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Published in:
Catheterization and Cardiovascular Interventions

DOI:
10.1002/ccd.30226

Publication date:
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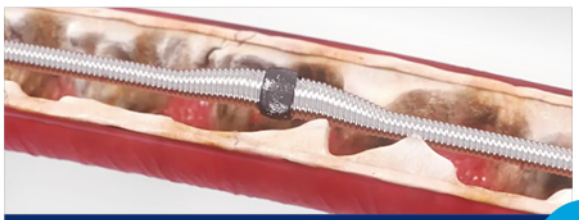

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Received: 16 March 2022 | Revised: 21 April 2022 | Accepted: 3 May 2022

DOI: 10.1002/ccd.30226

CASE REPORT

WILEY

Right ventricular hematoma: A rare but potentially fatal complication of percutaneous coronary artery intervention

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Abstract

Right ventricular hematoma secondary to coronary artery perforation during the percutaneous coronary intervention (PCI) is a rare complication. Nevertheless, with the growth of complex PCIs, including chronic total occlusion procedures, this complication may increase in frequency. We describe three cases of subepicardial right ventricular hematoma after complex right coronary artery PCI with different outcomes. Two cases were successfully managed with medication only. One case was managed with medication and pericardial drainage, unfortunately with a fatal outcome. All cases emphasize the need for awareness concerning this complication, which warrants prompt diagnosis and adequate therapy.

KEYWORDS

angioplasty, coronary artery disease, revascularisation, shock

1 | INTRODUCTION

Percutaneous coronary intervention (PCI) is one of the main treatments for coronary artery disease. Balloon or guidewire-induced coronary artery perforation secondary to PCI is a rare complication occurring in only 0.3%–0.5% of cases.[1,2] This complication is more commonly observed during PCI of complex lesions and chronic total occlusion (CTO) procedures.[1–3] Coronary perforation is associated with considerable morbidity and mortality, mainly due to the hemodynamic compromise of pericardial effusion ensuing cardiac tamponade.[1–3] In unusual cases, this perforation can result in a cardiac wall hematoma which is potentially life-threatening by inducing an acute obstructive shock. Here we report three cases of right ventricular (RV) hematoma after complex right coronary artery (RCA) PCI.

1.1 | Case 1

A 72-year-old woman with no cardiac history was evaluated for Canadian Cardiovascular Society (CCS) Class III angina pectoris. Myocardial perfusion scintigraphy demonstrated reversible ischemia in the basal and mid-inferior segments, after which an elective coronary angiography was performed. This revealed an ostial occlusion of the RCA. Multiple ad hoc attempts to cross the lesion with a wire were unsuccessful and a CTO procedure was scheduled. During this procedure, the occlusion was crossed with a hydrophilic wire (Fielder XT-A, Asahi Intecc), followed by dilatations with 1.5 and 2.5 mm balloons. Shortly after, extraluminal contrast was observed indicating a perforation of the proximal RCA (Figure 1A). Immediate transthoracic echocardiography (TTE) showed no pericardial effusion, but thickening of the RV wall with minimal compression on the right atrium and ventricle. Intravenous protamine and thrombocyte transfusion were given to reverse the effects of either heparin and acetylsalicylic acid and clopidogrel. The patient remained hemodynamically stable and therefore, the PCI procedure was stopped. One hour later progressive chest pain ensued, combined with decreasing blood pressure. Repeated TTE revealed a mass in the RV free wall, indicating an RV hematoma leading to an obstructive shock (Figure 1B). This was stabilized with intravenous (IV) fluids and vasopressors. An ad hoc heart team, including cardiologists and cardiothoracic surgeons decided to treat the patient conservatively instead of performing urgent surgery. Repeated TTE during the following days demonstrated no changes in the size of the hematoma, and the patient remained stable.

