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Complications of PCI and its Management

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ABSTRACT

Contemporary percutaneous coronary intervention (PCI) has few complications and enhanced safety on one hand and on the other hand, available modern tools have enabled interventional cardiologists to venture into more complex patient subsets and fresh challenges in tackling the ensuing newer complications. Individual operators may take several years of experience before being exposed to the complications and hence, it is important to learn from the collective experience on the detection and management of these complications and better equipped to handle them when the need arises. The complications of PCI can be access related or coronary intervention related. The common acute coronary complications include abrupt vessel closure, thrombus, slow flow, dissection, perforation, stent dislodgement, and guidewire fracture and embolization. The chronic complications include late stent thrombosis, in-stent restenosis, and aneurysm formation. The use of imaging, physiology, and plaque modification tools are associated with specific complications which need to be kept in mind while using them. The successful management of complications needs prompt recognition, involving help from others and continued team effort and vigilance.

Keywords: Percutaneous coronary intervention, Complications, Dissection, Perforation, No-flow

INTRODUCTION

At present, PCI has become a safe procedure and complications occur rarely. Improved hardware, operator skills and greater understanding of approach to PCI, potent anti-platelet agents, switch to radial access, use of non-ionic dye, adjunct imaging, and mechanical cardiac support (MCS) have all contributed to greater safety and better outcomes. On the other hand increasing complexity of procedures, complex and high-risk coronary intervention PCI, use of MCS, and chronic total occlusion (CTO) PCI are all associated with greater incidence of complications. The rarity of complications also means less experience of individual operators in handling various complications are scarce and becomes important to share experiences and learn from collective experience and literature.

COMPLICATIONS DURING PCI

Vascular complications of PCI occurred in 0.6% after diagnostic procedures, 2.6% after elective PCI, and 6% after complex interventions [Table 1].[1,2]

LOCAL COMPLICATIONS

Femoral access complications

Femoral puncture is done at the site of common femoral artery 2–3 cm below the line connecting the pubis symphysis and anterior superior iliac spine at the point of maximal pulse.

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A high puncture leads to risk of retroperitoneal hematoma and a low puncture is associated with pseudoaneurysm, arteriovenous (AV) fistula incidence. The contemporary use of fluoroscopy guidance with puncture over the mid-point of femoral head, ultrasound (USG) guidance and use of micro-puncture techniques, and vascular closure devices result in reduced femoral access complications. A meta-analysis of randomized and controlled trials of standard versus USG-guided puncture of femoral artery showed greater safety and improved efficacy with USG guidance. Successful first attempt was higher, number of attempts was decreased, and complications reduced 1.3% with USG guidance versus 3% in standard approach [Tables 2-4].[3-7]

MANAGEMENT

Retroperitoneal hematoma

Sudden hemodynamic collapse during or after PCI with flank pain and drop in hemoglobin should lead to suspicion of retroperitoneal hematoma. USG abdomen will show a collapsed inferior vena cava signifying volume depletion and fluid collection compressing bladder. Computerized tomography abdomen can detect leak from the femoral artery. It can be due to damage to inferior epigastric branch from the external iliac artery. A check shot at the completion of angiogram or after hypotension can lead to its early detection. The exsanguination can be very rapid and profound leading to significant blood loss and even death. The bleeding vessel can be occluded by micro coils through a contralateral femoral approach. Balloon occlusion of the femoral artery can also lead to sealing of the leak from a small arterial twig. A non-expanding retroperitoneal hematoma can be managed conservatively with blood transfusion and antibiotics. Collected blood can result in infection and sepsis. A small subset of patients with fall in blood pressure, hemoglobin, and hematocrit and not responding to fluid resuscitation may need urgent surgical intervention.

Pseudoaneurysm

Pseudoaneurysm is due to collection of blood from the arterial lumen through the tissue planes sealed off by surrounding fibrous tissue and leads to pain and risk of rupture. A small pseudo aneurysm can be resolved by USG mediated compression of the neck of the aneurysm. Small aneurysms <2 cm in diameter may resolve spontaneously with thrombosis. A direct percutaneous thrombus injection, saline/lidocaine injection, embolization by coils, covered stent, vascular closure devices like angio-seal, and VASCADE have been described in managing pseudoaneurysm. Thrombin injection carries very high success rates albeit with a small risk of embolization.[8] A large pseudo aneurysm needs a surgical intervention [Figure 1a and b].

AV fistula

AV fistula occurs due to a low femoral puncture below the bifurcation, detected by pain, and swelling of the groin post-PCI with the presence of a continuous murmur. It can be managed by manual compression, covered stent or a surgical intervention may be required [Figure 2].

Arterial dissection

The presence of tortuous and calcified vessels is a high risk for arterial dissection caused by guide wire or insertion of sheath. A non-flow limiting dissection can heal spontaneously, a large dissection may need a stenting of the iliac artery which is the most common site [Figure 3a-c].

Arterial thrombosis/Embolism

Artery thrombus can lead to limb threatening ischemia but is fortunately rare. A vascular surgeon must be involved to save the limb. Maintenance of activated clotting time (ACT), use of smaller sheaths, and early removal of sheaths can prevent it.

Infection

The use of aseptic precautions is important. The use of reused sheaths and presence of local hematoma are important causes of infection.

Radial access

Radial access results in reduced vascular complications. Radial artery spasm is the most important complication occurring in 7% of cases. The thrombosis and loss of radial pulse, radial artery occlusion (RAO) occurs in 1–10% of cases.[3]

RAO does not cause hand ischemia but makes it unavailable for AV fistula in chronic kidney disease (CKD) patients and conduit use in coronary artery bypass (CABG) patients. A radial artery to sheath size <1 is important in preventing RAO. The use of sheath less guide catheters and slenderized sheath can decrease the diameter of the access system up to 2 FR size and decrease complications. The use of 5000 units of heparin results in RAO in 4.3% as compared to 24% with 2000–3000 units.

The increased use of radial access has also led to decreased experience in femoral access and paradoxical increase in femoral complications called “Campeau Radial Paradox.”

Persistent pain in arm, loss of upper arm strength, pseudo aneurysm, AV fistula, and radial artery perforation are some of the rare complications. The presence of hematoma is also rare. Radial artery avulsion, compartment syndrome, and upper limb ischemia are the more serious complications.[9,10]

Distal radial access

Distal radial access, snuff box entry has been found safer with better hemostasis in the DISCO RADIAL trial.[11]

CORONARY COMPLICATIONS

Acute complications

Acute complications include abrupt closure, acute stent thrombosis, coronary dissection, coronary perforation and no flow or slow flow [Table 5].

