ADVANCED

CASE REPORT

CLINICAL CASE SERIES

Femoropopliteal Interventions

Popliteal Artery Entrapment Syndrome A Rare Cause of Interwoven Nitinol Stent Fracture After



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ABSTRACT

Stent fractures have been described after femoropopliteal interventions with relevant clinical sequelae, including restenosis and reocclusion. We report 2 cases of fractures of a novel intervoven nitinol stent with high radial strength and fracture resistance as a result of entrapment syndrome of the popliteal artery. (Level of Difficulty: Advanced.) (J Am Coll Cardiol Case Rep 2022;4:424-428) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

INTRODUCTION

Stent fractures after femoropopliteal intervention have been linked with adverse clinical outcomes, including higher in-stent restenosis and reocclusion rates.¹ Newer, laser-cut nitinol stents with improved conformability and higher radial strength were shown to exhibit lower but still clinically relevant rates of long-term stent fracture. The lowest stent fracture rates reported to date were recorded for the Supera peripheral artery stent system (Abbott). The unique stent design, consisting of braided, interwoven,

LEARNING OBJECTIVES

- To remind endovascular specialists to consider PAES as a rare cause of stent fracture in the popliteal area.
- To highlight that PAES could be the case, especially for dedicated fracture-resistant stents.
- To illustrate how such cases could be handled.

flexible nitinol wires with superior radial strength, fracture resistance, and flexibility, addresses the anatomical challenges of the high torsion and compression forces of the femoropopliteal segment. Previous studies using this device reported excellent primary patency rates for challenging femoral and popliteal lesions.^{2,3} Furthermore, the prospective SUPERB trial, which followed 325 patients after interwoven nitinol stent implantation, reported only 1 stent fracture after 3 years of follow-up.² In published reports, single cases of interwoven nitinol stent fracture have also been noted, but in most cases no explanatory additional risk factors could be identified.⁴⁻⁸ Here we present 2 cases of interwoven nitinol stent fracture secondary to popliteal artery entrapment syndrome (PAES).

HISTORY OF PRESENTATION AND MANAGEMENT

PATIENT #1. A 53-year-old man was admitted to our vascular center at the University Hospital Leipzig, Leipzig, Germany with severe claudication of the left

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lower limb (Rutherford III). Approximatively 1 year ago, a first interwoven nitinol stent implantation for focal popliteal atherosclerotic stenosis in the left lower extremity had been performed in another vascular center. Preinterventional work-up with duplex ultrasound showed high-grade restenosis of the left popliteal artery (PA), and the patient was scheduled for endovascular reintervention. The angiogram showed a fracture of the interwoven nitinol stent with high-grade restenosis between the 2 fragments (Figures 1A and 1B). Interestingly, the proximal femoral and distal infrapopliteal vessels did not exhibit signs of advanced atherosclerosis. Functional angiography was performed to intensify popliteal compression (images in dorsal extension, plantar flexion of the foot, and with the knee bent) for further clarification. In the angiogram with maximal knee extension and dorsal extension of the foot, blood flow stopped completely, thus confirming the diagnosis of PAES (Figure 1C, Video 1).

Next, magnetic resonance imaging (MRI), performed to reveal the underlying anatomical type of entrapment, showed functional occlusion of the PA, during provocation testing, that was caused by muscular tightness through the medial head of the gastrocnemius muscle (MHGM). Thus, surgical treatment was indicated. During the open vascular procedure, the MHGM was transected, and the 2 stent segments removed through a longitudinal arteriotomy. The PA was then reconstructed using a bovine pericardial patch (Figures 2A and 2B). The patient has now been free of claudication for more than 2 years.

PATIENT #2. A 74-year-old woman was admitted to our vascular department for worsening right lower limb claudication. The patient had a medical history of hypertension, diabetes mellitus, and previous popliteal (PI-PIII) bypass surgery for critical limb-threatening ischemia several years earlier. In addition, endovascular recanalization, with multiple stent implantations (1 interwoven nitinol stent, 2 drug-eluting stents) of the bypass as a result of reocclusion, was performed 1 year earlier.

Duplex ultrasound and the subsequent angiogram of the lower extremity revealed total occlusion of the femoropopliteal segment, including the bypass. The interwoven nitinol stent, located in the midsegment of the popliteal bypass, was fractured (Figures 3A and 3B). Given her previous artificial knee joint replacement, MRI could not be performed for further clarification, but the patient's clinical course was highly suggestive of PAES.

Next, complex endovascular revascularization of the occluded distal right femoral artery and popliteal

bypass was performed through the ipsilateral common femoral artery. Several antegrade and retrograde crossing attempts failed, but finally antegrade lesion crossing was successful using a dedicated recanalization and re-entry device. After predilation, 2 covered stents (Viabahn 6 \times 150 mm and 7 \times 100 mm stents, Gore Medical) were implanted throughout the bypass starting from the distal superficial femoral artery, followed by high-pressure balloon angioplasty of the entire stented segment. Finally, the area of the previous stent fracture was stabilized with another interwoven nitinol stent. Functional angiography after successful revascularization revealed extrinsic compression of the bypass indicating PAES. Thus, as part of a hybrid procedure, the bypass was decompressed by surgical transection of the MHGM. A control angiogram performed 7 days after the procedure showed a patent femoropopliteal segment without any residual stenosis (Figures 3C and 3D). To date, the patient has been free of any symptoms for more than 18 months.

