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Review Article

Internal Medicine Section

Coronary Perforation- A Nightmare in Cath Lab

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ABSTRACT

Percutaneous Coronary Intervention (PCI) is now the standard of care in patients with coronary artery disease. With advances in modern technology, the success of PCI has relatively increased, and so is its complication, specifically in complex coronary intervention. Coronary perforation is one of the most dreadful and life-threatening complications of PCI. The most vital step in the management of coronary perforation is its identification and quick action. Multiple methods for management are now recommended in the literature, but the mainstay of treatment is still prevention. This review discusses the incidence, risk factors, prevention, identification, and management of Coronary Artery Perforation (CAP).

Keywords: Ellis classification, Guidewire, Percutaneous coronary intervention

INTRODUCTION

Percutaneous Transluminal Coronary Angioplasty (PTCA), also known as PCI, is a minimally invasive process to open coronary artery stenosis or open blocked blood flow to the myocardium. Deposition of lipid-rich plaque within the arteries causes coronary stenosis leading to decreased coronary blood flow to the myocardium. CAP is a potentially fatal complication of PTCA. The vital step in the management of CAP is prompt identification and quick action. Although management varies with the type of perforation, a universal initial treatment approach can be followed. Risk of occurrence of CAP can be significantly reduced by carefully selecting the guidewire and baloon that is of an appropriate size. Intravenous anticoagulants need to be discontinued immediately. Activated clotting time should be measured and corrected with protamine. Management may vary as per the patient's haemodynamic status, type of perforation, and availability of resources in cardiac catheterisation laboratories.

INCIDENCE AND MORTALITY

CAP essentially involves an outward incision through the wall of the coronary artery. This becomes evident on fluoroscopy as an extravascular accumulation of the contrast dye used. CAP can range from a slight staining of the myocardium to more serious conditions, such as the rupture of a vessel. CAP complicates 0.1-0.6% of PTCA [1-4]. Its incidence is more (0.5-3%) in procedures requiring bulky athero-ablative devices like rotational atherectomy, transluminal extraction coronary atherectomy, directional atherectomy, or excimer laser angioplasty [5-8].

With the increasing boldness of the modern interventionalist, with increasing frequencies of complex coronaries and Chronic Total Occlusions (CTOs) being performed in the catheterization laboratory (cath lab), recent trends indicate a rise in CAP's incidence.

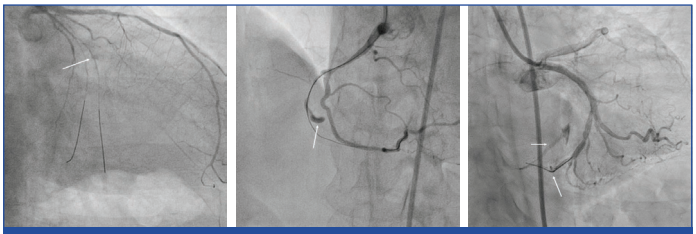
The overall in-hospital mortality rate in CAP is 16.7% [9]. Thus, the rapid recognition of CAP is essential to employ life-saving management techniques promptly. A 19-28% of CAP causes cardiac tamponade and has to be managed aggressively with pericardiocentesis, while the majority of CAP can be managed conservatively [10,11]. The incidence of pericardial tamponade and mortality adhered to the classification by Ellis [1], For example, type I-0.4% and 0.3%; type II-3.3% and 0.4%; type III-45.7% and 21.2%, respectively. It has also been observed that the occurrence of serious cardiac events are linked to type II CAP, which can occur over a prolonged period of time [10]. It has also been observed that 14.8% of deaths among hospitalised patients are associated with type III CAP as per Ellis classification [10]. Close observation with serial echocardiography and intensive cardiac monitoring is strongly advised postoperatively as small leaks caused by a guidewire or any late tear extension can lead to cardiac tamponade and sudden deterioration of the patient even up to 48 hours later [12].

CLASSIFICATION OF CORONARY ARTERY PERFORATION (CAP)

Ellis classification [1] is the most popular and widely used classification for CAP as shown in [Table/Fig-1-4].