Abrupt closure

The most important causes of abrupt vessel closure are dissection and thrombosis. Vasoconstriction can rarely cause abrupt closure. Acute coronary syndrome (ACS), multivessel disease, CKD, and female gender are associated with greater incidence of abrupt closure. The anatomical features causing risk of abrupt closure are proximal tortuosity, angulated lesion, diffuse lesion, large thrombus, and degenerated vein graft lesions. Patients presented with hypotension, bradycardia, chest pain, ECG changes, and arrhythmia. Vasopressors, inotropes, and intra-aortic balloon pump (IABP) may be required for hemodynamic stabilization. Imaging with intravascular ultrasound (IVUS) and optical coherence tomography (OCT) may be helpful in detecting the cause of abrupt closure. Balloon dilatation, optimization of ACT, and stenting to seal the dissection are the immediate remedial actions. Aspiration thrombectomy, glycoprotein (GP) IIb/IIIa inhibitor, and intracoronary lysis may be used in acute thrombosis.

Acute stent thrombosis (ST)

Acute ST occurs within 24 h of PCI. The predisposing factors of acute ST include ACS scenario, large thrombus burden long stent, diabetes mellitus (DM), CKD, anemia, diffuse coronary artery disease (CAD), bifurcation lesion, choice of antiplatelet regimen, and lower luminal area after stenting. The IVUS predictors of acute ST are final MLA <5 mm, malposition of stent struts, plaque prolapse, edge dissection, under expanded stent, and residual plaque. Management is by thrombus aspiration, angioplasty, stenting in cases of missed edge dissection or disease at the edge of previously deployed stent and switching to more potent antiplatelet drugs and use of GP IIb/IIIa inhibitors.

Coronary dissection

Coronary dissection can occur caused by guide catheter of ostium of left main coronary artery (LMCA) or right coronary artery (RCA), can be caused by guidewire, balloon, guide extension catheter, imaging catheters, fractional flow reserve (FFR) wires, rota burr, and other adjunctive devices. Coronary dissection should be suspected by ischemic ECG changes, hypotension, or bradycardia.

The incidence of coronary dissection is 0.2%. Coronary dissection, it is important to wire the true lumen, ballooning to establish flow and deployment of stent seals the dissection. The use of forceful dye injection should be avoided as it can result in propagation of the dissection. At the completion of PCI, it is important to check orthogonal views or imaging to diagnose significant edge dissection. Imaging by OCT is accurate in detection of edge dissection. OCT is highly sensitive and may diagnose all smaller dissections as well. A dissection of <3 mm length, arc <60 degrees, and limited to media only need not be stented [Table 4].

Guide catheter-induced dissection is usually due to deep throating, non-co-axial alignment of catheter, small, and diseased arteries. In a study of 56,968 patient cohort, catheter dissections occurred more commonly in RCA in 50% as compared to LMCA in 45% of cases. The predisposing factors were use of large guiding catheters, female sex, LMCA disease, complex lesions, deep seating, and use of AmPlatz catheter. The presence of decreased antegrade flow is associated with poorer prognosis. LMCA dissection can be life-threatening and prompt stenting of ostium or the main artery when the dissection extends into it is important to salvage the patient. The LMCA dissection can retrograde into the aortic root which occurs rarely in 0.02% of cases. Dissection extending <40 mm into the aortic root can be treated by LMCA ostial stenting. Aortic dissection extending beyond 40 mm needs surgical repair [Figure 5a and b].

Coronary perforation

Coronary perforation is defined extravasation of the contrast medium or blood from the coronary artery. The incidence is <1.0%. It is more common in calcified vessels, CTO. Women and elderly are more prone. It may be caused by guidewire perforation and vessel rupture.[12,13]

There are three types of coronary perforation described depending on the location of the perforation.

1. Large epicardial artery
2. Distal small vessel or branch
3. Collateral vessel.

RISK FACTORS FOR CORONARY PERFORATION

The major risk factors for coronary perforation include over sizing balloon to artery ratio >1.2, high pressure inflation, calcified vessels, small vessels, over sized stents, CTO with sub-intimal entry, retrograde epicardial collateral entry, and use of plaque modifying tools such as Atherectomy, Excimer laser and IVL use [Table 6].

CLASSIFICATION OF CORONARY PERFORATION

Ellis classification

- Type I – Focal extravasation -intimal crater without extravasation
- Type II – Pericardial or myocardial blush without contrast staining
- Type III – Contrast extravasation through perforation >1 mm
- Type III cavity spilling – Perforation into an anatomic chamber, coronary sinus, etc.

Grade 3 perforation is associated with tamponade, shock, MI and/or death, delayed tamponade may occur. The severity of perforation correlated with procedural mortality, starting from 0% for Ellis I, 1.7% for Ellis II and 21% for Ellis III. Ellis I perforations were more often wire induced and Ellis III perforations were due to balloon or stent deployment.[14]

Large vessel perforation should be managed emergently by prolonged balloon occlusion of 5–10 min up to a maximum of 20 min and sealed by covered stent. The ping pong technique is use of additional guide catheter, and the stent is delivered on another wire while transiently deflating the balloon on the wire through the first guide. Pericardiocentesis may be required. Anticoagulation reversal may be required in ruptures with rapid leak into pericardium but should be done after removing the hardware in the vascular system [Table 7].

