

Progression and interventional therapy of a coronary pseudoaneurysm: a case report

Svante Gersch 1, Hassina Baraki 12, and Karl Toischer 12*

¹Department of Cardiology and Pneumology, University Medical School Goettingen, Robert-Koch-Str. 40, 37075 Göttingen, Germany; and ²Department of Cardiovascular and Thoracic Surgery, University Medical School Goettingen, Robert-Koch-Str. 40, 37075 Göttingen, Germany

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Background	Coronary pseudoaneurysms (PSAs) occur as a rare complication following drug-eluting stent implantation and have been reported to occur between 1 week and 4 years after implantation. Most of them remain in a stable state, but progression of PSAs increases the risk of rupture and haemorrhagic cardiac tamponade.
Case summary	Here, we present a case of a 55-year-old patient, who developed a PSA of the proximal left circumflex artery after stent implant- ation of the left main artery, left anterior descending artery, and left circumflex artery. Within <1 year, the patient was readmitted to different hospitals due to cardiac decompensation and myocardial infarction. Thereafter, coronary angiography and computed tomography scans were performed, and progression of the PSA could be documented. Interventional therapy was chosen due to the high surgical risk of the patient. Implantation of a covered stent from the left main artery into the left anterior descending artery was chosen to treat the PSA, thereby silencing the chronically occluded left circumflex artery, followed by dilatation with a non- compliant balloon. The patient has remained asymptomatic in a 6-month follow-up.
Discussion	Coronary PSA should be controlled with respect to progression, and appropriate therapy can be chosen for treatment.
Keywords	Case report • Coronary artery disease • Pseudoaneurysm • Interventional therapy • Heart failure
ESC Curriculum	3.1 Coronary artery disease • 3.2 Acute coronary syndrome • 3.3 Chronic coronary syndrome • 3.4 Coronary angiography • 7.3 Critically ill cardiac patient

Learning points

- Coronary pseudoaneurysms (PSAs) are a rare complication following drug-eluting stent implantation and can become clinically relevant.
- Therapeutic decisions of PSAs closure should always be made on an individual basis taking into account pre-existing diseases and the coronary disease status.

Introduction

Coronary pseudoaneurysms (PSAs) have been described as a rare complication following drug-eluting stent (DES) or bare-metal stent implantation.^{1,2} Therapeutic measures comprise either a

surgical approach or an endovascular approach. The endovascular approach involves coil embolization or implantation of a covered stent. This case report addresses the clinical case, diagnosis, and interventional therapy of a fast-progressive PSA after DES implantation.

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^{*} Corresponding author. Tel: +49 551 3966318, Email: ktoischer@med.uni-goettingen.de

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Timeline

February	Coronary angiography of the right coronary artery with
2021	drug-eluting balloon implantation
April 2021	Coronary angiography with culotte stenting of bifurcation
	stenosis affecting the left main artery, left anterior
	descending artery, and left circumflex artery
May 2021	Admission to hospital due to chest pain, elevated troponin;
	no need for interventional therapy, a small
	pseudoaneurysm behind the left circumflex stent was
	detected.
June 2021	Computer tomography (CT), showing little progress of
	aneurysm
July 2021	CT and invasive proof of fast progression of the aneurysm.
	Emergency interventional occlusion via covered
	stenting of the left main artery

Case report

We report the case of a 55-year-old male with progressive coronary PSA after routine DES implantation in an external hospital in May 2021. The patient's medical record included coronary artery disease (CAD) with multiple stentings of the right coronary artery, ramus circumflexus (LCx), ramus interventricularis anterior (LAD), and the left main coronary artery (LMCA), an ischaemic cardiomyopathy with left ventricular reduced ejection fraction (40%), severe peripheral arterial disease with occlusions of the pelvic arteries, chronic kidney disease (Stage 3b, eGFR 33 mL/min/1.73), and furthermore, a broad spectrum of cardiovascular risk factors such as non-insulin-dependent diabetes mellitus Type 2 (current HbA1c 6.8%), arterial hypertension, and hypercholesterinaemia. The home medication included dual antiplatelet therapy with aspirin and clopidogrel, heart failure medication with an angiotensin-converting-enzyme inhibitor, beta-blocker, an aldosterone antagonist, and an antidiabetic therapy with metformin and sitagliptin as well as a statin.

In April 2021, a coronary intervention with stent implantation in the LMCA, LAD, and LCx (culotte-stenting) due to high-gradient bifurcation stenosis was performed in another clinic. No PSA was detected before or after percutaneous coronary intervention (*Figure 1*; Supplementary material online, *Videos S1* and *S2*).

