

# Unexpected Coronary Perforation Complicated by Cardiac Tamponade: A Case Report and How to Treat It

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**Abstract** Coronary artery perforation is a rare but fatal complication of percutaneous coronary intervention. Cardiac tamponade constitutes the most severe clinical consequence. If not properly and promptly treated, it is burdened by a high mortality rate. This article reports the case of an intra-stent coronary perforation after simple revascularization, followed by an immediate impairment of hemodynamic compensation, due to significant pericardial effusion and subsequent cardiac tamponade. While prompt surgical intervention may be lifesaving, expertise in the use of stent graft may provide a valuable rescue option for this serious complication. Interventional cardiologists should be able to recognize and know available treatment options of this serious complication.

**Keywords:** coronary perforation, percutaneous coronary intervention, prolong balloon inflation, stent graft

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## 1. Introduction

Coronary perforation is a rare but life-threatening complication of percutaneous coronary intervention (PCI). The incidence is among 0.2–0.6% [1,2]. It has been associated with high rate of major adverse outcomes such as cardiac tamponade (17 %), myocardial infarction or death (9 %) [3]. Albeit rare, the incidence of perforation has not decreased over time, probably reflecting the increased complexity of PCI practice. The predictors of perforation seen in the present study: complex coronary anatomy (type B2 and C lesions), chronic total occlusion (CTO), rotational atherectomy and intravascular ultrasound (IVUS) use [4]. Occasionally perforation can even be associated with simple stenting [5]. This case has demonstrated an intra-stent coronary perforation at the end of simple revascularization, followed by immediate impairment of hemodynamic compensation due to significant pericardial effusion and subsequent cardiac tamponade.

## 2. Case Report

A 55-year-old man with a history of smoking and exertional angina for 1 month was admitted to our department for anterior wall non ST elevation myocardial infarction (NSTEMI). The electrocardiogram (ECG) showed ST segment depression and T wave inversion in lead V1-4. The echocardiogram revealed normal left

ventricular ejection fraction (60%) with mild hypokinesia at anterior wall and mild mitral regurgitation. Other laboratory assessment revealed evidence of myocardial injury, with positive troponin I. Coronary angiogram shows significant lesion at proximal and mid part of left anterior descending (LAD) artery (Figure 1). Subtotal occlusion at left circumflex (LCX) artery but normal right coronary artery (RCA). The patient underwent percutaneous coronary intervention (PCI) after an informed consent. According to the clinical setting and electrocardiogram changes, LAD was considered to be the culprit vessel.

The left main (LM) artery was engaged with a 6 Fr Launcher EBU 3.5 guiding catheter (Medtronic, Minnesota, USA) via the right groin. A 0.014-inch Pilot 50 guide wire (Abbott Vascular, Santa Clara, CA, USA) was passed to distal LAD. Proximal and mid LAD lesions were predilated with Minitrek (Abbott Vascular, Santa Clara, CA, USA) balloon size 2.0 x 20 mm, and then Biometrix alpha (Biosensors, Shangdong, China) drug eluting stent (DES) 2.5 x 29 mm and 3.0 x 24 mm were deployed to mid and proximal part of LAD respectively. After postdilated mid LAD lesion with NC trek (Abbott Vascular, Santa Clara, CA, USA) balloon 2.75 x 15 mm up to 12 atm (Figure 2). Angiography confirmed the presence of Ellis grade 3 coronary perforation at the mid LAD stent with free flow of contrast in to the pericardial space with no coronary flow to distal LAD (Figure 3). Mechanical hemostasis was accomplished by inflating the same postdilatation balloon in the LAD proximal to the perforation at low atmosphere. The patient suddenly complained of severe chest pain and cardiac

