ABSTRACT: Coronary pseudoaneurysm after bare-metal stent implantation is a rare event. Observation, surgical resection, or implantation of another stent, bare or covered, are alternative and equivalent management options. Since no option prevails over the other, the most appropriate treatment should be evaluated in every single patient. We report the case of a pseudoaneurysm within a stent with diffuse restenosis, treated with implantation of a pericardium-covered stent, followed by postdilation with a paclitaxel-eluting balloon.

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Coronary pseudoaneurysm after bare-metal stent implantation is a rare event, occurring in 0.3%-3.9% of percutaneous coronary interventions.1 The implantation of a polytetrafluoroethylene (PTFE)-covered stent is a possible strategy to completely seal the pseudoaneurysm, but it is limited by the impaired navigability of this device and by a high restenosis rate (with restenosis occurring mainly at the stent edges).2 We report the case of a pseudoaneurysm arising from the proximal part of a stent with diffuse restenosis, treated with implantation of a pericardium-covered stent (PCS), followed by postdilation with a paclitaxel-eluting balloon.

Case Report. A 62-year-old woman with hypertension and hypercholesterolemia as coronary risk factors was admitted to our hospital on November 12, 2010 due to a non-ST elevation myocardial infarction (NSTEMI). Coronary angiography showed a 90% stenosis of the first marginal branch (Figure 1A), which was treated with implantation of a 2.75 x 25 mm bare-metal stent (Coroflex Blue; Braun) inflated at 14 atm (Figure 1B). The postprocedural course was uneventful. In August 2011, the patient started to complain of angina on effort. Stress electrocardiography showed signs of subendocardial ischemia in leads V3-V6 at a 100 Watt load. On August
A control coronary angiography yielded a diffuse, subocclusive in-stent restenosis of the first marginal branch with extraluminal extravasation of dye in the proximal part of the stent, suggestive of a pseudoaneurysm (Figures 1C and 1G). After administration of unfractionated heparin (5000 IU ia), the left coronary artery was selectively cannulated with a 6 Fr EBU 3.5 catheter (Zuma 2; Medtronic). The in-stent lesion of the left circumflex artery was crossed with a 0.014˝ Balance Middleweight guidewire (Abbott Vascular), which was exchanged for a Balance Heavyweight extra-support wire (Abbott Vascular). Three predilations with increased diameter balloon catheters were performed; the final balloon was a 2.5 x 20 mm Maverick (Boston Scientific) inflated at 14 atm. A 2.5 x 18 mm equine PCS (AneuGraft; ITGI Medical) was subsequently implanted at 12 atm in the first marginal branch, overlapping the previously deployed stent. Finally, a postdilation with a 2.75 x 26 mm paclitaxel-eluting balloon (Sequent Please; Braun) at 12 atm was performed. Final angiography showed the sealing of the pseudoaneurysm, no significant residual in-stent stenosis, and TIMI 3 flow (Figure 1D). A postprocedural optical coherence tomography (C7-XR; St Jude Medical) was performed, showing symmetrical stent expansion, without malapposition, and a minimal stent area (MSA) of 4.14 mm² in the proximal part of the stent.
The patient was discharged after 2 days on double-antiplatelet therapy with clopidogrel (75 mg/day) and aspirin (100 mg/day), to be continued for at least 12 months, and she remained henceforth asymptomatic. On April 5, 2012, a follow-up coronary angiography was performed, showing the persistent sealing of the pseudoaneurysm, and a moderate focal intimal hyperplasia in the proximal part of the PCS, leading to a 45% stenosis (Figures 1E and 1H) shown by quantitative angiographic analysis (QAngio XA; Medis). Optical coherence tomography was also repeated, showing a diffuse intimal hyperplasia and an MSA of 1.34 mm² in the proximal part of the stent (Figure 1F). Since no test of inducible ischemia was available, a measurement of the fractional flow reserve (FFR) in the marginal branch was performed in order to assess the hemodynamic relevance of the lesion and the need for revascularization. A 0.014” pressure wire (Certus; St Jude Medical) was positioned in the distal marginal branch and maximal hyperemia was induced by intravenous administration of adenosine (140 μg/kg/min) through a 6 Fr sheath in the right femoral vein. The FFR result was 0.87, showing no inducible ischemia and warranting no further interventions. At a telephone follow-up at 12 months (in August 2012), the patient was still free of angina and ischemic events.

