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EDITORIAL

Coronary Artery Perforation

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Coronary Artery perforation (CAP) remains one of the most dramatic and feared complications of percutaneous coronary intervention (PCI). The incidence of CAP is 0.43% with PCI but rises to 2.9% in chronic total occlusion interventions. Although uncommon, its consequences can be immediate and fatal if not recognized and managed promptly. In the era of increasingly complex PCI—chronic total occlusion interventions, heavy calcification, aggressive lesion modification, atherectomy, and advanced bifurcation techniques—CAP should no longer be viewed as a rare mishap, but rather as an anticipated procedural risk that demands constant vigilance and preparedness. A clear understanding of its mechanisms, early diagnosis, and structured management allows the operator to respond with confidence rather than panic. CAP is defined as an anatomical breach in the integrity of the tunica adventitia of an epicardial artery leading to extravasation of blood, either into the myocardium, pericardium or a cardiac chamber. Its clinical spectrum ranges from limited angiographic staining without hemodynamic consequence to frank contrast extravasation leading to hemopericardium, cardiac tamponade, shock, and death. The most common mechanism is guidewire-related injury, particularly with stiff, hydrophilic, polymer-jacketed, or CTO wires that inadvertently migrate distally or exit the true lumen. Balloon-related perforation results from oversizing (balloon: artery ratio>1.2), high-pressure or non-compliant balloon inflation, especially in calcified, tortuous, or small vessels. Additional mechanisms include aggressive stent deployment or post-dilatation, atherectomy-related trauma from wire bias or inappropriate burr-to-artery ratio, and injury from microcatheters or guide-extension devices. CAP risk is directly related to procedural complexity and may occur at any stage of PCI, including guidewire manipulation, lesion preparation, stent deployment, or post-dilatation. Notably, distal wire perforations may present late, despite an apparently satisfactory final angiographic result. The Ellis classification remains the most practical framework for describing CAP and guiding urgency of management. Type I CAP represents an extraluminal crater without contrast extravasation, Type II is characterized by myocardial or pericardial blush without contrast jet extravasation, Type III denotes frank contrast jet extravasation through a ≥1 mm perforation generally accompanied by tamponade, and Type III cavity spilling refers to perforation into a cardiac chamber or other anatomical cavity. Several factors increase the risk of perforation, including advanced age, female sex, chronic kidney disease, saphenous vein graft angioplasty, as well as lesion characteristics such as severe calcification, vessel tortuosity, CTO, and small (<2.6 mm) or distally tapering vessel. Procedural contributors include use of stiff or hydrophilic wires, balloon or stent oversizing, high-pressure inflations, atherectomy, intravascular ultrasound use and complex bifurcation strategies. Early diagnosis is important. Angiographic clues include contrast staining outside the vessel, myocardial blush, or frank contrast extravasation. Clinical warning signs such as chest pain, hypotension, and tachycardia should immediately raise concern. Echocardiography is mandatory once perforation is suspected, as pericardial effusion and tamponade may evolve rapidly or be delayed despite initial angiographic stability. Management hinges on a calm, rapid, and structured response. Immediate steps include cessation of further contrast injection, maintenance of wire position, prompt communication with the cath-lab team, and preparation for hemodynamic support. Definitive treatment depends on the size of the perforation, the degree of contrast extravasation, and the patient's hemodynamic status. Initial management should focus on prompt sealing of the perforation to prevent cardiac tamponade. This can be achieved using prolonged balloon inflation (PBI) at low pressure, 2-6 atm for 10 min (artery: balloon ratio 0.9:1.1) at the site of perforation, deployment of a covered stent, or distal embolization with coils, fat, or microparticles. When percutaneous strategies are unsuccessful or unavailable, emergency surgical repair becomes mandatory. PBI is successful in 60-70% of perforations. If sealing is not successful, start giving protamine in incremental doses of 25-50 mg over 10-30 min until ACT is <150 s; this should also be done in case of jet extravasation and cavity spilling. Heparin reversal with protamine should be considered in significant perforations, balancing bleeding control against the risk of acute stent thrombosis. If the case is to be discontinued, reversal of heparin is indicated, however it is deferred while equipment such as balloons & wires remains in the coronary artery. Intravenous IIb/IIIa antagonist & direct thrombin inhibitor should be avoided in case of perforation. PBI at or proximal to the site of perforation remains the first-line therapy for most proximal and mid-vessel perforations, with 5–10-minute inflations repeated as necessary. It is successful in 60-70% of perforations. If sealing is not successful, start giving protamine in incremental doses of 25-50 mg over 10-30 min until ACT is <150 s; this should also be done in case of jet extravasation and cavity spilling. Pericardiocentesis is mandatory in the presence of cardiac tamponade or hemodynamic compromise and should never be delayed; a pericardial drain should be left in situ for continuous monitoring and potential autotransfusion. Large proximal or mid-vessel perforations (Ellis Type III) are best treated with covered stent deployment, recognizing limitations such as reduced flexibility and risk of side-branch occlusion. Distal vessel perforations, most commonly wire-related, are effectively managed by coil embolization, gel foam, autologous fat embolization, platelet infusate, polyvinyl alcohol or thrombin injection, accepting sacrifice of a small distal vessel to save the patient. In resource-limited settings where dedicated covered stents are unavailable, an improvised makeshift covered stent using balloon material over a conventional stent or autologous vein-covered stents may be lifesaving. Larger guide catheters (7F–8F) and dual arterial access are often required, allowing one system to seal the perforation with a balloon while another delivers definitive therapy. Prevention remains the most effective strategy. Careful lesion assessment, appropriate device sizing, cautious wire escalation with early de-escalation, gradual balloon pressure increments, vigilant monitoring of distal wire position, and judicious use of atherectomy significantly reduce risk. Advance planning of access strategy and availability of emergency equipment are essential when undertaking complex PCI. Although infrequent, CAP represents an inherent risk of modern interventional cardiology and is associated with substantial morbidity and mortality. Early recognition and timely, structured management are crucial to optimizing patient outcomes.

Chayan Kumar Singha
Assistant Professor, Department of Cardiology, Bangladesh Medical University
Email-singhachayan@yahoo.com

MA Muqueet
Professor & Chairman, Department of Cardiology, Bangladesh Medical University

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