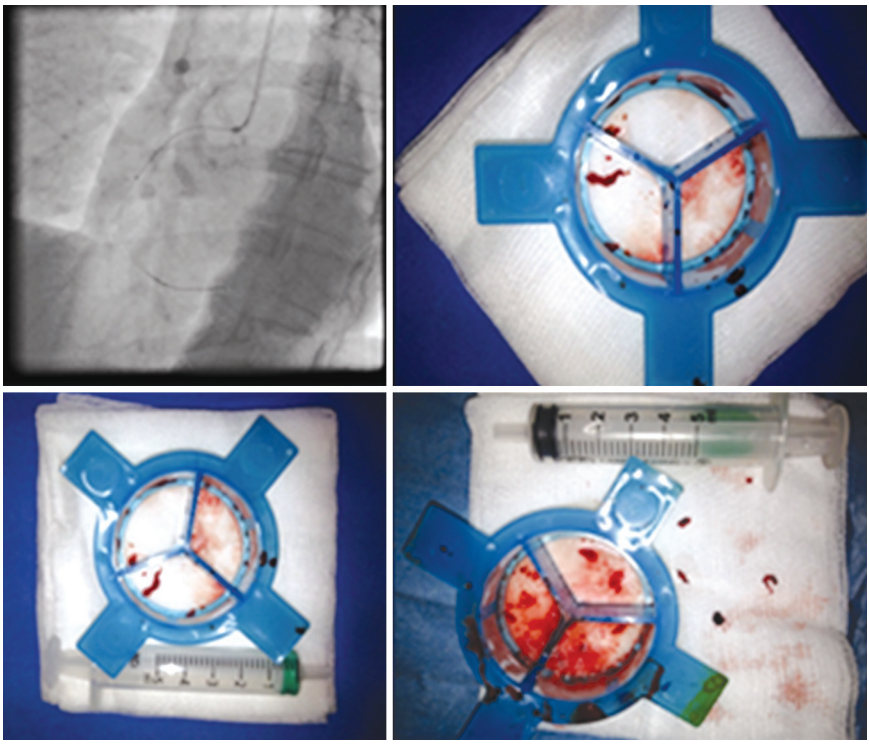

Fig. 9.1 Thrombus extracted after thrombosuction with eliminate device

symptoms and ST elevation started settling. However, despite apparent stabilization and because a huge quantity of thrombus was still persisting, inside the aneurysm, albeit without stenosis, it was decided to continue the IV infusion of abciximab for 12 hours and perform another coronary angiogram after a few days. Post-procedure, IV Dopamine and Dobutamine were needed for hemodynamic support the following days. Repeated echocardiograms showed RV dilatation and reduced LVEF (40–50%). Ticagrelor was switched to clopidogrel. Coronary angiogram 10 days after revealed persistent thrombus in the middle tract of RCA, (although reduced) with TIMI 3 flow. PL branch was still occluded, with collateral flow from the left coronary artery. A significant stenosis on proximal LAD was confirmed. Twenty days after the procedure a cardiac MRI was performed, showing a scar in the territory of the RCA, but viable tissue on every other area of both the left and right ventricles. After one month a coronary angiogram was performed and complete recanalization of RCA carried out without either residual stenosis or thrombus. Subsequently, PCI on LAD- diagonal branch, with implantation of three DES on the LAD in overlap (Xience Prime™ 3.5/18 mm + 3.0/15 mm + 3.0/12 mm) and balloon angioplasty only in the diagonal branch, with final kissing-balloon was performed. The patient was discharged (sent to a rehabilitation site) after one week from the last procedure. Echocardiogram: severely reduced

LV function (EF 30–35%), normal dimension of both ventricles and severely reduced RV function; moderate tricuspid insufficiency, PAPs 30 mm Hg. ICD implantation was planned if LV function continued to be reduced <305. Oral anticoagulation with Warfarin 5 mg was started in view of coronary aneurysm.

Analysis of the case: This case elegantly shows that the aim of primary PCI is to achieve as much flow as possible, and not to stent the artery. Once the prothrombotic state is over, the residual stenosis can be stented with low risk for no-reflow or slow flow at a later date.

Case 4

A 29-year-old euglycemic, hypertensive, gentleman, presented with choking associated with profuse sweating and difficulty in lying down for the last 2 hours. He had a recent history of undergoing strenuous exercises at the gym. ECG revealed ST elevation MI—inferoposterior STEMI with RVMI. He was taken up for PPCI after optimal pharmacological loading. Coronary angiogram revealed a severe thrombotic disease of proximal RCA, normal LAD and LCx. After obtaining informed consent, RCA was cannulated using 6F JR guide catheter. An 0.014 inch BMW™ universal II guidewire. Multiple runs of thrombosuction were done and intracoronary eptifibatide was given. Huge chunks of thrombus were removed. Check angiogram revealed no residual thrombus or stenosis or dissection with TIMI III flow. Hence, Stenting was not done. His initial biochemical parameters were within normal limits except for the mild anemia, raised plasma homocystine levels and dyslipidemia with elevated fasting glucose. Tests for thrombophilia were normal. His hospital stay has been uneventful. Predischarge LV function was normal and had no LV clots and was discharged with the advice to continue further medical management as outpatient and lifestyle modification.

Analysis of the case: "Stentless" PPCI is a reasonable option in many very young (<35 years) AMI patients and has the advantage of avoiding the use of stent in many of these primarily non-atherogenic occlusions. The total/near-total thrombus as the occlusive culprit lesion allows for the success of this strategy. This strategy can be refined by adjunctive use of imaging techniques like IVUS or OCT to assess any angiographically invisible underlying stent-needing dissections, etc.

Case 5

The case was of an acute MI in a smoker male, who presented late and as a consequence the thrombus was partially organized, hard and could not be sucked out using 7F Thrombuster™ catheter, even after double boluses of weight adjusted intracoronary eptifibatide injections. Finally, PTCA balloon was used to crush the thrombus with only partial success. Subsequent, manual suction after ballooning also did not help. Ultimately the whole length of LAD which contained thrombus was stented with good end result.

Analysis of the case: In many cases of resistant thrombus a thrombus trapping device is required. It could be an MGuard stent or Stentys device, but in case these devices are not available even routine stents can help (in some cases).

Case 6

A 54-year-old male, DM type 2–10 years, HT – 10 years, Smoker – 20/day for 10 years, presented to triage at 9 pm with acute severe retrosternal chest pain for last 4 hours. ECG revealed acute inferior MI. Echocardiography revealed severely hypokinetic inferior wall and posterior wall, akinetic apex with overall LVEF 35% with moderate MR. Coronary angiogram revealed LAD–80% proximal, LCx–OM 70%, RCA–70% mid–100% distal with thrombus (with a huge thrombus load). The 5F Spider™ distal protection device used and the lesion dilated with balloon catheter but the thrombus was still persisting. Therefore it was decided to stent; as such MGuard™ – 3.5 × 39 was deployed at 10 atm. Post-stent deployment, distal thrombus in healthy vessel was still persisting. Export™ thrombosuction was subsequently performed followed by multiple balloon dilatations. However, post Export™ thrombosuction, in-stent thrombus extending upto DPD was still visualized. DPD was removed. Check angiogram revealed a large thrombus persisting distal to the stent. Therefore, another MGuard™ 3.5 × 24 stent was depoloyed at 10 atm with a good result.

Analysis of the case: Sometimes persistence pays. MGuard™ is an embolus protecting stent which traps the embolus within its wire meshes. It is easy to use.

Case 7

A 52-year-old, diabetic, hypertensive, male presented with anterior STEMI, and was thrombolysed with injection tenecteplase. Patient had a resolution of ECG changes and angina relief following this. However, developed re-elevation of ST segments with angina the next day. He was taken for rescue PCI. Coronary angiogram revealed total thrombotic occlusion of proximal LAD, separate origins of LAD and LCX. During thrombus aspiration thrombus migrated into LCX, he developed angina with hypotension. LCx was wired with floppy wire, thrombus aspiration continued, flow established in LAD and LCx. Subsequently, intra-coronary tenecteplase was given via Clear way™ catheter. There was spontaneous dissection in proximal LAD which was stented with DES. Patient became stable post-procedure, discharged after 1 week.

Analysis of the case: This case once again demonstrates the importance of care while performing aggressive angioplasty especially in the context of thrombus containing lesion. In many cases of resistant thrombus use of just mechanical devices may not be enough and they will have to be supplemented with systemic anti-thrombotic/thrombolytic therapy. Intracoronary delivery of such an agent via Clear way™ catheter may be safe and effective in this context.

Case 8

A 35-year-old male smoker presented with recurrent chest pain for the last 2 days. He was taken to a peripheral center where he was diagnosed to have anterior wall MI and shifted to tertiary care center with cath lab facilities center. Patient had ongoing angina. He was taken up for primary PCI. Coronary angiogram showed a normal left main, LCx and RCA. The LAD was occluded at the ostium. Thrombosuction was done and a large amount of **white thrombus** was aspirated. Balloon dilatation was performed and stenting performed with a 3.5 × 33 mm Pronova™ stent. It was postdilated with 3.5 mm balloon. However, a large thrombus was noted prolapsing into the left main at the proximal send of the stent. Thrombosuction was attempted and postdilatation was done to squash the thrombus but without success. Hence another 3.5 × 13 mm Pronova™ stent was deployed in the left main and postdilated to 4.0 with good result. He was given ASA, clopidogrel and tirofiban infusion. After about 12 hours, patient had recurrence of chest pain with ECG changes and was taken to the cath lab. Thrombus was noted occluding the ostium of LCx and immediately POBA was performed. However, the thrombus prolapsed into the LM/LAD and could neither be aspirated nor squashed with balloon. It kept on moving to and fro and hence, stent was deployed in the LCx and kissing balloon dilatation done. Despite all efforts, the thrombus continued to be trapped at the carina and the guide was upgraded to 7F and postdilated to 4.0 in LCx and 3.5 in LCx (kissing balloon). The smashed thrombus persisted! And the procedure was abandoned. In next 2 days the patient stabilized and could be extubated and IABP removed. His oral antiplatelet agent was changed to prasugrel and aspirin along with tirofiban for 48 hours.

Analysis of the case: Persistent and reforming thrombus is one of the nightmares in catheterization laboratory. While all attempts must be done to resolve this problem, it must be realized that ***"Excellent is the Enemy of Good"***.

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That's me in the corner
That's me in the spotlight, Trying to keep up with it
And I don't know if I can do it
I'm Losing my religion

Chapter 10

Complications of Rotablation and its Management

Zehra Husain, Gianluca Rigatelli, Ramesh Daggubati

The potential complications associated with Rotablation (RA) are similar to those seen with all percutaneous coronary intervention (PCI) devices. *They can be classified into clinical, angiographic and device related*. However, because the Rotablator ablates plaque material, and delivers micro-particulate debris to the distal coronary bed, the possibility of impaired coronary perfusion during RA is greater than with other PCI devices.

CLINICAL COMPLICATIONS

The major clinical complications that may be associated with RA include death, need for emergent coronary artery bypass graft (CABG) surgery, or non-Q-wave myocardial infarction. The incidence of major complications from RA has ranged from 3% to 6%.[1-3] But prospective and retrospective studies have demonstrated the efficacy of RA in complex and often calcified lesions **(Table 10.1)**. Other clinical scenarios seen are bradycardia/AV block and hypotension.

The incidence of bradycardia is more common with right coronary artery (RCA) lesions, dominant LCx and can occur with proximal LAD when large burrs (2.25 mm) are used. It can happen instantly after activating the burr or can follow the slowing trend of the burr. The mechanism is unclear but various theories include microparticles interfering with vessels perfusing the AV node and the vibrations/heat of the burr causing reflex bradycardia.

Management includes limiting ablation times (<15–20 sec), deactivating the burr when slowing of heart rate is noted, asking the patient to cough and giving atropine. Some operators advance pacer wire into the inferior vena cava (IVC) and place it into the RV when needed. Present recommendations are the placement of temporary pacer in patients undergoing treatment of RCA, or dominant LCx or proximal LAD giving collaterals to the RCA.

Hypotension can occur due to the injection of various drugs (nitroglycerin, diltiazem, verapamil, adenosine) after each ablation run to reduce the risk of coronary vasospasm. Therefore, it is recommended that patients be well hydrated prior to the procedure and their systolic blood pressure be maintained above 100 mm Hg.

Non-Q-wave myocardial infarction (MI) after coronary interventions has been recognized as elevations of CK-MB as low as 1–3 times the normal. Development of myocardial ischemia is a significant concern during

Table 10.1 Clinical outcome after rotational atherectomy

Ref. (Year)	N	Lesion type	Major comp. %	Non-QMI comp. %	Rest. %
Dietz ('91)[32]	106	B, C with 44% CTO	1.9	4.7	42
Teirstein ('91)[33]	42	71% > 10 mm	4	19	59
Barrione ('93)[34]	166	Complex 63%	2.4	8.4	
Gilmore ('93)[35]	108	N/A	4.6	2.8	
Guerin ('93)[36]	61	$B_2$100%	3.2	6.6	
Safian ('93)[23]	104	A 20%, B 76%, C 4%	7.7	2.9	51
Stertzer ('93)[22]	302	A 7.5%, B/C 92.5%	3.6		37
Ellis ('94)[37]	316	A 24%, B 70%, C 6%	3.4	5.7	
Vandormael ('94)[38]	215	$B_1$15%, $B_2$72%, C 13%	2.3		62
Warth ('94)[21]	743	A 27%, B 59%, C 14%	3.4	3.8	38
MacIsaac ('95)[39]	2,161	Ca 50%, Non-Ca 50%	3.5	8.8	

Note: Major complications include death, emergent bypass surgery, or QMI. Non-QMI is CK>2 times the norm with positive creatine kinase-myocardial band. Missing data means that data were not available.

Abbreviations: A, B, C, American College of Cardiology/American Heart Association lesion classification; Ca, calcification; Comp., Complications; CTO, chronic total occlusion; N/A, not applicable; QMI, Q-wave myocardial infarction; Ref., reference; Rest., restenosis.

RA because the abrading mechanism of the burr results in delivery of atherosclerotic debris to the distal circulation. Additional concerns specific to rotational ablation and development of ischemia include activation of platelets and microcavitation during activation of the burr. Patients may develop significant symptoms of ischemia during RA, and these symptoms may persist after completion of burr runs, and in the face of normal angiographic coronary perfusion. It has been theorized that periprocedural CK-MB elevations may be related to platelet activation during PCI procedures. In several recent trials, glycoprotein IIb/IIIa antagonists have shown a benefit in reducing the incidence of CK-MB elevations after PCI procedures.

ANGIOGRAPHIC COMPLICATIONS

These include slow flow/no reflow and vasospasm, dissection, perforation and side branch occlusion. These can potentially be avoided by using proper procedural technique.

