Management of complications during percutaneous coronary intervention: a current appraisal

Tratamento de complicações durante intervenção coronariana percutânea: avaliação da situação atual

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ABSTRACT - More advanced technology, better drugs and more experienced operators have contributed to improved complex percutaneous coronary intervention outcomes. Complications with significant impact on patient survival and healthcare costs have not been completely eliminated by modern percutaneous coronary intervention procedures. Professional expertise and consistent preventive procedures are the only ways to avoid severe side effects of percutaneous coronary intervention. Dissections, abrupt arterial closure, coronary perforation, no-reflow, air embolism, stent deformation, device embolization, and rotating atherectomy burr entrapment are some of the periprocedural complications covered in this article.

Keywords: Percutaneous coronary intervention; Perforation; Non-reflow phenomenon; Device entrapment; Embolism

RESUMO - Tecnologias mais avançadas, drogas melhores e profissionais mais experientes contribuíram para desfechos otimizados das intervenções coronarianas percutâneas complexas. As complicações com impacto significativo na sobrevida do paciente e alto custo para o sistema de saúde não foram totalmente eliminadas pelos procedimentos modernos de intervenções coronarianas percutâneas. Os procedimentos preventivos e a experiência do operador são as únicas formas de se evitarem os efeitos colaterais graves das intervenções coronarianas percutâneas. As dissecções, o fechamento abrupto da artéria, a perfuração coronariana, o *non-reflow*, a embolia gasosa, a deformação do stent, a embolização do dispositivo e o aprisionamento da ogiva de aterectomia rotacional são algumas das complicações abordadas neste artigo.

Descritores: Intervenção coronária percutânea; Perfuração; Fenômeno de não refluxo; Aprisionamento do dispositivo; Embolia

INTRODUCTION

> It's a painful feeling to look at your problem and realise
> you're the only one who caused it.
> Ajax, Sophocles, 447 BC

With improvements in stents design, adjuvant technologies and development of more potent and effective anti-platelet drugs, higher patency rates may be associated with the safety of elective interventions. Periprocedural complications are still a major concern in percutaneous coronary intervention (PCIs), and treatment is often based on professional expertise and a small number of case studies, with little scientific support. Dissections, abrupt vessel closure, coronary perforation, no-reflow, air embolism, stent deformation, device embolization and rotational atherectomy (RA) burr entrapment are potential complications of PCI, which will be discussed in detail in this review. Prevention, early recognition and effective treatment will be emphasized.

DISSECTION

Coronary artery dissection is the medical term used to describe a condition that occurs when blood flow is interrupted due to a tear in the arterial wall. These complications were extremely common in the days of balloon angioplasty.[1] Although the incidence of clinically significant dissection was greatly reduced with the advent of stents,[2] this complication must be recognized as quickly as possible, since early treatment improves vascular patency and patient outcomes. Major dissections may lead to coronary flow obstruction, vessel occlusion, hemodynamic collapse and even death. According to the National Heart, Lung, and Blood Institute (NHLBI) system, dissection refers to an intraluminal filling defect or flap with hazy, ground--glass appearance. Luminal injury severity can be classified using the traditional Type A-F system (Table 1).[3] Operators specializing in chronic total occlusion (CTO) PCI are more often faced with dissections and may sometimes create dissections to use the subintimal space for dissection re-entry techniques.[4] Novel techniques and devices developed by CTO specialists to manage dissections may be used to treat iatrogenic dissections.[5,6]

The primary goal of the operator should be to establish and maintain an open artery. In cases of flow-restricting dissections, balloon dilatation should be followed by stent implantation (Figure 1). Anterograde contrast injection must be avoided to prevent propagation of the dissection. In case of uncertain guidewire position (true lumen or subintimal space), intravascular imaging test can be used to examine the dissection. In large intramural hematomas, a cutting balloon may be introduced to release the hematoma prior to stenting. In longer dissections, initial stenting of the distal margin is warranted to prevent downstream propagation.[7] Maintenance of the guidewire in the true lumen is critical. If guidewire position is lost and the true lumen cannot be regained by advancing a spring-coil wire (favored over polymer-jacketed guidewires), antegrade dissection-re-entry may be considered.[7] Retrograde wiring may facilitate anterograde stenting.[6] Emergency coronary artery bypass graft (CABG) surgery is required when these methods fail. Catheter-induced, spiral and stent-edge dis-section are the most prevalent causes of coronary dissection and may be iatrogenic.
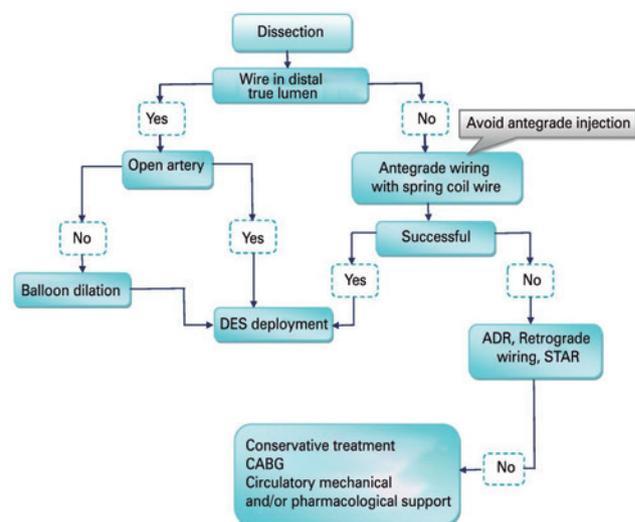
Guide catheter dissection