Covered stents available [Figure 6a-c]

A conservative strategy may be adequate in distal or small perforations but covered stents, coil embolization, and surgery are needed for brisk ruptures. Covered stents are bulky and there may be difficulty in delivering the stent and may need guide extension catheters.[15] Long-term DAPT therapy is advised with covered stents. In the CRACK registry data of covered stents for ELLIS 3 perforation complete sealing occurred in 83.2% but on 1-year follow-up had high risk of complications, cardiac death (7.1%), target lesion revascularisation (13.1%), myocardial infarction (MI) (11%), and ST in 6.1%.[14-21]

The Glasgow Natural History Study of Covered Stent Coronary Interventions study was a 18-year period study of incidence and outcomes of coronary perforations from a single center. The incidence was 0.37% of PCI and Ellis Type III perforations occurred in 35% of the total perforations. There was an increased incidence of coronary perforations over the years reflecting the increase in more complex interventions in the elderly, calcific lesions, and CTO. Rotational atherectomy was more commonly used in patients with Ellis's Type III/III B perforations. Over a period of 2.9 years, procedural mortality was 0% in Ellis Type I, 1.7% in Type II and 21% in Type III perforations, MACE was 47% in Ellis Type III/III B and 13.5% in Ellis Type I/II. Covered stents had a high incidence of ST of 9.1% versus 0.9% over the study follow-up of 2.9 years. Failure to deploy covered stents is associated with very high mortality, 47% within 24 h [Tables 7 and 8].[22]

When a covered stent is not available and perforation occurs, we can use two routine stents of desired size and make a low profile covered stent. Two stents of vessel size one with desired length to cover the perforation and the next, one size longer are wired in opposite directions on a single wire. The shorter one is inflated to more than nominal pressure. The inflated balloon gets attached to the stent. Both the edges were cut around separating the central tube. The assembly is transferred over the longer stent and hand crimped. Now, a routine stent over which there is a cover and hand crimped stent holds the entire unit together. The sandwiched stent is delivered over the perforation in routine manner. During the inflation, two stents with sandwiched balloon material in-between gets inflated sealing the perforation. There have been case reports of Grade III perforations handled in this indigenous manner.

A series of 24 patients with 31 self-made polyurethane covered stents has been described using a Polyurethane membrane cut from 3 M™ Tegaderm, to form a cover over standard DES by cutting it 2 mm shorter than the length of DES and width of 8 mm, wrapped twice around DES. The self-made stent had low profile with good deliverability and 79% success rate has been described with in-hospital mortality of 16% and 2-year MACE of 27% [Figure 4a-d].[23]

Stent loss

In the recent years, loss of stent has become rare but when it occurs, it is associated with high incidence of complications, such as MI, emergency surgery, stroke, and death. Coronary calcification, tortuosity, and poor preparation of the vessel before stent deployment are the major reasons leading to stent loss.[24] Stent loss occurred in 1.3% of PCIs in a meta-analysis of 71,665 PCIs. Recent studies report that an incidence of 0.3%. 66% of the stent loss was successfully retrieved, 12% deployed in the coronaries, 3% were crushed, and 3% left untreated. Clinical complications occurred in 19% of these 57% had surgery, 18% MI, 19% died, and 1 patient (0.6%) had stroke.[25] The key to successful retrieval is to maintain guidewire position securely. Stent may be dislodged in the coronary or in the aorta. Stent loss with guidewire in situ, snaring, multiwire and crushing techniques can be used. The loss of both stent and guidewire needs snaring, crushing technique or use of forceps/bioptome. Stent loss can occur in cerebral, carotid, or lodged in peripheral arteries above or below the femoral artery.

No flow or slow flow

No flow is defined as inadequate myocardial perfusion in the absence of mechanical obstruction of epicardial vessel.

It occurs in 0.6–3.2% of cases. The no-flow and slow flow phenomenon occur most commonly in ACS situation due to coronary micro-embolization, reperfusion injury, microvascular spasm, inflammation, and oxidative stress. It leads to increased MI, death, and heart failure. No flow is associated with chest pain ECG changes, arrhythmias, hypotension, MI or cardiogenic shock, left ventricular dysfunction, and may result in cardiac rupture.[26,27]

The predisposing risk factors for no-reflow phenomenon in ST elevation MI are advanced age, high grade thrombus, delayed presentation, Killip class 3-4, absent collaterals to infarct related artery, and high blood sugar levels, smoking, hypertension, hyperlipidemia, female sex, and CKD.[28]

The angiographic diagnosis of no-reflow is based on TIMI flow, myocardial blush grades (MBG), and TIMI frame count. TIMI flow <3 and MBG <3 is associated with increased mortality and left ventricular dysfunction.

It is more common to occur following rotational atherectomy and vein graft interventions.[29]

The use of direct stenting, thrombus aspiration and use of intracoronary vasodilators before stenting may prevent no-reflow.

The treatment of no flow includes vasodilators such as nicorandil, adenosine, verapamil, and sodium nitroprusside which improve microvascular dysfunction. Sodium nitroprusside in a doe of 50–300 μgm intracoronary administration had more sustained benefit than nicorandil. Intracoronary epinephrine has also been used in patients with hypotension and refractory no-flow.

GP2b/3a inhibitors and intracoronary thrombolysis can decrease thrombus load and improve no flow. IABP support may be required in shock state.

LATE COMPLICATIONS

Late and very late ST (VLST)

Late ST is defined as ST 1 month after PCI and VLST as occurring 1 year post-PCI. Late ST occurs at the rate of 0.6%/year with drug eluting stent (DES) and bare metal stent (BMS). The second-generation DES had a lower ST and have replaced BMS for all clinical situations. The bio absorbable stents came with a promise of absent risk of ST as the stent is completely absorbed over time, but the ABSORB II trial showed increased ST compared to the Everolimus stent at 3-year follow-up, leading to its world-wide withdrawal. ST presents as ACS, MI or sudden death and carries high mortality of close to 50%. ST causes 20 % of MI in patients with prior PCI and VLST carries a 4-fold higher mortality in comparison with non-stent related MI.

The causes of ST are patient-related, procedure–related, or secondary to problems in pharmacotherapy. The most important predisposing factors are DM, hypertension, CKD, ACS, multi vessel disease (MVD), bifurcation lesions, saphenous vein graft (SVG) graft lesions, prior CABG, long stents, small stent size, overlapping stents, residual stenosis, stent under expansion, malapposition, first generation DES use, DAPT discontinuation, hypo responsiveness of the drug or hyper platelet reactivity or hypercoagulable states, and lack of use of potent antiplatelet drugs. Recently, COVID infection has been reported to be associated with higher incidence of late ST. Imaging studies have shown that malapposition, under expansion, delayed endothelialisation with exposed struts, and plaque rupture due to neo-atheroma to be the important causes of LST and VLST.

