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CASE REPORT
Coronary heart disease

A case report of pseudoaneurysm of coronary artery within a month of percutaneous coronary intervention

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Background Coronary artery pseudoaneurysms (PSAs) are uncommon and have poorly understood natural history. Unlike true aneurysms, PSAs do not have all the three layers of the vessel in the aneurysmal wall. The PSAs are most commonly seen after an over-zealous percutaneous coronary intervention (PCI) which causes damage to the vessel wall. They usually develop slowly after PCI and PSAs within a month of a PCI are not so common. The PSA may be asymptomatic or present with recurrent angina.

Case summary Here, we report a case of symptomatic PSA to right coronary artery (RCA). The patient had a myocardial infarction for which a PCI was performed to deploy a drug-eluting stent (DES) in the RCA. The patient had in-stent restenosis (ISR) within a week of PCI for which plain old balloon angioplasty (POBA) was performed. The patient continued to have unstable angina and within a month of POBA was diagnosed as a case of PSA by intravascular ultrasound. A covered stent was deployed which effectively sealed off the PSA and resumed normal blood flow to distal vessel. Patient has been doing well on medication [aspirin 75 mg once daily, atorvastatin 80 mg once daily, and P2Y12 platelet inhibitor (Ticagrelor) 90 mg twice daily].

Discussion The PSAs usually take 6–9 months to develop. However, PSAs have been reported within 1–2 months of PCI. This case also shows that PSAs can occur within a month of PCI. It is possible that over-aggressive and/or high-pressure dilatation and/or deep engagement during POBA performed to open up the ISR could have damaged the struts of the DES and compressed it against the vascular wall. The resultant vascular wall injury could have been the cause of early PSA formation in this case. Hence, cardiologists should be vigilant enough to suspect PSA, especially in a patient presenting with angina. The case also shows that covered stents are a viable option to treat early presentations of PSA.

Keywords Pseudoaneurysm of coronary artery • Covered stent • Complex and high-risk coronary intervention • Coronary artery aneurysm • Case report

ESC curriculum 3.1 Coronary artery disease • 3.4 Coronary angiography

Learning points

- Coronary artery pseudoaneurysms (PSAs) can occur within a month of percutaneous coronary intervention (PCI).
- Treating physicians should suspect PSA in a patient complaining of angina after recurrent PCI.
- Polytetrafluorethylene (PTFE)-covered stents are a viable option to treat early presenting PSAs.

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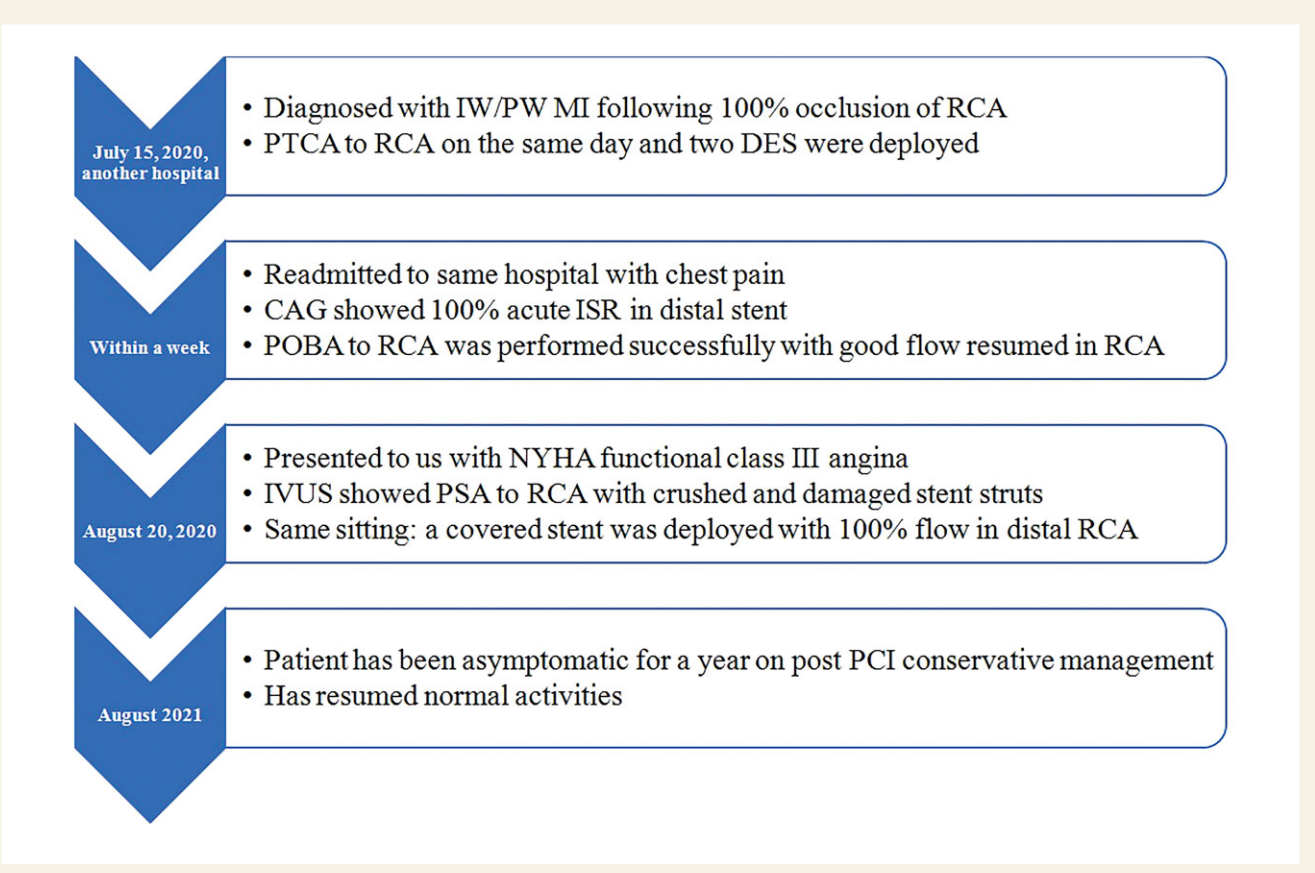
Introduction