Coronary dissection can occur with moderate to severe angulated lesions, because at angles, burr does not follow the natural course of the vessel. Unfavorable guide wire bias (divergence from the central axis of the vessel) can increase the chance of dissection and burning of normal tissue. Therefore, placement of the guide wire plays a paramount role in establishing the cutting vector of the device. Location of dissection plane usually remains in the calcified plaque, which is ablated and can be managed by deploying an oversized balloon at low pressure to tack up the tissue, followed by stenting.

Coronary perforation can occur in patients with extreme vessel angulation or tortuosity due to trajectory of guide wire, oversizing the burr, or advancing the burr too rapidly. Therefore, to avoid perforation, it is important to minimize guide wire bias by proper co-axial guide catheter and guide wire placement. Undersizing the burrs in severely angulated lesions, especially those that are straightened with guidewire or showing pseudolesions is crucial. Pecking technique should be used to avoid excessive cutting. Conventional treatment strategies for perforation include prolonged balloon inflation, covered stent implantation, coronary artery bypass graft (CABG) surgery, and coil embolization.[4]

Slow Flow/No Reflow

Rotablator atherectomy has this unique mechanism that relies on plaque pulverization and microparticle embolization to achieve luminal enlargement.[5,6] Experimental studies suggest that these particles are usually small enough to pass through the capillary circulation and are subsequently removed by the reticuloendothelial system.[7-11] Other experimental[7] and clinical[6,12,13] studies reported no significant impact on resting wall motion, myocardial perfusion, or other clinical markers of myocardial ischemia after uncomplicated Rotablator atherectomy. In contrast, the physiological effects of Rotablator-induced microembolization are unclear. Early trials with the Rotablator reported a low incidence of no reflow (1.2%) and vessel spasm (1.6%).[14] However, initial clinical experience with the Rotablator demonstrated that the incidence of vessel spasm and slow flow, or the no-reflow phenomenon, appeared to be greater than that observed with conventional PCI or other devices used in PCI.[15]

Detailed angiographic analyses after rotational atherectomy reported slow or no reflow in 7–8% of patients with associated myocardial infarction in 25%[15,16] and ECG evidence of ischemia in 43% of patients.[17]

Safian et al. demonstrated that angiographically successful Rotablator atherectomy is associated with increases in basal and hyperemic coronary blood flow velocity and normalization of the diastolic predominance of coronary blood flow (only in the left coronary artery) but persistent impairment in CFR despite increases in hyperemic blood flow.

Although the exact mechanism of slow flow and no reflow has not been elucidated, it appears to be multifactorial and includes particulate debris, platelet activation, and vasospasm. Others factors that may contribute include microcavitation, impaired local synthesis of EDRF, neurohumoral reflex and lower epicardial vessel pressure and increased LVEDP.

Technique modifications, in addition to adjunctive therapy have assisted in reducing the incidence of impaired distal coronary perfusion during rotational atherectomy.

ADJUNCTIVE THERAPIES

Advancements in adjunctive therapies are an area of significant progress in rotational atherectomy.

Glycoprotein IIb/IIIa Inhibitors

Recently, it has been recognized that platelets may contribute to the development of impaired coronary perfusion during RA. Reisman demonstrated increased platelet aggregability in an *in vitro* rotational atherectomy model.[18] Williams et al. using an *in vitro* model, reported that activation of platelets during rotational atherectomy is dependent upon burring speed and can be inhibited by abciximab.[19] Finally, Koch et al. evaluated 75 patients undergoing rotational atherectomy with or without abciximab.[20] All patients underwent Tc-99m Sestamibi scintigraphy before, during, and after treatment. The patients treated with abciximab had a significantly lower incidence of transient hypoperfusion during RA, resulting in a significantly lower incidence of periprocedural myocardial infarction and creatine phosphokinase (CPK) elevation **(Tables 10.2 and 10.3)**. Given the encouraging results of early evaluations of GP IIb/IIIa inhibitors with rotational atherectomy, and the consistently positive results with GP IIb/IIIa

Table 10.2 Influence of abciximab on rotational atherectomy-induced hypoperfusion

Group	*Incidence*	*Regions*	*Pre-R (%)*	*R (%)*	*Post-R (%)*
A (Abciximab)	10/30 (33%)*	1.4 ± 2.5†	76 ± 15	67 ± 14†	80 ± 13
B (Control)	39/45 (87%)	3.3 + 2.5	76 ± 15	56 ± 16	76 ± 15

Note: This study demonstrates the importance of the reduction of platelet activation in reducing the impact on the myocardial segment subtended by the treatment artery.
Incidence: Patients with perfusion defects; Region: number of regions with significantly reduced perfusion; per-R, R, post-R: myocardial perfusion in % of individual maximum before, during and after PTRA.
*$p < 0.001$ vs group B; †$p < 0.01$ vs group B.

Table 10.3 Impact of abciximab on creatine phosphokinase elevations

	Peak CK	*CK rise*	*1–3 XNL*	*>3 XNL*	*Any abn. CK*
ReoPro	145 ± 13	89 ± 18	10%	3%	13%
Control	238 ± 20	173 ± 45	17%	9%	25%
p value	0.02	0.03	0.15	0.07	0.02

Note: This study demonstrates the significant reduction in creatine phosphokinase for rotational atherectomy when using glycoprotein IIb/IIIa inhibitor.

inhibitors in multiple PCI trials, many operators use a GP IIb/IIIa inhibitor in the great majority of rotational atherectomy cases.

Vasodilators

Liberal use of coronary vasodilators improves coronary flow and reduces the incidence of vasospasm and no reflow. Intracoronary nitroglycerin (100–300 μg), verapamil (100–300 μg), diltiazem (100–300 μg), and adenosine (18–30 μg) are used prophylactically or therapeutically to improve coronary perfusion during RA. In cases of severe spasm or no reflow, selective administration of these agents by an infusion catheter may be more effective in improving distal blood flow. An additional benefit of selective administration is the ability to give larger doses of vasoactive agents without compromising systemic blood pressure and hemodynamics. In one study, the prophylactic use of intracoronary adenosine prior to RA of complex lesions was associated with a significant reduction in the incidence of no-reflow.[21]

Another significant change in technique is the addition of vasodilators and anticoagulants to the flush system that is attached to the sheath of the Rotablator system. The current additives to the flush system include verapamil 10 μg/mL, nitroglycerin 4 μg/mL, and heparin 3 U/mL. A small clinical study evaluating this drug cocktail in the Rotablator flush system showed good procedural success rates with low complication rates.[22] Recently, a number of operators have advocated the intracoronary injection of nitroprusside (50–100 μg) to treat vasospasm or no reflow. No formal evaluation of this intervention is available at present.

Technique Modifications

Techniques to reduce the incidence of slow flow/no reflow include:

- Gentle and slow advancement of the device with intermittent retractions (helps in preventing drop in <5000 rpm, significant generation of heat, and re-establishment of flow for particle clearance),
- Limiting the ablation time to 15–30 sec,
- Increasing the time between the ablations and
- Ablating at a slower speed of <150,000 rpm. Higher speeds have been associated with increased complications **(Table 10.4)**. Low speed also appears to have salutary effects **(Table 10.5)** on CPK elevation, and this is most probably based on the reduction seen in platelet activation **(Fig. 10.1)**.[18]

DEVICE COMPLICATIONS

Majority of device related complications happen due to the use of it outside the standard operations. These are burr entrapment, detachment, stalling and guidewire fracture.

Table 10.4 Advancement of the burr and clinical events

Predictors of MACE	*p(n = 216)*
Decelerations >5,000 rpm >10 s	0.03
Decelerations >7,000 rpm >5 s	0.02
Any decelerations >10,000 rpm	0.96
Mean time per run(s)	0.71
Total RA time(s)	0.61

Note: These data are derived from the chart recordings of rotational speed during treatment in STRATAS (Study to Determine Rotablator and Transluminal Angioplasty Strategy). It demonstrates a significant correlation between rotational speed and major adverse cardiac events (MACE)
Abbreviations: RA, rotational atherectomy; rpm, revolutions per minute.

Table 10.5 Retrospective comparison of low speed (<150,000) to conventional speeds (>160,000)

	STRATAS			
	LSRA n = 185	*Aggressive n = 53*	*Standard n = 51*	*ERBAC n = 215*
CKMB 3.8	8.6%	12.5%	14.7%	–
CKMB >8	4.8%	9.4%	7.8%	–
QMI	1%	1.9%	0%	3.2%
TVR	21%	37.5%	30.4%	46%

Note: Comparing the baseline data of several rotational atherectomy studies to a prospective low-speed registry demonstrated less frequent creatine phosphokinase elevations with the reduced speeds.
Abbreviations: CKMB, creatine kinase-myocardial band; ERBAC, excimer rotablator balloon angioplasty comparison; QMI, Q wave myocardial infarction; STRATAS, study to determine rotablator and transluminal angioplasty strategy; TVR, target vessel revascularization.

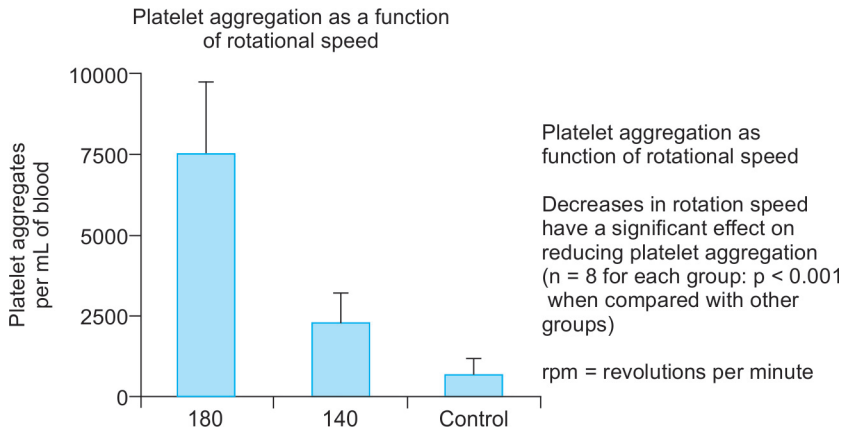


Fig. 10.1 Platelet aggregation as a function of rotational speed

Burr detachment is associated with excessive force applied to remove the nonspinning burr from the tortuous artery. To avoid this, it is important not to use burrs with <0.004″ clearance from the guide catheter. Verification of coaxial position of the guide catheter with the artery while exchanging the burr is essential to prevent trapping at the tip. If burr detaches from the drive shaft cable, the entire guidewire and catheter system should be withdrawn after giving adequate intracoronary vasodilators (the distal tip of the guide- wire is 0.017″ and will prevent the burr from exiting the end of the guidewire).

Burr stalling happens with significant resistance to rotation, kinking of the air hose, overtightening of "Y" connector, burr to artery ratio of 1, aggressive advancement in tight lesions, spasm in platform zone or operating without saline infusion. To prevent this, the DRAW pre-procedure test is performed.

Drip—saline drip from bottom of advancer and catheter

Rotate—burr is rotating and RPM's are stable

Advancer—free movement of advancer knob

Wire—is visible and brake is functioning.

Wire fracture results from excessive rotation of the burr in angulated and tortuous arteries, longer ablation times and formation of wire loops. To minimize the problem, the wire should be kept out of small branches, repositioned frequently during excessively long ablations, wire tip prolapsing should be avoided and contrast injected to demonstrate flow around the guidewire. Fractured portions may be retrieved with different types of snares and retrieval baskets. If unsuccessful and of no hemodynamic consequence, they can be left alone, and opting for conservative medical management.

Burr Entrapment: The Worst Nightmare for an Interventionalist

Entrapment of a rotablation burr is defined as the entrapment of the burr in a coronary lesion with the inability to rotate or retrieve it. It is a rare but very serious complication of RA. It can happen not only in tortuous calcified lesions but also in relatively straight proximal coronary segments.

In the literature, a total of 12 reports with 18 cases of a stuck rotablator burr have been identified.[23-33] In addition, Yokoi et al. reported on six cases of burr entrapment during RA in an experience of 1,212 RA procedures, but a full description of these cases is not available. The characteristics of all those patients and procedures are shown in **Table 10.6**.

The most popular solution described is surgical removal with coronary bypass grafting. The surgical approach is necessary because of the inability to remove the device with interventional techniques or because of coexisting vessel perforation.

Interventional approaches can be divided into two techniques:

1. Dilatation of the lesion with balloon angioplasty, and
2. Burr removal facilitated by deep catheter intubation.

In order to mobilize a stuck burr with conventional angioplasty devices, a second arterial puncture to gain additional access for a second Guide catheter

Table 10.6 Description of the characteristics of 18 patients with a stuck rotablator burr and their management

Case	Author	Demographics	Clinical presentation	Lesion	Calcification (visual estimation)	Burr (mm); Burr/artery ratio	Peak motion speed (rpm)	Management
1.	Alexiou et al.[9]	68 years, male	SA	RCA	N/A	N/A	N/A	Surgical removal
2.	Alexiou et al.[9]	70 years, female	SA	RCA	N/A	N/A	N/A	Surgical removal
3.	Endo et al.[10]	75 years, male	ACS	LAD, native	Severe	N/A	N/A	Surgical removal
4.	Gambhir et al.[11]	N/A	N/A	N/A	N/A	N/A	N/A	Surgical removal
5.	Kaneda et al.[12]	80 years, female	SA	LAD, native	Severe	1.25; 0.5	200,000	Surgical removal
6.	Shekar et al.[13]	73 years, male	SA	LCx, perforation in old proximal stent with stuck rotablator	N/A, CTO	1.25; N/A	N/A	Surgical removal
7.	Shekar et al.[13]	58 years, female	SA	RCA, in-stent restenosis	N/A, CTO	1.25; N/A	N/A	Surgical removal
8.	Sulimov et al.	46 years, male	ACS	LAD, freshly implanted stent	Moderate	1.75; 0.7	150,000	Second wire, post-dilatation in lesion
9.	Sulimov et al.	70 years, male	SA	RCA, native	Severe	1.75; 0.58	150,000	Second wire, post-dilatation in lesion
10.	De Vroey et al.[14]	67 years, female	ACS	LAD, native	Severe	1.5; 0.55	160,000	Second wire, post-dilation in lesion
11.	Grise et al.[15]	83 years, male	SA	LAD, native	Severe, CTO	1.25; N/A	150,000	Second wire, post-dilatation in lesion

Contd...

Contd...

