Coronary Perforation: An Inconvenient Complication

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EDITORIAL COMMENT

Coronary Perforation

An Inconvenient Complication*

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Coronary perforation is one of the most challenging and feared complications of percutaneous coronary intervention (PCI). The angiographic images of a large perforation are spectacular and are often debated during complication case review sessions at interventional cardiology meetings. Therefore, even if most interventional cardiologists have not been exposed personally, they are perfectly aware of this rare complication. Coronary perforation has been classified by Ellis et al. (1) in 1994, type III being the most severe form of perforation. It was originally defined as an active extravasation through a large breach (at least 1 mm) in the integrity of the adventitia of an epicardial artery in the pericardial space or in a cardiac chamber. This complication is rare (0.1% to 3.0%), often requires pericardiocentesis for tamponade, as well as combination of interventional techniques to seal the perforation, and occasionally surgical repair (2). The morbidity and mortality of large perforations is known to be high.

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In this issue of *JACC: Cardiovascular Interventions,* Al-Lamee et al. (3) reviewed the incidence, predictors, management, and clinical outcome of type III perforations during PCI in a large tertiary referral center. Over a 16-year period, this complication was noted in 56 of 24,465 consecutive patients undergoing PCI, thus giving a reported incidence of 0.23%.

A review of the literature reveals abundant data on perforation during PCI (1,2). The findings of the current observation are in accordance with common knowledge and provide some new, added value. Albeit rare, the incidence of perforation has not decreased over time, probably reflecting the increased complexity of PCI practice. This complexity is illustrated by the independent predictors of perforation seen in the present study: complex coronary anatomy (type B2 and C lesions), chronic total occlusion, rotational atherectomy, and intravascular ultrasound use. These predictors have all been identified previously (2,4). The highest risk was related to intravascular ultrasound use, reflecting PCI in complex lesion subsets, according to the investigators. They also speculate further in this context, on the role of higher balloon pressures or larger balloon sizes to optimize the angiographic result according to pre-defined intravascular ultrasound criteria. This is perfectly in line with the most common cause of perforation in the present study: compliant or noncompliant balloons and the stent-balloon delivery system. Interestingly, the second most frequent cause of perforation in the present study was guidewire exit (10 patients). Guidewire exit perforation usually causes "relatively small leaks" in the distal coronary bed or a side branch and is nowadays mostly successfully treated by coiling or particle embolization technology. It is therefore unclear why guidewire exit leads so frequently to type III perforation in this cohort.

Concomitant glycoprotein IIb/IIIa antagonist use increased dramatically the complication rate and decreased, despite applying different treatment modalities, hemostasis success. Even if the number of patients is too small to draw any firm conclusions, it makes common sense to pay particular attention to perforations in these patients, treating them more aggressively and reversing any antiplatelet activity if possible. It is known that guidewire exit and glycoprotein IIb/IIIa antagonist do not sit well with each other. Although enough information is not available in the present article, if frequently associated, this may possibly explain the high prevalence of guidewire exit as a cause of type III perforation in this study.

The treatment of a type III perforation remains a challenge for every catheterization laboratory team. It integrates continuous assessment of the hemodynamic status and the need for appropriate treatment of tamponade if needed, as well as an immediate attempt to seal the perforation. Both conditions are closely interrelated and failure to treat either will affect the immediate prognosis of the patient. Placement of a pericardial drain in the emergency setting is difficult particularly if tamponade is caused by the acute accumulation of a relatively modest amount of blood. Echocardiographic and fluoroscopic guidance are of critical importance in this situation. Any additional complication due to inadvertent myocardial puncture will further compromise the patient's outcome. The investigators observe currently that hemostasis often requires multiple treatment modalities: prolonged balloon inflation, implantation of a standard or covered stent, coil embolization, and/or surgical repair. Even if precise data are lacking, it seems that most patients could be treated by a combination of prolonged balloon inflation and implantation of a covered stent. This is important new information that is highlighted by a dramatic increase in procedural success and a clear reduction

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in in-hospital morbidity and mortality during this 16-year observation period. Without doubt, covered stents (84.6% in the present series) have the highest potential to seal large perforations. They are probably responsible in large part for the increased success rate in managing large perforation. There are, however, clear downsides. First, covered stents are bulky and proper stent delivery may be difficult or impossible. A helpful technique not described in the current article is the "double guiding catheter" technique. This consists of prolonged or intermittent (in patients with extensive ischemia) balloon inflation through the initial guiding catheter while preparing femoral access for an 8-F guiding approach. Upon balloon deflation through the initial guiding catheter, a guidewire is advanced through the second guiding catheter distal to the site of perforation to deliver the covered stent. The advantage of the technique is that temporary sealing of the perforation can occur while simultaneously preparing better support for stent delivery. The paper by Al-Lamee et al. (3) yields another important message and a second drawback of polytetrafluoroethylene-covered stents, in particular. The incidence of definitive stent thrombosis was 8.6% and only occurred after placement of polytetrafluoroethylene-covered stents. This is higher than with metallic noncovered stents suggesting a higher thrombogenicity of this platform. The role of possible suboptimal stent expansion of this double-layer metallic device is unknown. Furthermore, the type of long-term antiplatelet strategy may also have been important. Prasugrel, because of its lack of intrinsic resistance, might be considered as the thienopyridine of choice, although there are no data available.

The present article provides an algorithm for managing type III perforation. It is straightforward and comprehensive. The only uncertainty concerns the role of heparin reversal with protamine sulfate. Opinions clearly diverge on this subject. On the one hand, it may assist in sealing a perforation, but on the other, bleeding may persist and prolonged balloon inflation and simultaneous heparin reversal may lead to proximal vessel thrombosis. It is our belief that no universal guidance can be given based on the data available. Decisions should be pragmatic and individualized dependent on the hemodynamic status of the patient, concomitant IIb/III antagonist use, activated clotting time, and the duration of prolonged balloon inflation.

One particular form of type III perforation is termed *cavity spilling* (cardiac chamber, coronary sinus). Apparently, this complication was not encountered in this observational study. In general, management should be less aggressive because of the lack of associated acute hemodynamic compromise.

A large type III perforation is an inconvenient complication. In-hospital mortality was high at 14.8% and the rate of myocardial infarction was 42.9%. This proportion increased further during follow-up. Even in the experienced hands of these operators, the reported results are not better than what can be found in literature, indicating a need for better therapeutic devices.

In conclusion, this article has drawn our attention again on a dramatic but rare complication of PCI: type III perforation. The key messages are as follows. 1) Concomitant IIb/IIIa antagonist use increases the complications rate substantially and diminishes the ability to seal a perforation successfully. Its effect should be reverted if possible. 2) Polytetrafluoroethylene-covered stents have the greatest potential to seal type III perforations and probably explain increasing procedural success over the period observed. However, these potentially "life-saving" devices are definitely more thrombogenic. The investigators' conclusion is apposite: "This complication is still associated with poor adverse outcomes and there remains a need for improved technology in order to treat this dreaded complication."

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