Wire Perforation or Coronary-Cameral Fistula? A Diagnostic Dilemma Complicating a Case of ST-Segment Elevation Myocardial Infarction

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INTRODUCTION

Percutaneous coronary intervention (PCI) is the primary therapy for patients with histories of coronary artery bypass graft surgery (CABG) presenting with acute ST-segment elevation myocardial infarction (STEMI). Although PCI is relatively safe, with periprocedural complications typically <1%–2%, several complications have been associated with the technique. Guidewire-induced coronary dissection, perforation, and tamponade, with incidences of approximately 0.02%, 0.1%–3%, and 0.12%, respectively, are rare events that can occur in patients with STEMI undergoing PCI. If not promptly diagnosed and treated, these complications may lead to hemodynamic compromise and death.[1,2] Another event that can be observed in these patients is the discovery or creation of a coronary-cameral fistula. These anomalous connections between a coronary artery and any cardiac chamber are rare, with a prevalence ranging from 0.08%–0.3% of patients undergoing diagnostic coronary angiography.[3] We present the case of a 73-year-old male patient with a history of CABG who experienced an acute inferior STEMI due to thrombotic occlusion of a saphenous vein graft (SVG). His coronary intervention was complicated by extravasation of contrast media into an unclear potential space. Using contrast echocardiography and a right heart catheter, we were able to determine whether the observed extravascular flow represented a life-threatening coronary perforation or a relatively benign coronary-cameral fistula.

CASE PRESENTATION

A 73-year-old man with a history of three-vessel CABG presented with an acute inferior STEMI (Figure 1). CABG had been performed in the setting of angina approximately 20 years prior. He reported a history of hypertension, hyperlipidemia, and type 2 diabetes. His medical regimen before presentation included aspirin, losartan, metoprolol succinate, atorvastatin, and furosemide. He received loading doses of aspirin and ticagrelor in the emergency department and was taken emergently to the cardiac catheterization laboratory for coronary angiography.

Coronary and graft angiography was performed using a 6-Fr right common femoral arterial sheath. Native left coronary angiography was significant for a diseased left main coronary artery and a chronic total occlusion of the mid portion of the left anterior descending coronary artery. The left circumflex coronary artery had severe, calcific disease in its proximal portion before giving rise to a large, branching obtuse marginal system with evidence of competitive flow from a vein graft. The right coronary artery (RCA) was chronically occluded in the mid portion of the vessel. Left internal mammary angiography showed a patent left internal mammary artery–to–left anterior descending coronary artery graft. Two SVGs were identified: a patent SVG to obtuse marginal and an occluded SVG to the RCA, the latter demonstrating Thrombolysis in Myocardial Infarction (TIMI) grade 0 flow (Figure 2A). The occlusion appeared thrombotic and was felt to be the culprit lesion precipitating the patient's presentation with STEMI. Therefore, the decision was made to attempt PCI of the occluded vein graft.

A 6-Fr MB1 guide catheter (Medtronic, Minneapolis, MN) was seated at the ostium of the SVG-to-RCA graft. Bivalirudin bolus and infusion were initiated. A 300-cm PT^2 Moderate Support 0.014-inch guidewire (Boston Scientific, Marlborough, MA) was advanced across the thrombotic lesion into the distal vein graft. Balloon angioplasty was performed with a Trek 3.0 × 15-mm balloon (Abbott, Abbott Park, IL) in the proximal segment of the SVG at the site of the thrombotic occlusion. Repeat angiography revealed TIMI grade 2 flow in the vein graft and evidence of embolic material into the distal vein graft and native RCA (Figure 2B and 2C, Video 1). Repeat balloon angioplasty was performed on the distal portion of the SVG proximal to the RCA takeoff. Angiography then demonstrated TIMI grade 2 flow in the vessel and evidence of free-flowing contrast material originating from a small branch of the distal RCA (Video 2). In addition, angiography showed a wedge-shaped collection of extravascular contrast located roughly 2 cm inferior to the observed active flow downstream in the vessel, immediately causing concern for a wire perforation. The balloon was inflated in the distal SVG to occlude flow and limit extravasation of blood. The bivalirudin infusion was stopped.

We were initially concerned for a perforation into the pericardial space; however, angiography also raised the possibility of the presence of a coronary-cameral fistula. It was imperative to fully rule out tamponade physiology before proceeding. We accomplished this with a combination of right heart catheterization and transthoracic echocardiography.