Case	Author	Demographics	Clinical presentation	Lesion	Calcification (visual estimation)	Burr (mm); Burr/artery ratio	Peak motion speed (rpm)	Management
12.	Hyogo et al.[16]	55 years, male	SA	RCA, native	Severe	1.5; 0.52	170,000	Second wire, post-dilation in lesion
13.	Sakakura et al.[17]	67 years, male	SA	RCA, native	Severe	1.25; 0.42	217,000	Second wire, post-dilatation in lesion
14.	Prasan et al.[18]	77 years, male	ACS	LCx, native	Severe	1.5; N/A	180,000	Percutaneous snare, disassembly or rotablator
15.	Sulimov et al.	38 years, male	SA	LAD, freshly implanted stent	No	2.0; 0.5	190,000	Deep intubation with manual pullback
16.	Kimura et al.[19]	84 years, male	SA	LCx, native	Severe	1.25; 0.42	220,000	"Mother and child" catheter
17.	Cunnington and Egred[21]	78 years, female	ACS	LAD, native	Severe	1.25; 0.36	N/A	"Mother and child" catheter
18.	Sulimov et al.	72 years, female	SA	LCx, native	Severe	1.25	160,000	Second wire, post-dilatation in lesion, strong manual pulback

Abbreviations: N/A, data not available; ACS, acute coronary syndrome; CTO, chronic total occlusion; LAD, left anterior descending artery; LCx, left circumflex artery; RCA, right coronary artery; SA, stable angina.

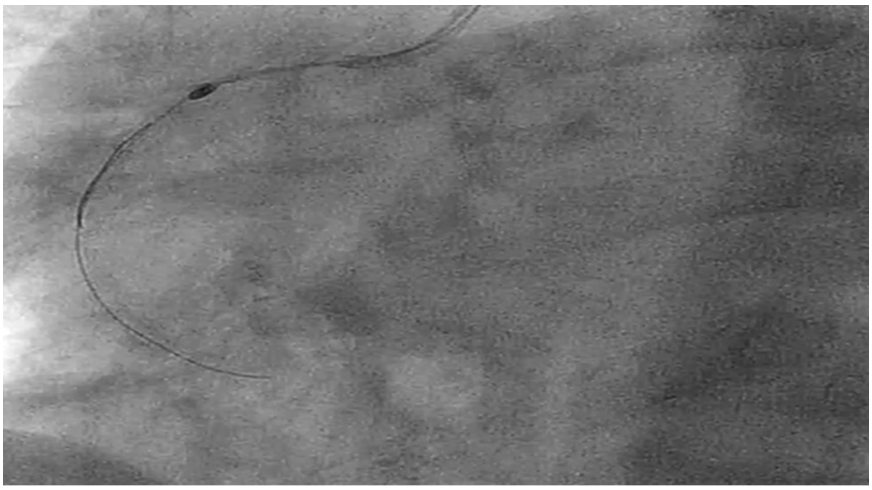


Fig. 10.2 Rotablator burr is stuck. A second wire is passed beyond the burr with attempt to dislodge the burr with an over-the-wire balloon

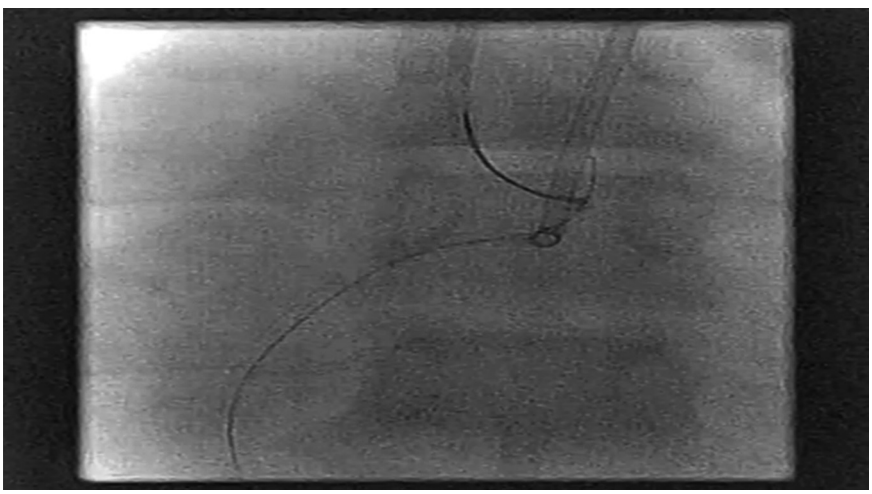
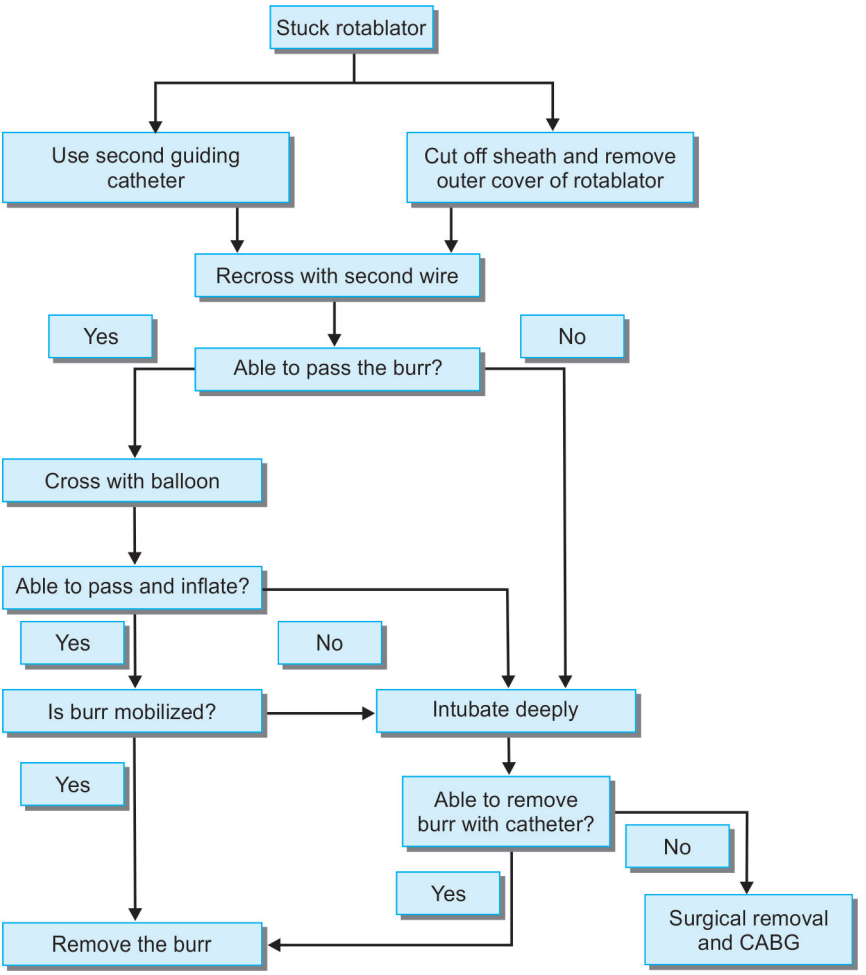


Fig. 10.3 Rotablator burr is pulled out forcefully with constant pressure. The broken wire is then snared out

is necessary **(Figs 10.2 and 10.3)**. This is due to the poor residual lumen in the guide catheter when the rotablator is still in. To overcome this obstacle and utilize the same guiding catheter for additional devices, the rotablator system can be cut off (disassembled) distal to the advancer (including sheath, driveshaft and rotawire). After removing the sheath and leaving only the slim driveshaft surrounding the rotawire in the catheter lumen, further devices can be advanced along the rotablator remnants through the same guiding catheter.[29-31]

Flow chart 10.1 A step-by-step algorithm for the management of a stuck rotablator



Deep intubation with subsequent pullback of all devices can be useful to focus the force on the burr and to protect the rest of the coronary artery. Once again this can be facilitated by cutting off the system and introducing a second smaller guiding or extension catheter over the drive shaft.

A step-by-step algorithm for the management of a stuck rotablator was proposed by Sulimov et al.[34] and is depicted in the **Flow chart 10.1**.

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"Any monkey can do an intervention but it takes a human brain how to get out of a complication!"

—Ramesh Daggubati

Chapter 11

Extremely Sick Patient: Role of Cardiac Assist Devices

Ramesh Daggubati, Gary Nash, Sundeep Adusumalli, Narasimha Swamy Gollol Raju

INTRODUCTION

Extremely sick patient is someone whom an experienced physician can easily identify standing at the foot end of the bed. Extremely sick patient is usually cold, clammy, in cardiogenic shock and in respiratory distress from congestive heart failure. High risk is defined as the probability and more importantly, the consequence, of abrupt closure of the dilated site, occlusion of large side or distal branches or widespread microvascular obstruction with spasm. Abrupt closure of a large epicardial artery or of a side or distal branch is recognized by standard angiogram while occlusion at the microvascular level is seen as slow flow or no-reflow secondary to showers of material from ruptured plaques. The risk of cardiogenic shock is higher in patients with left ventricular dysfunction (ejection fraction <30%), target vessel supplying more than 50% viable myocardium, circulation to both papillary muscles compromised and a high jeopardy score >3. Other conditions that can cause cardiogenic shock are incessant ventricular arrhythmias or severe aortic stenosis.

Cardiogenic shock (CS) is a state of inadequate end-organ perfusion primarily due to cardiac pump failure. It is characterized by persistent hypotension with a SBP less than 80–90 mm Hg or a MAP less than 30 mm Hg below baseline with a CI less than 1.8 L/minute/m^2 without support or less than 2.0–2.2 L/minute/m^2 with support with an adequate or elevated filling pressure.[1] Acute MI is the most common cause of CS. Approximately 5–8% of STEMI and 2.5% of non-STEMI are associated with CS. CS is the leading cause of death in patients with AMI with mortality rate of ≈50%.[1,3] Early revascularization, compared to initial medical stabilization, improves long term survival and is the recommended strategy for AMI patients presenting with CS due to LV failure.[2]

Percutaneous cardiac assist devices have been historically employed in AMI with CS for mechanical hemodynamic support.[3] In the setting of CS complicating AMI, Intra-aortic Balloon Pump (IABP) counter-pulsation is the most widely used form of mechanical hemodynamic support. IABP is a simple and safe device to insert. It relies on left ventricular function and a stable cardiac rhythm, which may not always be present in CS, to achieve its full potential. The estimated augmentation of cardiac output by 0.5 L/minute may not be sufficient to meet the demands of sick patients in CS without the additional use of deleterious vasoactive agents.[3] Randomized clinical trials

have not shown either 30 day or 1-year mortality benefit with IABP in patients with CS complicating AMI.[2,4,5]

Newer percutaneous ventricular assist devices (PVADs) that provide emergent and effective hemodynamic support in high risk patient population that overcome the limitations of IABP are increasingly being utilized.[1,3,6] The Tandem Heart™ and Impella 2.5/CP™ or extracorporeal membrane oxygenator (ECMO) are the more frequently used PVADs. These devices differ in their insertion technique and mechanism of action. Potential major complications include limb ischemia, bleeding, and transfusion requirements.[3,6]

The Tandem Heart™ is a left atrial to femoral artery bypass which can provide up to 3.5–4 L/Minute forward flow and active hemodynamic support. Device insertion, management and discontinuation require experienced staff. Clinical studies have shown that the Tandem Heart™ provides significantly better hemodynamic support compared to IABP in CS but at no difference in 30 day mortality.[6] Anecdotally several cases have shown effective utility of Tandem Heart™ in providing effective support in refractory CS and during high-risk cardiac interventions.[7]

The Impella 2.5™ device provides partial hemodynamic support that directly unloads the left ventricle. It requires one femoral artery access and, unlike the Tandem Heart, provides a maximal flow rate of ≈2.5 L/minute. It is considered a safe and easy to use, providing effective hemodynamic support during high-risk PCI[8] but has not shown superior outcome compared to IABP at 30 days.[9] Recently, Impella CP™ catheter has been introduced and it can provide flows averaging 3 L/minute.

Extracorporeal membrane oxygenation can assist both right and left ventricles. It can be used either venovenous or venoarterial depending on the placement of the cannulas and the ventricle that needs the support. ECMO has been used in various settings, including cardiogenic shock and postcardiac and respiratory arrest.[1]

Despite the lack of randomized clinical trials, higher rates of adverse events, and no proven survival benefits, the continued use of PVADs in CS is recommended.[6] The ability to initiate and maintain timely and effective hemodynamic support using PVADs in the catheterization laboratory has changed the way CS is approached in certain patient populations and is considered a major advancement in interventional cardiology. More clinical trials and studies are needed to further evaluate their effectiveness, comparative merits and adverse effects.[3,6,7]

CASE EXAMPLES

Case 1

Critical Aortic Stenosis Presenting in Cardiogenic Shock

Introduction: Aortic stenosis is one of the most common valvular abnormalities encountered in our practice. Once aortic stenosis becomes

symptomatic, the mortality rate is approximately 50% in the first 2 years. The treatment is typically valve replacement surgery, however in the setting of advanced age or multiple co-morbidities patients may be denied, secondary to increased surgical risk. Transcatheter aortic valve replacement (TAVR) has been proven to be beneficial in this subset of patients. That being said TAVI itself has certain contraindications including LVEF <20% and cardiogenic shock requiring inotropes.[1]

An 82-year-old male first presented to clinic with symptomatic aortic stenosis **(Fig. 11.1)**. His significant past medical history included: paroxysmal atrial fibrillation, chronic kidney disease stage I, prostate cancer in remission and GI bleed. He was initially randomized to the surgical arm of PARTNER II trial. Prior to scheduled surgery, he presented to the emergency department in cardiogenic shock. The patient was placed on mechanical ventilation, inotropes and vasopressors initially. Despite this, he remained in shock with multisystem organ failure. A repeat echocardiogram demonstrated and LVEF of less than 20% down from 55% to 60% a few months prior. Soon after a Tandem Heart™ was placed in the cardiac catheterization laboratory **(Fig. 11.2)**. A balloon aortic valvuloplasty was considered, however, since his cardiac output drastically improved with the Tandem Heart™ this was deferred. The patient was withdrawn from the PARTNER trial and received #26 Edwards Sapien™ valve via transfemoral approach. There was residual mild perivalvular leak shown on postdeployment transesophageal echocardiogram. Postprocedure, he was able to be weaned off of inotropes, vasopressors as well as the Tandem Heart™. At discharge, his organ function had returned to normal. He was seen in clinic several months after completion of cardiac rehabilitation and his symptoms had improved to NYHA class I.

