Stent Pseudorestenosis Due to Annular Calcification

To the Editor:

Inadequate stent expansion during implantation can be interpreted as an in-stent lesion during conventional angiography. This phenomenon is a lesser known mechanism than intimal proliferation, and is known as stent pseudorestenosis. Highly sensitive techniques, such as intravascular ultrasound,¹ are needed for the differential diagnosis between these entities, because of the implications for treatment. Nonoptimal stent apposition in the vessel wall has been related to revascularization complexity in some situations,²-⁴ and has been considered a factor associated with the subsequent development of events, such as stent thrombosis.⁵,⁶ Resolution of false restenosis may require the use of various revascularization techniques to increase stent expansion, sometimes with considerable difficulties and not always successfully.

A 45-year-old woman, ex-smoker as the single risk factor, went to another hospital for acute anterior myocardial infarction that was treated with systemic fibrinolysis. Since this failed, salvage angioplasty was performed. The right coronary and circumflex arteries showed no lesions, but a critical lesion was detected in the proximal left anterior descending artery (LAD), which was treated with implantation of a bare metal stent. At 2 months she was readmitted for unstable angina and electrocardiographic evidence of transient ST-segment elevation in the anterior territory in one of the crises. During hospitalization, the patient presented numerous angina attacks at rest with little therapeutic response. Repeat angiography showed a significant in-stent lesion that was interpreted as early stenosis. Surgical revascularization with a left mammary artery graft to the LAD was performed. At 4 months the patient again presented numerous angina attacks with no electrocardiographic changes and poor therapeutic response to the usual antianginal therapy. At that time she was referred to our center. Coronary angiography showed mammary graft occlusion and a critical lesion in the stent implanted in the proximal LAD. Intravascular ultrasound of the LAD lesion showed a lumen area of 2.5 mm² and a circumferential annular calcification that prevented stent expansion, with no intimal proliferation (Figure 1). Numerous attempts to break the annular calcification and expand the stent with various high-pressure balloons of different diameters and cutting balloons were unsuccessful. The angina symptoms persisted; hence, a second procedure was undertaken. Because the revascularization options were limited, rotational atherectomy in the lesion area was chosen. Following an unsuccessful initial attempt with a 2-mm burr, stent ablation was finally achieved with a 2.25-mm burr. After multiple dilations with high-pressure balloons, the results were reassessed with intravascular ultrasound, which showed rupture of the calcium plaque in the lesion and a final lumen area of 4.5 mm² (Figure 2). Because few options were available for revascularization and the procedure was difficult, the outcome

---

1. Intravascular ultrasound
2. Nonoptimal stent apposition
3. Revascularization complexity
4. Events associated with stent thrombosis
5. False restenosis
6. Intravascular ultrasound
7. Stent pseudorestenosis
8. Annular calcification
9. Mammary artery graft
10. Intravascular ultrasound
11. Rotational atherectomy
12. Stent ablation
13. Intravascular ultrasound
14. Calcium plaque rupture
15. Final lumen area

---

**Figure 1.** Baseline proximal left anterior descending artery.
was considered at the limits of acceptability. The cause of mammary artery graft occlusion was not determined. Various procedures showed TIMI 3 coronary flow, but no signs of embolization.

Elisabet Zamora, Eduard Fernández-Nofrerías, Oriol Rodríguez-Leor, and Josepa Mauri
Servicio de Cardiología, Hospital Universitario Germans Trias i Pujol, Badalona, Barcelona, Spain

REFERENCES


Idiopathic Chylopericardium.

A Case in Point

To the Editor:

A 41-year-old man presented dyspnea on moderate to heavy exertion 1 month before hospitalization. The examination showed normal vital signs, with no jugular vein enlargement or paradoxical pulse, low-intensity heart sounds, and pericardial rub. The laboratory workup revealed hemoglobin 18 g/dL, leukocytes 6.4, glucose 73 mg/dL, creatinine 1.1 mg/dL, and C-reactive protein 62.2 mg/dL. The echocardiogram showed severe pericardial effusion. A pericardial window was created, obtaining 1100 mL of a milky, nonfetid fluid, and a drainage tube was inserted. Fluid culture was negative. Cytochemical analysis of the pericardial fluid showed cholesterol 88 mg/dL, triglycerides 1830 mg/dL, lactate dehydrogenase 334 U/L, proteins 5.69 g/dL, and albumin 3.17 g/dL. Nonspecific inflammatory changes were observed in the pericardial biopsy. Chylopericardium was diagnosed and a diet based on medium-chain fatty acids was initiated; however, the drainage output did not decrease (Figure 1). This required thoracic duct ligation through the right posterolateral...