Our first strategy was to use a Swan-Ganz catheter as a marker. Femoral venous access was obtained. In an effort to determine the location of the contrast extravasation, a 7-Fr Swan-Ganz catheter was advanced via the venous sheath into the right atrium with the balloon just above the tricuspid valve. We then performed rotational angiography of the fistulous area (Figure 3A and 3B, Video 3). Simultaneously, our sonographer acquired an apical four-chamber view with transthoracic echocardiography while we deflated the coronary balloon and injected 1 mL of perflutren lipid microspheres echocardiographic contrast medium into the SVG via the guide

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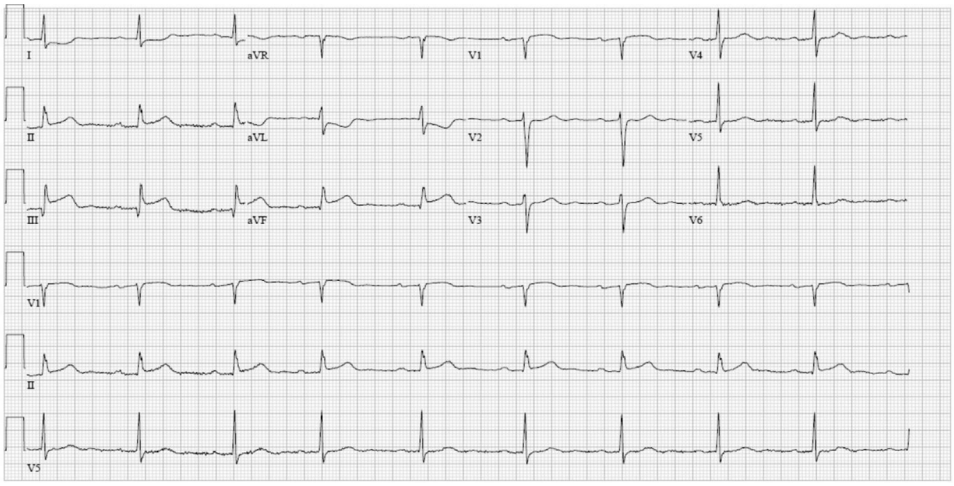

Figure 1 Presentation electrocardiogram showing ST-segment elevations in the inferior leads (II, III, aVF) and reciprocal ST-segment depression in lead aVL.

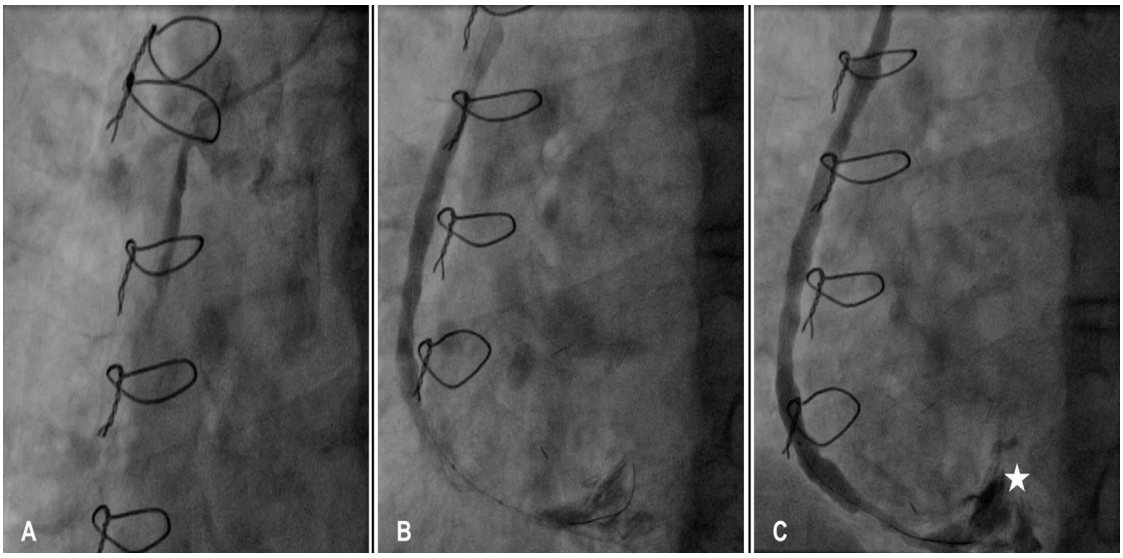

Figure 2 Angiographic still images of SVG-to-RCA graft. **(A)** Occluded SVG before intervention. **(B)** SVG following balloon angioplasty of the proximal portion with residual thrombus in the distal vein graft. **(C)** SVG following distal balloon angioplasty demonstrating extravasation of contrast (*star*).

catheter. The echocardiographic contrast medium immediately appeared in the base of the right ventricle, confirming the drainage of the fistula along the tricuspid annulus (Video 4).