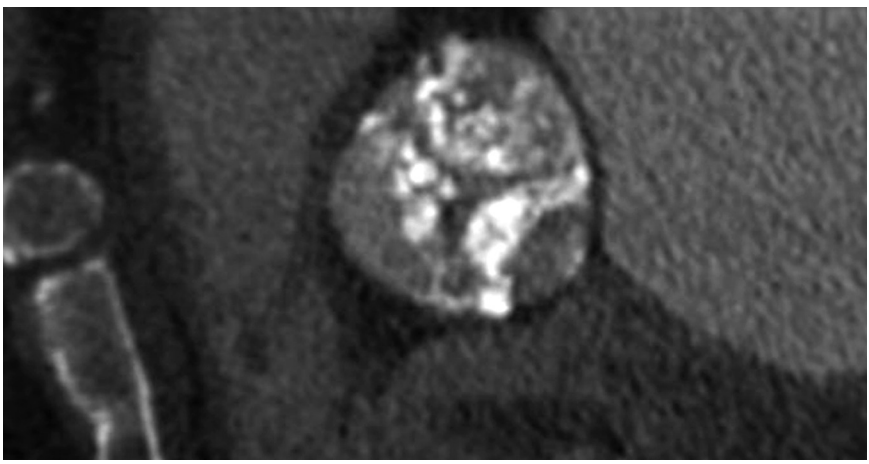


Fig. 11.1 Computed tomography scan of the chest showing severely calcified, trileaflet aortic valve with severe aortic stenosis

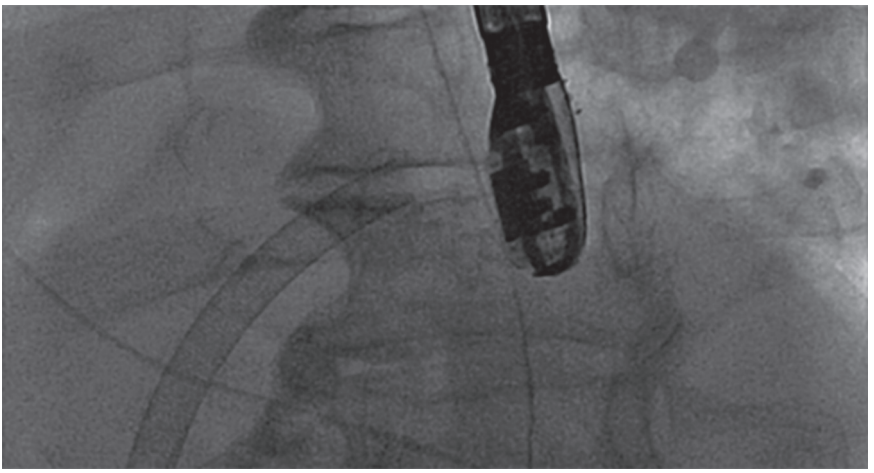


Fig. 11.2 Fluoroscopy showing transesophageal echocardiography probe behind which the transseptal cannula of the Tandem Heart is visible in the left atrium

Analysis of the case: We present a case of a patient with aortic stenosis in cardiogenic shock secondary to acute left ventricular systolic failure who ultimately went on to receive a TAVI and demonstrated good functional recovery. There is little data in the literature in this subset of patients. Fudim et al. all presented 2 case reports where TAVI was used successfully as a bailout in patients with cardiogenic shock secondary to bioprosthetic valve failure.[2] The most published data on the subject comes from D'Ancona et al. demonstrated technical feasibility and 19%, 30 day mortality in 21 patients with cardiogenic shock undergoing TAVI.[3] The Tandem Heart™ percutaneous ventricular assist device is a left atrial to femoral artery bypass system using a centrifugal pump that can deliver flow rates up to 4L/min.[4] It is particularly useful in this subset of patients since unlike Impella™, it does not involve crossing the aortic valve. Traditionally, balloon valvuloplasty has been the bailout procedure for patients in refractory cardiogenic shock with severe aortic stenosis. There is little data on performing TAVI in patients with cardiogenic shock, but with ventricular assist device placement this could be the next frontier for the procedure.

Case 2

Introduction: Hemodynamic support has been shown to be useful in recurrent arrhythmias secondary to STEMI.

A 52-year-old male with past history of CAD dyslipidemia, HTN, presented to a primary center with substernal chest pain and diaphoresis. He was on chronic clopidogrel therapy for a bare metal stent placed 4 months prior, and he discontinued it several days prior to presentation for an upcoming orthopedic surgery. His initial EKG showed an inferior STEMI **(Fig. 11.3)**. He was given thrombolytics and transferred to a tertiary care

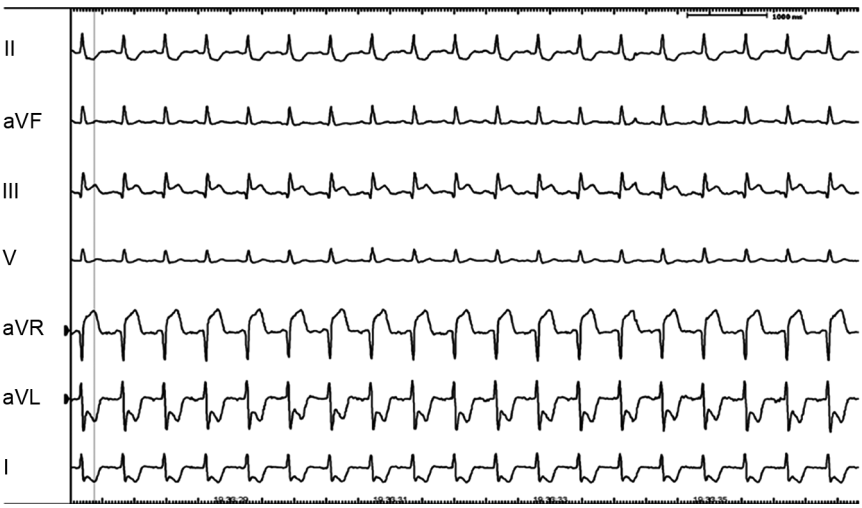


Fig. 11.3 Electrocardiogram showing sinus tachycardia with ST-elevation in lead III and aVR and reciprocal ST-depression in leads I and aVL

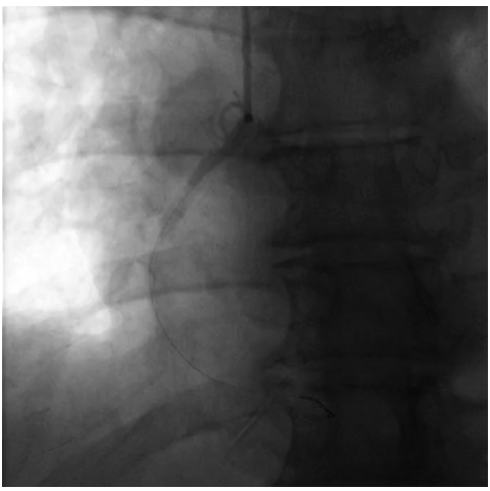


Fig. 11.4 Angiography of the right coronary artery showing 90% stenosis in middle segment with guidewire across the stenosis

facility. He underwent defibrillation for unstable ventricular tachycardia en route. Emergent angiography was performed with five defibrillations during the diagnostic portion of the procedure. Lidocaine and Amiodarone therapy was initiated without full resolution of his electrical instability. The RCA showed a mid-90% stenosis **(Fig. 11.4)**, and he underwent successful drug-eluting stenting to his RCA. Despite revascularization of his culprit vessel

and antiarrhythmic therapy, he continued to have episodes of ventricular tachycardia requiring defibrillations. At this point, an Impella 2.5™ along with a Swan-Ganz catheter was placed **(Fig. 11.5)** for hemodynamic support and electrical stability. At this point, he did not have any more episodes of ventricular arrhythmias, but his ST-elevations did not fully resolve. A repeat angiography revealed a thrombus and dissection in his mid-RCA. After aspiration of the thrombus, IVUS showed the stent to be underdeployed with a small distal dissection. At this point, a second stent was placed in his RCA with resolution of ECG changes **(Fig. 11.6)**. He was transferred to the cardiac intensive care unit. His Impella™ was weaned and removed in 24 hours without any recurrence of arrhythmias. He was discharged by hospital day 5.

Analysis of the case: Impella for hemodynamic support is a useful modality for electrical stability in unstable patients, which is illustrated in this case.

Case 3

Introduction: This case describes the successful use of an Impella CP™ device in a 60-year-old male who presented with acute ST-elevation myocardial infarction with cardiogenic shock.

A 60-year-old male with a past medical history of HTN, dyslipidemia, peripheral vascular disease, and CAD (CABG 20 years prior with known occluded vein grafts and patent LIMA-LAD) who presented to the emergency department at an outside facility with complaints of substernal chest pain. Initial ECGs did not show any signs of ischemia. He was given nitroglycerin

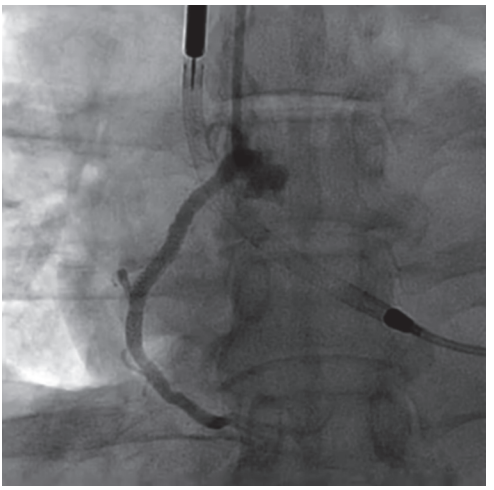


Fig. 11.5 Angiography of the right coronary artery status postplacement of drug-eluting stent in middle segment with good angiographic results. Also seen is the Impella™ device *in situ*

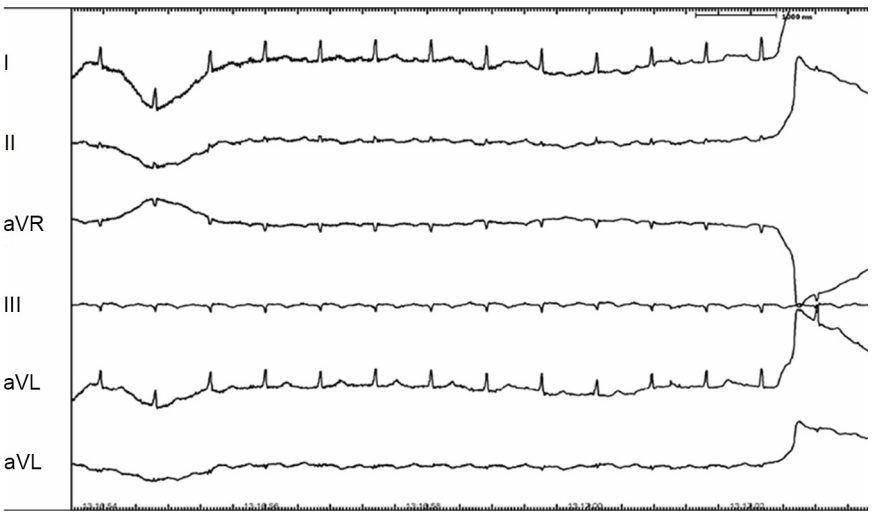


Fig. 11.6 Electrocardiogram showing sinus rhythm with improved ST-depression in leads I and aVL but still some residual ST-elevation noted in lead III

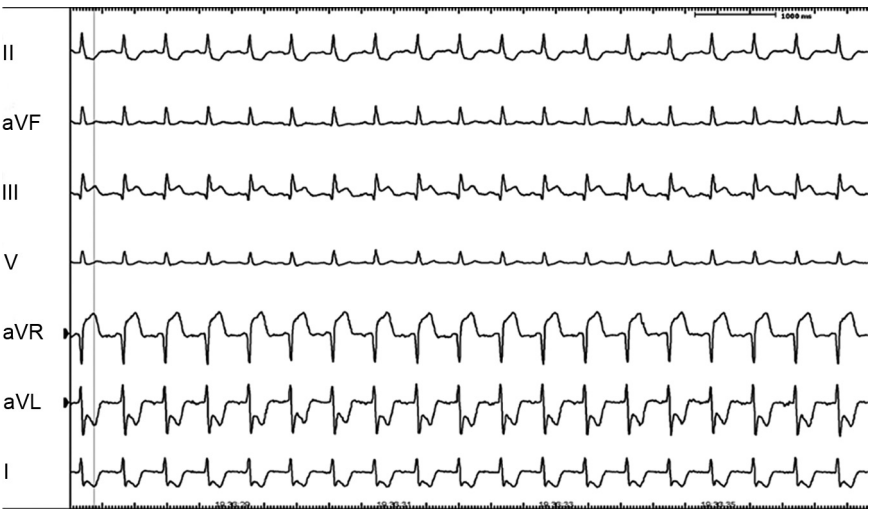


Fig. 11.7 Electrocardiogram showing sinus tachycardia with ST-elevation in leads III and aVR and reciprocal ST-depression in leads I and aVL

initially and became hypotensive. Repeat ECG showed ST-elevations in the inferior leads **(Fig. 11.7)**. He then developed ventricular fibrillation followed by 20 minutes of advanced resuscitation. He was started on dopamine and levophed for cardiogenic shock with persistent hypotension of 80/50 mm Hg. Patient was given TNKase prior to emergent transfer to a tertiary care facility.

Emergent angiogram showed severe native vessel disease (90% occluded left main, 100% proximally occluded left anterior descending with a small diagonal branch, 100% proximal left circumflex artery, and a 100% proximally occluded right coronary artery) and occlusion of 3 vein grafts with a patent LIMA to LAD. The native LAD after touchdown of the graft was a small vessel. An Impella CP™ catheter was then placed through the right femoral artery for hemodynamic support along with a cooling catheter in the right femoral vein. A Swan-Ganz catheter was placed in the left femoral vein for hemodynamic monitoring. Hypothermic protocol was initiated and the patient was transferred to the cardiac intensive care unit **(Fig. 11.8)**. Initial echocardiogram upon admission revealed an ejection fraction of 30–35% with mainly inferior akinesis. Initially, he exhibited decorticate posturing and was started on paralytic due to shivering on the hypothermic protocol. Within several hours of his CCU stay, re-warming protocol was initiated because he awakened and was able to follow commands. Levophed™ was used for vasopressor support upon arrival to the tertiary care facility and was weaned off by hospital day 2.

The use of the Impella™ and vasopressor support continued through hospital day 1. An echocardiogram was repeated on hospital day 2 which showed an improved EF of 45%. At this point, his vasopressors were weaned off while his Impella™ was weaned to P5. His Impella™ was removed by the end of hospital day 2 due to hemodynamic recovery. Echocardiogram on hospital day 6 showed an ejection fraction of 55–60%. He was extubated on hospital day 3. He was discharged to home on hospital day 10 with full recovery.