Coronary artery aneurysm (CAA) is a focal dilatation that is >1.5-times the normal size of the adjacent coronary artery segment.[1,2] The CAAs are classified into two types, true aneurysms and false or pseudoaneurysms (PSAs), based on the presence or absence of all the three coronary wall layers (intima, media, and adventitia) in the aneurysm.[3–5] Since PSAs usually have an adverse outcome, it is very important to differentiate between true and false aneurysms.[5]

Timeline



Coronary artery PSAs have an incidence of 0.3–6% after any percutaneous coronary intervention (PCI)[2,4] and 0.2–2.3% post-deployment of drug-eluting stent (DES).[4,6] The PSAs are most common in right coronary artery (RCA; 40.4%).[2]

The PSAs usually take 6–9 months to develop; however, PSA formation as early as 2 months after PCI has also been reported.[2,4,7,8] Here, we report the case of a patient who underwent percutaneous transluminal coronary angioplasty (PTCA) for inferior wall/posterior wall myocardial infarction (MI) and developed an in-stent restenosis (ISR) within a week. The patient then underwent plain old balloon angioplasty (POBA), but continued to have angina and a PSA was detected within a month of POBA during routine investigations for angina.

Case presentation

A 62-year-old male, with a history of chronic smoking and hypertension, presented with chest pain radiating to left arm at another hospital on 15 July 2020. Electrocardigram (ECG) showed inferior wall/posterior wall MI according to patient's discharge records. His coronary artery angiography (CAG) showed 100% occlusion of RCA (*Figure 1*). Patient underwent PTCA to RCA on the same day, and two cobalt–chromium DESs were deployed in the RCA. The patient was re-admitted to same hospital after 1 week of PTCA with chest pain. The ECG was suggestive of ST elevation according to the patient's discharge records. The CAG showed 100% acute ISR in distal stent. The POBA to RCA was performed in that hospital with thrombolysis in MI (TIMI)[9] Grade III flow.

However, in less than 1 month of POBA, the patient developed recurrent chest pain and presented at our hospital with The Canadian Cardiovascular Society (CCS) functional Class IV angina[10] (angina presenting with minimal activity; comfortable only at rest). On examination, his blood pressure was 120/80 mmHg, pulse was 72 b.p.m., and on auscultation of chest, S1 and S2 heart sounds were normally heard, there were no murmurs or other cardiac sounds; normal breath sounds. There were no signs of heart failure. The ECG did not show any dynamic changes. The patient had no history of fever and his total leucocyte count was normal at 7500 cells/μL (range 4000–11 000 cells/μL). Other inflammatory markers like