The management of LST and VLST is angiography and imaging followed by appropriate action of balloon dilatation and deployment of second DES if required and use of potent antiplatelet regimen for longer period of time. Armstrong *et al*., in their study of 7315 cases of ST, there was 70% use of stents in VLST as compared to 51% use in early ST.[30] OCT helps in studying the underlying mechanism of ST and helps in addressing the underlying problem such as under expansion, malappposition, uncovered stents, neo atheroma, or edge disease. Balloon angioplasty may be sufficient for under expansion and malapposition but neoatheroma and edge disease need a new stent to be deployed. Neo atherosclerosis within the stent can also be treated by drug coated balloon (DCB) and excimer laser therapies.[31,32] In a study of 370 patients with ST, LST and VLST were more common in patients undergoing hemodialysis (22.9% vs. 2.7%) and in-stent restenosis (ISR) was more common (25.9% vs. 9.4%) as compared to the early ST group.[33]

ISR

ISR rates have reduced after widespread DES use and the cause can be neo intimal hyperplasia, neoatherosclerosis, or geographical miss due to stent deployment in a diseased zone. It occurs in 5–10% of cases.

Four distinct angiographic patterns have been described by Mehran *et al*. [Table 9].[34]

In an analysis of 288 lesions, 42% were focal, 21% were diffuse intra stent, 30% were diffuse proliferative, and 7% total occlusions.

Waksman ISR classification of DES is based on the underlying mechanism of ISR, includes five types.

- Type I – Mechanical, IA – Under expansion and IB – Strut fracture
- Type 2 – Biologic, IIA – Intimal hyperplasia, IB – Neo atherosclerosis–non-calcified, and IIC – Calcified neoatheroma

- Type 3 – Mixed
- Type 4 – CTO
- Type 5 – DES-ISR treated previously with more than two layers of stents. Mechanical is related to factors such as stent under expansion, strut fracture, malapposition which can be initial or acquired late type, biological refers to inflammation and hypersensitivity reaction and neoatherosclerosis, and mixed refers to a combination of mechanical and biologic factors.[35]

Table 1: Complications of PCI.

1. Local access related complications
2. Coronary complications
   a. Acute –Stent thrombosis, Intra mural haematoma coronary perforation, cardiac tamponade, No flow, acute vessel closure, dissection, thrombus migration, branch vessel closure, stent loss, failure to deliver stent, under expanded stent, stent distortion, and entrapment of rota burr
   b. Chroni – ISR, LAST, Coronary aneurysm
3. Arrhythmic complications – VT, VF, Asystole, bradycardia, tachycardia, SVT, AF
4. Non coronary complications – Renal failure, acute respiratory distress, seizures, stroke
5. Death

PCI: Percutaneous coronary intervention, ISR: In-stent restenosis, VT: Ventricular tachycardia, VF: Ventricular fibrillation, SVT: Supraventricular tachycardia, AF: Atrial fibrillation, LAST: Late stent thrombosis

Table 2: Femoral access complications.

- Major complications
- Local hematoma
- Retroperitoneal hematoma
- Pseudoaneurysm
- Arteriovenous fistula
- Thrombosis, embolism
- Arterial dissection, rupture
- Infection
- Minor complications
- Minor hematoma
- Local pain
- Bruising and discoloration

Higher classes, shorter time to initial ISR, and recurrent ISR have been found to be powerful predictors of target vessel failure and a possible genetic predisposition has been proposed.

DM has also been associated with higher ISR rates.

The underlying cause for ISR may be stent under expansion which can be due to stent under-sizing, deployment of stent at low pressures, or inadequate lesion preparation in a calcific vessel.[36] The stent not covering normal to normal zone, geographical miss, stent fractures, and drug hypersensitivity are the other causes. The late ISR is usually due to neoatherosclerosis.

The management of ISR, imaging to know the underlying mechanism, and optimize the result is important, both DCB and DES can be used, DES gives better luminal gain than DCB but clinical MACE rates are similar. Paclitaxel eluting balloons and sirolimus eluting DEB are available. Scoring or cutting balloons before DCB is advisable. Rotational atherectomy or Excimer laser with contrast injection in ISR is useful in calcific neoatherosclerosis with under expanded stents. In patients with recurrent or diffuse ISR, CABG should be considered.

Coronary aneurysms

Local injury following stenting can lead to coronary artery aneurysms (CAA) but is rare following second- and third-generation DES but may occur more following biodegradable stents. Infection or immune complex deposition may also result in CAA. Many of the CAA are pseudo aneurysms following dissection and vessel rupture following rotablation, laser, and oversized balloon dilatation. The incidence is about 0.2–2.3% and can occur as early as 3 days or delayed up to 4 years.

CAAs have been classified into three types.

- Type 1 – Rapid presentation within 1 month of PCI due to vessel wall injury
- Type 2 – Chronic presentation due to metal, polymer or drug hypersensitivity
- Type 3 – Mycotic aneurysm secondary to infection and associated with fever. They can present with angina, ACS, MI, or rarely rupture and cause cardiac tamponade.

Table 3: The patient factors leading to excess femoral complications.

Advanced age	Females	Obesity	Small body surface area	Diabetes mellitus	Hypertension	Peripheral vascular disease	Chronic kidney disease	Thrombocytopenia anaemia	Calcific vessels	Tortuous vessels

Table 4: The procedural factors leading to femoral access complications.

Large arterial sheath	Long duration of in-dwelling sheath	Repeat arterial puncture	Inappropriate access site	Left femoral access	Excess anticoagulants and use of glycoprotein IIb/IIIa inhibitor

Table 5: Coronary complications of PCI.

• Abrupt closure
• Stent thrombosis
• Dissection
• No flow, slow flow
• Coronary perforation
• Stent loss

PCI: Percutaneous coronary intervention

Table 6: Classification of coronary dissection.

Type A-Minor radiolucency with dye persistence
Type B-Double lumen appearance
Type C-Extraluminal cap with dye persistence
Type D-Spiral defect
Type E-New filling defects
Type F-Total occlusion

Table 7: Factors causing risk of coronary perforation.

1. Over sizing balloon-balloon to artery ratio>1.2
2. High pressure inflation
3. Calcified vessels
4. Small vessels
5. Stenting of lesion with oversized stents
6. Chronic total occlusion with sub intimal entry
7. Retrograde epicardial collateral entry
8. Atherectomy, excimer laser, intravascular lithotripsy use

Table 8: Covered stents available.

1. Graft master, direct stent	PTFE, a polymer layer placed between layers of stainless steel-sandwich design, bulky, has difficult delivery, poor long term outcomes with high ISR 25% and ST 3-16%
2. BeGraft stent	cobalt chromium stent with an e PTFE membrane clamped at stent ends
3. PK papyrus stent	polyurethane covers the workhorse Biotronik stent, improved deliverability but high ISR-9% and ST-10% at 1 year
4. Aneugraft stent	Equine pericardium covered stainless steel stent-ISR is higher than graft master and papyrus stents.