arrested. Cardiopulmonary resuscitation (CPR) was done immediately and endotracheal tube was intubated. Return of spontaneous circulation (ROSC) was achieved after 5 minute of CPR. Echocardiogram revealed cardiac tamponade that required emergent pericardiocentesis and drainage, which yielded 500 ml of blood. Heparin was reversed with 20 mg of intravenous protamine sulphate. Multiple inflations were performed at low atmospheres for 4-5 min but leakage still persisted so the use of a coronary stent graft was decided. A 2.8 ×16 mm Graftmaster (Abbott Vascular, Santa Clara, California) was deployed over the perforation site (Figure 4). Test injections revealed no further extravasation, the flow into the pericardial space abolished (Figure 5). Due to rapid hemodynamic destabilization, after the placement of stent graft an intra aortic balloon pump (IABP) was placed, the patient remained hemodynamically stable thereafter. He was discharged from the hospital after one week with stable condition. During the subsequent monthly follow up visits, he was in a very good condition and follow up echocardiography did not show any pericardial effusion.

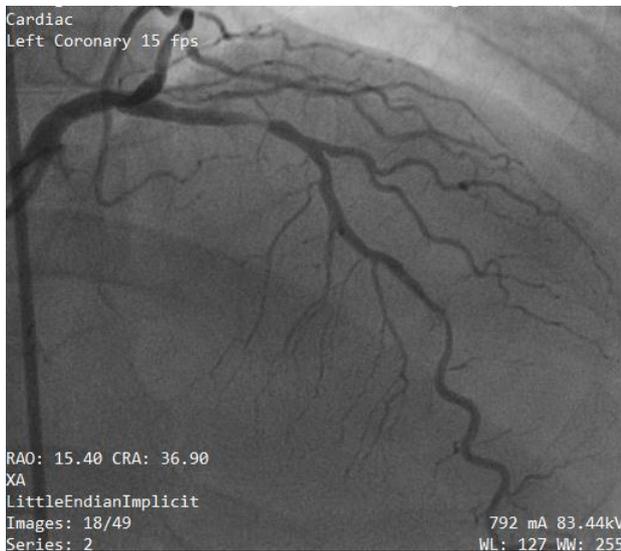


Figure 1. Initial coronary angiogram of left coronary artery

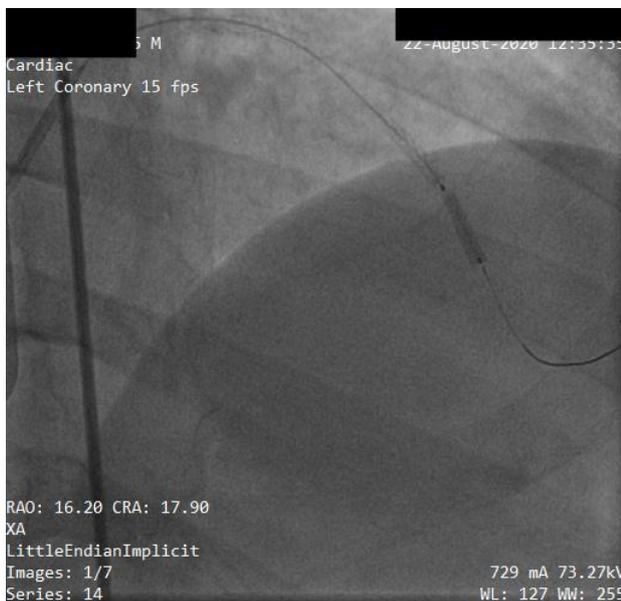


Figure 2. Postdilatation after stent deployment at mid LAD



Figure 3. Angiography confirmed the presence of Ellis grade 3 coronary perforation with free flow of contrast into the pericardial space