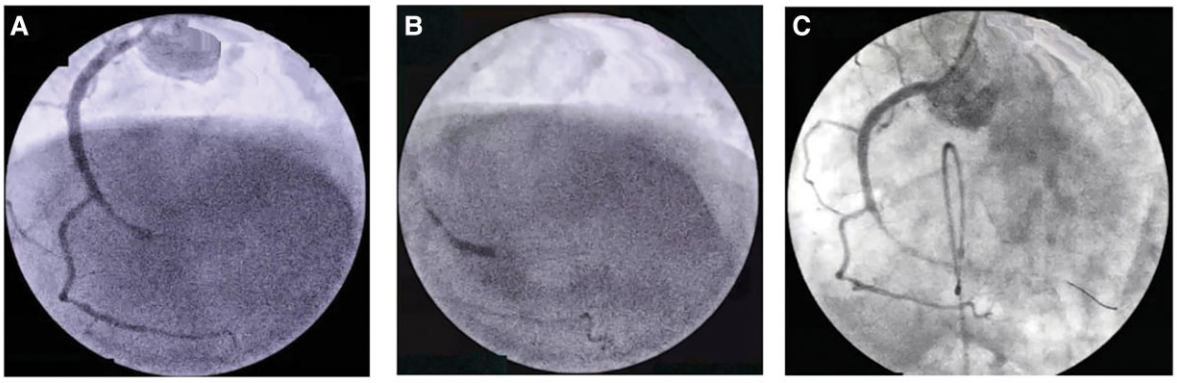


Figure 1 Initial angiogram showing 70% thrombus containing lesion followed by 100% occlusion of right coronary artery.

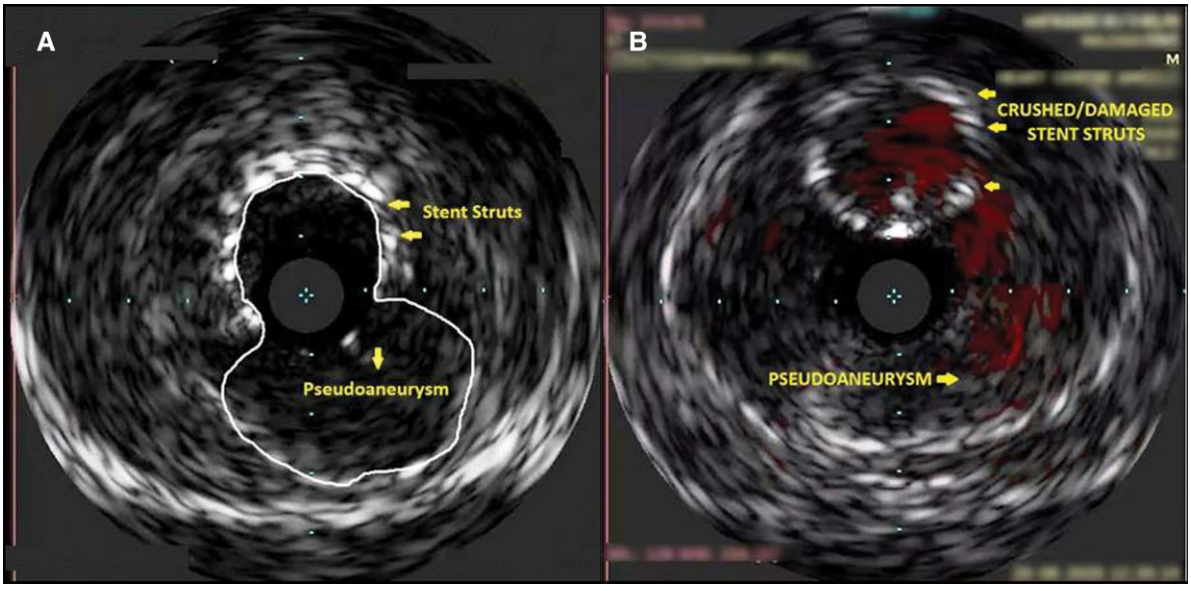


Figure 2 (*A*) Internal view of pseudoaneurysm and stent struts. (*B*) Internal view of pseudoaneurysm and crushed/damaged stent struts.

erythrocyte sedimentation rate (at 8 mm/h) and C-reactive protein (at 2 mg/L) were all normal. Cardiac enzymes were within normal limit: creatinine phosphokinase (CPK) 130 IU/L (range: 20–200 IU/L); CPK-MB 15 IU/L (range: 5–25 IU/L); Troponin T was negative (quantitative analysis is not done at our centre).