In May 2021, another coronary angiography was carried out in an external clinic due to chest pain and an elevation of troponin, suggesting a progression of the patient's coronary heart disease. Here, a small PSA in the proximal LCx behind the stent was detected. At that time, there was no need for a therapeutic intervention (*Figure 2A*; Supplementary material online, *Video S1*). Afterwards, the patient developed a groin haematoma, which needed surgical intervention.

In June 2021, the patient was readmitted to the external clinic due to *Staphylococcus aureus* bacteraemia, most likely caused by a femoral puncture, with subsequent spondylodiscitis without the need for surgical intervention. An anti-infective therapy with flucloxacillin was initiated. The overview computed tomography (CT) at admission showed the progression of his coronary PSA (*Figure 2B*). During hospitalization, the patient showed signs of cardiac decompensation with bilateral pleural effusions, followed by respiratory insufficiency, which led to intermittent intubation and the need for catecholamine therapy. Acute left ventricular heart failure and a worsening of the ejection fraction down to 30% in the echocardiography led to the suspicion of a progressing CAD, and a new invasive coronary imaging was indicated in the external clinic. The intervention revealed a progression in the size of the known coronary PSA (*Figure 3B* and *C*, Supplementary material online, *Video* S2) and LCx in-stent occlusion. In CT, the LCx PSA measured $40 \times 33 \times 24$ mm (*Figure 3A*).

The patient was referred to our hospital for evaluation of an emergency surgery. Due to multiple pre-existing diseases (*S. aureus* bacteraemia, cardiac decompensation with EF 30% with the need for catecholamine therapy, suspected bladder cancer with active bleeding, and his distinct peripheral artery disease with high-gradient stenosis of iliac arteries), an interdisciplinary heart team made up of interventional cardiologists and cardiac surgeons estimated that the intraoperative risk for the patient was too high. Therefore, this interdisciplinary conference agreed on an interventional procedure rather than a surgical approach. Since the proximal occlusion of the LCx was chronic, there was no indication for recanalization. A coronary angiography with brachial access was performed. Radial access was not possible, and femoral access was not chosen due to peripheral artery disease, haematoma from the previous coronary angiography. The guide catheter was

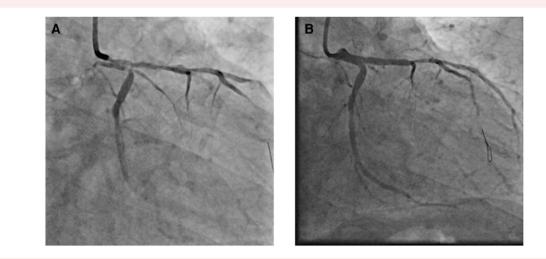


Figure 1 (A) Coronary angiography revealed a significant stenosis of the LAD–LCx bifurcation. (B) Final view after culotte stenting.

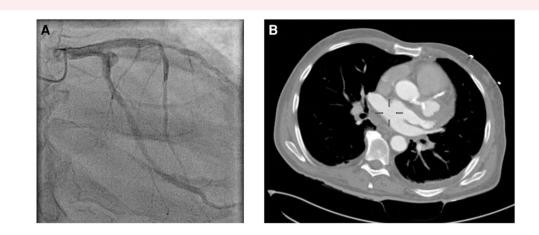


Figure 2 (A) A coronary angiography illustrates a small aneurysm after culotte stenting in May 2021. (B) Upon admission in June 2021, computed tomography shows the aneurysm of the LCx.

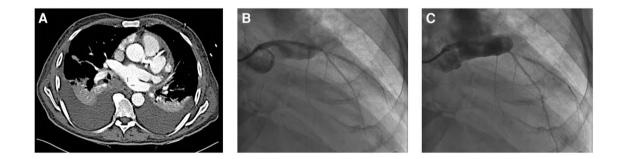


Figure 3 (A) In July 2021, computed tomography showed a progression of the aneurysm, measured $40 \times 33 \times 24$ mm. (B and C) Invasive coronary angiography shows the progression of the aneurysm preinterventionally.

