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Review Article

Complications encountered in coronary chronic total occlusion intervention: Prevention and bailout

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ABSTRACT

Despite the continuing developments of improved medical devices and increasing operator expertize, coronary chronic total occlusion (CTO) remains as one of the most challenging lesion subsets in interventional cardiology. Percutaneous coronary intervention (PCI) of CTO is a complex procedure carrying the risk of complications that are responsible for significant morbidity and mortality. The complications can be classified as coronary (such as coronary occlusion, perforation, device embolization, or entrapment); cardiac non-coronary (such as periprocedural myocardial infarction); extra cardiac (such as vascular access complications, systemic embolization, contrast-induced nephropathy, and radiation-induced injury). Further, certain complications (such as donor vessel dissection or thrombosis) are unique to CTO–PCI. There are also complications related to specialized techniques, such as dissection/reentry and retrograde crossing techniques. A thorough understanding of the potential complications is critical to mitigate risk during these complex procedures.

1. Introduction

Chronic total occlusion (CTO) remains one of the most difficult subsets for the interventionists because of the perceived procedural complexity. Over the past few years, tremendous improvement in percutaneous coronary intervention (PCI) materials and equipments, as well as growth of new strategies have enabled us to treat with success even complex CTO. As PCI of CTOs are among the most complex procedures, it is critical to understand the potential complications with these procedures, and steps that could be taken for mitigating risk. Even in high volume and experienced centers, death may occur in up to 1% of patients, and in-hospital myocardial infarction (MI) may occur in up to 5% of cases, despite the conviction that PCI of CTO is a low-risk procedure.[1] There has been a progressive decrease in periprocedural MI and rate of death reflecting the evolution of PCI techniques, the development of dedicated equipments, and the utilization of adjunctive pharmacological therapy.[2] A systematic review by Patel and colleagues[3] of 65 studies with 18,061 patients and 18,941 target CTO vessels also revealed low risk for death (0.2%), emergent coronary bypass graft (0.1%), stroke (0.01%), MI (2.5%), and contrast nephropathy (3.8%). An analysis of 3482 patients and 3493 target CTO lesions from a total of 26 studies[4] revealed the complications consistent with above-mentioned

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Table 1 – Complications of CTO–PCI.

Death	0.7%
Urgent CABG	0.7%
Cardiac tamponade	1.4%
Collateral perforation	6.9%
Coronary perforation	4.3%
Donor vessel dissection	2%
Stroke	0.5%
MI	3.1%
Q wave MI	0.6%
Vascular access complications	2%
Contrast nephropathy	1.8%
Wire fracture and equipment entrapment	1.2%

Table 2 – Proposed classification of coronary perforation.

Type	Definition
I	Focal extraluminal crater without extravasation
II	Pericardial or myocardial blush without an exit hole larger than 1 mm
III	Frank streaming of contrast through an exit hole larger than 1 mm
IV	Contrast spilling directly into anatomic cavity chamber such as coronary sinus and the right ventricle
V	Distal perforation related to the use of hydrophilic and/or stiff wires

study (Table 1). For didactic purpose, CTO-related complications could be classified according to timing (acute and long term) and according to location (cardiac coronary, cardiac non-coronary, and extracardiac).

2. Acute coronary complications

2.1. *Perforations*

Coronary perforation occurs when dissection or intimal tear propagates outward to completely penetrate the arterial wall. It is one of the most dreaded complications of CTO–PCI which can lead to cardiac tamponade necessitating emergency pericardiocentesis and rarely, cardiac surgery to be controlled. Although perforations are common in CTO–PCI (27.6%) in one series,[5] most of them do not lead to serious consequences, and the risk of tamponade is low (approximately 0.5%). The risk factors predictive of perforation include the use of oversized compliant balloons (balloon-to-artery ratio >1.2) coupled with relatively high inflation pressure and hydrophilic and stiffer wires, particularly in calcified and tortuous arteries.[6–9] Use of debulking such as rotational atherectomy is an additional risk of perforation. Balloon advancement or dilatation on a subintimal wire results in major blood extravasation which may cause pericardial effusion possibly leading to cardiac tamponade, intramural hematoma or iatrogenic fistula between the coronary and cardiac cavity.[10] Balloons and equipments are advanced in subintimal space in antegrade or retrograde dissection and reentry in CTO–PCI. To avoid complications, operators need to make sure that equipments are within the adventitia, not in side branches and to size balloons appropriately using intravascular ultrasound (IVUS). Ellis-graded perforations fall into severity, ranging from small endovascular leaks into the adventia (type I) to frank extravasation into the pericardial space (type III).[6] Type IV include perforations into an anatomic cavity, such as the coronary sinus and right ventricle. Muller and colleagues, introduced type V perforation: distal perforation related to use of hydrophilic and/or stiff wires.[11] A proposed classification of perforation is shown in Table 2.[1,11] Type I perforations rarely lead to tamponade compared to type III perforations that are associated with a high rate of hemodynamic compromise (due to tamponade).

2.1.1. *Prevention and treatment*

Prevention and minimization of the risk of bleeding is critical which can be accomplished in using unfractionated heparin for anticoagulation in contrast to bivalirudin and not using glycoprotein IIb/IIIa inhibitors during the procedure. With high activated clotting time (ACT), better anticoagulation can be achieved with heparin than bivalirudin. In addition, bivalirudin does not work in stagnant columns of blood which occurs in large bore guiding catheters leading to catheter thrombosis. Perforation has become important particularly with introduction of stiff wires for penetrating the proximal or distal cap of CTO. Dilatation of subintimal channel may result in vessel occlusion or perforation. It is also important to pay careful attention to tip hydrophilic wire (due to its propensity for subintimal passage and perforation of end capillaries) during attempts to deliver equipment; exchange a stiff or hydrophilic wire for a workhorse wire immediately after confirmation of successful crossing. The possibility of wire dissection can be avoided by meticulous angiography including dual injections and observing the wire path in orthogonal projections. Stiff guidewires induced perforation are of two types: perforation in the false lumen during wire advancement into false lumen; and distal small branch perforation after crossing the CTO lesions. Careful observation via angiogram is the most important consideration in diatal small branch perforations. These perforations very often cause delayed tamponade because operators are not able to detect them. It is advisable to use low balloon-artery ratio, high-pressure initial balloon inflation and over-sizing in heavy calcification. After initial balloon inflation, the deflated balloon should be kept in place; ECG should be watched for its reversal to baseline. The patient should be asked if there is relief of chest pain after balloon deflation. With a small contrast injection, if there is good distal flow without obvious extravasation of contrast, then the balloon should be pulled back in the guidewire to be reinflated should perforation occurs.[12]

In type I perforations, treatment is limited to careful observation for 15–30 min with repeated injections of contrast media. No further action is required if degree of extravasation does not increase or diminishes. However, increased extravasation is treated with reversal of anticoagulation and/or prolonged balloon inflation at or proximal to the perforated segment. Treatment of type II and III perforation should start with inflation of balloon over the site of perforation to occlude the flow (prolonged inflation of 10–30 min usually at 2 atm).[12] However, this attempt is sometimes difficult to continue because of ischemia (uncommon in CTO–PCI due to presence of collaterals). To relieve chest pain and avoid ischemia to distal area during balloon inflation, a microcatheter over another guidewire is positioned distal to site of perforation and

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