PTFE: Polytetrafluorethylene, IRS: In-stent restenosis

Table 9: Angiographic patterns of ISR.

Class I	Focal ISR	Lesions <10 mm in length, body of the stent or at the proximal or distal margin
Class II	Diffuse intrastent ISR, Lesions>10 mm within the margin of the stent	
Class III	Diffuse proliferative ISR	Lesions >10 mm extending beyond the stent margins
Class IV	ISR with total occlusion	

ISR: In-stent restenosis

Table 10: Complications of OCT/IVUS.

Complications	OCT (%)	IVUS(%)
Transient ST elevation	0.26	0.08
Coronary spasm	0.09	0.04
Bradycardia	0.18	0.04
Thrombus	0.09	0.16
Dissection	0	0.12
Deformation of stent	0	0.04

OCT: Optical coherence tomography, IVUS: Intra vascular ultrasound

Table 11: Complications of FFR.

Conduction disturbance	0.03%
Bronchospasm	0.02%
Dissection	0.03%
Ventricular arrhythmia	0.02%
Thrombus	0.01%

FFR: Fractional flow reserve

Table 12: Complications of rotablation.

• Acute no flow
• Major coronary dissection
• Athero embolism
• Hypotension
• Bradycardia
• Coronary perforation
• Entrapment of Burr
• Fracture of Rota wire

Covered stents can be used to exclude the CAA or coils can be used to close them when involving a side branch [Figure 7a and b]. A large CAA in a major vessel or left main may need surgical intervention.[37,38]

a. Coronary aneurysm of proximal left anterior descending (LAD) artery within 10 days of implantation, allergy to DES, polymer hypersensitivity, or infection being the possibility. Patient underwent CABG with the left internal mammary artery (LIMA) to LAD and discharged in stable condition.
b. IVUS image showing aneurysm with multiple air pockets communicating to the vessel.

Complications during Imaging and physiology measurements

Intravascular imaging is recommended in optimizing PCI and improving acute and long-term outcomes. Intravascular imaging helps in knowing the true vessel size, choosing the normal landing zone, selecting optimum stent length and size, assisting in decisions on lesion preparation, and correcting edge dissection and stent malposition.

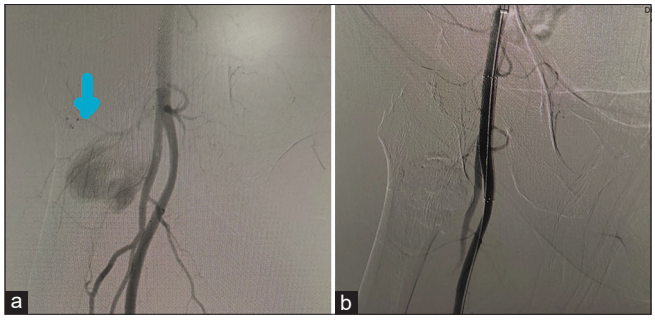

Figure 1: (a) A leaking pseudoaneurysm (Arrow). (b) A sealed pseudoaneurysm following balloon occlusion.

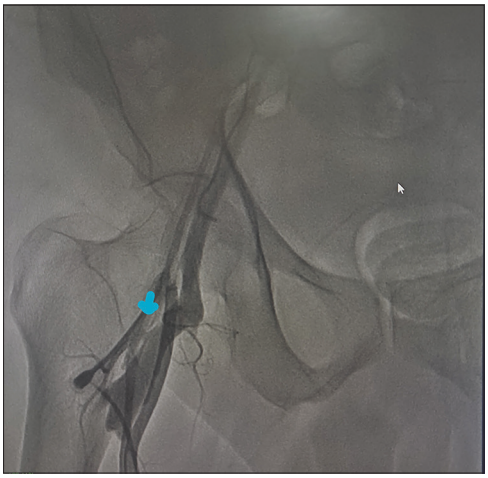

Figure 2: Arteriovenous fistula (Arrow).

The incidence of complications in a study of 1142 OCT and 2476 IVUS procedures was 0.5%.[39]

There were no fatal complications and most of the were self-limiting following removal of catheter.

A recent case report of OCT catheter stuck in distal LAD and derailed from guidewire, in a calcific LAD lesion with overlapping DES leading to deformation of stent in LAD lesion, resulting in slow flow, ventricular tachycardia, and death has been reported [Table 10].[40]

FFR

FFR is used to identify lesions of hemodynamic significance in stenosis between 30% and 90% and deferred stenting based on FFR leads to reduction of MACE as compared to routine angiographic stenoses based decisions on stenting. The IRIS-FFR registry included 5846 patients with FFR measurement. The incidence of complications in this registry was 0.1% [Table 11].[41]

COMPLICATIONS DURING PLAQUE MODIFICATION THERAPY

Rotablation[42]

Rotablation is required to prepare lesion in densely calcific lesions and balloon uncrossable lesions. The use of rotablation is limited by the fear of its complications and is used in 1–3% of cases.[43]

The most important complications encountered during rotablation.

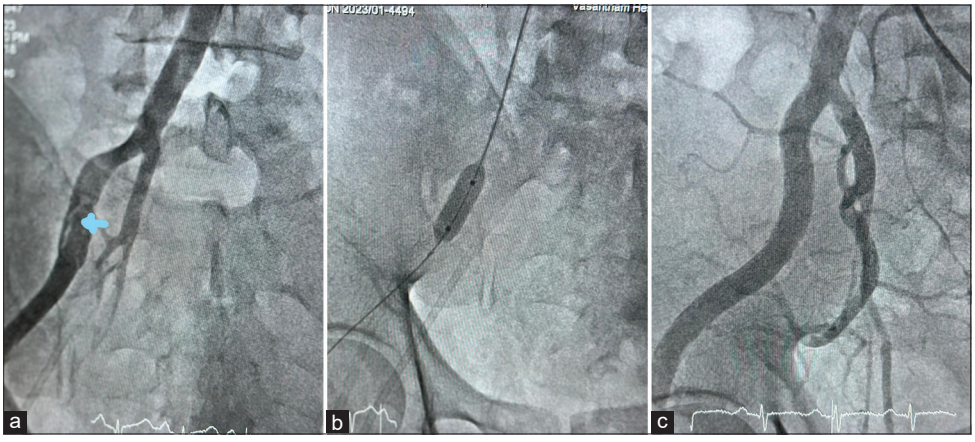

Figure 3: (a) Femoral Artery dissection producing hypotension, drop in hemoglobin, and shock during PCI (Arrow). (b) Balloon occlusion from contralateral femoral access. (c) Check angiogram showing completely sealed off dissection.