We did an intravascular ultrasound (IVUS) on 20 August 2020 to find the cause of angina. The IVUS showed crushed and damaged stent struts compressed to one side of the wall and PSA to adjacent RCA segment (*Figure 2A,B*). A polytetrafluorethylene (PTFE)-covered stent was deployed which effectively sealed off the PSA and resumed good TIMI Grade III blood flow to distal RCA (*Figure 3A,B*).

The post-PTCA stay in hospital was uneventful. The patient did not have symptoms of angina post-covered stent to date and is being managed on aspirin 75 mg once daily, atorvastatin 80 mg once daily, and P2Y12 platelet inhibitor (Ticagrelor) 90 mg twice daily until the date of this publication. Additionally, the patient continued on his prescribed anti-hypertensive medications (an angiotensin-converting enzyme inhibitor and a beta-blocker) with good blood pressure control.

Discussion

We present a case of Type I PSA that follows an acute course and forms within a month of PCI.[4] Vascular injury during PCI can be caused by atherosclerosis, atheroma burden, slow dissolution of thrombotic pieces lodged in stent struts, necrosis or apoptosis of vascular wall, or anatomical anomalies of coronary artery.[2,4,5] Procedural factors include catheter type used, improperly placed catheter, stent malposition, dissection of stent edge, rapid injection of contrast agent, deep engagement during POBA, or spontaneous dissection not visible during CAG.[2,4,5] The DES deployment further

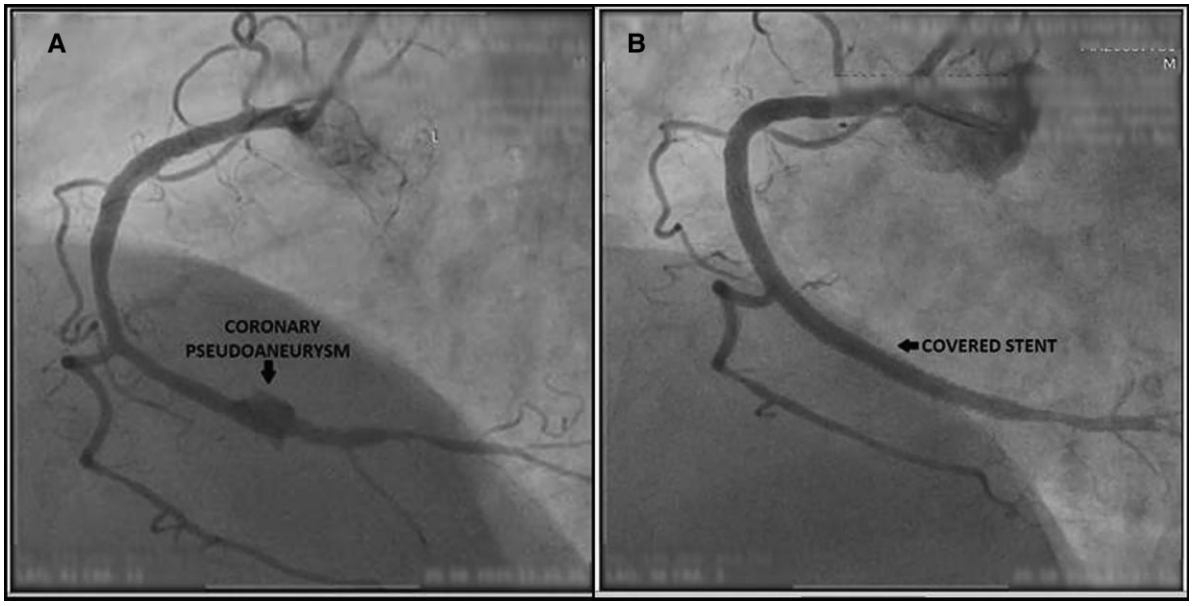


Figure 3 (*A*) Angiogram with the pseudoaneurysm before treatment. (*B*) Angiogram after covered stent was deployed and normal blood flow resumed.

prevents or delays vascular healing due to inhibition of neointimal hyperplasia and induction of hypersensitivity reactions in the coronary artery endothelium.[2,4] In this case, the early PSA formation could have resulted from vascular injury caused by over-aggressive and/or high-pressure dilatation and/or deep engagement during POBA. As in this case, PSAs are usually discovered while assessing recurrent post-PCI angina.[5] Assessment methods usually include CAG and IVUS. Of the two, IVUS is more precise in diagnosing PSA than CAG as the latter provides only luminal information and does not throw light on wall conditions.[4,5] Hence, we used IVUS to rule out PSA. When we performed IVUS, the PSA showed all the characteristic findings seen on IVUS. The PSA appeared as a large, saccular, thin-walled aneurysm communicating with the arterial lumen through a ruptured arterial wall.[11] The stent was crushed and pressed to one side causing an aneurysm on the opposite side (*Figure 2B*). There was an abrupt and angled transition from a three-layer vessel wall to a mono-layer vessel wall.[3] Along with the loss of integrity of the vessel wall, the adventia and perivascular tissue was damaged.[3]