Catheter-induced dissection occurs in less than 1% of PCI procedures and may be caused by deep intubation of larger catheters into smaller diseased arteries. Abrupt vessel closure may result from proximal dissection. Patients with localized dissections may benefit from conservative treatment.[8,9] Guidewire introduction into major bifurcating vessels should start as soon as a left main coronary artery (LMCA) dissection is detected. If the correct lumen cannot be wired, use of an alternative catheter curve with a spring-coil guidewire is recommended. Whenever the dissection propagates to one of the major branches of the artery, emergency stenting should be performed to preserve remaining branches. Once major arteries have been stented, a second angiography in different planes should be performed prior to guide catheter withdrawal. When removing other devices from the vascular lumen, the guide catheter should be retracted to prevent this complication. Also, the guide catheter should not remain deeply engaged for extended periods of time.

Spiral dissection

Spiral dissection is rare in contemporary PCI, but may be iatrogenic or spontaneous. Contrast delivery into an unrecognized tissue plane after a catheter-induced injury may lead to iatrogenic dissection. Once the correct lumen has been identified, a guidewire should be inserted and stenting accomplished as quickly as possible. Intracoro-

Table 1. Classification of coronary dissections

| Classification	Description
Type A	Minor radiolucency within the coronary lumen without dye persistence
Type B	Parallel tracks or double lumen separated by a radiolucent area during angiography without dye persistence
Type C	Extraluminal cap with dye persistence
Type D	Spiral luminal filling defects
Type E	New persistent filling defects
Type F	Dissection leading to total occlusion

Source: Huber et al.[3]

Source: Doll et al.[7]
DES: drug eluting stent; ADR: antegrade dissection and re-entry; STAR: subintimal tracking and re-entry; CABG: coronary artery bypass graft.
Figure 1. Coronary dissection management algorithm.

nary imaging is useful to confirm guidewire position (luminal or subintimal). An extremely uncommon (0.02% of cases) but potentially disastrous consequence is retrograde aortic dissection following coronary dissection. Retrograde extension of this dissection into the sinuses of valsalva and cusp, the ascending or aortic arch or the descending aorta may affect other coronary ostia, depending on where it occurs first. This may result from stent insertion into a guide catheter, balloon catheter extraction or guide catheter manipulation (particularly the Amplatz catheter). Aggressive contrast injection, stiffer guidewires, eccentric LMCA angulation and lack of expertise have been associated with increased risk of dissection, among other factors.[9] It must be expeditiously ruled out when there is unexplained chest pain or hypotension after PCI of any ostial or proximal lesion. Dissection extension is key to overcome this problem. Stable patients with aortic root dissections less than 40mm in length may benefit from coronary ostial stenting (Figure 2). Emergency surgical intervention is required whenever the index dissection extends 40mm into the aortic root, has severe aortic regurgitation or involves supra-aortic arteries. Follow-up chest CT imaging can be used to distinguish between stable patients who do not require additional treatment and complex patients who may require surgical intervention.[10]

Stent edge dissection

Dissections and intramural hematomas are one of the most common causes of periprocedural narrowing after stent implantation. Dissection may occur in the intima or media. Dissections within the media may lead to intramural hematomas, which often lack a re-entry point. In these cases, intravascular imaging is critical to determine key factors, such as extent of disease, length and depth of the intima or media, circumference and length of the extension, side branch involvement and residual lumen. Intramural hematoma due to dissection at the proximal edge of the stent generally is less liable to propagate as dissection is retrograde, and the flap may be suppressed by antegrade flow. However, the distal edge dissection may turn into flap
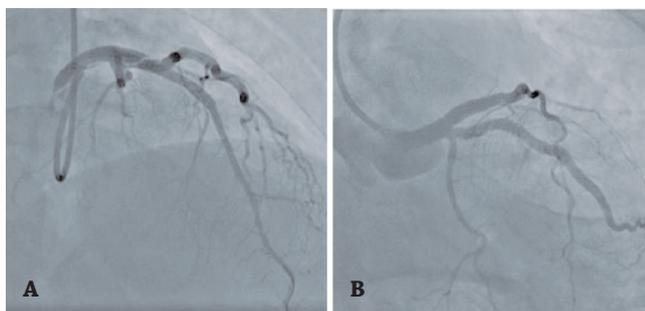
Source: Dash et al.[9]

Figure 2. Coronary angiography. Iatrogenic aorto-left main dissection. (A) Left main dissection with retrograde extension into aorta. (B) Final outcome following stent deployment and sealing of the dissection.

dissection and ineluctably leads to abrupt closure which can be managed by stenting.[11]