The coronary balloon was removed from the vein graft. After approximately 30 min of monitoring with no clinical deterioration, repeat angiography revealed TIMI grade 3 flow in the vein graft with persistent staining of myocardium in the inferior base. The patient was then admitted to the cardiac intensive care unit.

The patient remained hemodynamically stable throughout the rest of his admission. His troponin T peaked at 3.1 ng/mL approximately 12 hours after presentation. He received guideline-directed medical therapy following STEMI. Postcatheterization echocardiography showed preserved left ventricular systolic function with areas of hypokinesis in the inferior and inferoseptal walls, with no pericardial effusion. He was discharged home 2 days later. At 1-month follow-up, the patient was doing well and remains asymptomatic.

DISCUSSION

STEMI in patients who have previously undergone CABG poses a diagnostic and therapeutic challenge. Not only is the interventionalist constrained to the 90-min door-to-balloon time window for these patients, but he or she also often must intervene without full knowledge of the graft anatomy. Patients with STEMI with previous CABG have a greater 90-day mortality rate compared with those with with no history of CABG, and they are less likely to achieve reperfusion in the artery causing infarct following PCI.[4,5]

The differential diagnosis for extravasation of contrast outside of a coronary artery during coronary catheterization is limited. Iatrogenic coronary artery perforation with a guidewire is a rare but well-known complication, with an incidence ranging from 0.1%–3%.[2] Spontaneous fistulous formations have been associated with heavy atherosclerotic burden and myocardial infarctions.[6] CABG-associated

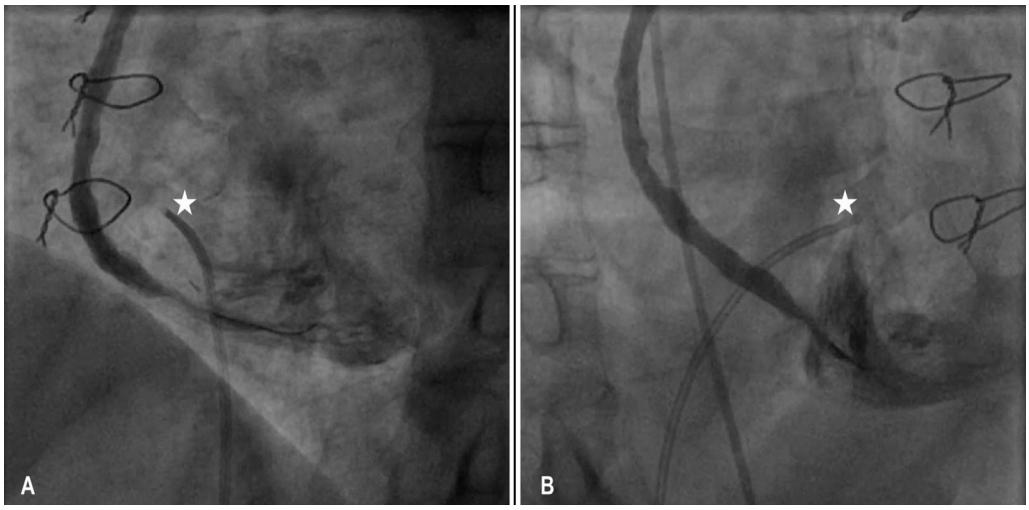

Figure 3 Angiography of SVG-to-RCA graft with Swan-Ganz catheter in place for positional reference. **(A)** Left anterior oblique view and **(B)** right anterior oblique view of coronary-cameral fistula with Swan-Ganz catheter in position (*star*) in the right atrium acting as a point of reference for spatial orientation. Extravasation of dye can be observed into the right ventricle.

