Complications of coronary intervention: device embolisation, no-reflow, air embolism

ABSTRACT

The introduction of drug-eluting stents, better equipment, stronger antiplatelet drugs, and higher levels of operator experience has led to markedly improved patency rates for complex percutaneous coronary interventions (PCIs). The evolving techniques of contemporary PCI have been unable to completely eliminate complications. However, rigorous preventive measures pre-empt the appearance of complications. During traversal of severely diseased coronary arteries and manipulating equipment, particularly devices with detachable components, the opportunity for loss or embolisation of material in the coronary circulation presents itself. Device embolisation is associated with periprocedural myocardial infarction and emergent referral to surgery, particularly if the device is not retrieved. The coronary no-reflow phenomenon is a feared complication of PCI. It is associated with a worse prognosis and has been shown to be an independent predictor of death, myocardial infarction and impaired left ventricular function. Air embolism can be prevented by flushing of catheters during equipment exchanges.

DEVICE EMBOLISATION

Incidence

Embolisation of devices such as coronary stents and guide wire fragments is a catastrophic complication of percutaneous coronary interventions (PCIs). Stents are the most common devices embolised, with an incidence ranging from 3% for the first-generation hand-crimped devices to much lower, 0.32%, for current stent delivery systems.[1 2] The incidence of retained components of percutaneous angioplasty equipment is reported to be 0.2% in single-centre series.[3] The successful recovery of balloons and guide wire fragments uses techniques similar to those for retrieving embolised stents.[4 5]

Mechanisms

A tortuous and calcified coronary anatomy increases the rigidity of the vessel making it prone to a stent peeling off the balloon as increased force is applied to advance the device. For this reason, stents are more commonly lost in the right coronary and left circumflex arteries and less commonly in the left anterior descending artery.[1 6] In attempts to direct stenting, unexpected difficulties in advancing the stent may be encountered. In these cases, the stent should be gently retracted back into the guide catheter, removed, and the lesion predilated. If the distal tip of the stent has engaged the lesion while advancing, the stent may be stripped off the balloon and remain embedded in the lesion when the balloon is retracted. Stent loss occurs when it cannot be deployed and, on withdrawal, is caught by the edge of the guide, dislodging it from the balloon platform. This complication is more likely when the guide is not coaxial with the coronary ostium.

Treatment

When a stent cannot be delivered to the target area because of proximal tortuosity or tight lesion, it (the unembolised stent) has to be withdrawn into the guide. Then the tip of the guide should be made coaxial. If a satisfactory coaxial relationship cannot be made between the guide and the stent, then the guide should be retracted until a favourable alignment between the guide and stent can be achieved. Sometimes, the guide might have to be retracted up to the tip of the femoral sheath in order to straighten the tip of the guide. There are numerous approaches to the retrieval of an embolised stent (box 1).

Balloon technique

This technique is practicable only if the stent remains on the angioplasty guide wire. After withdrawal of the stent balloon from which the stent has become dislodged, a second smaller 1.5 or 2 mm balloon is advanced over the wire and through the stent, and is inflated distal to the stent. Then it is retracted back into the guide. When the balloon cannot be advanced all the way through the stent, low-pressure inflation of the balloon when it is at least partially within the stent will suffice. In some cases, the stent may be contained within the distal tip of the guide, but the inflated balloon cannot be retracted into the guide. In this scenario, the guide and balloon should be removed as one unit over the wire. Two balloons are required if the stent diameter is too large for a single balloon to retract.

Wire braiding technique

This method is useful if the stent has come off the balloon but remains on the wire. A second, soft-tipped wire is navigated strategically through the side struts and not the central lumen. It is then passed into a branch distal to the stent and is separate from the first wire. Torque is then applied to both wires, and the twisting action results in them wrapping around both sides of the stent, trapping the stent in between.[7 8] With gentle and persistent pulling, the whole system (guide, stent twisted between two wires) can be withdrawn.[5]

Removal of stent with a snare

This is probably the most widely used tool for stent retrieval.[9 10] Many snares are available commercially. Once a stent slips off the delivery balloon, the indwelling wire is advanced as far as possible into the distal vasculature. The loop of the snare is passed over the angioplasty wire, encircles it, and is advanced up to the coronary ostium. It arrives at the right position as its loop is encircling the proximal end of the stent. The loop is tightened by advancing the transport catheter, and the whole stent–snare–wire complex is withdrawn as a unit.