ABRUPT VESSEL CLOSURE

One of the most common complications of PCI is abrupt vessel closure. With the advent of novel, more effective antiplatelet drugs, the incidence of abrupt vessel closure dropped from 3% to 0.3% since the balloon angioplasty era.[12] Abrupt vessel closure may result from dissection, intracoronary thrombus formation, native vessel thrombus embolization, air embolism or spasm.[3,13,14] Acute ischemia associated with abrupt vessel closure might present as chest discomfort, electrocardiographic abnormalities, hypotension or arrhythmias. In more than half of cases, causes are unclear.[15,16] Use of over-the-wire (OTW) balloon catheters or micro catheters ensures minimum contrast media infusion, whilst checking intraluminal placement of the coronary guidewire. Intracoronary imaging may be used to confirm subintimal guidewire position. To prevent the propagation of the dissection, extensive antegrade injection after the guidewire has penetrated the subintimal layer should be avoided.

Dissection or intraluminal thrombus formation in response to intraluminal guidewire placement is the most common cause of abrupt vessel closure. Antegrade flow may be restored by inflation of several balloons. Dissection stenting with or without thrombus aspiration is common in this situation. If the first one fails, a second guidewire should be used to re-enter the true lumen. The subintimal wire must be left in place. Anticoagulation therapy is critical to prevent thrombotic occlusion of the stented artery. When heparin resistance is the suspected cause of abrupt vessel closure, heparin levels should be monitored at 30-minute intervals and resistance investigated.[17] Bivalirudin or other direct thrombin inhibitors, such as glycoprotein IIb/IIIa antagonists,[8] can be used. If certain parameters are met, abrupt vessel closure induced by thrombus may be treated with glycoprotein IIb/IIIa antagonists. Hemodynamic stabilization and ischemia reduction should be a priority. If hemodynamic deterioration is severe enough, use of intra-aortic balloon pumps (IABPs) or ventricular assist devices should be considered. Emergency CABG may be required in persistent abrupt vessel closure and hemodynamic compromise. Vasopressors and diuretics may be used to restore antegrade coronary blood flow in cases with acute vagal reactivity. Abrupt vessel closure induced by dissection must be distinguished from no-reflow as soon as possible. No-reflow is often associated with distal embolization or microvascular blockage. No-reflow vessels may not benefit from stent placement, which may even be counterproductive.

PERFORATION

Leaking of contrast medium or blood out of the coronary artery may result from dissections or an intimal tear

that extends outwards and perforate the arterial wall. Perforation is one of the most feared consequences of CTO PCI procedures (4.8%) and occurs in 0.19% to 1.46% of patients.[18,19] Perforation rates as high as 10% to 15% have been reported in some studies. Perforation is associated with a five-fold increase in in-hospital mortality risk.[18,20,21] It most commonly results from the balloon or stent oversizing, categorically when the balloon: artery ratio is more than 1.2:1 or when the semicompliant balloons are inflated at very high pressure,[15] may infrequently be caused by an appropriately sized catheter in the context of extensive dissection or lack of vessel wall integrity, can occur in the presence of arterial calcification, or be caused by inadvertent guidewire tip migration. Additional risk factors include use of atherectomy devices and cutting balloons, CTO PCI, older age, female sex and prior CABG. Ellis-graded perforations fall into types, ranging from small endovascular leaks into the adventitia (type I) to frank extravasation into the pericardial space (type III).[22] In type IV perforations, anatomical cavities, such as the coronary sinus and the right ventricle, may be involved (Table 2). Muller et al. introduced type V perforation: distal perforation associated with use of hydrophilic and/or stiff wires.[23] Type I perforations are less dangerous than type III perforations, which may lead to cardiac tamponade and hemodynamic collapse, myocardial infarction (MI) and death.

Coronary perforation and respective hemodynamic consequences can be estimated according to the volume of extravasated contrast medium during coronary angiography. At the earliest opportunity, coronary extravasation must be controlled, and hemodynamic stability restored. In cases with rapid development of severe chest discomfort during balloon inflation or stent deployment, angiography should be repeated to confirm or rule out perforation. Intravenous protamine (recommended dose, 1mg per one hundred units of unfractionated heparin) may be used to achieve a clotting time of 150 seconds in cases of established perforation.[24] However, the risk of acute stent thrombosis must be accounted for in therapeutic decision-making.[25] For patients on bivalirudin, the only alternative for partial reversal of anticoagulation is infusion of fresh frozen plasma.

