




Giant Intramural Right Ventricular Hematoma after PCI in a Patient with Condition after CABG

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Abstract

Keywords

- ▶ cardiac catheterization/intervention
- ▶ coronary artery bypass grafting
- ▶ CABG
- ▶ myocardial injury
- ▶ reoperation
- ▶ surgery
- ▶ complications
- ▶ cardiology/cardiologist

Coronary artery perforation secondary to percutaneous coronary intervention (PCI) is a rare, but a potentially life-threatening complication. There is a misconception that cardiac tamponade rarely occurs in patients with prior coronary artery bypass grafting (CABG). We first describe a giant right ventricular intramural hematoma following PCI via a saphenous vein graft to treat a distal stenosis of the right coronary artery, and its successful treatment with redo cardiac surgery. Complex elective PCIs on patients after CABG should be performed in specialized centers with a well-established heart team that has the expertise to treat any of the potential complications.

Introduction

Coronary artery perforation (CAP) secondary to percutaneous coronary intervention (PCI) is a rare complication that occurs in ~0.4 to 0.7% of PCIs,^{1,2} with clinical tamponade developing in 50.7% of the patients and associated with an overall 30-day mortality of 10.7%.² There is a misconception that cardiac tamponade cannot occur in patients after coronary artery bypass grafting (CABG) due to postoperative adhesions. Bleeding-associated tamponade may, nevertheless, occur in various intrathoracic compartments. As a result, this opinion is gradually changing due to several reported cases.³

Here we describe an extremely rare case of a giant intramural hematoma of the right ventricle (RV) following elective PCI of the right coronary artery (RCA) via a saphenous vein graft (SVG) in a patient with a history of CABG, and its successful surgical treatment.

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Case

A 73-year-old male with three-vessel coronary artery disease and elective CABG 16 years ago (left internal thoracic artery [LITA] to left anterior descending [LAD], SVG to RCA), and PCI of the Ramus interventricularis posterior (RIVP) after acute coronary syndrome 8 years ago, presented with new-onset palpitations and progressive dyspnea. Coronary angiography demonstrated an open LITA-LAD bypass, a chronic total occlusion of the proximal native RCA, a patent SVG to medial segment 3 of the RCA, and a significant new stenosis of the crux cordis (transition from segment 3 to 4). The new high-grade stenosis in the area of the crux cordis was then treated

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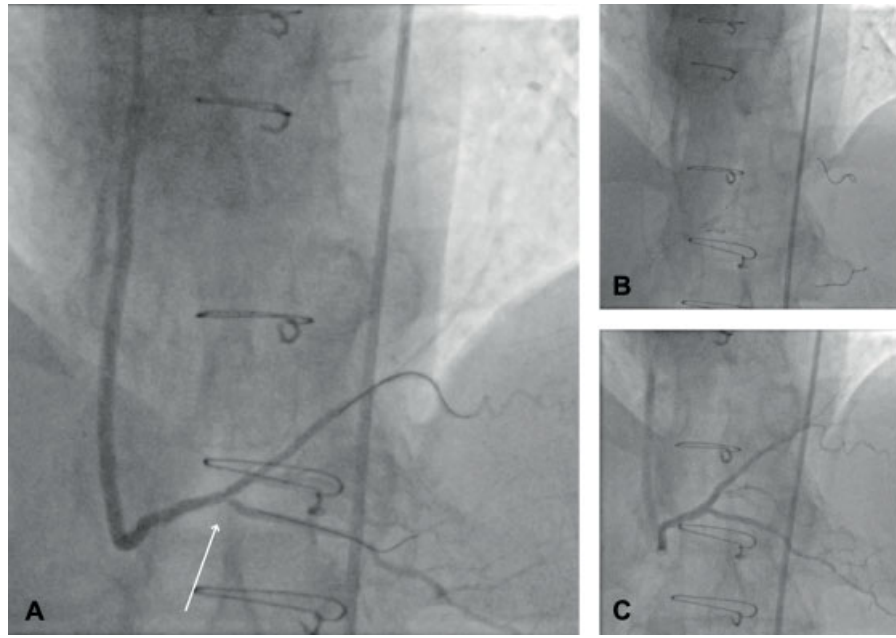


Fig. 1 Bifurcation stenting from the saphenous vein graft into the right coronary artery into the Ramus posterolateralis dexter and Ramus interventricularis posterior as mini-crush technique. Before (arrow = native bifurcation stenosis) (A), during (B), and after (C) the intervention.