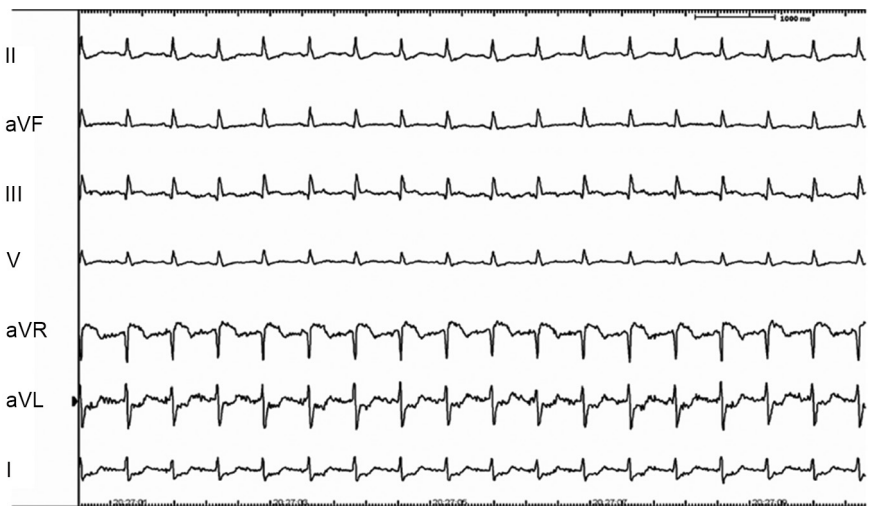


Fig. 11.8 Electrocardiogram showing sinus tachycardia with improved ST-segment deviations in inferior and lateral leads after Impella placement

Analysis of the case: When one is taking care of an extremely sick patient, there is sparse evidence-based medicine to decide the choice of cardiac device to be used. ABCDs of resuscitation efforts have to be followed first that include airway, breathing, circulation and defibrillation, if needed. Once patient's respiratory status is stabilized, attention should be turned to stabilize the patient hemodynamic status. Cardiogenic shock is a spectrum, still carrying high mortality in the range of 40–50% with all currently available left ventricular support devices. One should first assess the patient and classify shock into mild, moderate or severe shock.

LEVELS OF CARDIOGENIC SHOCK

- *Mild*: Low levels of inotropic support (Dopamine ≤5 μg/kg/min, epinephrine or norepinephrine ≤0.03 μ/kg/min)
- *Moderate*: Modest levels of inotropic support (Dopamine ≤10 μg/kg/min, epinephrine or norepinephrine ≤0.05 μg/kg/min)
- *Severe*: Maximal inotropic support (Dopamine >10 μ/kg/min, epinephrine or norepinephrine >0.05 μ/kg/min, vasopressin at any dose.

Suggested Devices

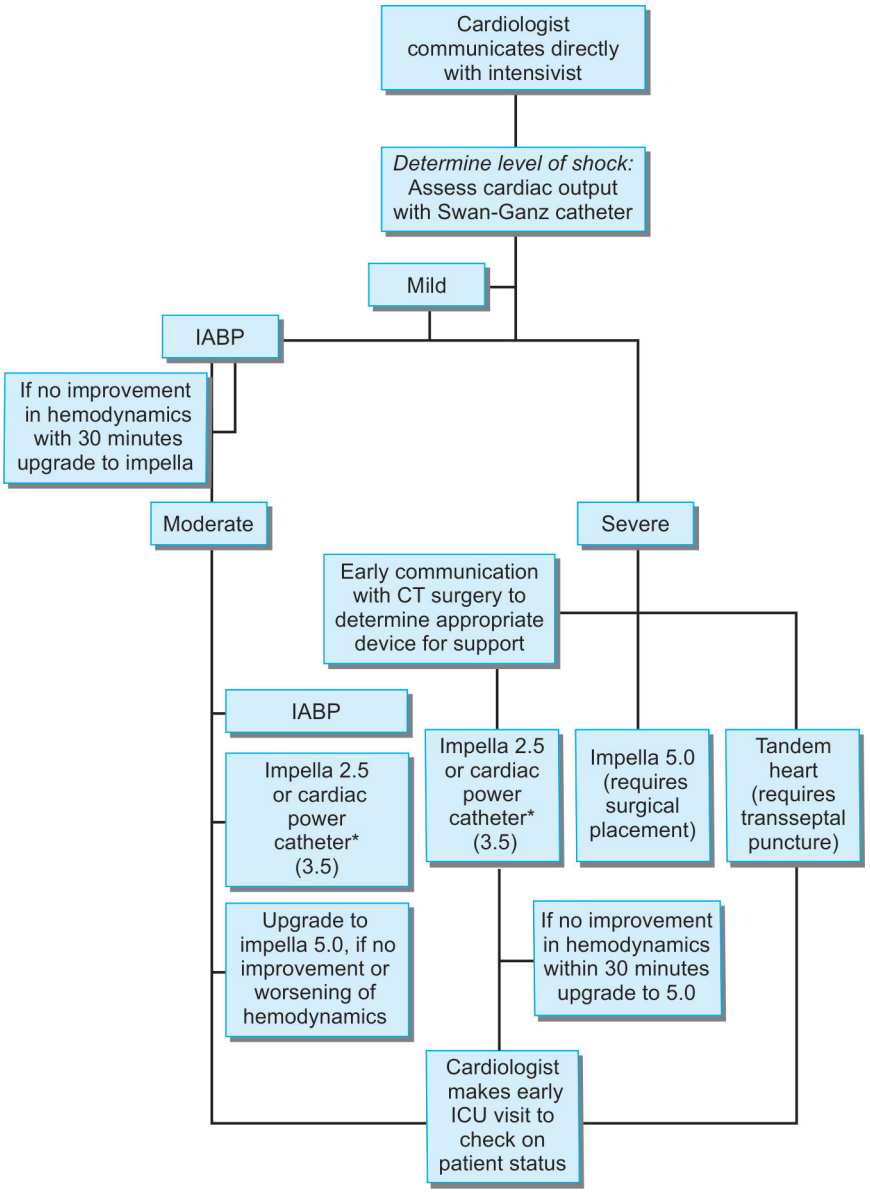
Mild Shock: Intra-aortic balloon pump

Moderate Shock: Impella CP™ catheter

Severe Shock: Impella CP/Impella™ 5.0, Tandem Heart™ or ECMO.

The choice of device depends on local expertise and comfort of the operator. The right ventricular support devices such as the Impella RP™ and Tandem Heart™ right ventricular support cannula are available now. However, there have been no randomized studies to support one device over the other to improve mortality. In our own registry of 42 patients with acute myocardial infarction and cardiogenic shock, quick escalation protocol of devices has reduced in-hospital mortality from 44% to 24% **(Flow chart 11.1 and Table 11.1)**. These results have to be further confirmed in larger sample size. The role of cardiac assist devices in extremely sick patients is a changing landscape due to the paucity of data, cost of the devices, patient factors and support from insurance companies. Extracorporeal membrane oxygenation (ECMO) has been used in refractory cardiogenic shock patients for decades with and without cardiac or respiratory arrest.[10-12] However, survival in most ECMO trials or series is less than 40%. ECMO can be performed at bedside with femoral venous and arterial access. Complications include bleeding, ischemia and venous thrombosis. Poor predictors of mortality with ECMO include age >60 years, pH >7.30, inotropic score >20, recent history of CPR, ECMO implantation during CPR and oligoanuria.[13]

Flow chart 11.1 Suggested cardiogenic shock protocol at East Carolina Heart Institute, Greenville, NC, USA



*Cardiac power catheter can pump up to 3.5 L/min and is inserted percutaneously in the lab.

Table 11.1 Outcomes of East Carolina Heart Institute Cardiogenic Shock Registry

Cardiogenic shock	*Group I (18)*	*Group II (24)*
Incidence	7.7%	8.4%
Mortality	44.8%	24.4%
Impella insertion	5.5%	85%
Tandem heart	11%	21%

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Chapter 12

Complications of Radial Access: Children of a Lesser God

S Anandraja

Coronary angiograms and interventions using the radial artery are being increasingly done. The advantages of radial access include patient comfort and fewer access site bleeding complications. There was a 73% reduction in major bleeding complications in the radial route as compared to the femoral route.[1] The (RadIal Vs femorAL access for coronary intervention trial) RIVAL study reported 1.4% incidence of major vascular complications with the radial route as compared to 3.7% in the femoral access group.[2] The first radial access coronary angiogram was first performed in 1989 by Campeau[3] and since then there has been an exponential growth in the number of procedures done through the radial route. However, challenges and complications do occur with the radial access site and it is important to have a thorough understanding to avoid potential complications.

RADIAL ARTERY SPASM

Radial artery spasm is the most common complication associated with the radial access and it occurs in about 5–10% of procedures. Radial artery spasm may occur either during initial cannulation attempts or during the procedure. Radial artery spasm at the beginning of the procedure is mainly due to multiple puncture attempts with repeated entry with the wire. This will result in transient loss of pulse and changeover of the route to femoral artery. Radial artery spasm during the procedure is commonly seen in women, anxious patients, multiple catheter exchanges, a small radial artery and with use of a larger sheath size compared to the radial artery.[4] Methods to avoid radial artery spasm include adequate sedation of the patient, use of nitroglycerine in the local anesthesia around the artery, radial artery cannulation with minimal attempts and use of nitroglycerin and calcium channel blocker in the antispasmodic cocktail after cannulation. Use of a smaller size radial sheath with hydrophilic coating as compared to the artery size will prevent radial artery spasm.[5] During procedure, minimal manipulation of the catheter and guidewire and avoiding multiple catheter exchanges will help in reducing the spasm. Spasm of the radial artery will cause immense pain to the patient during catheter manipulation and aggressive catheter withdrawal during spasm can lead to avulsion of the radial artery. During catheter exchanges, an additional dose of nitroglycerine can be given to prevent spasm. In case

of spasm, in addition to extra dose of nitroglycerine, a smaller size catheter should be used to continue the procedure. If there is catheter entrapment due to intense vasospam, it is best to leave the catheter in place for a while and sedate the patient. Attempts to remove the catheter should be made after 30–60 minutes and are usually successful. Rarely general anesthesia may be required to relieve the spasm and retrieve the entrapped catheter.

CASE EXAMPLES

Case 1

A 43-year-old male was brought to the cath lab for performing angiogram through the radial route. Modified Allen's test was consistent with a patent palmar arch. The patient was administered 25 μg of Fentanyl and 1 mg of Midazolam intravenously. Radial artery cannulation was difficult and was possible after multiple attempts. There were multiply wire entry episodes during the attempts at cannulation. After cannulation, a 6 French sheath was inserted and diagnostic angiogram done using the 5F Tiger catheter. Diagnostic angiogram showed a proximal 80% lesion in the left anterior descending artery. During withdrawal of the diagnostic catheter there was some resistance in the forearm. A 6 F EBU coronary guiding catheter was advanced into the sheath. However, there was some resistance at the forearm but the wire has crossed easily and the patient experienced some pain. The catheter was withdrawn and additional 500 μg of nitroglycerine given. After a few minutes a 5F EBU coronary guiding catheter was advanced, which negotiated now smoothly and the procedure was completed.

Analysis of the case: If there is spasm and prevents catheter advancement, additional bolus of nitroglycerine can be administered and a smaller size catheter should be selected.

Case 2

A 40-year-old lady was taken up for coronary angiogram using the radial approach. Modified Allen's test suggested a patent palmar arch. She was anxious and her heart rate was 110 bpm. In the cath lab she was given 25 μg of Fentanyl and 1 mg of Midazolam intravenously. The tissue around the radial artery was infiltrated with 10 mL of 2% lignocaine and 1000 micrograms of nitroglycerine. Radial artery was cannulated after multiple attempts and a hydrophilic sheath inserted. After administration of 5000 U of heparin, coronary angiogram was performed using 5F Tiger catheter. However, engagement of the ostia of left coronary artery (LCA) and right coronary artery (RCA) could be done only after considerable manipulation of the catheter. During the manipulation, there was resistance and patient experienced pain at the elbow. Withdrawal of the catheter was difficult and there was considerable resistance indicating spasm of the radial artery and possible entrapment of the catheter. Patient was administered additional

dose of Fentanyl and Midazolam, and shifted to the patient holding area. After 30 minutes, the catheter could be withdrawn successfully with little bit of resistance.

Analysis of the case: Radial artery spasm can be precipitated by aggressive catheter manipulation especially in anxious women and can result in catheter entrapment. Forced catheter withdrawal can result in radial artery avulsion. Additional sedation has to administered and attempts to withdraw the catheter has to be made after a variable period of time.

RADIAL ARTERY OCCLUSIONS

Asymptomatic Radial Artery Occlusion

Due to the presence of palmar arch and dual blood supply to the hand, radial artery occlusions following radial artery cannulation are mostly asymptomatic. However, it is prudent to check the patency of the palmar arch before radial artery cannulation by using the modified Allen's test. The incidence of asymptomatic occlusion ranges from 5% to 15%.[6] Though there are generally no major long-term consequences, the site may not be suitable for further access and the radial artery may not be useful for coronary bypass surgery as free arterial grafts. Factors that are more commonly associated with radial artery occlusion include use of large sheath size (compared to the radial artery size),[7,8] inadequate anticoagulation after cannulation,[9] radial artery spasm, multiple cannulation attempts,[6] prolonged duration of compression during hemostasis,[10] compression technique used[11] and delayed sheath removal.[12]

Acute Symptomatic Occlusion

Acute occlusion of the radial artery following radial artery procedures leading to ischemia and gangrene of the hand are very rare. Factors that can cause acute symptomatic occlusion include absence of a patent palmar arch and presence of underlying vascular disease like Raynaud's disease.[13] Confirmation of the patency of the palmar arch by performing the modified Allen's test can prevent such disastrous complications.[14]

Nonocclusive Radial Artery Injury

Following radial artery procedure, there is sometimes tissue proliferation in the radial artery leading to partial loss of lumen. Trauma to the vessel wall during catheter and guidewire insertion and manipulation is postulated to trigger intimal proliferation.[15] This results in difficulty during attempts at later cannulation and catheter advancements.[16] Also such a disease makes the artery unsuitable as arterial grafts for coronary bypass procedure because of lower long-term patency rates.[17] Smooth advancement of the catheter and guidewire and minimal manipulation can reduce this complication.

Hematomas

Hematomas can occur at the puncture site or in the forearm. Hemostasis is quite simple to achieve after radial artery cannulation. Radial artery being a superficial structure and lying over the radius bone, it is very amenable to compression. Both manual compression and compression using devices (Radial TR band) can be used to achieve hemostasis. For this reason, the incidence of hematomas and bleeding complications at puncture site are much less as compared to the femoral site. There seems to be a significant reduction in local bleeding complications with the radial access as compared to the femoral access.[18]

Hematomas in the forearm remote from puncture site are related to perforation of the small branches of the radial artery or the radial artery itself. Perforation can occur during radial artery cannulation or during catheter advancement. Hydrophilic wires used during initial cannulation can inadvertently perforate small branches. When there is even slight resistance to advancement of the wire or the wire catheter assembly, the wire tip should be checked under fluoroscopy. If the wire has entered a small branch, it has to be withdrawn and rerouted to the correct main lumen. Doing an angiogram with the catheter withdrawn proximally also can help in identifying the main lumen and appropriate adjustment of the wire direction. Perforation of the radial artery is rare and occurs in less than 1% of cases.[19]

Hematomas in the forearm are also easy to control, if identified early. Application of pressure bandage over the forearm can arrest the hematoma. Initially intermittent inflation of the sphygmomanometer cuff over the forearm can be used to arrest the bleeding. Inflation of the pressure to just above the systolic blood pressure is successful in arresting the bleeding. Alternately the cuff can be tied to the ipsilateral arm while manual compression or compression bandage is applied to the forearm to assist in achieving hemostasis. Anticoagulants and intravenous antiplatets (GpIIb/IIIa inhibitors) used during coronary interventions are important factors that contribute to the occurrence of forearm hematomas. Forearm hematomas are rarely ever seen in case of diagnostic coronary angiograms.