In confirmed cases of coronary perforation, a balloon should be immediately placed at the perforated site as a transient measure to ensure rapid hemostasis. The balloon should be inflated at the lowest possible pressure to promote hemostasis as verified by contrast injection at conventional intervals: customarily inflations to 2 to 4 atm for approximately 5 to 10 minutes are adequate, depending on localization and extent of the perforation and on the tolerability of the patient with occlusion of the coronary vessel with a categorical focus on the development of myocardial ischemia and hemodynamic instability. Balloons should be appropriately placed and submitted to high pressure inflation for up to 20 to 30 minutes if partial sealing occurs.[26,27] Covered stents and perfusion balloons (Ryusei, Kaneka Medix, Osaka, Japan, for example) are first-line treatment alternatives for LMCA perforation.[21]

Intravenous fluids, atropine, vasopressors and mechanical circulatory support may be required even if the patient's hemodynamic may normalize with balloon occlusion. Emergency pericardiocentesis is vital in cases of cardiac tamponade. Aspirated blood should be immediately reinfused into a vein to promote hemodynamic stability. Treatment of type I perforations consists of 15 to 30 minutes of careful visual inspection and repeated injection of contrast media. If extravasation decreases, no additional interventions are needed. However, if extravasation increases, anticoagulation should be reversed and/or the balloon further inflated around or next to site of perforation. In more extensive perforations (Ellis grades II to III), extravasation is often persistent in spite of prolonged balloon inflation. In some cases, alternative methods, such as subcutaneous fat embolization, or intravenous injection of thrombin, occlusive coils, beads, or the implantation of covered stents should be attempted (Figure 3 and 4). Emergent surgical intervention may be required under certain circumstances. To reduce chest discomfort and prevent distal ischemia, balloon inflation is accomplished using a microcatheter attached to a guidewire. In such cases, occurring during balloon inflation, a microcatheter over another guidewire is placed distal to site of perforation and the patient's own arterial blood via microcatheter is injected (microcatheter distal perfusion technique).[27,28]

The advent of covered stents has revolutionized coronary perforation therapy. Introduction of these devices has led to a substantial drop of cardiac tamponade, requirement for emergency CABG,[29] and death rates associated with coronary perforation.[30] These stents are coated with polytetrafluoroethylene (PTFE) and therefore are extremely heavy, stiff and difficult to deliver. Distal anchor technique and catheter extensions, such as GuideLiner® (Vascular Solutions, Minneapolis, Minnesota) and Guidion (IMDS, The Netherlands) may be required to advance beyond previously deployed stents. Deployment of double-layered PTFE-covered stents requires vigorous post-dilatation at inflation pressure >20 atm. Intracoronary imaging should also be used to ensure proper stent expansion. This is par-

Table 2. Classification of coronary perforations

| Type	Morphology
I	Focal extraluminal crater without leak
II	Pericardial or myocardial blush without an exit hole larger than 1mm
III	Frank streaming of contrast through an exit hole larger than 1mm
IV	Contrast spilling directly into anatomic cavity chamber, such as coronary sinus and right ventricle
V	Distal perforation associated with use of hydrophilic and/or stiff wire

Source: Ellis et al.[22] e Muller et al.[23]

ticularly important, given the increase in stent thrombosis (32%) and in restenosis rates (5.7%).[31] The stiffness of double-stents may make their deployment difficult even in the presence of minimal tortuosity. Placement of covered stents using a "ping-pong" approach, in which two catheters are used to reduce the risk of leak into the pericardium, is common. The stent is deployed using a different guiding catheter via a contralateral access. At this time, the balloon employed to seal the perforation is deflated and removed to allow replacement with another guidewire and covered stent.[32] Perforations involving tortuous arteries and hemodynamic compromise should be treated with this method. Covered stents may neglect side branch arteries at the perforation site, resulting in periprocedural MI. In some cases, deep intubation is needed to accelerate hemostasis at the perforation site and enable stent deployment without using the "ping-pong" approach. As a rule, this occurs when the right coronary artery is involved.[21]
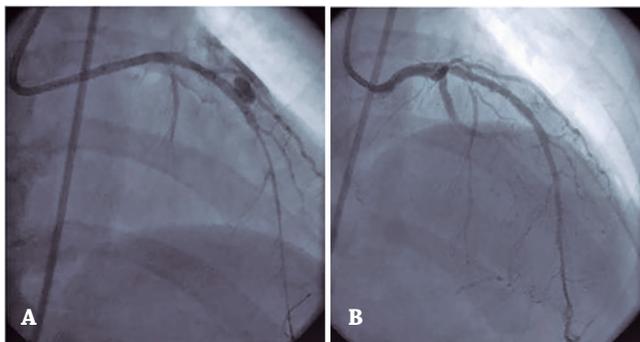
Source: Dash et al.[8]
Figure 3. Coronary angiography. Grade III perforation in a native left anterior descending coronary artery. (A) Large perforation caused by direct stenting. (B) Final outcome following sealing of the perforation using a covered stent.
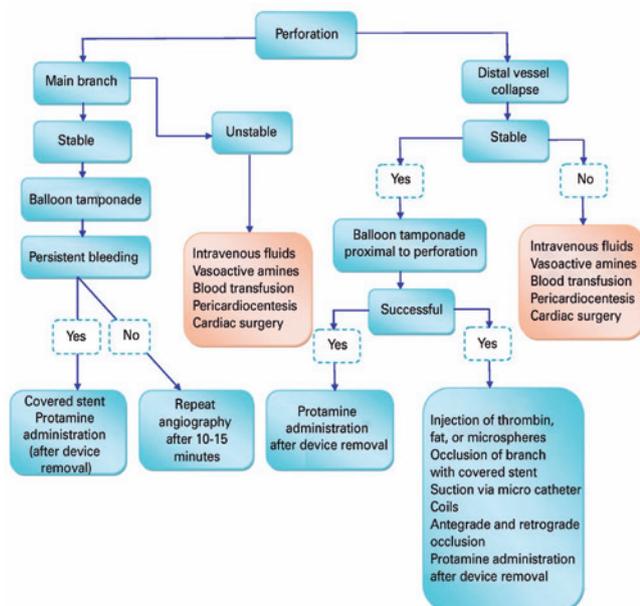
Source: Doll et al.[7]
Figure 4. Coronary perforation management algorithm.