Deployment of an embolised stent

Even if the stent is not advanced completely through the lesion, it should be expanded where it is to its fullest possible dimension using the deployment balloon. If the deployment balloon

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> **Box 1 Strategies for managing stent loss**
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> - ▸ No treatment for peripherally embolised small stents
> - ▸ Deployment of the embolised stent with balloon or second stent
> - ▸ Crushing of the stent into vessel wall with stent or balloon
> - ▸ Retrieval of the stent with a snare
> - ▸ Removal of the stent with two twisted wires
> - ▸ Inflation of a small balloon distal to the stent and removal of the whole system

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cannot be advanced through the stent, a balloon with a lower profile should be inserted. If a normal sized balloon does not pass through, a very small (1.5–2.0 mm) balloon should be used to facilitate subsequent larger balloon entry.

CORONARY NO-REFLOW
Incidence, pathophysiology and diagnosis

Coronary no-reflow is the inability to perfuse the myocardium after opening a previously occluded or stenosed epicardial coronary artery.[11] Two types of no-reflow are encountered in clinical practice: reperfusion-related no-reflow (RNR) and primary no-reflow (PNR).[12–14] In RNR there is impaired myocardial perfusion due to epicardial coronary obstruction, as in the setting of acute ST-elevation myocardial infarction (STEMI). Even if the obstruction is relieved with PCI or pharmacological intervention, distal myocardial tissue remains compromised. Left ventricular systolic dysfunction and infarct size are significantly increased in the setting of RNR. In PNR, the intervention precipitates no-reflow on a vessel with normal perfusion. The pathophysiology of no-reflow appears to be multifactorial. It has been hypothesised to result from a combination of cellular and interstitial oedema, endothelial damage, platelet–fibrin embolisation, and vasospasm overwhelming the coronary microcirculation. Despite the benefits of epicardial coronary recanalisation, the act of reperfusion contributes to no-reflow via infiltration of microvasculature with activated neutrophils and platelets. In PNR, PCI-related barotrauma is suspected of precipitating distal embolisation of atheroma and thrombus. It has been observed that obstruction of more than 50% of the microvasculature is required to decrease myocardial blood flow irreversibly.[15] The incidence of no-reflow ranges from 0.6% to 2% and is more commonly observed with the use of stents, aterectomy and PCI in saphenous vein grafts (SVGs).[16 17]

Diagnosis of no-reflow is made angiographically by delayed antegrade flow, often accompanied by ischaemic ECG changes and angina. It can be quantified by thrombolysis in myocardial infarction (TIMI) flow grade, TIMI frame count and TIMI blush score. Other ways of assessing microcirculatory myocardial perfusion are positron emission tomography, cardiac magnetic resonance imaging and myocardial contrast echocardiography. Risk factors for no-reflow include large thrombus burden with more than 5 mm of thrombus proximal to the obstruction, presence of mobile thrombus, a cut-off pattern of obstruction without taper morphology, SVG intervention, diffuse coronary ectasia intervention, rotational atherectomy (RA), increased reperfusion time, leucocytosis and hyperglycaemia.[18–20]

Prevention

The most efficacious approach to no-reflow is to prevent its occurrence in the first place. It can be prevented or minimised with adequate pharmacological and mechanical pre- and post-treatment (box 2). Whenever possible, native vessel PCI should be attempted rather than tackling a severely degenerated SVG with large thrombus burden. One must minimise the door-to-balloon time in acute MI, which is likely to reduce chances of RNR. During stenting of high-risk lesions, barotrauma and distal embolisation can be minimised by limiting the number, diameter and pressure of balloon inflations. Pretreatment with intracoronary (IC) calcium channel blockers is a useful adjunct to the treatment of SVGs.[21] The SAFER (Saphenous Vein Graft Angioplasty Free of Emboli Randomised) trial established that routine use of a distal protection device is associated with reduced incidence of no-reflow in the treatment

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Box 2 Prevention of no-reflow

- ▶ Avoidance of severely degenerated saphenous grafts when possible
- ▶ Use of distal protection device in treating saphenous graft lesions
- ▶ Use of medication and appropriate device selection while performing rotational atherectomy
  - – Reduction of burr/artery ratio to 0.6 or less
  - – Lowering of rotational speed to 140 000 rpm or less
  - – Use of nitroglycerine, verapamil in combination with flush solution
- ▶ Minimise door to balloon time in ST elevation myocardial infarction
- ▶ Minimise balloon inflation and consider direct stenting in patients with bulky atheromas or saphenous venous grafts
- ▶ Pretreatment with vasodilators, eg, verapamil or adenosine

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of degenerated SVGs.[22] In another study, equivalence of proximal occlusion with the distal protection approach was reported.[23] During RA, no-reflow can be prevented by avoiding lesions with thrombus, reducing the burr/artery ratio to 0.6 or less, and lowering rotational speed to ~140 000 rpm. IC adenosine and nitroglycerine during RA decreased the rate of no-reflow from 11.4% to 1.4%[21 24] in one study. Pharmacological preventive measures in RA include abciximab,[25] IC adenosine,[26] and a drug cocktail in the flush solution including nitrate, verapamil and heparin.[27]