The actual course of PSAs is largely unknown. Its pathophysiology points towards inflammation, endothelial dysfunction, platelet aggregation, and adhesion, and can therefore trigger thrombosis with distal embolization.[12] Untreated PSA can rupture, cause cardiac tamponade, or can become infected.[12] The PSA is more likely to rupture in patients ≥40 years of age, or in those presenting with angina, infective endocarditis, congestive heart failure, or distal embolization.[2] Therefore, risk of rupture was high in our patient who was 62 years old and had CCS. Class IV angina. The PSAs are unlikely to resolve without prompt treatment, especially in symptomatic patients (i.e. those with angina as seen the presented case). Untreated PSAs in symptomatic patients can cause complications.[12] Hence, it is very important to timely diagnose and treat PSAs.

There is no standard treatment guideline for PSAs occurring after a PCI.[5,12] Conservative observational approach with pharmacological therapy (anti-platelets, anti-coagulants, calcium channel blockers, or long acting nitrates and/or beta-blockers) may be used for asymptomatic patients with no evidence of angina/myocardial ischemia on biochemical and ECG parameters.[2] Symptomatic PSAs may be treated by deploying PTFE-covered stents, Papyrus-covered stents, pericardial-covered stents, vein graft-coated stents, bare metal stents (BMSs), coil embolization, or coronary artery bypass grafting and aneurysm ligation.[6,13] The use of stents is postulated to reduce blood flow into the PSA and eventually close it through neointimal proliferation and thrombus formation within the struts.[6] Papyrus-covered stents are not available in India and hence, we used a PTFE-covered stent.

The PTFE-covered metal stents are easy to deploy in PSAs formed after DES implantation and are therefore emerging as a viable option in this scenario.[14] The PTFE-covered metal stent is a single-layered stent that can be rolled into a thin multi-layer cover, further expanded to four to five times its original diameter without causing laceration and with no likelihood of shrinkage.[14] Another advantage is that the negative charge on the stent polymer prevents platelet aggregation and thrombus formation.[14]

The clinical outcomes of PTFE-covered stents may be same as those of BMS, and PTFEs may not be superior to BMS in preventing early thrombosis and restenosis.[14] However, further trials and evidence are required to conclusively prove these points, especially since PTFE-covered stents have many advantages in the treatment of PSA.[14]

We used a PTFE-covered stent because of its advantages, especially in a non-flow limiting PSA formed after DES deployment. We were able to achieve 100% flow in distal RCA. Patient has been doing well on pharmacological management (aspirin, statin, and anti-platelet) with no symptoms since then.

Patient's perspective

Patient's perspective is important in this case because of the trauma of undergoing multiple PCIs in a short span of time. The patient was initially apprehensive because he was being asked to undergo a third procedure within a very short span. We counselled the patient and explained all possible scenarios and treatment options. He understood the need for undergoing IVUS and subsequent PTFE-covered stent, and gave written informed consent for the procedure in good faith. On follow-up visits, patient was free of angina. He resumed normal activities with no chest pain or discomfort.

Conclusion

We present a case of a PSA, detected within a month of POBA, treated effectively with a covered stent, and doing well since then on pharmacological management. Over-aggressive and/or high-pressure dilatation and/or deep engagement during POBA could be a possible explanation for early PSA formation in this patient. The case also shows that PTFE-covered stents are a viable option to treat early presentations of PSA.

Lead author biography

Dr Raghav Sharma is the main treating cardiologist of the case and was assisted by Dr Aditya Vikram Ruia during the case management. Both the cardiologists have jointly written the case report in discussion with each other.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

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All named authors for this manuscript meet the International Committee of Medical Journal Editors (ICMJE) criteria for authorship. All authors take full responsibility for the integrity of the work and have given final approval for the published version.

Slide set: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary material.

Consent: Written informed consent was taken from the patient for publication of this case including images and associated anonymous text for promoting scientific knowledge, and in line with COPE 5 guidance.

Ethics approval: No ethical compliance was required.

Conflict of interest: None declared.

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