In cardiac emergencies, a pericardium covered highly deliverable ballon-expandable stent (PCS, ITGI Medical or Akiva, Israel) can be immediately inserted.[33] The sixth-generation bare metal stent (PK Papyrus; Biotronik, Berlin, Germany) is one of the most flexible and deliverable. Therefore, there is no need to change access catheters if anything goes wrong. It is also compatible with both 5-F and 6-F catheters.[27]

Unresponsive type II and III perforations require emergency surgical intervention. In most cases, these perforations require no treatment. Small, self-limiting defects caused by hydrophilic guidewires can be addressed using extended balloon occlusion in the vicinity of the injured segment. Embolization with microcoils, Gelfoam, thrombin, polyvinyl alcohol, or subcutaneous tissue are useful alternatives if leakage persists. The insertion of an inflated OTW balloon into the distal lumen, or inflation of a balloon prior to injection, can prevent materials from spreading into the adjacent arteries.[27] Should these treatments fail, a covered stent may be inserted across the origin of the vessel towards another vessel.

Chronic total occlusion PCI is more likely to result in coronary artery perforations due to the use of stiff, hydrophilic guidewires or subintimal dissections. In the absence of collateral vessels, endovascular coils or subcutaneous fat embolization can be used. However, these are more expensive. To reduce the risk of complications, donor artery collateral flow should be addressed using microspheres or beads, endovascular coils, local thrombin injections, subcutaneous fat embolization, or covered stent implantation. Anterograde and retrograde collaterals from donor arteries should be thoroughly examined for perforations.[21] Intravascular imaging and prevention of subintimal stent oversizing are critical to decrease the risk of perforation. Early detection of additional leaks or development of pericardial effusion/tamponade requires continuous patient monitoring, rigorous hemodynamic assessment and serial echocardiography.[34]

NO-REFLOW

Coronary no-reflow occurs when an epicardial coronary artery can no longer perfuse the myocardium after opening an occlusion or stenosis[35] and is associated with increased risk of mortality, MI and left ventricular function deterioration.[36] The overall incidence ranges from 0.6% to 2.2%. Primary PCI (8% to 11.5%), saphenous vein graft (SVG) PCI (8 to 15%) and RA (≤16.5%) account for the largest percentages of cases.[37] Reperfusion-related no-reflow (RNR) and primary no-reflow (PNR) are the two forms of no-reflow seen in clinical practice. Reperfusion-related no-reflow occurs when myocardial perfusion is compromised due epicardial coronary blockage, as in acute ST-elevation MI (STEMI).[38-40] Even if the obstruction is palliated with PCI or pharmacologic intervention, distal myocardial tissue remains compromised which leads to left ventricular

systolic dysfunction and significantly increased infarct size in setting of RNR. In PNR, the intervention precipitates no-reflow on a vessel with mundane perfusion. No-reflow results from an amalgamation of cellular and interstitial edema, endothelial damage, platelet-fibrin embolization, and vasospasm overwhelming the coronary microcirculation. It occurs when activated neutrophils and platelets infiltrate the microvasculature after reperfusion, in spite of benefits provided by epicardial coronary recanalization. In PNR, distal atheromatous or thrombus embolization are thought to be precipitated by PCI-related barotrauma. According to optical investigations, at least half of the microvasculature must be blocked to permanently reduce myocardial blood flow.[41]

When antegrade flow is delayed, no-reflow is confirmed angiographically. Abnormal ECG findings and angina symptoms are often present. *Thrombolysis in Myocardial Infarction* (TIMI) flow grade,[42] TIMI frame count and myocardial blush grade (MBG) can be used to assess no-reflow. Traditional definitions of no-reflow include TIMI grade 0 or 1 and MBG between 0 and 1.44.[43] The precision of these three methods depends on several factors, such as the quantity of contrast injected, length of injection, and fluoroscopic time, as well as systemic blood pressure. Myocardial perfusion can also be quantified using positron emission tomography (PET), cardiac magnetic resonance imaging (CMRI) and myocardial contrast echocardiography (MCE). Regardless of imaging modality, an angiography is required to diagnose delayed coronary flow and no-reflow after PCI. No-reflow should be suspected whenever the ST segment elevation is not resolved after PCI. Heavy thrombus burden with more than 5 mm of thrombus proximal to the obstruction, presence of mobile thrombus, a cutoff pattern of obstruction without a taper morphology, SVG PCI, RA, increased reperfusion time, leukocytosis and hyperglycemia are risk factors for no-reflow.[44-46]