Forearm hematomas if not identified and treated early can sometimes lead to compartment syndrome endangering limb viability. Prompt fasciotomy is indicated to prevent adverse consequences. Early identification and anticipation is the key to successful management of complication related to perforation of the artery.

CASE EXAMPLE

Case 3

A 67-year-old lady was taken up for coronary angiogram for non ST elevation MI through the radial route. Modified Allen's test indicated a patent palmar arch. The patient was administered 25 μg of Fentanyl and 1 mg of Midazolam intravenously. The radial artery was cannulated and 6 F

hydrophilic radial sheath inserted. However, during the initial cannulation attempts, the guidewire would not go freely. Diagnostic angiogram revealed a culprit lesion in a large obtuse marginal branch and it was subsequently stented using a drug eluting stent. During the procedure a total of 7500 U of heparin was administered and patient was started on GpIIb/IIIa inhibitor infusion. The sheath was removed and hemostasis achieved manually. In the coronary care unit (CCU), the patient complained of pain in the ipsilateral forearm and a hematoma was observed. Immediately the GpIIb/IIIa infusion was stopped and the sphygmomanometer cuff was inflated around the arm and the pressure was intermittently maintained about the patient's systolic pressure while manual compression was given to the forearm. After about 10 minutes of intermittent inflation, the hematoma did not progress and a pressure bandage was applied to the forearm.

Analysis of the case: Prompt identification of the forearm hematoma is crucial for successful management and to prevent compartment syndrome. Difficult radial artery cannulation with multiple wiring and aggressive anticoagulation are factors associated with forearm hematoma.

Arteriovenous Fistula

Arteriovenous fistulas are very rare with the radial access as compared to the femoral access. Significant large veins are absent adjacent to the radial artery and compression applied generally occludes the adjacent small veins and fistula track if any. However, rarely it can occur and will result in a swelling that gradually increases over time. Patient will present with swelling and pain near the puncture site. Color Doppler will confirm the presence of the fistulous channel and angiogram will reveal the anatomy of the arteriovenous fistula. Management include surgical ligation or coil embolization of the fistula.

CASE EXAMPLE

Case 4

A 75-year-old male, diabetic and hypertensive was admitted with acute coronary syndrome and was brought to the cath lab for coronary angiogram. After confirming patency of the palmar arch with a modified Allen's test, radial artery cannulation was done after multiple attempts. Patient underwent successful angioplasty and stenting of the left anterior descending artery. Patient received 7500 U of unfractionated heparin and Tirofiban, with an initial bolus of 25 μg/kg followed by infusion 0.15 μg/kg/min for 24 hours. Radial artery sheath was removed immediately after completion of PCI and hemostasis was achieved by application of a tight bandage. After an uneventful hospital stay, patient was discharged with medications. Two months later patient came back with complaint of swelling and unusual

sensation over the access site. On examination a palpable thrill was observed over the access site and arteriovenous fistula was diagnosed with the help of Doppler ultrasound. Coil embolization was planned and a 5 mm coil was placed in the AV fistula and closed. Following the procedure, the swelling and turbulence at the radial access site decreased. At one month follow-up, the patient was symptom free and the thrill over the access site had disappeared.

Analysis of the case: AV fistula following radial artery cannulation is a rare complication and can be managed with coil embolization.

Pseudoaneurysm

Pseudoaneurysms are again very rare with the radial access for coronary interventions with a reported incidence of less than 1%.[20] It is commonly associated with multiple puncture attempts and aggressive wiring. Perforation of the small vessels in the forearm can lead to pseudoaneurysm especially in the presence of aggressive anticoagulation, inadequate compression and delayed bleeding. In the presence of pseudoaneurysm, the hematoma continue to expand despite compression of the hematoma. Patient presents with a delayed swelling which may be painful. Color Doppler evaluation will reveal the presence of pseudoaneurysm usually with a small neck. Management of pseudoaneurysm include Doppler guided compression of the neck of the aneurysm or injection of thrombin into the aneurysmal sac.[21,22] Usually compression of the neck of the aneurysm is successful in closure of the aneurysmal sac and resolution of the hematoma. In some cases surgical interventions may be required to ligate/repair the pseudoaneurysm. If there is suspicion of perforation, angiogram during the procedure or at the end will help to localize the site of extravasation if any and pressure at that site can prevent pseudoaneurysm formation.

Difficulty in Catheter/Guidewire Advancement

Catheter/guidewire advancement can be difficult sometimes; there will be resistance while advancing either the wire or the catheter. Catheter and guidewire advancement has to be extremely gentle to avoid major complication. Even a slight amount of resistance has to be taken seriously and the cause identified. The main causes include arterial spasm, entry into a side branch, arterial loops, entry into a collateral and tortuous subclavian artery (especially in the elderly). Prevention and management of arterial spasm has been discussed previously. Entry into side branch can be easily identified by fluoroscopy whenever the guidewire meets resistance.

Tortuous subclavian artery are quite common in the elderly and can cause substantial difficulty during guidewire advancements. Instructing the patient to take deep breath will help to make the artery less tortuous and guidewire can be passed. If difficulty persists, hydrophilic wires are helpful. Sometimes asymptomaic subclavian artery occlusions can be present and

doing an angiogram using hand injection will help to delineate the course of the artery.

Arterial loops are important cause of resistance to catheter and guidewire advancement. Some loops get straightened once guidewire has negotiated through them and further catheter advancement will be easy. These loops are mainly due to redundant arteries and seen in elderly. Congenital loops are present near the elbow joint and these are attached to the underlying tissue. Guidewire often enters a branch from the loop and soon will face resistance. Inadvertent force will lead to perforation of the branch and result in hematoma formation. Usually, the guidewire especially hydrophilic ones will successfully negotiate the loop and but the catheter may still not be able to negotiate it. Forcing the catheter can result in avulsion and rupture of the artery and has to be avoided. Often these loops are bilateral, it is better to resort to femoral access for completing the procedure.

Rarely the guidewire at the elbow will enter collateral and then reenter the main vessel above. The guidewire movement and course will appear normal. However the catheter advancement will be difficult. Prompt angiogram will reveal the true course of the guidewire and help in rerouting the guidewire in the correct lumen.

CASE EXAMPLES

Case 5

A 50-year-old lady was taken up for coronary angiogram using the right radial artery route. Modified Allen's test indicated a patent palmar arch. In the cath lab, patient was anxious and had a heart rate of 110 bpm. She was given 25 μg of Fentanyl and 1 mg of Midazolam intravenously. Radial artery was cannulated and a 6F hydrophilic radial sheath was inserted and 5000 U of unfractionated heparin given. A 5F Tiger catheter over a 0.035 inch J tip teflon coated guidewire was advanced. After some distance there was some resistance and fluoroscopy showed looping of the guidewire. The guidewire was withdrawn and again advanced. There was consistent coiling of the guidewire and the patient experienced pain. At this juncture Teflon guidewire was exchanged for a hydrophilic guidewire which was advanced under fluoroscopic guidance. The wire pass was smooth and the catheter was advanced over it. Subsequent procedure was completed without any difficulty. Post-procedure an angiogram was done through the radial sheath and did not show any extravasation at the site of Teflon guidewire coiling. The sheath was immediately removed and manual compression applied and hemostasis achieved.

Analysis of the case: If there is resistance with the Teflon guidewire, it should be promtply changed to a hydrophilic wire. The Teflon wire might be seeking a branch preferentially and may be adjusted by gentle manipulation. However persistence of the problem needs guidewire change to avoid potential perforation.

Case 6

A 72-year-old man was taken up for coronary angiogram through the radial artery route. Modified Allen's test showed patency of the palmar arch. In the cath lab, he was administered 25 μg of Fentanyl and 1 mg of Midazolam intravenously. A 6F radial sheath was inserted and 5000 units of heparin administered. A 5F Tiger catheter was advanced over a Teflon guidewire through the sheath. However, the guidewire started coiling in the subclavian artery and could not be advanced. The patient was instructed to take a deep breath and hold. Now the guidewire could be negotiated into the arch of aorta and down below and the catheter could be advanced over it. Right coronary artery was hooked and RCA angiogram done. However, engagement of the left main ostium was unsuccessful in spite of multiple manipulations. The catheter was exchanged for a 5F EBU guiding catheter and now the LCA could be successfully cannulated.

Analysis of the case: In elderly anticipate tortuous subclavian artery and difficulty in negotiating the guidewire and difficulty in cannulation of the LCA. To overcome the tortuous subclavian artery, advancement during breath held in deep inspiration and use of hydrophilic wires is advocated.

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Trumpets and violins I can hear in distance
I think they're calling our names
Maybe now you can't hear them, but you will
If you just take hold of my hand
But first, are you experienced?
Have you ever been experienced?

Chapter 13

Momentary Lapse of Reason: Mortality in Cath Lab

Sundeep Mishra, Neeraj Parakh

INTRODUCTION

Since the introduction of cardiac catheterization, in the 1940s, mortality has been occasionally encountered in catheterization laboratory (cath lab). The first large study which reported on this event, recorded a mortality of 0.16% for an active cath lab.[1] Classically, several factors have been found corelative of the rate of death from cardiac catheterization.[2] Over the period of time, while the use of heparin, low-osmolar contrast media and better equipment as well as experience, has decreased the mortality; on the other hand, increasing frequency of older and sicker patients, patients presenting with acute coronary syndrome (ACS) and especially those in with co-morbid conditions, has increased it. With the advent of interventional procedures however, death rates significantly increased.[3-12]

DEFINITION

Death from catheterization is any death occurring in patients (except moribund patients: those supported with intermittent or continuous chest massage before entering the laboratory) within 24 hours after the procedure or later if causally related to the catheterization.

BACKGROUND

The PCI is fraught with its own hazards.[13] Coronary dissection and thrombosis resulting from plaque disruption occur in about 5% of patients. Compared to angiography there is a difference between the etiologies contributing to mortality. While, angiography-related deaths largely occurred in patients with left main-stem artery or triple-vessel disease, or both, with minimal remaining reserve in the coronary circulation or with extensive extracoronary atherosclerosis and multiple comorbidities, in patients undergoing elective PCI the anatomic details of the coronary arteries are already established (and thus possibility of taking prophylactic steps), death from PTCA is largely confined to intervention during evolving coronary events, when ongoing thrombosis was likely or cardiogenic shock is well established. Other possibilities are technical errors during the procedure and even late complications after the procedure.

The issue of mortality in cath lab can be addressed at 2 levels: At the organization level and individual level.

From organizational stand-point, improvement in mortality rates can occur by strictly adhering to guidelines, avoiding errors of commission (inappropriate use of investigations or stents) and omission (failure to adhere to safety procedures), by ensuring cath lab quality (both structural and procedural), by appropriate accreditation of not only cath lab but cath lab personnel including physicians and paramedical staff but most importantly by evaluating and adjudicating outcomes. In other words mortality can be reduced by choosing the right patient, taking the right procedural decisions, executing the procedure properly and finally keeping a tab on the outcome. Currently the mantra for reimbursement at least in some part of West is not pay-for-service but pay-for-performance.

From individual standpoint, mortality can be understood correlative of temporal issues; predominantly factors prior to procedure, intraprocedural factors or factors after the procedure.

FACTORS BEFORE THE PROCEDURE

It is important to remember that 90% of interventional procedure is performed in mind and only 10% is in physical realm. That means 90% of mortality can be prevented even before the procedure begins by proper selection of procedure, proper selection of strategy and hardware and proper selection of the medical team. The crux of matter is the sickness of the patient. In patients with sole surviving vessel, very poor ejection fraction, calcific, thrombotic and tortuous lesions, left main bifurcation with triple vessel disease, cardiogenic shock, renal failure, diabetes mellitus, with comorbid conditions and particularly patients who are already intubated or arrested and on cardiac massage the results are expectedly poor. These cases can again be divided into two groups. In the ones which are elective such as PCI of sole surviving vessel wherein there is a huge advantage of supporting the ventricle (which may be at risk at the time of procedure). Depending on the severity of illness various types of cardiac assist devices can be chosen intra-aortic balloon pump (IABP) and the left ventricular assist devices Tandem Heart and Impella. Each of these devices has its advantages and disadvantages, and an understanding of the role each plays in various pathophysiologic conditions is necessary, as this will assist the interventionist in making the correct decision as to which device will optimize patient outcomes.[14] In PROTECT-II trial undertaken in patients undergoing high-risk PCI, hemodynamic support with Impella compared with IABP resulted in improved event-free survival at 3-month follow-up; this finding was further supported by multivariate analyses.[15] Ongoing clinical trials will provide further insight into the comparative advantages and disadvantages of each and whether one or more is beneficial over another in reducing cardiovascular events and mortality. With continued refinements in device technology, technique, and application, it is anticipated that percutaneous device-based procedures will continue to improve patient outcomes in the most critically ill and highest-risk patients.[16]

In patients who are sick because of acuity of vessel occlusion such as AMI with cardiogenic shock or AMI with occlusion of left main or arrested patient on cardiac massage there may be a benefit of opening the artery as soon as possible. But here again there may be sudden increase in oxygen consumption once reperfusion occurs which may in fact adversely affect the outcome by predisposing to risk of arrhythmic event. Thus even in these cases prior stabilization and ventricular support by an appropriately chosen Cardiac Assist Device such as IABP may be of great value.[17]

Further, several other strategy chosen may be of utmost importance in these patients. In really sick patients with hypotension and pulmonary edema, minimal use of contrast is mandatory. In sicker patients trying to keep the procedure as simple as possible helps for example direct stenting or in very sick patients with bifurcation lesion, choosing simultaneous kissing stent over complex cullotte, double crush etc. may be more realistic. On the other hand in cardiogenic shock, a strategy of as much (complete) revascularization as feasible and safe instead of opening only infarct related artery may be appropriate.

Besides these technical details the preparedness of cath lab to deal with complications is also mandatory. Simple precautions; before starting the procedures in cath lab: checking whether the defibrillator is working, whether intubation equipment, IABP is available close at hand. In addition certain adjunctive devices; covered stent, glue, micro-coils, perfusion balloons should be available in lab. Further, it is a good idea to prepare a Crash Cart (with essential drugs and resuscitation equipment) and keep it readily available.