Discussion. The treatment of coronary pseudoaneurysm occurring after stent implantation is not universal and different therapeutic options were available for this case. Medical therapy and observation were denied, as the patient was symptomatic, with reported inducible ischemia, and the concern for a possible rupture of the pseudoaneurysm was justified. Surgical treatment was excluded after discussion with the cardiac surgeon, since a single-vessel disease, without involvement of the left anterior descending coronary artery, would have not deserved a surgical revascularization if a percutaneous option were available. The percutaneous treatment had to fulfill two different needs, ie, the sealing of the pseudoaneurysm and the treatment of the diffuse in-stent restenosis. The implantation of another bare-metal stent has shown to be effective in reducing the size of pseudoaneurysms occurring after stent implantation, although without complete disappearance.³ Vein-graft coated and PTFE-covered stents have also been successfully used for complete sealing of coronary aneurysms following bare-metal stent implantation.⁴⁻⁶ However, PTFE-covered stents have two drawbacks: a low trackability, due to the bulky profile produced by a sandwich-like structure, and a high rate of restenosis, occurring in roughly 30% of the cases, mainly at the stent edges. In the cases reported in the literature, the procedural success of PTFE-covered stents was enabled by a large (>3.0 mm) vessel diameter and by the lack of in-stent intimal hyperplasia; furthermore, in these reports, no data about the long-term patency of covered stents are available. In our case, the implantation of a PTFE-covered stent was impossible due to the small vessel diameter (a 2.75 mm stent had been implanted during the index procedure, whereas 3.0 mm is the lowest diameter available for a PTFE-covered stent). Moreover, the advancement of the stent to the lesion would have been hampered by the vessel tortuosity and by the severe in-stent intimal hyperplasia. We decided, therefore, to use an equine PCS, which has improved deliverability compared to PTFE-covered stents, due to its single-layer structure.⁷ In order to facilitate the navigability, a stent that was shorter in length than the previously implanted stent, but sufficient to seal the aneurysm, was intentionally chosen. The effective treatment of the diffuse in-stent restenosis and the prevention of a recurrence would have required the implantation of a drug-eluting stent. The overlapping implantation of a drug-eluting stent, before or after PTFE-covered stent implantation, is a strategy associated with a reduced restenosis rate.⁸ In this specific setting, however, the implantation of a third overlapping stent would have been a challenging task due to tricky navigation in a tortuous, small diameter vessel; even in the case of a successful advancement, the presence of multiple strut layers would have potentially led to inadequate expansion of the stent, and to an increased risk of thrombosis. Pretreatment of the lesion with a paclitaxel-eluting balloon has also been considered as an option to reduce in-stent restenosis of PCS.⁹ The concern of a possible paclitaxel development in the pseudoaneurysm induced us to reverse the sequence of the procedure and to perform in-stent postdilation with a paclitaxel-eluting balloon of the same length as the stent implanted at the index procedure, in order to inhibit the development of further intimal hyperplasia inside the stent, and to ensure a good drug delivery at the edges of the PCS. At angiographic follow-up, a focal intimal hyperplasia occurred at the proximal part of the stent, at the site of the pseudoaneurysm, resulting in a moderate stenosis at quantitative angiography, without hemodynamic relevance.

Conclusion. PCS showed an outstanding performance in a challenging anatomy. The use of a paclitaxel-eluting balloon limited further neointimal growth and only a focal, non-significant stenosis was detected at follow-up. Although this strategy was the result of a heuristic approach to the restenosis issue of PCS, it should be further evaluated in clinical studies in order to assess its efficacy and safety.

References