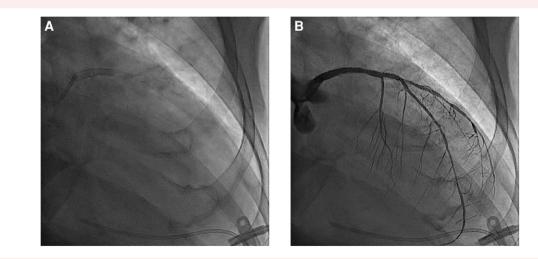


Figure 4 (A and B) Coronary angiography displays the main steam with a covered stent, silencing the chronically occluded LCx and the aneurysm.

placed in the left main stem ostium, and a covered stent (Graftmaster 2.8/19 mm) could be implanted from the LMCA into the LAD. After post-implantation dilatation with non-compliant balloons (NC Quantum 4.5/12 mm" in LMCA and NC Quantum 3.5/12 mm in the LAD), a complete sealing of the coronary PSA could be achieved (*Figure 4*; Supplementary material online, *Video S3*). Finally, the ostium of the left main stem was treated with a drug-eluting balloon catheter (SeQuent Please NEO 4.0/20 mm). Intravascular ultrasound confirmed the successful closure of the LCx (see Supplementary material online, *Video S3*).

Initially elevated troponin levels were declining the postinterventional period, and the patient's dyspnoea improved after a forced diuretic therapy. After the implantation of a covered stent, a prolonged triple therapy consisting of aspirin 100 mg, clopidogrel 75 mg, and apixaban 2.5 mg bid for 3 months (due to the newly diagnosed atrial fibrillation), followed by 9 months of dual therapy of clopidogrel 75 mg and apixaban 2.5 mg, was recommended³. A relocation of the cardiopulmonary-stable patient for further treatment to the referring hospital was possible 2 days after the intervention. The patient has remained asymptomatic in a 6-month follow-up.

Discussion

Coronary aneurysms are commonly based on arteriosclerotic changes and normally develop due to the destruction of arterial media, thinning of the arterial wall, increased wall stress, and progressive dilatation of the coronary artery segment. Pseudoaneurysms are also a very rare complication after DES implantation, which, in our opinion, are the most likely underlying mechanisms in this patient's case report.^{1,2,4} They have been described to occur between 1 week and 4 years after implantation, while a reliable estimate for the rupture incidence is currently lacking.⁴ The mechanisms of PSAs following DES include traumatic deep-vessel injury of the arterial wall with perforation of at least one vascular layer (intima and/or media), delayed re-endothelialization, or mediating inflammatory changes of the medial wall.^{5,6} We postulate that the bacteraemia and the progression of PSA occurred incidentally in this case. Since PSA progression already presented itself before the bacteraemia and no infection developed after PSA closure, it is more likely that the progression of the PSA was due to haemodynamic stress and not due to infection.

In this presented case, the PSA showed progress over 3 months with no necessity for intervention in May 21. Theoretically, two possible mechanisms seem reasonable: It can be discussed whether progression of the PSA is due to closure of the LCX and thereby increased pressure on the PSA entry or whether—vice versa—the progress of the PSA leads to stent thrombosis/in-stent restenosis of the LCX.⁷

The presented PSA can be classified according to Díaz-Zamudio et al. as a fusiform giant PSA and should be treated due to the rupture risk.⁸ In principle, four treatment options were conceivable: (i) a surgical approach with coronary artery bypass followed by ligation or resection of the aneurysm,⁹ (ii) a coil embolization,¹⁰ (iii) an interventional approach by applying a covered stent,¹¹ and (iv) conservative or at least medical treatment.¹² Because of the rarity of the cases and as a lack of outcome data comparing the possible options with one another, no standardized management has been established, and the therapy is mainly decided on an individual basis. For all four options, successful approaches have been described. In this case, we ruled out a conservative treatment because of the fast, radiologically observed progression with a high risk of rupture and did not consider the surgical option due to the high operative risk. Coil embolization would have been challenging, on the one hand, due to the size of the PSA, and on the other hand, in case of coil migration—a rare complication—LMCA and LAD would have been at risk.¹³ Alternative covered stent implantation can be used for PSA treatment.^{14,15} Since LCx was chronically occluded and a recanalization was not indicated, we decided to implant a covered stent over the LCx ostium with complete closure of the PSA.

Collectively, in this case, we could show a timeline of PSA progression and an interventional strategy for PSA closure. Therapeutic decisions of PSA closure should always be made on an individual basis considering pre-existing diseases and the coronary disease status.

Lead author biography



Svante Gersch is a third-year resident at the Department of Cardiology and Pneumology at the University Medical Center of Göttingen.

Supplementary material

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

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

Consent: The authors confirm that consent for submission and publication of this case report has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

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