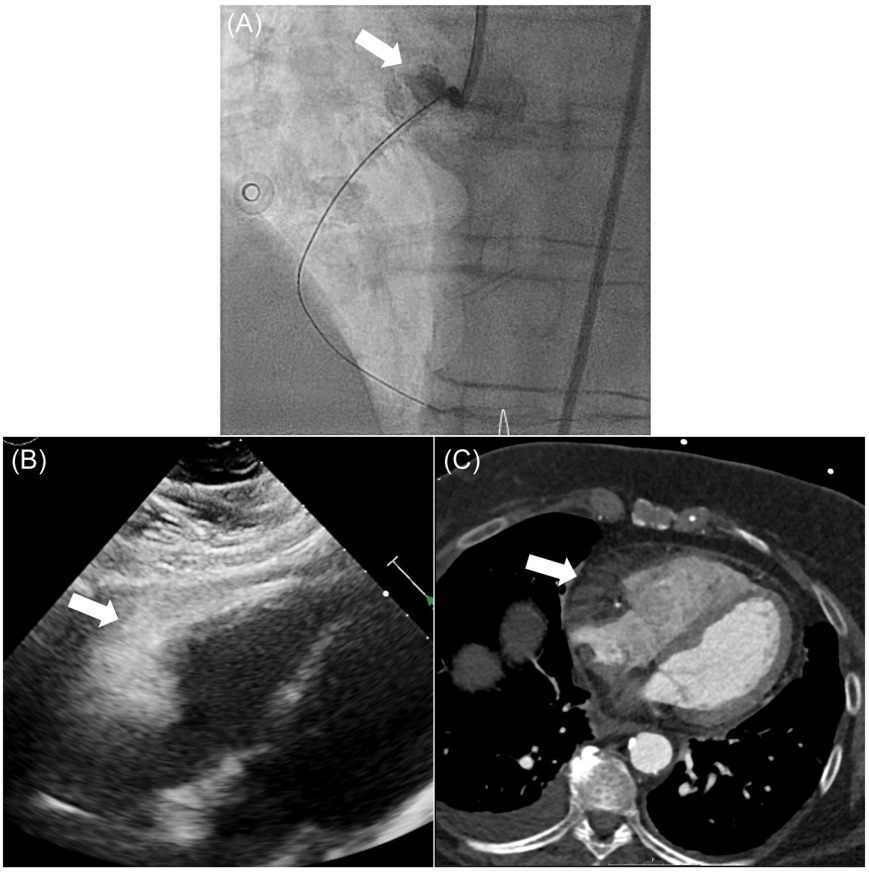

FIGURE 1 (A) Coronary angiogram revealing extravasation of contrast indicating perforation of the proximal right coronary artery. Subcostal echocardiography (B) and axial cardiac computed tomography (C) demonstrating the subepicardial hematoma in the right atrial and ventricular wall.

Cardiac computed tomography (CT) performed after 4 days confirmed the diagnosis and demonstrated a subepicardial hematoma in the right atrial and ventricular wall. (Figure 1C) The patient was discharged on the fourth postprocedural day. At 2 weeks follow-up, the patient was stable but still experienced angina pectoris for which a successful and uncomplicated CTO PCI was performed. At 3 months follow-up the patient reported no angina pectoris and was doing well.

1.2 | Case 2

A 85-year-old man with a history of CABG 7 years before, in which the left internal mammary artery (LIMA) was used as a bypass to the left anterior descending artery (LAD), was evaluated for CCS Class III angina pectoris. Coronary angiography demonstrated a patent LIMA graft, a subtotal ostial occlusion of the native RCA, and stenosis in the posterior descending artery (PDA), whereafter an elective PCI was planned. During this procedure, the lesions were crossed with a standard workhorse wire (SION, Asahi Intecc), and after predilatation with a 2.5 mm balloon, two drug-eluting stents (DES) were implanted with satisfactory results. Postprocedural angiography showed extravasation of contrast caused by distal wire perforation of the PDA (Figure 2A). Prolonged balloon inflation to seal the perforation was not successful. Emergent TTE revealed no pericardial effusion. Because of hemodynamic stability and absence of pericardial effusion, it was thought to be a perforation to another cavity (Ellis type IV). Further interventions such as coils or fat embolization were therefore not performed, and a conservative management was elected. Six hours after the procedure, the patient experienced sudden-onset chest pain. Electrocardiogram showed ST-segment elevation in the inferior leads. Repeated angiography revealed diffuse spasm of the PDA, new contrast extravasation from the RV branch, and persistent contrast extravasation from the PDA. TTE demonstrated a 5.3 × 6.5 cm RV wall hematoma with compression of the right ventricle resulting in hemodynamic instability. (Figure 2B,C) Therapeutic options were urgently discussed in a multidisciplinary heart team. Although there was rapid hemodynamic deterioration, it was decided to pursue maximal conservative management with IV fluids and vasopressors, based on the patient's age and previous cardiothoracic surgery. Heparin was balanced by protamine and thrombocyte transfusion was considered despite the risk of stent thrombosis. Even though hemodynamic instability persisted and a pericardiocentesis was performed along with the placement of two covered stents in the RCA. Despite these interventions and the administration of high dose vasopressors and IV fluids, stabilization was not achieved and the patient died soon afterward.