Type	Morphology
Type I	Limited to the vessel wall and forms a crater on the outer luminal surface without any leakage
Type II	Blush observed in the pericardium or myocardium without the leakage of contrast dye (perforation <1 mm)
Type III	Jet extravasation (perforation >1 mm) within the space of the pericardium or into some other spac

[Table/Fig-1]: Ellis classification for Coronary Artery Perforation (CAP).



[Table/Fig-2]: Coronary angiogram showing wire induced type I coronary perforation (arrow) in the obtuse marginal branch of the left circumflex artery in a patient with chronic thrombotic occlusion. The patient was haemodynamically stable and managed conservatively.
[Table/Fig-3]: Coronary angiogram showing balloon induced type II coronary perforation (arrow) in the right coronary artery in a patient with chronic thrombotic occlusion. The patient was haemodynamically stable and managed conservatively.
[Table/Fig-4]: Coronary angiogram showing wire induced type III coronary perforation (arrow) in the distal left circumflex coronary artery with extravasation of contrast till coronary sinus. (Images from left to right)

Type II and III CAP can result in life-threatening complications. Out of 12900 procedures conducted by Ellis SG et al., [1], it was observed that Type I CAP caused cardiac tamponade in 8% and no deaths or Myocardial Infarction (MI); Type II CAP caused cardiac tamponade in 13% and MI in 14%; Type III CAP caused cardiac tamponade in 63% and mortality in 19%.

RISK FACTORS FOR CORONARY PERFORATION

(A) Non Modifiable Risk Factors:

Elderly patients and patients with previous CABG or past procedures involving the coronary arteries have increased propensity to develop CAP [13]. Some other minor factors that increase the risk of developing CAP include a history of congestive heart failure, hypertension, diabetes mellitus, females sex, and chronic kidney disease [14].

(B) Modifiable Risk Factors:

(i) Angiographic characteristics:

Complex angiographic characters like type B or type C lesion [8], (CTO) arteries [8,15], calcified lesion [8,15], tortuous, and angulated lesions increase the risk of CAP. In small coronary size, especially in right coronary or circumflex arteries, or the presence of multivessel coronary disease, PCI is associated with higher CAP.

(ii) Use of atheroablative devices:

Ellis SG et al.. found a higher incidence of CAP with devices that removed rather than displaced tissue [1]. CAP rates were 1.3% following the removal of atheroma from the lumen of the coronary arteries and 2% following coronary angioplasty using a laser. Lloyd-Jones D et al., reported an incidence of CAP of 3% [16], while Ajluni SC et al., reported CAP in 0.39% of patients using excimer laser in coronary angioplasty [5]. Class III CAP is more likely to develop in the procedures where instruments that remove atheromatous plaque are used. The increased occurrence of CAP has been linked to procedural training using these instruments. However, development of CAP can be prevented by using steeply angulated segments with a burr size of <0.8 [10].

(iii) Hydrophilic wires:

Hydrophilic wires have equipped cardiac surgeons with a crucial tool to effectively treat CTO and other complications associated with coronary artery damage. In a study by Javaid A and Kiernan TJ et al., it was reported that using hydrophilic guidewires were responsible for 65%-87% of CAP cases [9, 15]; however other studies have reported conflicting data [17] so, it is still a matter of debate if CAP is actually caused by the use of hydrophilic guidewires or it is just perceived that the higher incidence of CAP arises from their increased use.

(iv) Balloon angioplasty and stents:

Although wires cause most CAPs, CAP is less frequently caused by balloons or stents. CAP can arise from inserting stents into severely damaged coronary arteries, inflating the balloon under high pressure, using large-sized balloons, and using balloons that do not comply with the specified procedural criteria. In fact, balloons and stents are responsible for 0.05% to 0.15% of CAP incidence during procedures involving PCI [5-19]. A balloon to artery ratio of >1:1 is linked to a two to three-fold rise in the incidence of CAP [5]. During inflation, rupture of the balloon predisposes to perforation of the artery by production of pin-hole orifices, due to the jet under high pressur.

(v) Use of adjunctive anti-thrombotic therapy:

There have been conflicting reports between the possible association of the use of anti-thrombotic therapies like glycoprotein (GP) IIb/IIIa inhibitors and the incidence, severity, and outcomes of CAP.

In Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction (HORIZONS-AMI), therapy of CAP patients using antithrombotics, such as bivalirudin, didn't worsen the prognosis, compared to heparin plus GPIIb/IIIa inhibitors [11,12].

It has been reported by Ramana RK et al., that using GPIIb/IIIa inhibitors didn't disturb the hemodynamic condition of CAP patients [19]. In a series of 39 CAP cases, Witzke et al., reported that the use of GPIIb/IIIa didn't influence the outcome [20]. It has also been reported in a group of 6214 patients (of whom 39 had CAP), that adjunctive therapy with abciximab neither elevated the risk of CAP, nor had any adverse effects on the clinical prognosis of patients already suffering from CAP [4].

It has been reported by Gunning MG et al., that pericardial drainage was needed in 9 out of 10 CAP patients who were on GP IIb/IIIa inhibitors. Moreover, in 4 of these CAP patients, signs of tamponade were observed over two hours following PCI [21]. In these 4 cases, it was presumed that the underlying cause of the tamponade was due to puncturing of the coronary arteries by a guidewire, resulting in bleeding, which was aggravated by GPIIb/IIIa inhibitors. It was reported by Fasseas P et al., that in 33.3% of CAP patients on GPIIb/IIIa inhibitors, there was a need for placement of covered stent or emergency heart surgery, compared to 3.2% of patients who didn't [12]. However, the type of perforation and clinical prognosis, including tamponade, MI, and death, between the two groups were the same. A study by Stankovic G et al., found that there was a higher incidence of CAP with the concurrent use of GPIIb/IIIa inhibitors [13]. In another study by Kini AS et al., it was observed that tamponade arising from guidewire-related CAP, occurred less frequently when bivalirudin was used, compared to heparin due to its reversibility and shorter half-life [22].

Sequel of coronary artery perforation (CAP)

CAP can lead to distal ischaemia, blood loss, cardiac tamponade, cardiogenic shock, and death. Even the management strategy can cause myocardial ischaemia by acute vessel closure or may require immediate surgery, adding morbidity and mortality

DIAGNOSIS

Besides the typical symptoms associated with inflation of the balloon, there may be some atypical symptoms, including nausea, vertigo, and severe chest pain. Following deflation of the balloon, there may be persistence of the ST-T changes. Vasovagal attack might also occur perforation, along with slow heart-rate (bradycardia) and low blood pressure (hypotension). In these instances, a high-degree of suspicion is a prerequisite for the timely detection of even a minute perforation caused by a guidewire, which may otherwise result in pericardial tamponade being delayed. There may be abnormal migration of wire tip. Once the perforation occurs, the key step is to remain calm and call for additional help.

PREVENTION AND MANAGEMENT OF CAP

Prevention of Perforation

The best way to manage a CAP is to prevent it. Morphologic assessment of complex lesions should be done by Intravascular Ultrasound (IVUS) (to analyse location, depth, and eccentricity of calcification). Unfractionated Heparin (UFH) is preferred in long procedures as it is easily reversible, and adjuvant anti thrombotic like GP IIb/IIIa inhibitors should be used with keeping a watch on optimal Activated Clotting Time (ACT). Final angiograms in multiple views should be seen with dual injections and delayed views in suspected cases.

Meticulous attention to the following steps during angioplasty is a must:

- Guidewire advancement and positioning: Resistance to guidewire advancement, buckling of the wire, or restricted tip movements of wire should be exercised with caution as the wire may be subintimal. In such cases, withdraw and reposition the wire. If in doubt, a gentle dilute contrast injection can be delivered through a microcatheter or the central lumen of an Over-The-Wire (OTW) balloon after removing the wire. Persistent contrast staining

indicates a position within a false lumen, indicating the need to reposition the wire.

- Balloon choice: Noncompliant balloons should be used for postdilatations and for high-pressure balloon dilatations in nonyielding lesions.

Device sizing: Only in case of PTCA, a balloon-to-artery ratio of 1.0 and device-to-artery ratio of 0.5-0.6 in case of lasers and rotablaters, should be used. Always appropriately pretreat a calcified lesion with rotational atherectomy rather than aggressively trying to postdilate with an oversized noncompliant balloon to expand an under-expanded stent optimally.