DISCUSSION

We reported 2 cases of PAES, which typically manifests with exercise-induced leg pain but also can be a rare cause of stent fractures. Compression syndromes of the popliteal fossa are reported with a prevalence of 3.5% on the basis of autopsy studies, whereas the reported clinical prevalence is even lower.⁹

PAES, first described by Stuart in 1879, is often misdiagnosed but should be considered, especially in younger men presenting with typical claudication but smooth-walled vessels on ultrasound or angiography and without classic risk factors for atherosclerosis. PAES is more common in athletes than in the general population because stronger muscles exacerbate the anatomical tightness created by misaligned muscles. However, repeated compression of the PA can cause trauma to the arterial wall and lead to premature local atherosclerosis.

PAES results from an abnormal relationship between the PA and the surrounding myofascial structures. Both embryonic and acquired (functional) causes have been described, but most PAES cases are caused by embryologic anomalies, with compression of the popliteal artery by the MHGM inserting at the level of the knee.⁹ PAES is typically classified into 6 types. In type 1, the PA is medially displaced, with an aberrant course around a normal MHGM. In type 2, the MHGM is attached laterally on the distal femur. In type 3, a deviated accessory slip from the MHGM

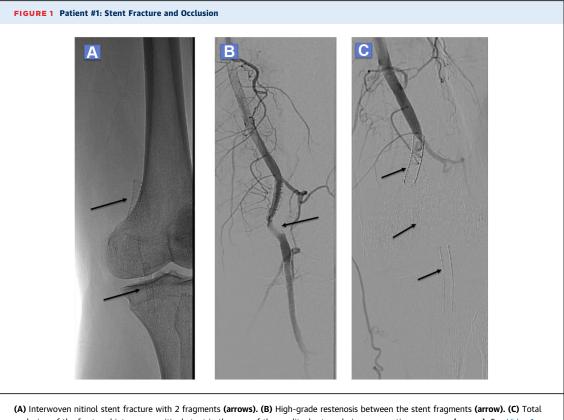
ABBREVIATIONS AND ACRONYMS

MHGH = medial head of the gastrocnemius muscle

MRI = magnetic resonance imaging

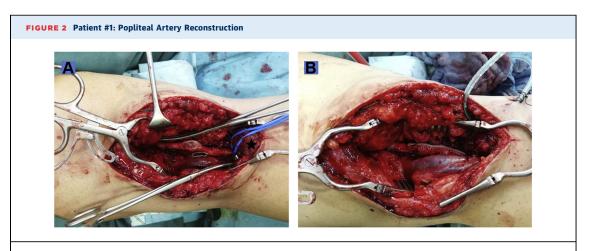
PA = popliteal artery

PAES = popliteal artery entrapment syndrome

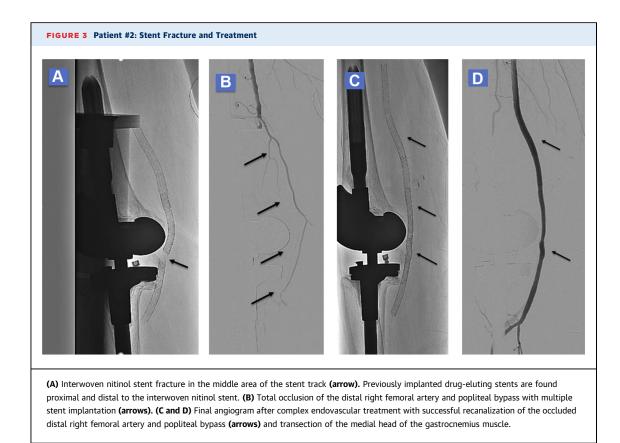


(A) Interwoven nitinol stent fracture with 2 fragments (arrows). (B) High-grade restensis between the stent fragments (arrow). (C) Total occlusion of the fractured interwoven nitinol stent in the area of the popliteal artery during provocation maneuver (arrows). See Video 1.

wraps around the PA. In type 4, the PA lies deep in the popliteal muscle or beneath fibrous bands in the popliteal fossa. Types 5 and 6 comprise primarily venous and functional (without aberrant anatomy) entrapments, respectively.⁹ If PAES is identified, decompressive surgery is the treatment of choice and is usually performed through a lateral or medial approach to the popliteal fossa. Musculotendinous division aims to release the entrapment, and arterial reconstructive procedures may be



(A) Posterior view of the popliteal fossa. The **star** shows the distal part of the transected medial head of the gastrocnemius muscle. Longitudinal arteriotomy of the popliteal artery revealing the atherosclerotic material between the 2 fractured stent segments. (B) Final surgical result showing the popliteal artery after removal of the stent and reconstruction using a bovine pericardial patch.



necessary, including replacement of the affected vessel section with an autologous vein or prosthetic bypass.¹⁰

Advanced imaging modalities, consisting of MRI, duplex ultrasound, or angiography, combined with maneuvers to provoke compression are essential to identify the underlying cause of entrapment. In most cases, a combination of MRI and duplex ultrasound is preferred because it has the advantage of avoiding radiation exposure.

CONCLUSIONS

PAES can be a rare cause of femoropopliteal stent fractures and should be considered, especially when stents designed with high fracture resistance are affected. To avoid incorrect treatment, PAES should be ruled out using provocation tests in combination with dedicated imaging techniques, including MRI and angiography.

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KEY WORDS peripheral vascular disease, stents, treatment, ultrasound

APPENDIX For a supplemental video, please see the online version of this paper.