The easiest way to avoid no-reflow is to prevent it from happening in the first place. Pre- and post-pharmacological and mechanical treatments are the most effective preventive strategy. No-reflow prevention and control strategies are shown in table 3. Whenever possible, native vessel PCI should be performed instead of attempting to repair a badly degenerated SVG. To reduce the risk of RNR, the time from STEMI to wiring must be shortened. In patients with high-risk lesions, balloon inflations should be limited in number, diameter, and pressure to avoid barotrauma and distal embolization. Pretreatment with intracoronary calcium channel blockers is an useful adjunct to the treatment of SVGs.[41] The SAFER (Saphenous Vein Graft Angioplasty Free of Emboli Randomized) trial revealed treatment of degenrated SVG with distal protective devices reduces the probability of no-reflow.[47] Research has also indicated that proximal occlusion and the distal protection method are equivalent.[48] In RA, no-reflow can be avoided by simply keeping the burr-to-artery ratio below 0.6 and decreasing the rotation speed to 140,000 revolutions per minute (rpm).

No-reflow rates decreased from 11.4% to 1.4% with intracoronary use of adenosine and nitroglycerine during RA.[49] Pharmacologic preventive measures in RA include abciximab,[50] IC adenosine,[51] and a drug cocktail in the flush solution including nitrate, verapamil, and heparin.[52]

In the ADMIRAL (Abciximab afore Direct Angioplasty and Stenting in Myocardial Infarction Regarding Acute and Long-Term Follow-up) trial,[53] the number of patients with TIMI III flow both before and after PCI increased in response to pre-PCI treatment with abciximab. However, primary PCI outcomes were not improved by distal protection or rheolytic thrombectomy.[54] No-reflow and perfusion optimization in STEMI using manual thrombectomy have been recently reported. In the TAPAS (Thrombus Aspiration after Percutaneous Coronary Intervention in Acute Myocardial Infarction Study) trial, aspiration thrombectomy improved MBG and ST-segment elevation resolution after 30 days. Within 1 year, cardiac mortality decreased from 6.7% to 3.4% compared to standard PCI.[55] Benefits of adjunctive thrombectomy during STEMI were reproduced in both meta-analysis and in a pooled patient analysis of 11 RCTs for both 30 day and 1 year major adverse cardiovascular and cerebrovascular events respectively.[56]

In spite of meticulous preventive measures, pharmacological treatment of no-reflow remains the mainstay of therapy in these cases (Table 4). Coronary spasm, thrombus formation and dissection are some of the mechanical causes of reduced blood flow that must be ruled out. Contrast media may be injected into the channel via the second lumen of the OTW balloon to monitor flow rate. No-reflow occurs when the vessel fills with contrast beyond the suspected point of obstruction, but does not resolve spontaneously. If a mechanical obstacle is present, the vessel will be refilled to the site obstruction. No-reflow may be treated by placing a catheter in the coronary artery using this method. If the stenosis is widely patent on angiography or intracoronary imaging, supplemental coronary artery stenting is not required. Intra-aortic balloon counterpulsation (IABP)

Table 3. No-reflow prevention

Avoidance of severely degenerated saphenous grafts whenever possible
Use of distal protection devices to treat saphenous graft lesions
Use of medication and appropriate device selection in 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 and verapamil in flush solution
Minimization of door to balloon time in ST elevation myocardial infarction
Minimization of balloon inflation and consider direct stenting in patients with bulky atheromas or saphenous venous grafts
Pretreatment with vasodilators (e.g., verapamil or adenosine)

Source: Dash et al.[41]

may be indicated in cases with hemodynamic compromise. Angiographically confirmed no-flow associated with SVG or primary PCI may be reversed using intracoronary adenosine.[57,58] In most cases of no-reflow, TIMI flow grade, angina and ST-segment elevation can be improved with intracoronary verapamil.[59] No-reflow may also be effectively treated with intracoronary nicardipine and diltiazem. No-reflow improvement with intracoronary nitroprusside[60,61] has been reported, with no significant side effects.[62] Other successful treatment alternatives for no-reflow patients include nicorandil, papaverine and epinephrine.

AIR EMBOLISM

The incidence of coronary air embolism (often iatrogenic) ranges from 0.1% to 0.3%. Immediate cardiovascular assistance is critical due to sudden vessel obstruction in response to *air lock*, cardiac arrest and/or MI. Aspiration failure, balloon rupture and air infiltration are contributing factors. When air enters the heart through an intracardiac defect, paradoxical embolisms rarely occur.[63,64]

The consequences of an air embolism vary according to the amount of air entering the coronary flow. Myocardial perfusion compromise may ensue as soon as air is delivered, leading to symptoms, such chest discomfort, hypotension, transient ECG changes consistent with myocardial ischemia, arrhythmias and other signs (bradycardia, heart block, ventricular tachycardia and traumatic inflammation). Fluoroscopic and angiographic techniques can be used to break up the bolus of air into smaller bubbles, as it progresses distally within an epicardial conduit, delaying the migration of air.[64]

Air embolisms may be prevented by careful catheter aspiration and coronary device flushing. Since tiny air bubbles had no discernible effects, the great majority of emboli detected do not require treatment. Air lock therapy is aimed to maintain coronary blood flow whilst attempting to eliminate air bubbles. Supply of 100% oxygen should be started immediately (Table 5). To overcome air locks in the coronary microcirculation, the mean arterial pressure (MAP) must be raised. Air lock may be dissipated using forced contrast or saline injection, or by placing a guidewire. Balloons are excellent alternatives to pulverize large bubbles. Bubbles can be removed using OTW balloon or thrombectomy catheters. Vasopressors and IABP may be required to treat severe hypotension.