1.3 | Case 3

A 74-year-old man with a history of anterior myocardial infarction was admitted with angina pectoris CCS Class III. Elective coronary angiography revealed a CTO of the RCA. Magnetic resonance imaging (MRI) revealed viability of the inferior wall and it was decided to perform a PCI.

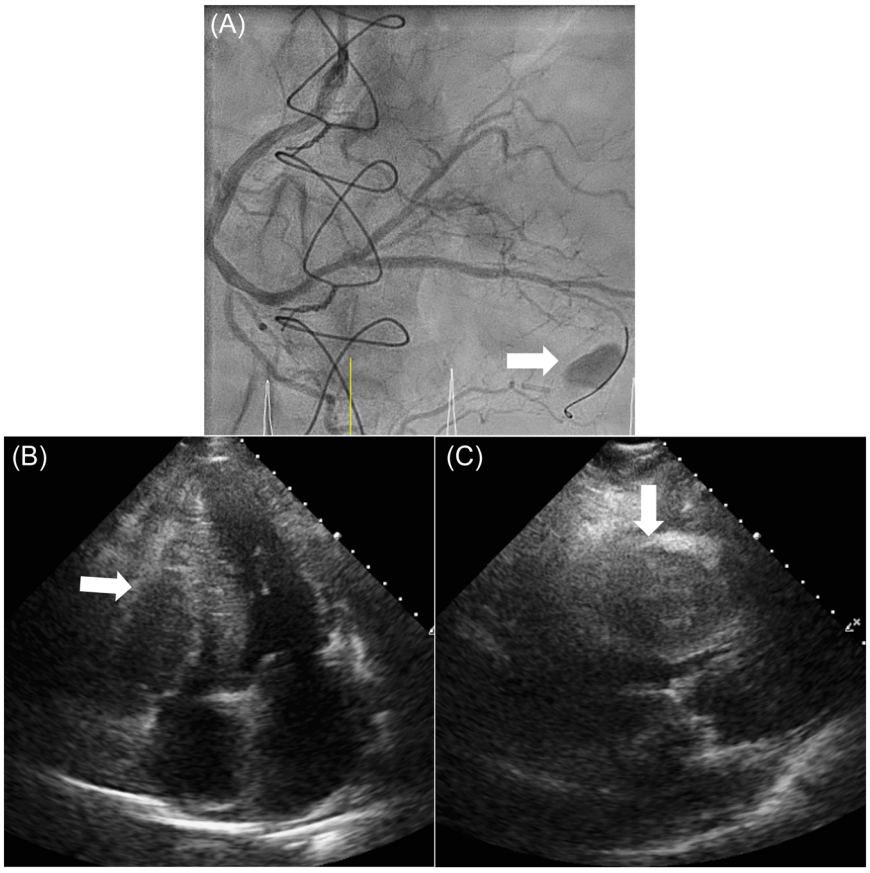

FIGURE 2 (A) Control angiogram post-PCI demonstrating contrast extravasation caused by wire perforation of the distal posterior descending artery. Apical (B) and parasternal long axis (C) echocardiographic views reveal a large right ventricular wall hematoma. PCI, percutaneous coronary intervention.