Administration of abciximab before PCI for STEMI increased the proportion of patients with TIMI 3 flow both before and after PCI in the ADMIRAL(Abciximab before Direct Angioplasty and Stenting in MI Regarding Acute and Long-Term Follow-up) trial.[28] Interestingly, distal protection and rheolytic thrombectomy[29] have failed to improve outcomes in primary PCI. Adjunctive manual thrombectomy is the most recent development in preventing no-reflow and optimising perfusion in the setting of STEMI. Moreover, in the TAPAS (Thrombus Aspiration during Percutaneous Coronary Intervention in Acute Myocardial Infarction Study) trial, aspiration thrombectomy improved myocardial blush score and ST-segment resolution at 30 days. At 1 year, it had reduced the rate of cardiac death to 3.4% compared with 6.7% in association with routine PCI.[30] Benefits of adjunctive thrombectomy during STEMI were reproduced in both meta-analysis and a pooled patient analysis of 11 randomised clinical trials for both 30-day and 1-year major adverse cardiovascular and cerebrovascular events, respectively.[31 32]

Treatment

When no-reflow is encountered despite diligent preventive efforts, the mainstay of treatment is pharmacological (table 1). The interventionist must exclude a mechanical cause, such as coronary spasm, dissection or thrombus, as a cause of impaired flow. Injection of contrast through the second lumen of an over-the-wire balloon will provide information about flow in this part of the vessel. If the vessel fills with contrast past the suspected occlusion into the proximal segment and the contrast does not clear, the diagnosis of no-reflow is made. In the case of mechanical obstruction, the distal flow will be normal and the vessel will fill back to the point of obstruction. This approach also allows treatment of no-reflow with the catheter positioned
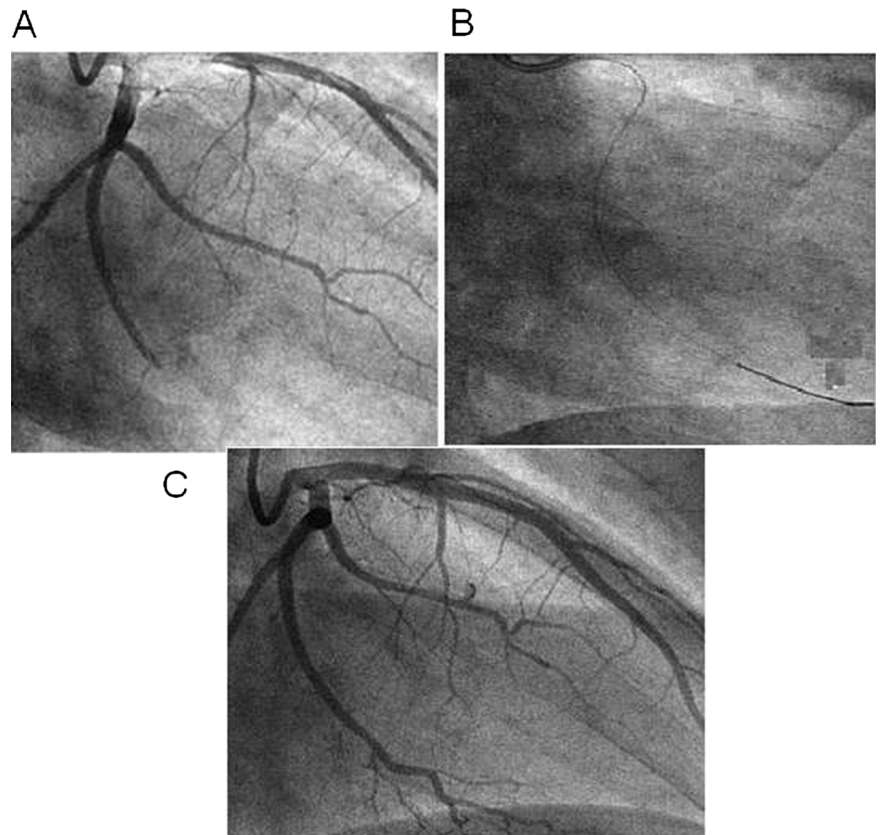
Table 1 Drug regimen for treatment of no-reflow

Drug	Intracoronary administration
Adenosine[17]	Boluses of 24 μg up to four doses
Verapamil[34]	Boluses of 100–200 μg up to four doses
Nitroprusside[38]	Boluses of 100 μg up to 1000 μg
Papaverine[40]	8–12 mg single bolus
Diltiazem[36]	2.5 mg over 1 min
Nicardipine[37]	100–200 μg boluses
Nicorandil[39]	2 μg bolus
Epinephrine[41]	50–200 μg bolus

subselectively in the coronary artery. Emergency coronary artery bypass and thrombolytic therapy do not play any role. If the site of the original stenosis is widely patent by angiography or intravascular ultrasound, additional coronary artery stenting is not helpful. Cases of cardiogenic shock may require intra-aortic balloon counterpulsation (IABP).