LONGITUDINAL STENT DEFORMATION

Contemporary cobalt-chromium or platinum-chromium stents with thinner struts that provide greater trackability, pushability and delivery than older generations stents are met with an accelerated risk of longitudinal stent deformation.[65] Ostial, bifurcation, calcified, tortuous and long, diffuse lesions are the most common angiographic features associated with stent deformation.[66] Multiple balloon post-dilations, stents, aggressive guide catheter manipulation and use of mother-child catheter systems are independent predictors of this syndrome.[67] Longitudinal stent deformation may disrupt flow and increase the risk of stent thrombosis due luminal invasion by the stent and significant strut malapposition.

During PCI procedures involving ostial lesions, placement of guiding catheters or extension systems into previously stented segments must be accomplished with extreme caution. Care must be taken once the proximal portion of a long stent has been purposefully underexpanded in a tapered vessel. Whenever post-dilation balloons or imaging catheters cannot be delivered following stent implantation,[21] longitudinal stent deformation should be suspected. Novel maneuvers (such as leaving an additional guidewire in the sinus of Valsalva) can be used to prevent guide catheters from entering and exiting the coronary arteries with respiratory or cardiac movements. In cases of suspected LSD, the stented portion must be examined radiographically, ideally using StentBoost (Philips Andover, Massachusetts, United States). A small compliant balloon should be used in the first instance, followed by a high-pressure noncompliant balloon aiming to make sure adequate expansion and apposition of the deformed stent struts. Implantation of a second stent may be deemed necessary in case of an inadequate result. The use of intracoronary imaging is strongly advocated.[21]

Table 4. Drug regimens for no-reflow treatment

Drug	Intracoronary administration
Adenosine	Boluses of 24mg, up to 4 doses
Diltiazem	2.5mg over 1 minute
Epinephrine	50-200mg bolus
Nicardipine[3]	100-200mg boluses
Nicorandil[37]	2mg bolus
Nitroprusside	Boluses of 100-1,000mg
Papaverine	8-12mg, single bolus
Verapamil	Boluses of 100-200mg, up to four doses

Source: Dash et al.[41]

Table 5. Treatment of coronary air embolism

Ventilation with 100% oxygen
Guidewire or balloon catheter to induce *air* lock dissipation
Catheter aspiration of air embolus
Intravenous fluids, atropine or vasopressors for hemodynamic support
Intra-aortic balloon pump hemodynamic support
Treatment of no-reflow phenomenon with standard vasodilators (adenosine, verapamil, nitroprusside)

Source: Dash et al.[41]

DEVICE EMBOLIZATION

When devices such as stents and guidewires, catheter fragments or misplaced intravascular coils are lost in the coronary vascular system, embolization may result, which is an unprecedented but devastating complication of PCI. With decreasing stent embolization rates (3% to 0.32% from the first generation to the era of modern stent delivery methods), stents have become the most commonly embolized devices.[68,69] PCI element retention rates of 0.2% have been reported in single-center studies.[70] Successful retrieval of embolized stents may be accomplished with the same approaches used to retrieve balloons, microcatheters and guidewire fragments.[71,72]

Vessels with complex anatomy (excessively tortuous or angled) and calcifications are more prone to stent dislodgment due to potential detachment of stents from the delivery balloon catheter. Hence, stents may be lost in the right coronary or left circumflex arteries.[41] In an attempt to direct stenting, when an unexpected difficulty in advancing a stent is encountered, the stent should be gently retracted back into the guide catheter, removed and the lesion pre-dilated. If the balloon is inflated and the distal tip of the stent becomes entrapped, the stent may be lost. When the balloon is retrieved, stents can become entrapped at edge of the guide catheter, then break free and detach from the balloon platform. This is more likely to occur when guide is not in coaxial alignment with the coronary ostium. It may bring about systemic or intracoronary embolization which may cause cerebrovascular events and a high risk of coronary thrombosis and subsequent MI.[21]

Leaving the guidewire in place does not cause vascular occlusion. Intracoronary imaging is needed to rule out guidewire unraveling as a potential cause of thrombus formation. In some instances depending on the possibility of retained fragments, the operator may consider stenting the fragment to prevent late distal migration that would cause perforation and tamponade.[21] Stents that cannot be deployed at the target site due to the proximal tortuosity or narrow lesions must be removed from the guide. Coaxial alignment of the guiding catheter tip should be ensured at all costs. If perfect coaxial connection between the guide and the stent cannot be established and the stent cannot be aligned with the guide, guide withdrawal should be considered. Embolized stents can be retrieved in a variety of ways (Table 6).[41]