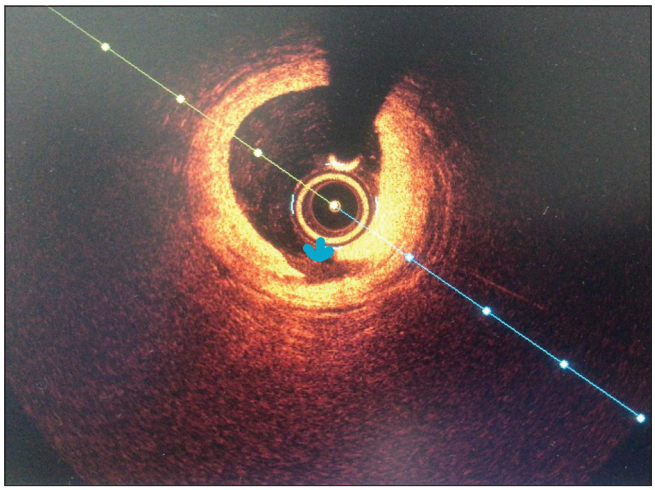

Figure 4: OCT image of edge dissection (Arrow).

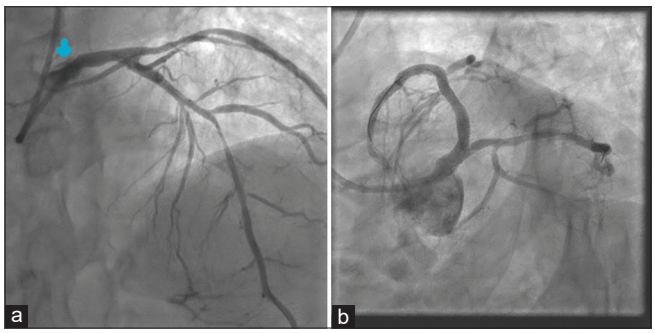

Figure 5: (a) Guide induced left main dissection due to non-coaxial catheter (Arrow). (b) Post-stenting of left main after early recognition.

The complications of rotablation are major and difficult to handle and may result in fatality. Hence, rotablation is generally considered contraindicated in thrombus containing lesions, vessels predilated and having major dissections, saphenous vein grafts, and only remaining vessel.[43]

The important steps to adopt to prevent complications include sizing the burr to 0.4–0.6 of vessel size, gentle pecking to and fro motion, short runs of <20 s, and avoiding burr deceleration.[44]

In a study of 13,335 cases from the J-PCI registry, the incidence of n-hospital death, tamponade, and emergency surgery was 1.31% following rotablation. Death in 0.6%, tamponade in 0.6%, and emergency surgery in 0.18% of cases. The use of rotablation in emergency PCI is associated with 4-fold higher risk than elective PCI; hence, it is preferable to accept TIMI III flow with balloon dilatation in primary PCI and perform elective rotablation at later date. Advanced age, CKD, previous MI, MVD, and low institutional volume are other risk factors for increased complications.[45] Coronary perforation rates are higher with rotablation 2% versus 0.4% in other cases. Extreme tortuosity, angulation, RCA or left

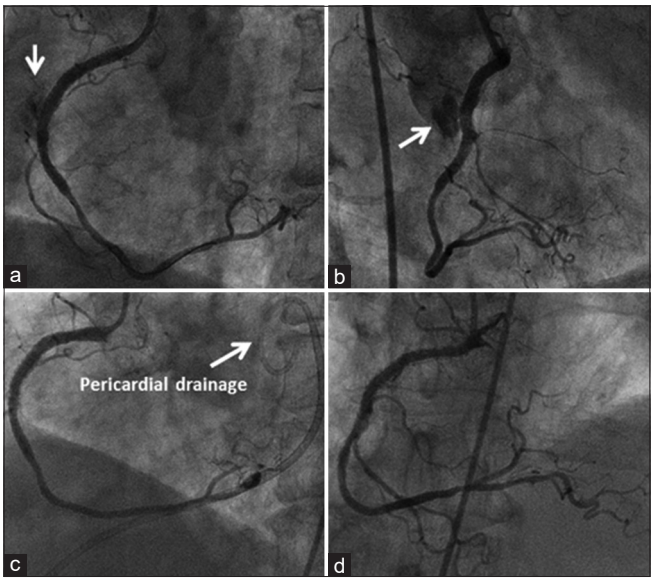

Figure 6: (a) ELLIS Grade III Perforation, (b) Extravasation, (c) Pericardial drainage, and (d) Sealed perforation after balloon occlusion (Arrow).

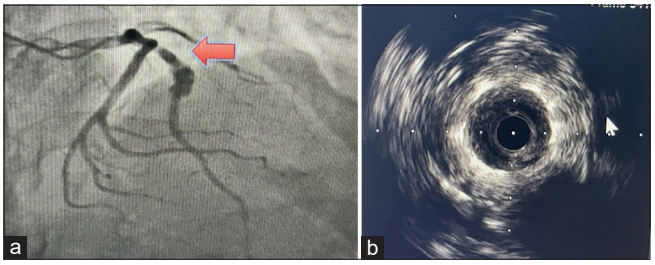

Figure 7: (a) Coronary aneurysm of proximal left anterior descending artery (LAD) within 10 days of implantation, allergy to drug eluting stent, polymer hypersensitivity, or infection being the possibility. Patient underwent coronary artery bypass with the left internal mammary artery (LIMA) to LAD and discharged in stable condition (Arrow). (b) Intravascular ultrasound image showing aneurysm with multiple air pockets communicating to the vessel.

circumflex lesions, and long lesion length are predictors of increased complications [Table 12].[46]

Excimer laser angioplasty

Excimer laser is used in moderate calcific vessels, ISR lesions, balloon uncrossable and un dilatable lesions, CTO, ostial lesions, and SVG graft thrombus containing lesions.[47] The PELCA registry of 1521 patients had complications of dissection – 22%, vasospasm – 6.1%, perforation – 2.4%, arrhythmia – 0.7%, and aneurysm formation – 0.3%. Death occurred in 0.7%.[48] Excimer laser use has been found to be safest in ISR lesions and worst complication rates in CTO lesions. The recent modification has made laser catheters smaller and safer. In a recent study of 119 patients, the dissection rate was 3% and perforation of 1.7%.