CASE EXAMPLES

Case 1

A 60-year-old hypertensive female presented with the complaint of atypical chest pain of six-month duration. Cardiovascular system examination was normal. Routine lab investigations were also normal. Baseline ECG and transthoracic echocardiography were within normal limits. However, during the course of TMT examination patient developed severe chest pain and hypotension in recovery phase with down-sloping ST segment depression in leads II, III, avF, V4-V6 with ST segment elevation in avR, TMT was reported strongly positive. Patient was shifted to ICU but was profusely sweating with a pulse rate of 40/min, BP: 70 systolic and bilateral basal fine rales on chest auscultation. Patient was started on inotropic support and shifted to cath lab immediately. However, while being shifted patient developed cardiac arrest. CPR was instituted; patient was intubated and put on a ventilator. Femoral venous and arterial access was immediately obtained. A left guiding catheter was taken and 0.014 floppy wire was introduced into the left coronary system using sinus injection and all the while CPR was continued. Left coronary angiogram revealed the ostial LMCA stenosis. Auto-perfusion of coronary system was performed with guiding catheter and intracoronary adrenaline was given. Driver™ stent was deployed in the ostial LMCA.

Post-stenting coronary angiogram revealed good blood flow in the left system. However, patient continued to be in cardiogenic shock. Arterial blood gas analysis showed severe acidosis (pH 6.8). Acidosis was corrected and the patient was maintained on inotropic support. Chest X-ray revealed aspiration pneumonitis. Over the course of 24 hours the patient stabilized and regained consciousness completely. The inotropic support was tapered, urine output was adequate. Ventilatory support was continued due to aspiration pneumonitis. Renal function test was normal but TLC was high possibly due to aspiration. Unfortunately, next day early morning (4AM) the patient had cardiorespiratory arrest and died.

Analysis of the case: It was a remarkable case of save (at least initially) performed by application of a good skill and technique to a patient who had already undergone a cardiac arrest and was on CPR. Only support by a cardiac assist device like IABP was not performed and this could have contributed to the ultimate mortality because it is possible that providing a cardiac support at the time of PCI could have helped salvage more myocardium which could give that little extra boost to hemodynamics which perhaps could have saved life.

Case 2

Patient was a 62-year-old male, COPD with restricted activity, postoperative CABG about 10 years back, exertional angina NYHA class IIb, TMT was strongly positive at 4 minutes with hypotension. Native left coronary injection revealed a tight stenosis in mid LAD after huge diagonal. Left circumflex and RCA were completely occluded. SVG to RCA and LCx/OM were also occluded. It was decided to do PCI to LAD. The lesion was wired and directly stented. Post-stent deployment cine-angiogram looked OK; however, some under-expansion was detected on stent boost. The stent was postdilated with a non-compliant balloon. Subsequent cine run postdilatation showed a good result. To confirm the end-result a post stent IVUS was also performed which revealed a well opposed stent. Patient discharged on 2nd day post-procedure on clopidogrel 150 mg and ASA 150 mg. Three days later at 4 AM morning patient was found collapsed. He was brought immediately to casualty, but by the time he reached the hospital, ECG was showing a flat line. Multiple shocks given, patient was intubated but the rhythm never returned.

Analysis of the case: It is mandatory to be very careful in doing patients with Sole Surviving Vessel. In this particular case aggressive antithrombotic regimen could have been given. However, the real cause of death lay perhaps in the strategy chosen. He was treated as sole surviving vessel. It may have been prudent to try to open one of the occluded CTOs and attempting LAD only when at least one vessel was recanalized.

Case 3

Patient was a 70-year-old male, hypertensive, with type II diabetes and was having chest pain since 7–8 hours. An initial diagnosis of COPD with acute

exacerbation, accelerated hypertension along with left ventricular failure was made at a primary care center. At that point BP recorded was 170/110 and heart rate 132/min. He was managed with IV NTG and nebulization and administered aminophylline and efcorlin. However, over the course of time patient deteriorated and was then referred to a tertiary health-care center where he presented in a gasping state to the ER at 3 am. BP was found 50 mm Hg systolic, heart rate 34/min, RR 10/min, SpO_2 60%. Patient was immediately intubated, inotropes instituted and bedside TPI instituted in view of AV dissociation. ABG revealed a pH of 6.65, pO_2 84.9, pCO_2 59, HCO_3 6.1. Blood sugar was 480 mg%, although urine ketones were negative. Trop T was strongly positive. ECG revealed a complete AV dissociation with subtle ST depression in anterior leads and ST elevation in inferior leads. Chest X-ray revealed pulmonary edema with bat-wing appearance. Bedside echocardiogram revealed a left ventricular EF of 25% with mild mitral regurgitation and severe hypokinesia of inferior, lateral and posterior walls. Patient was immediately shifted to cath lab. Initially right femoral pulse could not be felt therefore left femoral artery was used for access. At this point arterial BP was recorded 40 systolic. Coronary angiogram revealed LCx 100% proximal occlusion, LAD 80% mid long segment stenosis and RCA was nondominant with mild disease. Initially, radial access was attempted for angioplasty but was unsuccessful in two attempts. Therefore angioplasty was carried out via left femoral approach. LCA hooked with XB 3.5 7F Guide-catheter and LCx lesion crossed with Whisper™ wire with a slight difficulty. Thrombectomy was attempted but Export™ could not be advanced, hence lesion crossed with 2.5 × 15 mm balloon and dilated at 6 atm. The OM was also crossed with BMW™ wire. Subsequently, thrombus from LCx and ostium of large major OM was extracted. Finally LCx was stented with 2.75 × 23 Vision™ deployed at 12 atm. After PCI of LCx, IABP was inserted via left groin and an augmented BP of 110 systolic on high inotropes (augmented from 90 systolic) was obtained. At this point right femoral access could also be obtained (right femoral artery could now be felt but difference of 30 mm Hg pressure noted between left and right femoral artery possibly suggesting peripheral vascular disease as well) and check angiogram revealed TIMI 3 flow in LCx and OM although ostium of OM was found somewhat pinched. It was decided to accept this outcome (excellent is the enemy of good). Swan Ganz catheter was floated for hemodynamic assessment; pulmonary capillary wedge pressure found 20 mm Hg on table. Procedure was completed with 70 mL Visipaque™ and patient shifted to ICU with augmented blood pressure of 100 mm Hg.

In ICU the ABG revealed pH 7.018, pCO_2 45.6, pO_2 90, HCO_3 13.6. Lab reports revealed creatinine 2.2, BUN 38, CKMB 96, SGOT 238, SGPT 205, Hb 15, TLC 17300, platelet 230000, Na 128, K 4.9. Two hours later patient had sudden bradycardia and asystole and died despite aggressive CPR.

Analysis of the case: Patient had several adverse features, advanced age, multi-vessel disease, cardiogenic shock, delayed presentation, hyperglycemia, severe metabolic acidosis, severe LV dysfunction, baseline nephropathy, and

lower limb peripheral vascular disease. However, the factors which probably pushed the patient finally to mortality were:

- Initial wrong diagnosis and management in nursing home
- Time wasted in attempt to gain a radial access when femoral access was already available
- IABP was inserted only after the procedure
- In cardiogenic shock all possible vessels should be revascularized (and not only infarct related vessel)
- A sub-optimal result was accepted after the procedure.

FACTORS DURING THE PROCEDURE

Several factors can go wrong during the procedure; they can be human, material or strategic. In general strict adherence to technique, proper optimization of procedure, and skill and level-headedness of the operator is of utmost importance. At the time of PCI, abrupt closure of vessel is generally the most important cause of mortality. Abrupt closure is a heterogeneous entity and may be caused by acute stent thrombosis, thrombus extension, dissection, emolizations (air, thrombus or other) contributing to slow flow or no re-flow phenomenon or something as innocuous as coronary spasm. Coronary perforations and strategies employed to deal with the problem can also contribute to on-table mortality. Stent (or other cath lab material) entrapment or embolizations and attempts to retrieve it can also contribute to mortality. Rarely guide-catheter trauma, aorto-ostial dissections, cerebrovascular accidents, pericardial effusion and tamponade can also predispose to a fatal event.

CASE EXAMPLES

Case 4

Patient was a 64-year-old male, diabetic, hypertensive who presented with NYHA class II angina for 2 years which aggravated recently. TMT was strongly positive. Angiogram done showed that LAD was normal, OM2—totally occluded, small vessel RCA—totally occluded in mid part, but micro-channels seen. Retrograde filling of PDA and PLV was also observed. Right coronary artery was intubated by JR guide-catheter and lesion attempted to be crossed with Fielder FC™ wire. Initially the wire went sub-intimally but ultimately distal wire could be parked in PDA. However, several balloons of lower profile were tried but could not cross. Assuming that guide-support was not enough, the guide-catheter changed to AR. Further to increase support to guidewire, proximal RCA was dilated and subsequently even stented. Subsequently, Tornus microcatheter was tried but could not cross. Finally, to further increase the guide-support even Guideliner™—Mother and Child catheter was employed but still the guidewire would not cross. Therefore, the operator decided to stop the procedure. Meanwhile, patient continued to

have refractory angina. Thus, a repeat PCI was planned by a more experienced physician. He intuited that perhaps the wire had gone sub-intimally and recrossed into true lumen, which is why balloon could not cross in the prior attempt. Therefore, the team including a senior operator in the tertiary center initially tried same JR guide and same Fielder FC™ wire but was unsuccessful. Subsequently, they attempted two solutions; a better guide-wire and better procedural guidance by retrograde injection to see PDA and PLV. Tried Miracle 6™ wire but could not cross, rather wire kept entering the subintimal space easily. Attempted to change the direction of tip but was still not successful. Ultimately, took Conquest Pro 12™ wire which enabled crossing into the true lumen. Tried crossing with 1.25 × 6 Sprinter™ balloon but could not cross. Finally, Corsair microcatheter was used to cross (although during the maneuver wire kept getting pulled back and forth). Finally the wire was nicely parked in PLV, Corsair™ removed and 1.25 × 15 balloon passed and inflated. This was followed by 2.5 × 15 mm balloon which was like-wise passed and inflated. During the course of these maneuvers, the guide-wire came out of PLV and was now parked into PDA. It was then decided to stent the lesion. Meanwhile a very small leak was detected in PLV but it was decided to stent first. With that view, Nobori™ 3 × 28 and Nobori™ 3.5 × 18 stents were deployed. Meanwhile perforation in the PLV branch increased. Patient also developed chest pain. At this point, Conquest Pro™ was removed and exchanged with Fielder XT™ wire. A 2 × 15 balloon was taken up to the site of perforation in PLV and inflated for 2 minutes at one point and slightly distal also. However, patient developed hypotension and cardiac tamponade. The perforation was attempted to be sealed by gel foam first and then by fat delivered through 1.5 × 10 mm OTW balloon to PLV. However, the leak still continued and patient arrested on cath table and could not be revived.

Analysis of the case: There are several learning points during the procedure. After using Corsair™, the stiff wire should have been exchanged for a softer one. The wire tip movement should have been watched carefully, especially when a stiff wire was used and when Corsair™ was being withdrawn. Attention to both proximal and distal fields should have been paid at all times, so that even a minor perforation (or dissection) could have been picked early. Once perforation was noticed, instead of proceeding with stenting, management of perforation should have been a priority. Finally, managing the perforation, inflation for 2 minutes is not enough; balloon should have been inflated for 10 minutes or more. Finally, heparin was not reversed by protamine for fear of stent thrombosis, which should have been done. Another point, microcoils and PVC alcohol particles were not available in the lab.

Case 5

Patient was a 65-year-old male, who presented with a complaint of intermittent chest pain for last 5–6 days, angina still continuing. ECG revealed ST-T changes in lateral leads. Troponin T was positive. Echocardiography revealed essentially normal LV function. Coronary angiogram revealed;

LAD was type III large size vessel and normal, LCx was a normal vessel well but OM 1 shows 100% occlusion after a small branch. RCA was large size vessel and was normal. PCI of OM1 was planned. Left radial route was taken and sheath less 7F EBU 4 guiding catheter taken to intubate left coronary system. A Hi-Torque™ hydrophilic extra support wire was taken and lesion dilated with 1.5 × 10 mm balloon. Meanwhile, guiding-catheter got slightly disengaged and was reengaged. Post-dilation, there was TIMI III flow in OM. However, suddenly patient arrested with zero blood pressure and developed VF. DC shock given and CPR started. Check shot revealed normal left main, normal LAD and unchanged LCx/OM1 but with slight stagnation of blood flow. Urgently, femoral puncture was done. Guiding-catheter reengaged during massage. At this point check shot taken during massage revealed adequate flow in left main, LAD and LCx but patient was still hypotensive and otherwise critical. Massage was continued with injection epinephrine and multiple shocks. Temporary pacemaker was inserted, patient intubated, massage continued and auto perfusion started from femoral vein to coronary artery. Cardiac tamponade was suspected but echocardiography revealed no evidence of cardiac tamponade. Again check shot revealed that left main, LAD and LCx were still normal. Massage was continued, meanwhile, fresh check shot revealed a thrombus forming at left main bifurcation. Immediately same guide-wire was taken and crossed into LAD, left main was dilated and stented with 4 × 23 mm stent. Adequate result seemed achieved at least angiographically. The massage was continued but surprisingly there was no response at all although repeated check angiograms revealed adequate flow in left system. Finally, distal LAD showed sluggish flow. Ultimately, a check shoot after disengaging the catheter from left main revealed an aortic root dissection starting from left main ostium and extending into ascending aorta (occluding right coronary artery) up to carotids. By this time, it was already too late. ECG developed flat line and patient could not be revived.

Analysis of the case: It is essentially a case of hypotension during PCI of left coronary artery despite good PCI result and normal flow. There could be several reasons for this problem; most common to happen is a vaso-vagal response or contrast reaction. The other possibilities which should however be ruled out include distal wire perforation (which indeed was ruled out), ostial left main dissection, aortic root dissection (which turned out the cause in this situation). Another possibility could be air-embolism to RCA or carotids or other kind of embolizations in carotid leading to CVA. In this particular case, during the performance of PCI guiding had got disengaged many times possibly leading to aortic root dissection.

Case 6

Patient was a 73-year-old male with type II DM, old anterior myocardial infarction with moderate LV dysfunction, presented with recent class II, effort angina and right upper limb claudication. Coronary angiogram revealed triple vessel disease and right subclavian total occlusion. PCI to LAD was

carried out with 2 Endeavor Resolute™ stents and TIMI III flow was achieved. PTA to right subclavian artery was planned and a 6Fr VITEK™ vertebral catheter was advanced into aortic arch. However, during the procedure the catheter tip got broken and got embolised into right middle cerebral artery. First attempt was made thus; A 0.014 BMW™ wire was passed through the broken fragment, then a 2 × 10 Ryujin™ balloon was threaded over the wire beyond the embolized fragment, inflated and pulled. However, this procedure failed in bringing out the embolized fragment, so the balloon was fished out. Another attempt was made by passing another 0.014 BMW™ wire along the side of fragment distal to it and then inter-twinning the two wires and then pulling the whole assembly; entangled two wires and the broken fragment out. However, this procedure too failed. Meanwhile, patient started showing signs and symptoms of raised intracranial tension and check angiogram revealed no antegrade flow in right MCA [M1segment] with extravasation of contrast in the right MCA territory. Patient was put on mechanical ventilator. CT scan revealed hematoma. To emergently salvage the situation a right decompressive craniectomy and lax duroplasty were carried out. However, despite all emergency efforts patient's neurological status deteriorated followed by vasomotor failure. Ultimately, patient became non-responsive to maximal therapy and succumbed to death.