Adenosine, a purine nucleoside, can be administered via an intravenous or IC route, and, because of its short half-life, the duration of any adverse effect is limited. IC adenosine has demonstrated efficacy in reversing angiographic no-reflow in primary PCI and in SVG intervention.[33][34] IC verapamil improves TIMI flow grade and angina and ST-segment elevation in the majority of no-reflow cases.[17][35] Other calcium channel blockers shown to be effective in no-reflow include IC nicardipine and diltiazem.[36][37] IC nitroprusside has been shown to improve no-reflow without significant adverse effects.[38] Even though nitroglycerine is commonly administered to exclude underlying spasm, there is no evidence to support its use in reversing no-reflow. The other agents found to be useful in the treatment of no-reflow are nicorandil,[39] papaverine[40] and epinephrine.[41]

CORONARY AIR EMBOLISM
Incidence and aetiology
Although it is rare and preventable, coronary embolism is still one of the inevitable complications of PCI. Almost always iatrogenic, the incidence of this complication ranges from 0.1% to 0.3%.[42] Introduction of air into the coronary artery is potentially fatal, resulting in an 'air lock', causing abrupt occlusion of the vessel, possible cardiac arrest, and MI or both, often making immediate cardiovascular support critical. It usually results from incomplete aspiration of catheters, entry of air into the manifold system during the change of contrast, balloon rupture, and insinuation of air with balloon catheter introduction (especially, when a previously expanded balloon catheter is put through a Touhy Borst opening, ie, fully opened with no good backflow). Occasionally, introduction of air through an intracardiac defect can lead to paradoxical embolism into the coronary circulation.

Diagnosis
The consequences of air embolism depend on the amount of air introduced into the coronary vessels. Once air is injected, an air lock can develop preventing perfusion of the distal coronary bed (figure 1). Air embolism may manifest as pain, hypotension, transient ECG changes consistent with myocardial ischaemia, or arrhythmias (bradycardia, heart block, ventricular tachycardia and fibrillation). The diagnosis of air embolism is made fluoroscopically and angiographically, and the bolus generally divides into smaller bobbles as it progresses distally in the arterial tree causing slow flow in epicardial vessels.[43]

Figure 1 (A) Air embolism 'air lock' created by large air embolus causing cessation of flow in obtuse marginal artery. (B) Extraction of air by aspiration catheter. (C) Restoration of thrombolysis in myocardial infarction (TIMI) 3 flow.

Box 3 Treatment of air embolism

▶ Ventilation with 100% oxygen
▶ Intravenous fluids, atropine or vasopressors for haemodynamic support
▶ Consideration of intra-aortic balloon pump counterpulsation for haemodynamic support
▶ Wires or balloon catheter to induce 'air lock' dissipation
▶ Catheter aspiration of air embolus
▶ Treatment of no-reflow phenomenon with standard vasodilators (adenosine, verapamil, nitroprusside)

Treatment

The most efficacious means of dealing with air embolism is prevention of its occurrence by careful aspiration of catheters and meticulous flushing of coronary equipment. The vast majority of observed emboli are extremely small volumes of air that do not result in symptoms or have haemodynamic consequences; therefore they do not require therapy. When an air lock develops, therapy is directed at maintaining coronary blood flow while attempting to promote dissipation of the air bubbles. Administration of 100% oxygen should be initiated immediately (box 3). Increasing the mean arterial pressure will force air bubbles into the coronary microcirculation and overcome the air lock. Forceful injection of contrast and saline can also assist in dissipating an air lock. Guide wire disruption of distal air bubbles may be useful. Balloons can be used to pulverise large bubbles. Thrombectomy catheters or over-the-wire balloon catheters can be used to extract bubbles. IABP and ionotropic agents should be initiated when the patient is profoundly hypotensive.

CONCLUSION

With better equipment, novel stent design, stronger antiplatelet drugs, and higher levels of operator experience, the incidence of device embolisation, no-reflow and air embolism is rare. Prevention is far superior to treatment, but once these complications are recognised, treatment should be initiated rapidly and aggressively to achieve the best outcome. No matter how experienced the operator, complications can never be completely avoided, and even the best operator will always be humbled by complications and continue to learn from these experiences to become an even better operator.

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