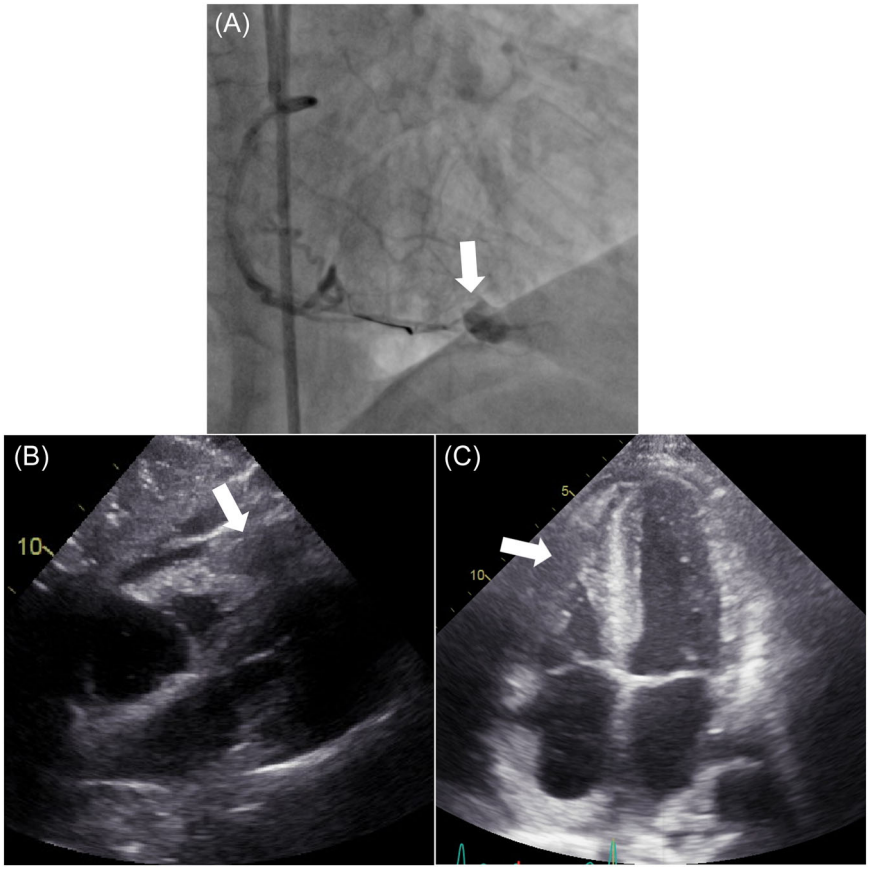

FIGURE 3 (A) Postprocedural angiogram revealing contrast extravasation from the distal PDA indicating coronary perforation. Subcostal (B) and apical (C) echocardiographic views showing the right ventricular wall hematoma.

An initial retrograde approach through collateral with a microcatheter was unsuccessful. Thereafter, an antegrade approach using wire escalation was successful and two drug-eluting stents were deployed. Control angiography demonstrated contrast extravasation from the distal PDA toward the septum (Figure 3A), but no significant pericardial effusion was detected on TTE. Due to suspicion of septal hematoma and it is often self-limiting nature, the patient was treated conservatively. After 2 hours there was a rapid hemodynamic deterioration and TTE showed mild pericardial effusion and a RV hematoma with signs of inflow obstruction resulting in obstructive shock. (Figure 3B,C) A new angiography demonstrated patency of the stents in the RCA and no contrast extravasation. Thereafter, a multidisciplinary heart team decided to pursue a conservative echo guided management. After administration of high dose vasopressors and IV fluids, hemodynamic stability was achieved and the patient remained stable. On the fifth postprocedural day the patient could be discharged, the recovery was further uneventful. A TTE performed 2-months later showed almost complete hematoma resolution.

2 | DISCUSSION

Right ventricular hematoma as a consequence of coronary artery perforation is a rare complication of PCI. This complication has various clinical outcomes as demonstrated in this case series and in previous reports.[4–8] It may be asymptomatic and self-limiting, but it may also lead to an obstructive shock due to inflow or outflow obstruction of the right ventricle with rapid circulatory collapse and sometimes even fatal outcome. Coronary perforation cannot only lead to RV hematoma, but also to left ventricular, left atrial, and septal hematomas with similar consequences.[9–12] With the increasing number of complex PCIs including CTO procedures, these complications might be inevitable and will continue or even increase in frequency.[2] Therefore, raising awareness for this complication is important.

In the first place, it is of importance to understand the location of a cardiac wall hematoma caused by coronary perforation. In previous reports, different definitions are used, such as "intramural hematoma," "intramyocardial hematoma," "subepicardial hematoma," or "localized hematoma."[4–10] On echocardiography the exact location is difficult to discriminate. Based on the CT scan in Case 1, the hematoma is localized between the visceral pericardium (=epicardium) and myocardium, in other words, "subepicardial." A previous report demonstrated an entire intramyocardial hematoma on cardiac CT and MRI after coronary perforation during RCA PCI.[4] Technically speaking both hematomas are subepicardial, so this would be the most accurate definition in our opinion. More importantly, both subepicardial and intramyocardial hematomas do not lead to pericardial effusion which develops between the visceral and parietal pericardium. Nowadays the Ellis classification is used to grade coronary perforations based on angiographic appearance. Type I indicates an extraluminal crater without extravasation, Type II indicates pericardial or myocardial blush without contrast jet extravasation, Type III represents frank extravasation with contrast jetting into the pericardial space, and Type IV (or Type III cavity spilling) indicates contrast jet extravasation into another anatomic cavity.[13] Normally, when PCI is complicated by coronary perforation, a TTE is performed to exclude pericardial effusion. However, subepicardial hematomas are caused by Type II perforations that result in blood entering directly into the myocardium or into the subepicardial space without producing pericardial effusion. Therefore, this complication could be missed by only focussing on the presence of pericardial effusion on TTE.

