COVERED STENTS IN IATROGENIC CORONARY ARTERY FISTULA; A CASE REPORT
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Abstract

BACKGROUND: Coronary artery fistula is an abnormal communication between a coronary artery and a cardiac chamber or major cardiac vessels, mostly congenital but some of them are acquired as a consequence of coronary artery perforation.

CASE PRESENTATION: We report a case of cavity spilling coronary artery perforation during percutaneous coronary intervention 7 years ago. Because of continuing symptoms and risk of developing heart failure and pulmonary hypertension we were ought to treat this iatrogenically formed coronary artery fistula. We used stent graft implantation to treat it with acceptable results.

CONCLUSION: Beside their application as a rescue for acute coronary artery perforations, stent grafts can be used with acceptable results in iatrogenically acquired coronary artery coronary artery fistula

Keywords: Coronary artery perforation, Coronary artery fistula, Stent graft.

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Background

Coronary perforation occurs when a dissection or intimal tear propagates outward sufficient to completely penetrate the arterial wall.1-3 Balloon to artery ratio > 1.3, balloon rupture (especially those associated with pinhole leak as opposed to longitudinal tears), use of debulking devices, complex lesion (AHA type 2B or C lesions), old age and female gender are risk factors for coronary artery perforations.4-9 In a multicenter registry of 12,900 percutaneous coronary intervention (PCI), 62 (0.5%) perforations were reported and categorized as: Type I, extraluminal crater without extravasation; Type II, epicardial fat or myocardial blush without contrast jet extravasation; Type III, extravasation through frank (> 1 mm) perforation; and Type III "cavity spilling" (CS) referring to Type III perforations with contrast spilling directly into either the right or left ventricle, coronary sinus or other anatomic circulatory chamber. The higher the grade, the more mortality and morbidity (tamponade, myocardial infarction, need for emergency bypass surgery) except for cavity spilling coronary artery perforation which despite its frank nature has less catastrophic consequences.10 With passing time these forms of coronary artery perforation become and gain all features of coronary artery fistulae we report one of these cases with reviewing treatment options.1-9

Case Presentation

The patient was a 63-year old lady with a suspicious history of systemic lupus erythmatosus (SLE); despite discontinuing medical treatment for 8 years, she had no symptoms of skin rush and artheralgia for which SLE was once diagnosed and chloroquine was once started.

The patient also had frequent episodes of palpitation. In referral to cardiologist in 2001 the diagnosis of paroxysmal Supraventricular tachycardia (PSVT) (AVNRT) was made and the patient was referred to cardiac electrophysiologist for radiofrequency (RF) ablation. During (based on records) her successful modification of slow pathway, the
patient developed excruciating chest pain. Coronary angiography (CAG) at hoc showed severe stenosis at Left Anterior Descending (LAD) artery mid-portion. Since the symptoms were dissipated on simple medical measures like TNG spray and low doses of narcotics no invasive intervention had been done and the patient had been discharged home then.

But it was now previously undiagnosed LAD lesion which made her symptomatic. During the months after her index ablation procedure, the patient developed typical exertional chest discomfort FC 2-3.

In March 2002 she was hospitalized with the intention of coronary angiography and probable angioplasty of LAD lesion. During coronary angioplasty after placement of Cypher stent 3 \times 23 in LAD midportion the procedure was complicated by coronary perforation unexpectedly to right ventricle (RV) cavity (Figure 1). Since the patient hemodynamics and symptoms were stable in catheterization laboratory, the procedure was terminated and this newly formed fistula was left untreated.

The presence of this fistula was reconfirmed in another angiography done on July 2003 and again no specific treatment was done. At that angiogram her left main stem, circumflex and right coronary arteries were all clear (Figure 2). A MIBI scan in the same year (on December), showed apical infarct with periinfarct ischemia.

The patient had been discharged on medical treatment but year by year her symptoms (mostly exertional chest pain and dyspnea) were aggravated. On July 2008, her symptoms once confined to walking upstairs and aggravated by cold weather was already severe enough to impede her light physical activity in moderate temperature. Another session of coronary angiography was scheduled, and fistula sealing by covered stent was planned.