Stent-related perforations can be avoided by appropriate stent position. When the distal part of the dissection can be ascertained by angiography, only then stents should be used.

Management

The strategy for management of perforation has to be individualised and depends on the site of perforation, the severity of insult, persistent leak, and haemodynamic stability of the patient.

Supportive therapy includes O_2, Intravenous (IV) fluids, analgesia/sedation, atropine for bradycardia, inotropic support and/or Intra-aortic balloon pump for hypotension

The most vital step in management is prompt identification and quick action. Although management varies with the type of perforation, a universal initial treatment approach can be followed. Valuable time may be gained by prolonging inflation of the balloon proximally (1:1 balloon: vessel size). However, it should be kept in mind that distal ischaemia can occur if balloon dilation is prolonged. This is especially so when downstream collaterals are absent. In contrast, the perforation can be sealed by perfusion balloons, at the same time as distal vessel perfusion. Intravenous anticoagulant and antiplatelet therapy should be stopped and reversed immediately. Activated clotting time needs to be estimated, following which, protamine should be administered to reverse the effects. Moreover, there may be a need for platelet transfusions if GPIIb/IIIa antagonists were used.

Type I CAP

Intermittent injection of contrast dye, accompanied by continuous monitoring is a good approach for minor breaches of the coronary vessels. However, if continuous oozing occurs or if 'contrast blush' is prolonged, heparin reversal with protamine may be warranted. Importantly, before shifting the patient to a Cath Lab, a 2D echo should be done, which should be repeated intermittently for 24 hours, following the procedure.

Type II CAP

(a) The action of heparin and GPIIb/IIIa inhibitors may be reversed with protamine and platelet infusions, respectively. Bleeding is usually reduced by gradually reducing the blood pressure in a controlled manner. In case of patients who don't have hypertension, there is a need to maintain the Mean Arterial Pressure (MAP) at 50-65 mmHg. However, in patients who have hypertension, the MAP should be kept 30% below the baseline value. Over 40% reduction in bleeding has been reported in case of most studies [18,23].

(b) Balloon occlusion to halt blood flow into the side branch or proximally to the perforation may be needed in case of a side branch perforation. This is possible with hardly any risk of ischaemia. As per standard procedure, low pressure (2-4 atm) is applied for 15-20 minutes. If the site of perforation is a distal main vessel, a balloon occlusion method should be used. If the perforation is more proximal in the main vessel, such as in a balloon-induced perforation, it requires immediate balloon-tamponade at the site. There may be significant ischaemia in the distal arterial bed in case of proximal balloon tamponade. Therefore, in order permit limited flow, there is a need to inflate intermittently with controlled inflation pressures. This type of procedure has been effective in over 50% of patients [10]. However, sometimes, it may not be possible to seal the perforation with balloon occlusion and heparin reversal. In such a scenario, artery size and origin may decide the approach.

(c) For a side branch wire perforation in a smaller vessel (<2.5 mm diameter), a persistent leak can be tackled by arterial occlusion with embolic material, including microcoil [24], gel-foam [25] or Polyvinyl Alcohol Particle (PVA) embolisation [26]. Trisacryl (acrylic co-polymer), microspheres linked to gelatin, collagen, thrombin, fibrin glue, cyanoacrylate glue, or subcutaneous fat should be considered in order to close the perforation in the distal coronary artery at the cost of a minute infarction. Slightly oversized microcoil (1.5 times more than the caliber of the target vessel) can be deployed with the help of microcatheters in order to seal the site of perforation mediated by their thrombogenic properties [Table/Fig-5]. Other less common percutaneous treatment modalities that can be tried are autologous clot injection [27] and subcutaneous tissue injection [28]. Comparatively small-sized distal CAP (<1 mm diameter) can be treated more effectively by gel foam embolisation, than by coil embolisation or PVA foam [25]. However, there is a need to exercise extreme caution as reflux of embolic gel into the proximal vessel must be averted. It has been observed that PVA foam embolisation results in permanent sealing, whereas gel foam embolisation produces temporary occlusion that reverts back within 3-4 weeks [29,30].