Intravascular lithotripsy (IVL)

IVL is used to treat calcific lesions by delivering pulsatile sonic pressure waves**.** In the disrupt CAD II study on 121 patients, IVL catheter delivery was successful in all the patients, the incidence of in hospital MACE was 5.8% and there were no procedure-related deaths.[49]

IVL can be used *de novo* in calcific lesions or secondary use after non-compliant balloon failure or stent under expansion. The complications include dissection, balloon rupture and coronary perforation. The disrupt III trial showed no perforations, abrupt closures and slow flow events, there were only dissections which were managed successfully.

In a study of 71 patients by Aksoy *et al*., IVL was used in three groups, Group 1 – *de novo* in calcific lesions, Group 2 – Failure of non-compliant balloon, and Group 3 – Stent under expansion. IVL success was achieved in 84.6% in Group 1, 77.3% in Group 2, and 64.7% in Group 3. Balloon rupture occurred in seven patients without any adverse outcomes.[50]

Complications with IVL are rare and are mostly coronary dissections and balloon rupture. Perforations, slow flow, and death are extremely rare. They have a short learning curve for their use and are useful in both superficial and deep calcium containing lesions.

CONCLUSION

Complications of PCI have become rare but being mindful of prevention of complications by adopting safe practice and skills is critical to patient safety and outcomes. Early recognition of complications and team effort to quickly stabilize the situation is instrumental to avoid adverse outcomes. The post PCI follow-up is crucial to detect and address the late complications.

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Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Doll JA, Hira RS, Kearney KE, Kandzari DE, Riley RF, Marso, SP, *et al*. Management of percutaneous intervention complications. Circ Cardiovasc Interv 2020;13:e008962.
2. Godino C, Colombo A. Complications of percutaneous coronary intervention. In: Pan Vascular Medicine. Germany: Springer; 2002. p. 2297-2322.
3. Sherev DA, Shaw RE, Brent BN. Angiographic predictors of femoral access site complications: Implication for planned percutaneous intervention. Catheter Cardiovasc Interv 2005;65:196-202.
4. Dencker D, Pedersen F, Engstrøm T, Køber L, Højberg S, Nielsen MB, *et al*. Major femoral vascular access complications after coronary diagnostic and interventional procedures: A Danish register study. Int J Cardiol 2016;202:604-8.
5. Rashid S, Hughes S. Risk factors for femoral arterial complications and management. Br J Cardiol 2016;23:155-8.
6. Bangalore S, Bhatt DL. Femoral arterial access and closure. Circulation 2011;124:e147-56.
7. Sorrentino S, Nguyen P, Salerno N, Polimeni A, Sabatino J, Makris A, *et al.* Standard versus ultrasound-guided cannulation of the femoral artery in patients undergoing invasive procedures: A meta-analysis of randomized controlled trials. J Clin Med 2020;9:677.
8. Dalvin M, Dessecker B, Vitvitsky E. Treatment of common femoral artery pseudoaneurysm: A novel approach utilizing a VASCADE percutaneous closure device. Case Rep Surg 2019;2019:1397981.
9. Roy S, Kabach M, Patel DB, Guzman LA, Jovin IS. Radial artery access complications: Prevention, diagnosis and management. Cardiovasc Revasc Med 2022;40:163-71.
10. Sandoval Y, Bell MR, Gulati R. Transradial artery access complications. Circ Cardiovasc Interv 2019;12:e007386.
11. Aminian A, Sgueglia GA, Wiemer M, Kefer J, Gasparini GL, Ruzsa Z, *et al*. Distal versus conventional radial access for coronary angiography and intervention: The disco radial trial. JACC Cardiovasc Interv 2022;15:1191-201.
12. Giannini F, Candilo L, Mitomo S, Ruparelia N, Chieffo A, Baldetti L, *et al*. A practical approach to the management of complications during percutaneous coronary intervention. JACC Cardiovasc Interv 2018;11:1797-810.
13. Rogers JH, Lasala JM. Coronary artery dissection and perforation complicating percutaneous coronary intervention. J Invasive Cardiol 2004;16:493-9.
14. Fischman DL, Vishnevsky A. Management of iatrogenic coronary artery dissections: Failing to prepare is preparing to fail. JACC Case Rep 2021;3:385-7.
15. Lemmert ME, Van Bommel RJ, Diletti R, Wilschut JM, de Jaegere PP, Zijlstra F, *et al.* Clinical characteristics and management of coronary artery perforations: A single-center 11-year experience and practical overview. J Am Heart Assoc 2017;6:e007049.
16. Kinnaird T, Kwok CS, Kontopantelis E, Ossei-Gerning N, Ludman P, deBelder M, *et al.* Incidence, determinants, and outcomes of coronary perforation during percutaneous coronary intervention in the United Kingdom between 2006 and 2013: An analysis of 527121 cases from the British cardiovascular intervention society database. Circ Cardiovasc Interv 2016;9:e003449.
17. Umar H, Sharma H, Osheiba M, Roy A, Ludman PF, Townend JN, *et al.* Changing trends in the incidence, management and outcomes of coronary artery perforation over an 11-year period: Single-