In the case of subepicardial hematoma post-PCI, early recognition and diagnosis are essential. This way adequate treatment can be started in time, preventing shock-based organ failure. Interventional cardiologists should be aware of this complication when PCI is complicated by coronary perforation with an active blush and without pericardial effusion detected. In this case series, all patients were initially stable directly after coronary perforation, but experienced sudden-onset chest pain and hemodynamic collapse a few hours post-PCI. Despite initial hemodynamic stability, this complication could have already been diagnosed in the catheterization laboratory with the right expertize. This emphasizes the importance of awareness concerning this complication. TTE is the first imaging modality, which should be performed acutely after every coronary perforation. It enables to distinguish between pericardial effusion and wall hematoma and focus should be on both. RV hematoma appears as a hypoechoic mass in or on the RV wall, mostly without significant pericardial effusion. Cardiac CT or MRI could clarify the exact location of the hematoma, but usually has no direct clinical consequences. However, both CT and MRI are adequate modalities to monitor the course of this complication or are useful as alternative when the patient is not suitable for echocardiography.

There are different therapeutic options to treat coronary perforation leading to a subepicardial hematoma. First, it is important to attempt to seal the perforation. This can be achieved by prolonged balloon inflation or deployment of a covered stent at the perforation site. In the case of distal coronary artery perforation, balloon inflation proximal to the perforation site or embolization of the distal vessel with thrombin, coils or autologous subcutaneous fat can be performed.[1] Progression of the hematoma can be prevented by administering protamine to reverse the effects of heparin. Thrombocyte transfusion to neutralize antiplatelet therapy, although it increases the risk of stent thrombosis, could be considered. Second, when the patient exhibits signs of shock, treatment with IV fluids should be started. If needed, this should be combined with vasopressors or inotropes. Third, if no hemodynamic stabilization can be obtained through this conservative management, more invasive techniques should be considered. Pericardiocentesis is usually ineffective because the hematoma is not located in the pericardial cavity. Urgent surgical intervention might be indicated. However, previous reports indicate that surgical evacuation of a wall hematoma is challenging and often has a poor outcome.[7–9] In case of refractory shock, a temporary mechanical circulatory support device can be applied such as percutaneous left or right ventricular assist devices or venoarterial extracorporeal life support.[11,12] Specifically

for RV hematoma, there might be a role for Impella RP to stabilize hemodynamics by pumping blood from the inferior vena cava to the pulmonary artery, thereby temporarily bypassing the compressed right ventricle. Fortunately, if conservative management is successful, complete recovery of the right ventricle may be expected as described in our case and also previously by Kajander et al.[4] During the entire diagnosis and treatment process, close monitoring and a multidisciplinary approach are essential. In this case series, all patients were discussed in an ad hoc heart team, leading to an expeditious and well-considered treatment strategy.

3 | CONCLUSION

Subepicardial hematoma as a complication of PCI is rare, but its incidence increases with the complexity of the procedure. Because complex PCIs including CTO procedures are becoming more prevalent, this complication may increase in frequency and therefore should be brought to awareness. Subepicardial hematomas develop from a coronary artery perforation with an active blush. Immediate TTE with specific focus on the ventricular wall is required to diagnose this complication. Recognition and prompt diagnosis are essential to prevent progression and irreversible hemodynamic deterioration.

ACKNOWLEDGMENTS

The authors would like to thank dr. Teeuwen, dr. van der Werf, dr. Houthuizen and dr. ter Woorst for their clinical expertize and help in the preparation of this manuscript.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

PATIENT CONSENT STATEMENT

Informed consent from patient 1 was obtained. Unfortunately patient 2 and 3 passed away before this case series was conducted.

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How to cite this article: van Hattem VAE, Otterspoor LC, Lipsic E, Vlaar P-JJJ. Right ventricular hematoma: a rare but potentially fatal complication of percutaneous coronary artery intervention. *Catheter Cardiovasc Interv*. 2022;100:100-104. doi:10.1002/ccd.30226