electively via the patent SVG. The bifurcation intervention was performed as a mini-crush technique in the standard fashion, with a 2.25×28 mm Sirolimus-eluting stent from the RCA into the Ramus posterolateralis dexter (RPLD) and an additional 2.25×16 mm Sirolimus-eluting stent into the RIVP, whereby a good result was achieved (**► Fig. 1**). Following PCI, a dual-antiplatelet therapy (DAPT) was initiated by continuing acetylsalicylic acid (ASA) and adding a loading dose of clopidogrel. The patient was transferred to the intensive care unit for routine monitoring. Eight hours after PCI, the patient complained of new-onset chest tightness. On electrocardiography (ECG), there were ST-segment elevations indicating an early

in-stent thrombosis, which required reintervention. The bifurcation stent implanted in the crux cordis was thrombosed in the RIVP portion. This occlusion was successfully interrogated with a guidewire, and subsequently, the bifurcation stent was dilated with a 2.5×8 mm semicompliant balloon catheter in the RIVP and a 2.5×12 mm semicompliant balloon catheter in the RPLD using an alternating kissing balloon technique. Final fluoroscopy following successful reintervention revealed minimal contrast agent accumulation in the surrounding tissue via a parenchymal branch emerging from the proximal third of the native RIVP at the distal end of the stent (**► Fig. 2**), without hemodynamically significant extravasation (Ellis grad II).

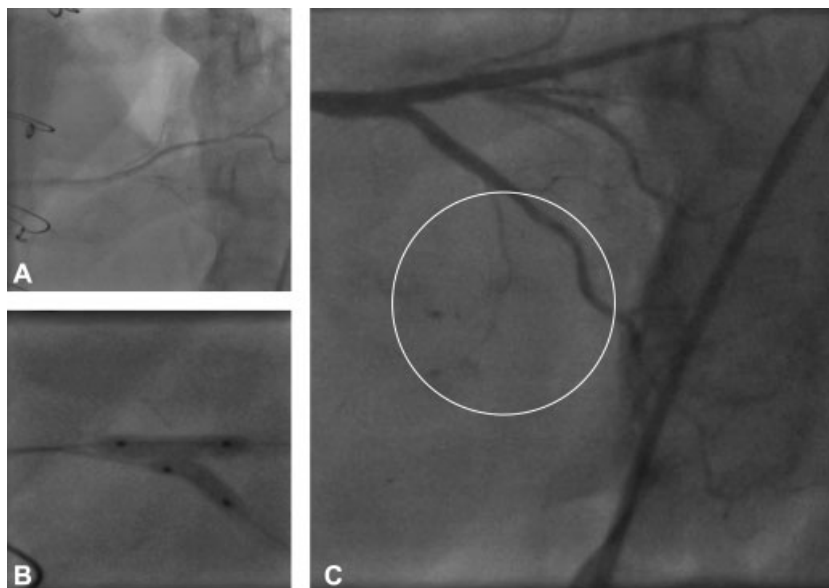


Fig. 2 Reintervention as alternating kissing balloon technique for early in-stent thrombosis. Before (A), during (B), and after (C) the intervention. Circle = contrast agent accumulation.

Following the reintervention, the patient was transferred back to the intensive care unit for monitoring and the DAPT was converted to ASA and ticagrelor. Two hours later, the patient reported recurrence of dyspnea, which was accompanied by ECG changes. Bedside echocardiography was performed and demonstrated a new mass of unclear origin adjacent to the RV resulting in near total RV obstruction. Emergency computed tomography scan revealed a giant isolated intramural RV hematoma (→ Fig. 3). The patient was taken emergently to the operating room for a reoperative cardiac procedure via a median sternotomy with cardiopulmonary bypass (CPB) standby. After exposing the groin vessels for a possible emergency CPB connection, resternotomy was performed and the anterior aspect of the heart was dissected. The entire mediastinum was hemorrhagic and densely adhered rendering identification of individual tissue planes extremely challenging. Two areas of a contained RV wall rupture were identified, dissected, and a hematoma of ~200 mL of coagulated blood was evacuated without entering the RV chamber. The RV was noted to be held together only by scar tissue from the prior surgery, and following hematoma evacuation, the right ventricular wall was noted to be remodeled as a result of the intracavitary pressure. To achieve a secure repair, the RV wall was reconstructed in a beating heart fashion, as further dissection was impossible secondary to the dense adhesions and significant hemorrhage. The small intramural cavity that developed secondary to hematoma evacuation was repaired using the native friable RV myocardium and two layers of bovine pericardial patch. This was achieved using a 4-0 polypropylene suture, which was run in a continuous fashion taking deep bites to incorporate the following deep-to-super-

