

Complications of CTO Intervention

Managing perforation and dissection.

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Chronic total occlusion (CTO) remains as one of the most challenging lesion subsets in interventional cardiology, even with the continuing developments of improved medical devices and increasing operator expertise. Complications related to CTO intervention include, in particular, coronary perforation, guide catheter dissection, inadvertent occluding of collateral circulation, arrhythmia, distal embolism, intramural hematoma, and extensive dissection. This article focuses on the management of coronary perforation and dissection.

CORONARY PERFORATION OR RUPTURE Incidence and Causes

Coronary perforation occurs when a dissection or intimal tear propagates radially outward to completely penetrate the arterial wall. It is undoubtedly one of the most serious complications in the catheterization laboratory because of its associated morbidity and mortality rates.

Numerous studies have reported a low incidence of coronary perforation ranging from 0.1% to 3% of lesions treated with various intervention techniques.¹⁻⁹ However, in the setting of CTO intervention, the increasing use of stiff wires for penetrating the proximal and distal caps of the total occlusion may lead to an increased rate of perforation in this subset of lesions. In a study of 172 patients with de novo CTO lesions and in which percutaneous coronary intervention (PCI) was attempted, the overall incidence of perforation was 1.7%. However, in patients in whom the initial attempt had failed, the investigators found the occurrence of frank perforation (type II or III) to be 6.7%.¹⁰ In another series of 7,443 procedures, the incidence of perforation was 0.93% (69 of 7,443), but 64% (44 of 69) occurred when treating a CTO.¹¹

Demographic risk factors for coronary perforation include older age and female gender. Equipment-related

risk factors include the use of oversized compliant balloons (balloon-to-artery ratio, >1.2–1.3) coupled with relatively high inflation pressure and hydrophilic-coated and stiff wires, particularly in calcified and tortuous arteries.^{1,5,6,8} The use of atheroablative devices is an additional risk.^{3,4,6,8,12,13}

Distal migration of the guidewire (especially in the presence of glycoprotein IIb/IIIa inhibition) is also an important risk factor, in one study accounting for 51% of all the cases of coronary perforation.⁹

Classification of Coronary Perforations

In 1994, Ellis et al evaluated a novel angiographic classification scheme for coronary artery perforations as a predictor of outcome. In a multicenter registry of 12,900 PCIs, 62 (0.5%) perforations were reported and categorized as (1) type I, crater extending outside the lumen only in the absence of linear staining angiographically suggestive of a dissection; (2) type II, pericardial or myocardial blush without an exit hole ≥ 1 mm; (3) type III, frank streaming of contrast through an exit hole ≥ 1 mm perforation; and (4) type IV, perforations with contrast spilling directly into either the left ventricle, coronary sinus, or other anatomic circulatory chamber.¹ Numerous studies have since confirmed that the angiographic classification of perforation can be used as a tool to predict outcome and as a basis of management.^{6,9} For instance, type I perforations rarely result in tamponade or in myocardial ischemia. Type II perforations usually have a high treatment success rate when managed conservatively, resulting in a low incidence of adverse outcome. On the other hand, type III perforations are associated with a high rate of hemodynamic compromise (due to tamponade). They also carry a high risk of mortality due to the subsequent need of emergent surgery, despite conservative strategies. This classification has recently been completed by Muller and colleagues, who added a fifth type of perforation: distal perforation related to the use of hydrophilic and/or stiff wires.¹⁴

Management and Treatment of Coronary Perforations

Due to the significant mortality risk associated with coronary perforation, their management and treatment is quite important and should be initiated very rapidly. As stated previously, the best strategy is determined by the angiographic type and hemodynamic findings. The treatment algorithm is summarized in Figure 1.

In type I perforations, management is commonly limited to careful observations for 15 to 30 minutes with repeated injections of contrast media. If the degree of extravasation does not increase or if it diminishes, no further action is required. If the extravasation increases, intravenous heparin-neutralizing protamine sulfate should be administered. Direct antithrombin agents (such as bivalirudin) may be more problematic because there is no antidote for this class of agents.

In type II perforation, the first step in management is the rapid placement of a standard balloon catheter to seal the perforation. Echocardiographic assessment should be performed without delay. In patients with clinical or echocardiographic signs of tamponade, reversal of anticoagulation with protamine sulfate and platelet transfusion should be performed if they have received abciximab along with urgent pericardiocentesis. In type III perforations, an immediate, aggressive treatment strategy is required, including adequate volume resuscitation, administration of catecholamine, and, frequently, urgent pericardiocentesis. Immediate reversal of anticoagulation with intravenous protamine and platelet transfusion in abciximab-treated patients should be performed.