(d) Persistent leaks in the main vessel (>2.5 mm diameter) are preferably managed using polytetrafluoroethylene (PTFE) coated covered stents. PTFE stents have both advantages and disadvantages. The major advantages are that these stents are inert, easy to deliver, and biocompatible. However, the major disadvantage is that these are difficult to insert into curved segments, often leading to thrombosis and occlusion of the side branches. Recent advancements in science has led to the development of electrospun polyurethane covered stents with better flexibility and deliverability, thereby greatly reducing chances of thrombosis [31-33].

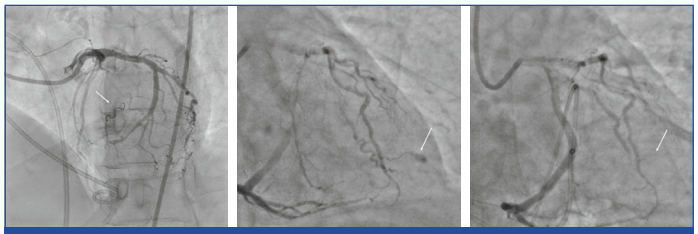
Type III CAP

Type III CAP is life-threatening events and often horrifying to the primary operator. Extravasation of coronary blood causes the vicious cycle of ischaemia and tamponade physiology. Balloon tamponade at the site of perforation and pericardiocentesis with autoperfusion should be done. Simultaneous fluid resuscitation and inotropic backup are recommended. Precious time can be saved by having a balloon in a catheter, ready for insertion. Prolonged (5-10 min) balloon inflation should be alternated with brief periods of deflation to prevent MI. This type of alternate cycling is capable of slowly sealing the perforation. However, it should be noted that covered stents are usually required for most type III CAPs, which are effective in more than 90% of cases. A 'double guide catheter technique' has been developed to save time during balloon deflation. In this procedure, the coronary artery is hooked sub-electively using a 7F guide catheter through second femoral access. The first guidewire is removed, keeping the balloon catheter in place in an inflated state. The balloon is then slowly deflated while the second guiding catheter positioned. The covered stent is placed OTW at the coronary perforation. Once effectively managed, before taking a final check angiogram, it may take a few minutes for stabilisation and to regain haemodynamic balance. In case of all type III CAPs, the CTVS team must be informed, as surgical intervention might be required for closure, although this is usually very rare, as it is needed only when all interventional options have failed.

Use of Polyvinyl Alcohol (PVA) in Coronary Perforation

PVA particles are used for vascular embolisation by an interventional radiologist for embolisation of arteriovenous malformations, lower

gastrointestinal bleeding, and even hypervascular tumours [29,30]. PVA particles have a smooth surface and are inert and biocompatible. They come is various sizes, ranging between 45 microns to 1180 microns and are used for occlusion of arteries having small bore. They are prepared in contrast medium and produce dilute suspensions that are slowly infused via the balloon catheter or microcatheter to occlude small vessels. It is important to utilise OTW balloon catheter for PVA particles having a small diameters, usually below 300 microns, which are capable of passing through a lumen of 0.014 inch. Microcatheter can be considered as a carrier when larger sized PVA particles (>300 microns) are used [Table/Fig-6,7].



[Table/Fig-5]: Coronary angiogram showing wire induced type III coronary perforation in the distal left circumflex coronary artery with extravasation of contrast till coronary sinus managed by microcoil (arrow).
[Table/Fig-6]: Coronary angiogram showing wire induced type III coronary perforation (arrow) in the ramus intermedius coronary artery with extravasation of contrast.
[Table/Fig-7]: Coronary angiogram showing wire induced type III coronary perforation in the ramus intermedius coronary artery managed with Polyvinyl Alcohol Particles (PVAs) (arrow). (Images from left to right)

CONCLUSION(S)

Pre-procedure planning with respect to patient characteristics/ lesion is required for prevention.

It is imperative that there is proper awareness of guidewire selection criteria, the various associated risks, and the importance of avoiding balloon overexpansion. The likelihood of distal CAP can be lessened by fluoroscopic monitoring of the distal wire-tip position during balloon catheter exchanges as well as during contrast injection. After wire removal, final angiographic views should profile the distal vascular to detect subtle, delayed contrast extravasation. It is of vital importance that patients are monitored uninterruptedly, both in the cath lab, as well as the Intensive Coronary Care Unit (ICCU), especially in case of high-risk groups.

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