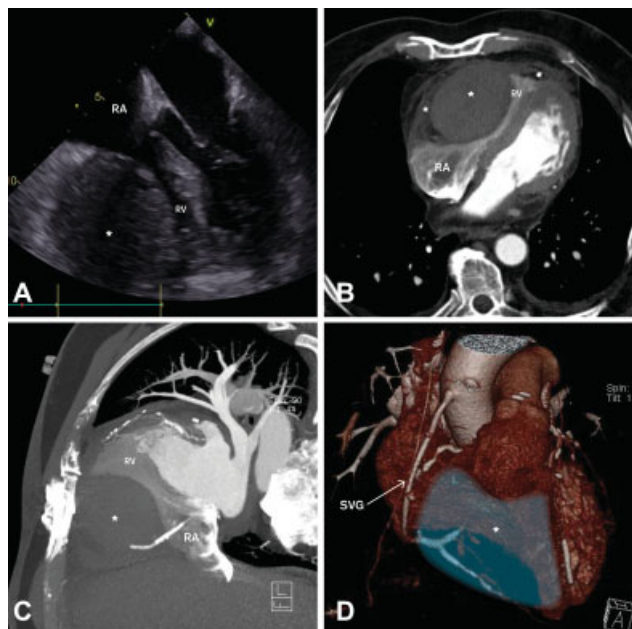


Fig. 3 Giant intramural hematoma (*) after elective percutaneous coronary intervention via a saphenous vein graft (SVG). Transesophageal echocardiography (A); computed tomography (CT) scan: axial (B), sagittal (C); three-dimensional-volumerendering CT with an artificially created area to visualize the size of the hematoma (D). RA, right atrium; RV, right ventricle.

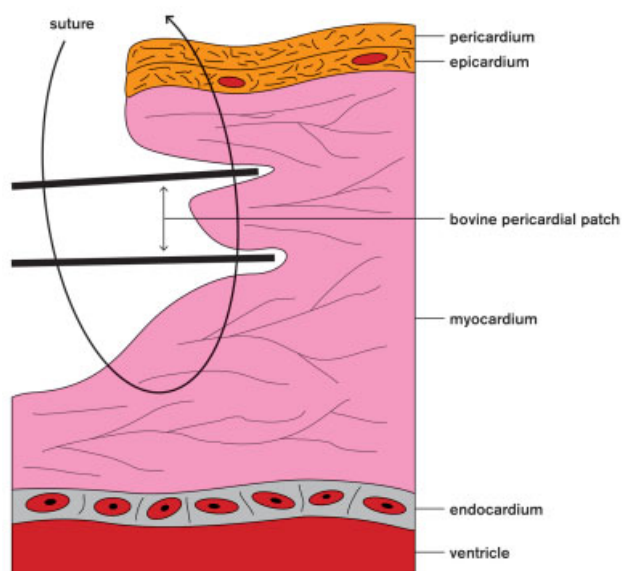


Fig. 4 Reconstruction of the right ventricular wall. Pericardium (1), epicardium (2), bovine pericardial patch (3), myocardium (4), endocardium (5), ventricle (6) and suture direction (7).

ficial layers: bovine pericardium patch, myocardium, tattered myocardium with the patch layers in between, epicardium, and pericardium (→ Fig. 4). Fibrin glue was then applied to fill the remaining cavity, which was then covered by a third bovine pericardial patch. Thereafter, the outer edges of the defect that were reinforced by the patch were sewn together with two suture lines of running 4-0 polypropylene sutures. Postoperatively, the patient developed significant hemorrhage, requiring a total of 4 units of packed red blood cells, 6 units of fresh frozen plasma, 2 units of platelets, and 1,437 mL of cell saver filtered operative field blood. The patient's in-hospital postoperative course was complicated by pneumonia, acute respiratory distress syndrome, critical-illness-myopathy and -polyneuropathy. He was discharged on postoperative week 12 from the regular cardiac surgical ward to a rehabilitation facility.

Discussion

An intramural hematoma following elective PCI in patients with previous CABG is a severe and potentially life-threatening complication.^{2,3} Previously described intramural hematomas in these patients were always located in the left atrium.^{1,4-6} In our case, we describe an intramural hematoma of RV in a patient after PCI of the RCA via a SVG.

CAP with its potential complications should not be trivialized in patients after CABG. Approximately 25% of CAPs were not detected during PCI in patients without a history of a prior CABG.² In the case of preoperated patients, almost twice as many (43%) were noticed after the intervention.³ In addition, the overall 30-day mortality rate for patients with tamponade after PCI and previous CABG is approximately twice as high as for patients who have not undergone surgery before (22 vs. 10.7%).^{2,3}

Intramural hematoma is easily detectable by echocardiography.⁴⁻⁶ Therefore, a cardiac tamponade can usually be

diagnosed by transthoracic echocardiographic examination in the intensive care unit. Depending on the individual situation, transesophageal echocardiography and/or computed tomography scan with intravenous contrast are appropriate examinations in the case that transthoracic echocardiography does not reveal any relevant information and the patient's hemodynamic status continues to deteriorate, enabling rapid detection and subsequent effective treatment by the heart team.

For this reason, in our opinion, such complex interventions should only be performed in centers with high interventional expertise. Onsite cardiac surgical service would be desirable. In addition, these patients must be monitored in an intensive care unit for at least 24 hours after the intervention, especially when a contrast agent extravasation is evident during the intervention.

Conclusion

An intramural hematoma of the RV after PCI of the RCA via an SVG can occur and may lead to a life-threatening situation. Complex elective PCIs in patients with prior CABG should only be performed in specialized centers that offer all treatment options to manage any potentially life-threatening complication and should be monitored in an intensive care unit for at least 24 hours.

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