centre experience. Open Heart 2022;9:e001916.
18. Abdalwahab A, Farag M, Brilakis ES, Galassi AR, Egred M. Management of coronary artery perforation. Cardiovasc Revasc Med 2021;26:55-60.
19. Al-Mukhaini M, Panduranga P, Sulaiman K, Riyami AA, Deeb M, Riyami MB. Coronary perforation and covered stents: An update and review. Heart Views 2011;12:63-70.
20. Mikhail P, Howden N, Monjur M, Jeyaprakash P, Said C, Bland A, *et al*. Coronary perforation incidence, outcomes and temporal trends (COPIT): A systematic review and meta-analysis. Open Heart 2022;9:e002076.
21. Das D, Acharya D, Singh J, Pramanik S. A detailed review of management of coronary perforations by gelfoam closure. J Pract Cardiovasc Sci 2021;7:230-5.
22. Jacob D, Savage MP, Fischman DL. Novel approaches to coronary perforations: Everything but the kitchen sink. JACC Case Rep 2022;4:142-4.
23. Ford TJ, Adamson C, Morrow AJ, Rocchiccioli P, Collison D, McCartney PJ, *et al*. Coronary artery perforations: Glasgow natural history study of covered stent coronary interventions (GNOCCI) study. J Am Heart Assoc 2022;11:e024492.
24. Song X, Qin Q, Chang S, Xu R, Fu M, Lu H, *et al*. Clinical outcomes of self-made polyurethane-covered stent implantation for the treatment of coronary artery perforations. J Interv Cardiol 2021;2021:6661763.
25. Alomar ME, Michael TT, Patel VG, Altomare CG, Rangan BV, Cipher D, *et al*. Stent loss and retrieval during percutaneous interventions: A systematic review and meta-analysis. J Invasive Cardiol 2013;25:637-41.
26. Kostantinis S, Karacsonyi J, Simsek B, Brilakis ES. Complications of stent loss during treatment of a heavily calcified and tortuous chronic total occlusion. Cardiovasc Revasc Med 2022;40S:293-7.
27. Ramjane K, Han L, Jin C. The diagnosis and treatment of the no-reflow phenomenon in patients with myocardial infarction undergoing percutaneous coronary intervention. Exp Clin Cardiol 2008;13:121-8.
28. Gupta S, Gupta MM. No reflow phenomenon in percutaneous coronary interventions in ST-segment elevation myocardial infarction. Indian Heart J 2016;68:539-51.
29. Rezkalla SH, Stankowski RV, Hanna J, Kloner RA. Management of No-Reflow phenomenon in the catheterization laboratory. JACC Cardiovasc Interv 2017;10:215-23.
30. Armstrong EJ, Feldman DN, Wang TY, Kaltenbach LA, Yeo KK, Wong SC, *et al*. Clinical presentation, management, and outcomes of angiographically documented early, late and very late stent thrombosis. JACC Cardiovasc Interv 2012;5:131-40.
31. Serruys PW, Daemen J. Late stent thrombosis-a nuisance in both bare metal and drug-eluting stents. Circulation 2007;115:1433-9.
32. Wang X, Chen X, Sun W, Tian T, Zhou S, Zhang Z, *et al*. Very late stent thrombosis in drug-eluting stents new observations and clinical implications. Cardiol Rev 2019;27:279-85.
33. Konishi A, Mitsutake Y, Ho M, Handa N, Koike K, Mochizuki S, *et al*. Patient and lesion characteristics in late/very late stent thrombosis with everolimus-eluting stents from real-world adverse event reporting. J Cardiol 2020;75:255-60.
34. Mehran R, Dangas G, Abizaid AS, Mintz GS, Lansky AJ, Satler LF, *et al*. Angiographic patterns of in-stent restenosis: Classification and implications for long-term outcome. Circulation 1999;100:1872-8.
35. Her AY, Shin ES. Current management of in-stent restenosis. Korean Circ J 2018;48:337-49.
36. Kawsara A, Nunez Gil IJ, Alqahtani F, Moreland J, Rihal CS, Alkhouli M. Management of coronary artery aneurysms. JACC Cardiovasc Interv 2018;11:1211-23.
37. Aoki J, Kirtane A, Leon MB, Dangas G. Coronary artery aneurysms after drug-eluting stent implantation. JACC Cardiovasc Interv 2008;1:14-21.
38. Nagaraja V, Kalra A, Puri R. When to use intravascular ultrasound or optical coherence tomography during percutaneous coronary intervention? Cardiovasc Diagn Ther 2020;10:1429-44.
39. Chhikara S, Datta R, Shrivastava A, Gupta A. Stuck OCT catheter: An unanticipated complication. J Invasive Cardiol 2022;34:E149-50.
40. Ahn JM, Park DW, Shin ES, Koo BK, Nam CW, Doh JH, *et al*. Fractional flow reserve and cardiac events in coronary artery disease: Data from a prospective IRIS-FFR registry (Interventional cardiology research incooperation society fractional flow reserve). Circulation 2017;135:2241-51.
41. Chiang MH, Yi HT, Tsao CR, Chang WC, Su CS, Liu TJ, *et al*. Rotablation in the treatment of high-risk patients with heavily calcified left-main coronary lesions. J Geriatr Cardiol 2013;10:217-25.
42. Gallagher SM, Jones DA, Smith EJ. Complications rotablation. In: Chikara LA, Di Mario C, editors. Complications of Percutaneous Coronary Intervention. London: Springer; 2016.
43. Sakakura K, Inohara T, Kohsaka S, Amano T, Uemura S, Ishii H, *et al*. Incidence and determinants of complications in rotational atherectomy. Insights from the national clinical data (J-PCI Registry). Circ Cardiovasc Interv 2016;9:e004278.
44. Gupta T, Weinreich M, Greenberg M, Colombo A, Latib A. Rotational atherectomy: A contemporary appraisal. Interv Cardiol Rev 2019;14:182-9.
45. Mahmood U, Hajj G. Excimer laser coronary angioplasty. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2022.
46. Baumbach A, Bittl JA, Fleck E, Geschwind HJ, Sanborn TA, Tcheng JE, *et al*. Acute complications of excimer laser coronary angioplasty: A detailed analysis of multicentre results. Coinvestigators of the U.S. and European percutaneous excimer laser coronary angioplasty (PELCA) registries. J Am Coll Cardiol 1994;23:1305-13.
47. Sintek M, Coverstone E, Bach R, Zajarias A, Lasala J, Kurz H, *et al*. Excimer laser coronary angioplasty in coronary lesions: Use and safety from the NCDR/CATH PCI registry. Circ Cardiovasc Interv 2021;14:e010061.
48. Ali ZA, Nef H, Escaned J, Werner N, Banning AP, Hill JM, *et al*. Safety and effectiveness of coronary intravascular lithotripsy for treatment of severely calcified coronary stenoses: The disrupt CAD II study. Circ Cardiovasc Interv 2019;12:e008434.
49. Keeiakes DJ, Virmani R, Hokoma JY, Illindala U, Mena-Hurtado C, Holden A, *et al*. Principles of intravascular lithotripsy for calcific plaque modification. JACC Cardiovasc Interv 2021;14:1275-92.
50. Aksoy A, Salazar C, Bacher MU, Tiyerili V, Weber M, Jansen GF, *et al*. Intravascular lithotripsy in calcified coronary lesions-a prospective, observational, multicentre registry. Circ Cardiovasc Interv 2019;12:e008154.

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