Treatment of type III perforation should start with standard balloon catheter inflation at the site of perforation for at least 5 to 10 minutes, and pericardiocentesis should be performed as needed. Subsequent serial prolonged balloon inflation (for 5–10 min) may successfully seal a type III perforation or can provide time to prepare a polytetrafluoroethylene-covered stent.¹⁵ The site of coronary perforation must be completely sealed by these therapeutic modalities and confirmed by an angiogram, obtained at least 10 minutes after

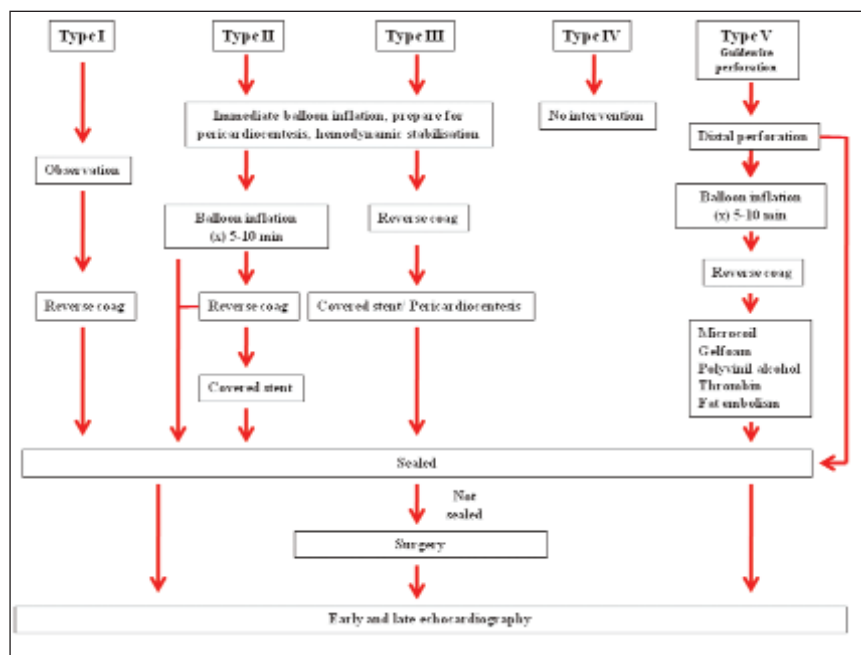


Figure 1. An algorithm for the management of coronary perforation. The traditional Ellis classification has been extended by a type V perforation, encompassing distal coronary perforation.

treatment. In type II and type III perforations, emergent cardiac surgery is reserved for patients who do not achieve hemostasis with these conservative measures.

Type IV perforation usually requires no treatment.

Type V coronary perforations are inflicted by distal guidewire manipulation and should be treated first by proximal balloon inflation. Alternatively, and particularly in case of persistent leakage, embolization of the artery may be considered in order to seal type V perforations. The use of microcoils, gelfoam, clotted autologous blood, subcutaneous tissue, as well as polyvinyl alcohol and thrombin have been described to manage this complication.¹⁶⁻²¹

In each type of perforation, early echocardiography is mandatory to establish a baseline examination, followed by late (24 hours) echocardiography to capture persistent leaks, which may complicate distal perforations and become clinically relevant only after several hours.

CORONARY DISSECTION

Mechanical dilatation of the artery with a balloon or a stent, as well as with use of atheroablative devices, is associated with plaque fracture, intimal splitting, and localized medial dissection. These tears (coronary dissections) may extend into the media for varying distances and may extend through the adventitia, resulting

in coronary perforation. When treating CTO lesions, the use of stiff or hydrophilic wires, along with the absence of direct visualization of the artery lumen when trying to cross the occlusion, may lead to a high occurrence rate of coronary dissections. In one study of 172 patients with de novo CTO and in which PCI was attempted, dissection of the artery wall was recognized in 7.6% of the entire cohort, and in up to 20% of patients in whom the attempted opening ultimately failed.¹⁰ The National Heart, Lung, and Blood Institute had, in the pre-stent era, developed a classification of dissection types after balloon angioplasty, based on their angiographic appearances (types A through F) (Figure 2).²²