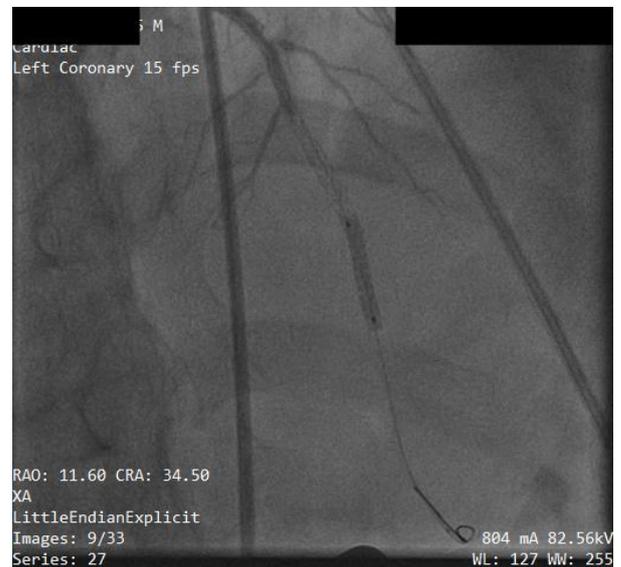


Figure 4. A 2.8 × 16 mm GraftMaster (Abbott Vascular, Santa Clara, California) was deployed over the perforation site



Figure 5. Test injections revealed no further extravasation, the flow into the pericardial space abolished

### 3. Discussion

Coronary artery perforation is a rare complication. Its incidence varies according to patient, lesion and procedure characteristics. Ellis et al. classified coronary perforations based on their angiographic appearance [6]. Type I perforations are limited to the vessel wall and produce an intramural crater without extravasation on the angiogram. Type II perforations show limited extravasation with pericardial or myocardial blushing on angiography, whereas in 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. 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 [4]. Both conditions are closely interrelated and failure to treat either will affect the immediate prognosis of the patient.

Therapeutic strategies include prolonged balloon inflation, stents graft, reversal of anticoagulation, embolization of the distal vessel and surgery, the choice depending on the site and severity of the perforation, the patient's hemodynamic status and the equipment available in the catheterization laboratory. Echocardiography should be performed as soon as a perforation is identified. The sudden accumulation of blood in the pericardial space may lead to cardiac tamponade with resulting cardiogenic shock and high mortality rate. If hemodynamic collapse occur pericardiocentesis should be performed immediately [7]. Administration of fluids is recommended. A balloon should immediately be placed with inflations lasting up to 5- 10 minutes to block extravasation. If the perforation can not be sealed, repeated inflations should be made. Distal ischemia being a concern, perfusion balloons can be used without blocking distal blood flow. Reversal of anticoagulation can be achieved with protamine sulfate. As previous study shown, the use of protamine sulfate is safe and does not predispose to stent thrombosis [8,9]. Deployment of stent graft is an important role as bail-out treatment, especially when the perforation was located in the proximal vessel segments with a diameter > 2.75 mm., that is more likely caused by oversized or aggressive use of balloons or devices [10]. The main objective of a stent graft is to seal the perforation with a layer impermeable to blood but there still have some limitation. Firstly, all stents carry the inherent risk of side branch occlusion. Second, there are concerns about late thrombogenicity of Polytetrafluoroethylene (PTFE) covered stents that might be overcome with prolonged dual-antiplatelet therapy. Newer generation covered stents contain graft material made from autologous veins or equine pericardium to improve stent flexibility and decrease thrombogenicity [11,12].

Alternative therapies used in selected cases include coil embolization, thrombogenic particles including gelfoam, thrombin, embolic agents like N-butyl cyanoacrylate glue, and autologous blood clot [13], these agents carry a risk of loss of the vessel lumen and subsequent infarction so these should be used as a last resort of distal perforation. If a

large perforation causes severe ischemia or hemodynamic deterioration or cannot be sealed with the available techniques, emergency surgery is indicated.

In this case, coronary artery perforations are caused by a mismatch between the balloon size and the artery diameter. A balloon artery ratio of greater than 1.1 has been associated with a 2–3 fold increase in coronary artery perforations [14]. Since balloon dilation failed to cease the leakage so the pericardiocentesis and the emergency stent graft implantation were performed to treat the perforation site. Although Coronary artery bypass graft surgery (CABG) is an excellent treatment in type III Ellis perforation, the use of stent graft represented an alternative and quicker treatment.

### 4. Conclusion

Coronary artery perforation is a rare complication of percutaneous coronary intervention, but morbidity and mortality are considerable. Early recognition and adequate management are very importance. Interventional cardiologists should be able to recognize and know available treatment options of this complication.

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