Ballon technique

This procedure is most effective when the stent is attached to the guidewire. Smaller balloons (1.5 or 2mm in diameter) are then deployed distal to the stent and inflated, followed by withdrawal into the respective guide. If the balloon cannot be passed through the stent, low-pressure inflation may be used. In some cases, the stent may be contained in the distal tip of the guide, but the inflated balloon can not be retracted into the guide. In this situation, the guide and balloon ought to be withdrawn as one unit over the wire. Two balloons are required if the stent diameter is just huge for a single balloon to retract.[41]

Wire braiding technique

This method may be used when the stent has detached from the balloon, but remains attached to the wire. A second, soft-tipped wire is navigated strategically alongside the embolized stent through the side-struts and not the central lumen. When the stent is removed, the second twine is directed to a different department, which is not connected to the first one. With a twisting movement, both wires can be wrapped around the sides of the stent to encase it. The whole system is then withdrawn with a constant and gentle pull (guide and twisted stent and wires).[73]

Snare technique

The snare is still the most commonly used instrument for stent retrieval.[73] Once the stent has been removed from the delivery balloon, a distal wire is advanced as far as possible into the vascular system. The loop of the snare is passed over the wire, encircles it, and is advanced up to the coronary ostium. Once the loop has been tightened using a Convey catheter, the whole system (stent, snare and wire) may be simultaneously withdrawn.

Embolized stent implantation

Stents that cannot be advanced all the way through the lesion must be expanded to maximum size. If balloon deployment cannot be accomplished, a low-profile balloon must be used. Small (1.5 to 2.0mm) balloons should be used whenever normal-sized balloon cannot be passed.[41]

Rotational atherectomy burr entrapment

Burr entrapment occurs when a RA burr cannot be advanced through or removed from a significant coronary artery lesion, especially if vessel tortuosity or coronary spasm is present. Techniques such as gentle pecking and 15-second rotablation runs can help prevent burr entrapment. Entrapped burrs can be removed by simply pulling the spinning system backwards, applying manual traction, or using rotation without a Dynaglide (Boston Scientific, Marlborough, Massachusetts, United States). Aggressive

Table 6. Stent loss management strategies

No treatment for peripherally embolized, small stents
Deployment of embolized stent with balloon or second stent
Crushing of the stent against vessel wall using stent or balloon
Stent retrieval using a snare
Stent removal using two twisted wires
Inflation of a small balloon distal to the stent and removal of the whole system

Source: Dash et al.[41]

retrieval techniques should be avoided to prevent vascular perforation or burr shaft breakage. An additional vascular access is needed when manual traction fails. If the second guiding catheter is used, the guidewire can be advanced past an impermeable burr. Subsequent balloon dilatation of the lesion may facilitate entrapped burr removal.[74-76] Disassembly of Rotablator (Boston Scientific) parts to reveal the burr shaft and advancement of a snare just proximal to the burr are other alternatives to release entrapped burrs.[77] Child-in-mother catheters may also be used.[78] Cardiac surgery is required when treatment attempts are unsuccessful (Table 7).[79]

Table 7. Troubleshooting for rotational atherectomy burr entrapment

Manual pull
Setting rotational speed to up to 200,000rpm and trying to negotiate the burr into the distal true lumen
Cutting off the rotalink shaft distal to the advancer and removal of outer sheath
Navigation of a second guidewire (dual catheter technique) beyond the entrapped burr and inflation of a balloon proximally
Advancement of a snare over the exposed drive shaft as close to the burr as possible and simultaneous retraction of the snare and guide
Negotiation of a child-in-mother catheter
Subintimal tracking and re-entry with balloon dilatation adjacent to the entrapped burr
Emergent cardiovascular surgery

Source: Dash et al.[79]

CONCLUSION

Advances in device design, adjunctive technology, pharmacotherapy and hemodynamic support systems, combined with judicious use of percutaneous coronary intervention, have led to a dramatic drop in percutaneous coronary intervention-related complication rates. Periprocedural complications may occur even in the hands of the most skilled operators. In worst case scenarios, these complications may lead to severe hemodynamic compromise or even death. Given the randomized clinical trials have failed to provide sufficient evidence for general recommendations, these potential consequences can only be avoided with operator expertise and preventive strategies. To ensure the best possible outcome, therapy should be initiated as soon as these complications are detected. Comprehensive understanding of effective treatments, team-based communication skills and multidisciplinary teamwork are also required. Patient outcomes will continue to improve with more training in the developing field of percutaneous coronary intervention.

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CONFLICTS OF INTEREST

The authors declare there are no conflicts of interest.

CONTRIBUTION OF AUTHORS

Conception and design of the study: DD; data collection: DD; data interpretation: DD, RM, NA, JT, SRM and BM; text writing: DD, RM and SRM; approval of the final version to be published: DD, RM, NA, JT, SRM and BM.

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