Table 1 Ellis classification for coronary artery perforation

	Type I	Type II	Type III	Cavity-spilling perforations
Description	Extraluminal crater without extravasation	Pericardial or myocardial blush	Extravasation through >1 mm of perforation	Perforation into an anatomic cavity chamber or vessel
Management	Observation/balloon tamponade/covered stent deployment	Balloon tamponade/coil embolization ± pericardiocentesis	Balloon tamponade/coil embolization ± pericardiocentesis	Balloon tamponade/coil embolization ± pericardiocentesis

fistulous formations have been reported and must be considered in this subset of patients undergoing coronary angiography.[7] Congenital coronary-cameral fistulas are rare but make up an integral part of the differential diagnosis. Finally, coronary arterial dissection is a well-known complication of coronary instrumentation that results in extravasation of contrast outside the true lumen of the vessel. In some cases, this extravasation into the intimal space is immediately evident on angiography. Therefore, iatrogenic dissection should always be considered.

The presented case is notable for a few reasons. First, approximately 3%–7% of all patients with previous bypass surgery have acute myocardial infarctions each year.[4,8,9] SVGs are used to bypass the RCA or left circumflex coronary artery in >70% of patients requiring CABG despite the fact that it has been shown to be the most common graft to fail, occurring in approximately 34% of cases of acute myocardial infarction.[8-10] Second, the concomitant myocardial staining and presence of fistula were concerning for a catastrophic complication, namely, a large coronary perforation. It was imperative to localize the fistula, assess for evolving hemodynamic compromise, and rule out tamponade immediately.

The Ellis classification system was developed to assist in grading perforation severity in order to predict the likelihood of major complications, specifically, tamponade (Table 1). Type I perforations (extraluminal crater without extravasation) are fully contained perforations that rarely lead to myocardial ischemia or induce tamponade. Type II (pericardial or myocardial blush without contrast jet extravasation) perforations also rarely lead to unfavorable complications if treated promptly with balloon inflation. Type III perforations involving extravasation through >1 mm of perforation are more likely to lead to tamponade and therefore must be managed in an expeditious fashion. Finally, cavity-spilling perforations involve perforation into an anatomic cavity chamber or vessel.[11] We were concerned for a type III versus cavity-spilling perforation, because either would be catastrophic and would potentially require immediate surgical intervention.

Coronary-cameral fistulas are typically congenital in nature and are present in approximately 0.002% of the general population. The RCA has widely been the most reported site of fistulous origin, with 50%–60% of cases originating from this artery, 25%–42% from the left anterior descending coronary artery, and 18.3% from the left circumflex coronary artery.[3] Acquired coronary-cameral fistulas are rare and usually result from trauma, aortic dissection, chest irradiation, Takayasu arteritis, or iatrogenic injury.[12] Most fistulae are small and do not result in symptoms; however, large fistulae may have hemodynamic consequences, such as volume overload and pulmonary hypertension.[3]

The use of echocardiography contrast for the localization of coronary-cameral fistulas has been previously described.[13,14] It is important to note that this is an off-label use of echocardiography contrast and that intra-arterial injection is listed as contraindicated on the contrast product label. This simple diagnostic procedure confirmed the coronary-cameral fistula location and excluded a life-threatening complication of infarct angioplasty.

The etiology of the coronary-cameral fistula in our patient was not entirely clear. We did not have access to the patient's prior

angiography, so we were unsure if he had a known congenital fistula. The presence of coronary-to-cardiac chamber connection on imaging is not in itself diagnostic of coronary perforation. A previously present congenital coronary-cameral fistula could also result in these findings. Therefore, our differential diagnosis for possible etiologies in this patient included a congenital fistula, iatrogenic injury from the guidewire, iatrogenic postsurgical fistula related to previous bypass graft surgery, or a coronary-cameral fistula that resulted from myocardial injury related to the ongoing myocardial infarction. The site of the fistula distal to the location of our wire perforation and the large size of the fistula made us suspect that the fistula may have been present before our procedure.

CONCLUSION

Coronary artery perforation is a rare complication of PCI that may have catastrophic outcomes if not identified and managed swiftly. In some cases, such as the one presented, the initial angiographic appearance of contrast extravasation is not completely diagnostic of coronary perforation. In these complex cases, the use of supplementary imaging techniques such as contrast echocardiography and repeat rotational angiography may facilitate timely characterization of the contrast extravasation. In this case, supplementary imaging techniques supported the presence of a concomitant coronary-cameral fistula, which was managed conservatively.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2018.04.005.

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