Type A dissections represent minor radiolucent areas within the coronary lumen during contrast injection, with little or no persistence of contrast after the dye has cleared. Type B dissections are parallel tracts or a double lumen separated by a radiolucent area during contrast injection, with minimal or no persistence after dye clearance. Type C dissections appear as contrast outside the coronary lumen (extraluminal cap), with persistence of contrast after dye has cleared from the lumen. Type D dissections represent spiral (barber shop pole) luminal filling defects, frequently with excessive contrast staining of the dissected false lumen. Type E dissections appear as new, persistent filling defects within the coronary lumen. Type F dissections represent those that lead to total occlusion of the coronary lumen without distal antegrade flow. In general, types A and B dissections are considered clinically benign and do not adversely affect procedural outcome. However, types C through F are considered major dissections and carry a significant increase in morbidity and mortality. Follow-up percutaneous transluminal coronary angioplasty (PTCA) of mild dissections as early as 6 weeks after the procedure often demonstrates complete healing, although occasional late localized aneurysm formation has been described. In contrast, larger dissections are associated with an increased risk of progression to total occlusion (abrupt closure) of the treated arterial segment. In one series of 691 dissections after PTCA, 543 were type B. This study showed that these patients had a course similar to those without dissections. In comparison, patients with type C to F

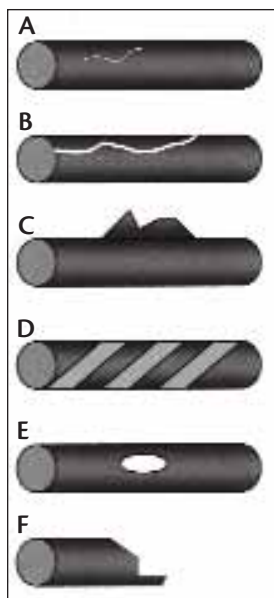


Figure 2. An angiographic classification of coronary dissection: type A, luminal haziness; type B, linear dissection; type C, extra luminal contrast staining; type D, spiral dissection; type E, dissection with reduced flow; type F, dissection with total occlusion.

dissections had a high incidence of acute vessel closure (31%), myocardial infarction (13%), and emergency coronary artery bypass grafting (37%).²³ The increased risk of abrupt closure and myocardial infarction associated with a large dissection has led to the routine use of stenting for any dissection. However, a stent may not be always be necessary for a nonocclusive dissection. This finding was illustrated in a study comparing the outcome of 45 patients with 49 nonocclusive dissections (type A to D) and good distal flow (TIMI grade 3) who did not receive a stent, with 60 patients with a dissection who underwent stenting. At 6 months, there were no clinical adverse events in the unstented group, although the restenosis rate was lower than in those who underwent stenting (12% vs 25%).²⁴ The most feared complication of coronary dissection is acute vessel closure. In the pre-stent era, this complication occurred in up to 11% of all elective PTCA.²⁵⁻²⁸ With the advent of coronary stents, the incidence of acute closure in elective PCI has decreased to less than 1%.²⁹ These days, ischemic complications usually occur as manifestations of edge dissections after stenting, which may predispose to stent thrombosis.

Given the potential adverse outcome related to these edge dissections, most are treated with additional stent deployment. Providing that the guidewire is seating in the true lumen, dissections can usually be managed by deployment of stents sufficient to seal off the dissection flap. Taking great care not to lose guidewire position in these situations is of major importance.

Given the propensity of dissections to propagate distally, special care should be taken to contain and cover, as soon as possible, the distal extent of the dissection with a stent to prevent further extension.

Risk factors for the development of coronary artery dissection include the presence of calcified, eccentric, and long lesions; complex lesion morphology; and vessel tortuosity.³⁰⁻³² In addition, aggressive guiding catheters, such as the Amplatz catheters (Cook Medical, Bloomington, IN), are often used to enhance support in CTO procedures. However, these catheters often engage the coronary ostium deeply, and there is an increased potential for proximal dissection.³³ Therefore, careful manipulation of these catheters is required by experienced operators. Special care must also be taken when using hydrophilic

wires to treat CTO, due to their propensity for subintimal passage. These wires should always be used carefully and never pushed against resistance. To avoid wire dissection, we recommend meticulous angiography (ideally with dual injection) in order to observe the path of the wire in both orthogonal projections.

Finally, the interventionist should also consider that in certain CTO procedures, wire dissections can help the operator find the true lumen by using the "parallel wire" technique or can provide a unique opportunity for exiting the occluded vessel proximally and entering it distally via the STAR (subintimal tracking and re-entry) technique.³⁴

CONCLUSION

Despite major advances in the treatment of CTOs, certain complications still persist. One of the most common and feared complications during CTO procedures is coronary perforation. The angiographic type of perforation will largely determine the outcome and the type of treatment. In this article, we have attempted to provide a treatment algorithm to guide the interventional cardiologists in the management of this life-threatening complication. Finally, the operator should always remember that performing a gentle and careful procedure will help avoid most of these complications. ■

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