Analysis of the case: Usually only coronary embolizations need treatment and most noncoronary embolizations can be left alone because extraction of embolized fragments is not as innocuous as it seems and often leads to a lot of complications. However, intracranial embolization may be an exception. If embolized fragment has to be dealt, crushing the fragment is safest and retrieving is only the second choice. However, if stent is the embolized material, trying to re-wire and re-deploy is often the safest strategy.

Case 7

Patient was a 63-year-old gentleman, hypertensive, current smoker who presented with acute anterior wall STEMI; window period of 4 hours. Echocardiography revealed regional wall motion abnormality in the interventricular septum with moderate LV dysfunction. A loading dose of clopidogrel 600 mg was given immediately and patient shifted for primary PCI. Coronary angiogram revealed that LAD was calcified ostial thrombotic total occlusion with ultra-short left main (LM). LCx revealed proximal 75% lesion while RCA revealed mild proximal disease. The lesion was crossed and thrombosuction carried out. Subsequently this lesion was balloon dilated which achieved a TIMI II flow but lesion still looked underdilated. While trying to pass a longer balloon it could not be tracked. Thus a buddy wire approach was taken to increase wire support. Meanwhile patient developed slow flow phenomena with hemodynamic instability. At this moment IABP was instituted and TPI support obtained. During all the maneuvering the thrombus was identified in left main and ostial LCx.

Subsequently, thrombosuction in LCx and left main coronary artery which achieved a good flow in these vessels but no reflow in LAD, Subsequently patient arrested and could not be revived.

Analysis of the case: Ultra short LM with thrombotic, calcified, true ostial LAD always pose significant challenges during primary PCI. Resistance to thrombosuction catheter and balloon is common. Slow flow and no reflow is also more common than a garden variety of PCI. There could be several reasons for thrombus visualization in proximal vessel: Inadequate heparinization is perhaps the commonest. However, there could be several other causes as well: Thrombus proximal migration when the thrombosuction or balloon catheter is withdrawn is another. Other reasons could be proximal thrombus migration when the distal lesion is dilated or even injury to proximal segment by cath lab hardware such as thrombosuction catheter or balloon catheter or even guide-catheter trauma. In this case any of the factors (or all) could be operative. Further, this patient was loaded with clopidogrel (which requires at least 5 hours for optimal effect). The patient should have been pre-loaded with prasugrel or ticagrelor or alternately GpIIb/IIIa inhibitors should have been administered. Institution of prophylactic IABP and TPI might be useful adjuncts but by themselves may not be enough. Likewise prophylactically using filter devices may be useful in individual case but as a strategy have not shown consistent benefit.

FACTORS AFTER THE PROCEDURE

While most cases of mortality are related to either prior to procedure or at least during the time of procedure but there are some situations which contribute to mortality after the procedure, particularly in early post-PCI period. As such setting up a standard operating procedure (SOP) in the early post-procedure period after PCI is critically important part of all cath lab protocols. The key is careful monitoring, anticipation of problems and taking timely steps to ameliorate them. While stent thrombosis can occur at any stage after PCI the possibility decreases temporally. Other problems like groin complications look innocuous but may contribute to mortality especially retroperitoneal hematoma which should be clinically suspected early, confirmed directly by ultrasound or CT scan or even angiography and suitable steps including surgery if required undertaken, otherwise many a times it can be fatal. Cardiac tamponade is another dreaded complication occurring during early post-procedure period (typically within 6 hours), is often related to guidewire perforation. Here again anticipation (and thus avoidance of GpIIb/IIIa antagonists) and early diagnosis by clinical suspicion followed pericardial tap and other interventions could be life-saving. Massive internal bleeding; gastrointestinal or cerebrovascular could be another fatal event. Rarely other factors may complicate an otherwise completely successful PCI such as contrast induced nephropathy, late free wall rupture, arrhythmic storm, etc.

CASE EXAMPLES

Case 8

Patient was a 86-year-old active male, hypertensive, dyslipidemic, COAD-on inhalers. During the past one year he had been admitted for ischemic LVF 3 times. He presented with unstable angina (Trop I +ve) with ischemic LVF. In view of recurrent ischemic LVF, patient was advised coronary angiogram. Coronary angiogram revealed–Triple Vessel Disease with a Syntax score of 23. Patient underwent PCI of all three arteries in the same setting with a successful outcome. Nearly 2 days after PCI, patient developed hypotension. Echocardiography revealed a hyper-contracting LV with no pericardial effusion. Intravenous fluids were immediately rushed and patient started on low dose of dopamine. At the same time ECG revealed sinus tachycardia with ST elevation in inferolateral leads. Patient was taken up for check angio with a possibility of sub-acute thrombosis. Check angiogram was normal but groin examination revealed a high femoral artery puncture and a hematoma which was intermittently compressed during and after procedure. ACT was 270 sec, hence sheath could not be removed, hence tight bandage given. There was a drop in Hb 4.5 g/dL. Meanwhile, Ryle's tube aspiration revealed 500 mL of coffee colored aspiration. Seeping of blood was also detected in medial/posterior aspect of thigh, therefore 5 units of blood transfusion administered. Over the course patient developed anuria, i.e. acute renal shut down. However, hemodialysis could not be performed due to persistent hypotension. Ultimately, patient expired.

Analysis of the case: Patient had a higher femoral puncture, which should not only have been avoided in first place but the problem quickly recognized and corrective action taken. Proper technique would have been to recognize the hematoma and give manual compression immediately and intermittently, watch out for recurrence. It is important to look not only at site but also in medial/posterior aspect of thigh, and quickly intervene if accumulation is detected there. If patient develops persistent hypotension with tachycardia post PCI, common causes are guidewire perforation (easily detected by echocardiography) or bleeding from any site; retroperitoneal bleed/local site hematoma/GI bleed. In elderly, anticoagulation regimen with low bleeding risk such as bivalirudin may be considered.

Case 9

Patient was a 50-year-old postmenopausal hypertensive female with metabolic syndrome but normal renal function. She had past event of ACS and was treated as NSTEMI. At the time of presentation patient had a TIMI risk score 4, Killip class I, persisting rest angina and resting LVEF 30%. Patient was loaded previous night with DAPT (ASA 325 mg and 600 mg of clopidogrel). On angiogram, LMCA was normal, LAD was Type 3 vessel with proximal tubular type B 90% lesion, grade III hetero-collaterals from right system were apparent. Ramus Intermedius was a small caliber vessel with

diffuse disease. LCx was nondominant with distal diffuse 50–60% disease. OM 1 was 2.5 mm vessel with a proximal 80% tubular type B1 lesion. RCA was dominant with mild plaquing. Right femoral approach was obtained, 7F XB 3.5 guide-catheter was taken and lesion crossed with Cougar XT™ 0.014″ @ 190 cm wire. After predilation with 2 × 10 mm @ 10 atm lesion was stented with—BMS 2.75 × 15 mm stent, deployed at proximal LAD. Subsequently, PCI of OM1 undertaken. Lesion was crossed with Cougar™ wire and lesion predilated with 2 × 10 mm balloon; serially at 6 to 8 atm. Finally, lesion stented with BMS 2.5 × 20 mm stent deployed at 12 atm. Patient remained asymptomatic till next morning 6 AM when he started developing chest pain. ECG revealed down-sloping ST depression infero-lateral leads. Patient did not respond to sorbitrate given sublingually. Patient was taken up for chest angiogram. Re-injection done while continuing chest pain with BP of 165/83 (117) mm Hg. Check angiogram revealed thrombosis of both proximal LAD stent (BMS 2.75 × 15 mm) and OM stent BMS (2.5 × 20 mm) with no ante-grade flow across either stent. LAD crossed with 0.014″ @ 180 wire and thrombus aspiration via thrombus aspiration catheter (Pronto™) performed. Subsequently, POBA was done across entire length of occluded segment from distal towards proximal end of stent with 2 × 10 mm @ 6 atm thrice with a 2.5 × 15 mm balloon at 6 atm with a view to crush the thrombus. The same Pronto™ catheter used again to suck thrombus from LAD system with 5 passes across the thrombus containing lesion. On examining a white thrombus (? Platelet–rich) was detected on the aspirate from Pronto™ aspirator. Again POBA to the entire length of the occluded segment was carried out with 2.75 × 13 mm balloon @ 8 atm for 60 sec across proximal edge of stent. Further, since haze persisted at proximal edge of stent, BMS 2.75 × 10 deployed in proximal LAD with 3 cell overlap with previous 2.75 × 15 mm stent. After that LCx lesion was crossed with 0.014″ @ 180 cm guide-wire. Again POBA to the entire length of the occluded segment was performed with 2 × 10 mm balloon @ 6 atm thrice from distal towards proximal part of stent and then with 2.5 × 15 at 4 atm × 4 times from distal towards proximal part of stent. The lesion decreased from total occlusion but 20% lesion with layered organized thrombus still persisted with in stent margin. Patient reloaded with prasugrel 60 mg. However, during PCI of LCx, it was evident that more thrombus was again forming across LAD stent. Again thrombus was repeatedly aspirated with Pronto™ catheter but it kept reforming with amazing speed. However, after multiple thrombus aspiration attempts, new thrombus load seemed to be reducing and when <10% thrombus was left lining the inner edge of the stent, the result was accepted. Weight based injection eptifibatide was administered intracoronary followed by IV injection for 4 hours. However, post-procedure ejection fraction had dropped to 15–20%. Patient was kept intubated and continued to be ventilator dependent. LMWH, DAPT (ASA+ Prasugrel), statins were continued after intravenous eptifibatide dose was over. However, 10 hours after the procedure patient again developed severe chest pain, developed sudden cardiac arrest and despite CPR delivered in CCU patient could not be revived.

Analysis of the case: The patient represents a case early (acute) stent thrombosis. The cause of acute stent thrombosis is mostly procedural. The stent 2.75 × 15 mm in LAD could have been under-deployed so could have been the OM stent (2.5 × 20 mm). Alternately, the stent could have been malapposed (anyway they were deployed at only 12 atm pressure and postdilatation was not performed). At the same time patients were preloaded with clopidogrel; loading with prasugrel or ticagrelor may have been a better idea in a patient presenting with ACS. The second sub-acute thrombosis could have been due to injury of the second procedure when entire segment was ballooned but all the areas were not covered with stent. Another remote possibility could have been ASA or clopidogrel resistance which led to recurrent thrombosis as soon as the effect of heparin and IIb/IIIa inhibitor wore off.

HOW TO PREVENT MORTALITY

From organizational standpoint: Currently, one of the biggest factors which can help reduce mortality in cath lab is reducing the door-to-balloon time during primary PCI.[18-20] Other ways of reducing mortality include having monthly meetings between hospital clinicians and emergency medical services (EMS) to review PCI cases, having cardiologists always on site to carry out emergency procedures without delay, having pharmacists round on patients with AMI, but also importantly having an organizational environment where clinicians are encouraged to creatively solve problems without fear or guilt and where the effectiveness of changes in care are consistently evaluated (focus on outcomes). Further, cross-training critical care nurses for the catheterization laboratory and having nurse champions(s) are also was associated with improving outcomes.[21]

From individuals standpoint: Preventing mortality is like waging war against complications. The pre-requisites as defined by Chinese philosopher Sun Tzu applies equally well to fight against complications as well. He proposes that a good prince who wants to win a war should undertake 5 crucial steps: choose the weather, choose the terrain, choose the general, choose appropriate war strategy but most importantly have peoples benefit at heart. Likewise a good cath lab operator should:

1. *Choose the cath lab/cath lab center*: It should be a properly equipped cath lab with which the operator is well conversant and well equipped with all the hardware (including the Crash Cart). Cath lab staff should be well trained and stand-by CTVS support should be available.
2. *Timing of complex procedures well chosen*: Difficult cases should be scheduled properly; they should not be the first case (check everything is working properly), they should not be at the end of the day (everybody is tired and in a hurry to finish the case), the operator should neither be hungry, nor full, complex cases should not overlap with change-over time of staff. They should be scheduled at a time when at least some OTs are free and surgeons available as stand-by.

3. The operator should have personality attributes akin to a fighter pilot; he/she should be a stickler of method, be skilled/trained—especially to deal with complications, possess a cool temperament—should keep in control once things go wrong. He/she able to make reasonable main strategy but also have Plan B, Plan C ready. He/she be able to think on the run—modify strategy as and when required. In addition, he/she should possess leadership qualities; should be the only person giving commands, he/she should carefully organize duties of others—everybody should know what one is supposed to do in times of emergency. He/she should maintain a clear and proper command and communication chain. Finally, he/she have attributes of a monk; have no fear or pride. Should be able to call for help when needed and should be able to hand over the procedure to somebody more experienced (whether junior or senior).
4. Choose a proper strategy which includes proper selection of case, proper selection of diagnostic modalities, proper selection of hardware **(Table 13.1)**, judicious following of steps and optimization of technique **(Table 13.2)**, proper visualization before finishing the case **(Table 13.3)** and lastly proper monitoring of the patients early and late after the procedure.
5. Finally, the only outcome for which the cath lab operator should be working towards is the welfare of the patient.

Table 13.1 Proper selection of hardware

- Non-invasive imaging (CT)
- Guide-catheter
- Guidewire (Penetrating GW, Gaia wires etc)
- Balloon Catheter (OTW, NC, low profile balloons etc)
- Stent (covered stents)
- Microcatheters
- Imaging catheters
- Distal/proximal protection devices
- Thrombectomy devices

Table 13.2 Lesion preparation and PCI optimization techniques

- Calculate the dose of contrast before starting the procedure (4 × creatinine clearance, max dose 6 × creatinine clearance)
- Plan how long you will continue (fluro time)
- Bilateral access
- Proper vessel preparation (balloon dilatation, rota/cutting balloon)
- Use of thrombus extraction, DPD as required
- Use of imaging pre/post deployment
- Post-deployment optimization (dilatation with NC balloon)

Table 13.3 Proper visualization and monitoring after PCI

- Both proximal and distal parts of total operative field properly visualized before ending the procedure – From distal part where wire had gone, to site of lesion, to ostial part of vessel (guide catheter) to the access site
- Procedure ended in at least 2 views
- Guard should never be lowered till patient discharged and after ----.

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