On admission to catheterization laboratory, the patient was in good general condition, vital signs were normal. Cardiac exam revealed apical continuous murmur 2/6. ECG had incomplete right bundle branch block (RBBB) without significant ST-T change. Echocardiographic findings before coronary angiography revealed apical fistula with about 3m/s shunt flow, mild right atrium (RA) and RV dilatation. Right ventricular systolic pressure was estimated to be 60 mm Hg using tricuspid regurgitation (TR) spectrum. Mild to moderate mitral regurgitation (MR) and mild TR was detected as valvular abnormalities. Left ventricle (LV) systolic function was mildly reduced at 45-50% but LV apex appeared severely hypokinetic. Coronary angiography was done. Left circumflex artery (LCX) and proximal right coronary artery (RCA) appeared intact and normal. LAD was ecstatic before the fistula and LAD flow after the fistula was very poor but no prominent stenosis was detected in LAD course. Floppy Guidewire (AVE) was placed across LAD and LAD was directly stented by a 3.5 \times 16 covered stent. In final injection there was no significant stenosis, fistula was completely sealed by the covered stent and Distal LAD flow was reinstated.

The patient was discharged home in a very
good general condition. Chest pains were mostly alleviated and she was able to achieve her daily activities without any symptoms. Another echocardiography six days after the procedure, showed fistula and RV systolic pressure was declined to 48 mm Hg.

Discussion
Our patient was a suspicious case of SLE. In reviewing literature we did not find any relation between coronary endarteritis of SLE and susceptibility to coronary perforation during conventional PCI. Since the patient symptoms were quiescent despite discontinuing her previously light medical treatment, it is difficult to assign any role to her not completely proven SLE disease.

Most cases of symptomatic coronary artery perforations occurs on the pericardial side of the artery with blushing of blood in the not previously prepared low compliant pericardial sac with its divesting sequela. Sometimes blushing become contained in myocardium and in rare circumstances finds its way to one of cardiac cavities mostly right ventricle (the so called cavity spilling). Cardiac cavities e.g. right ventricle are much more compliant than pericardial sac and hence this type of perforation has much more benign acute course than pericardial one and it seems to be well tolerated in acute settings (So we were lucky to encounter this type of perforation!!). Its long-term sequela and management is somewhat resemble to coronary artery fistula and in long-term it attains all the features of a typical coronary artery fistula like prefistoulous ectasia (as in our case). The clinical sequela of coronary artery fistula include myocardial ischemia from coronary steal, heart failure from longstanding cardiac shunts, endocarditis.

Because of severe symptoms and rising pulmonary artery pressure of left to right shunt we could not leave the patient untreated and we had three treatment options as follows a: CABG b: transcatheter embolization of fistula c: sealing of the fistula by implanting stent graft. CABG was not a logical treatment option because the majority of patient symptoms were due to the steal effect of fistula and CABG did not address this issue.

Although ligation of fistula by open surgery was a viable option, its invasive nature with all its perioperative morbidities made us reluctant to do the procedure.

Transcatheter coil embolization of coronary fistula was a novel treatment option we considered.

Ante grade embolization of coronary artery fistula can sometimes be technically challenging if the origin of the fistula is relatively distal, especially with marked tortuosity of the proximal segment of the affected coronary artery. In the latter case, proper seating of the delivery catheter may not be achieved and this can potentially lead to embolization of a coil into the distal coronary artery bed. Moreover, there has been a report of possible thrombus forming in the residual cul-de-sac of the occluded fistula. But what made our case even more challenging for coil embolization were its small neck and the presence of stent struts adjacent to the ostium of coronary fistula (11-15).

Any way we considered stent graft implantation as the therapy of choice for our patient’s fistula and treatment of any residual stenosis. However, there are several points to remember when this technique is being considered. First, one of the major drawbacks of stent grafting, is its potential to inadvertently obstruct flow into a major sidebranch. Second, it comprises of two stents sandwiching a graft, and may require a high-pressure deployment to ensure adequate stent expansion, and more importantly, a good apposition to the wall to completely seal off the fistula. We have no major sidebranch adjacent to the proposed site of stent graft implantation. And although we did not check stent apposition using IVUS study, stent expansion was complete in conventional fluoroscopy.

Conclusion
Stent graft implantation is a novel approach for treatment of coronary artery fistula and as a treatment approach is comparable with coil embolization and surgical ligation.
References


