

CASE REPORT

Trapped by the Entrapment

Alban Longchamp^{a,b,†}, Justine Longchamp^{a,†}, Sara Manzocchi Besson^{a,c}, Daniel Danzer^{a,d,*}^a Department of Vascular Surgery, Sion Hospital, Sion, Switzerland^b Department of Vascular Surgery, Centre Hospitalier Universitaire Vaudois and University of Lausanne, Lausanne, Switzerland^c Division of Angiology and Haemostasis, University Hospitals and Faculty of Medicine, Geneva, Switzerland^d Division of Cardiovascular and Vascular Surgery, University Hospitals and Faculty of Medicine, Geneva, Switzerland

Introduction: Popliteal entrapment syndrome results from extrinsic compression of the popliteal artery by the surrounding musculotendinous structures and is a rare cause of limb ischaemia. The purpose of this report is to highlight potential mistakes in the management of popliteal entrapment.

Report: In 2000, a 23 year old man underwent a popliteal to popliteal artery bypass surgery for what was initially diagnosed as a traumatic popliteal artery thrombosis. After being initially lost to follow up for 13 years, this “unspecified traumatic” thrombosis led to several inappropriate endovascular and open procedures misinterpreted as being caused by late graft failure. These included thrombectomy, aneurysmorrhaphy, polytetrafluoroethylene covered stent graft, a redo femoropopliteal bypass, and bypass thrombolysis. The diagnosis was reached 19 years after the initial surgery, when the patient underwent a redo bypass using a retrogeniculate approach. An abnormal lateral insertion of the gastrocnemius muscle medial head, and its accessory slip, constricted the artery, and also involved the popliteal vein (Type V), thus explaining previous revascularisation failures. Surgery consisted of resecting the accessory slip and the aneurysmal bypass. The artery was reconstructed with the cephalic vein. The patient was discharged on clopidogrel 75 mg, with no further complication, and a patent bypass at six months. Based on post-operative imaging (duplex ultrasound and magnetic resonance imaging), with forced plantarflexion and dorsiflexion, asymptomatic popliteal entrapment was also present on the contralateral side.

Discussion: The finding of an isolated popliteal artery lesion in a young individual should be considered to be caused by popliteal artery entrapment, unless proven otherwise. Definitive surgical release of the popliteal artery should be favoured over other strategies.

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INTRODUCTION

Popliteal artery entrapment syndrome (PAES), is an uncommon limb threatening vascular entity affecting 0.2%–3.5% of the general population.¹ PAES is defined by abnormal compression of the popliteal artery by nearby musculotendinous structures. PAES, which was first described in 1879 by Stuart,² and later classified into six different types, depending on the impinging structures or concomitant venous compression. While duplex ultrasound (DUS), magnetic resonance imaging (MRI), computed tomography (CT) scan, and arteriography, with or without stress positional manoeuvres, can be valuable, diagnosis is

often challenging if not actively suspected.^{3,4} In previous studies, the mean duration of symptoms before surgical treatment ranged from two to 216 months,⁵ and there are reports of misdiagnosis for up to 15 years.⁶ Prompt diagnosis is important. When PAES is recognised early, and the artery remains undamaged, myotomy alone can be performed. In more complex cases, when arterial stenosis, occlusion, or aneurysm is present, saphenous vein bypass with entrapping structure release or extra-anatomical tunnelling is the gold standard, with good long term patency (83%–100%).⁷

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

CASE REPORT

In 2000, following knee trauma, and popliteal artery thrombosis, a 23 year old sedentary man, with no past medical history except for smoking, underwent a right limb popliteal to popliteal artery bypass surgery. Nineteen years later, a careful medical history revealed a blunt, non-

[†] Shared first authorship.

* Corresponding author. Department of Vascular Surgery, Sion Hospital, Sion, Switzerland.

E-mail address: daniel.danzer@hopitalvs.ch (Daniel Danzer).

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penetrating bull foot trauma to the knee. At the time, the patient reported immediate swelling but was otherwise asymptomatic. He only complained of progressive limb pain, and paraesthesia six months later, leading to the discovery of popliteal artery thrombosis. This history of arterial trauma misled multiple surgeons into inappropriate surgical procedures, which will be described in chronological order.

The first surgical procedure consisted of an infrageniculate anatomical femoropopliteal bypass, using a medial approach, and a short segment of ipsilateral saphenous vein. The procedure was uneventful. The patient was discharged with aspirin 100 mg daily. The patient remained asymptomatic and was lost to follow up. Thirteen years later, the patient underwent surgical thrombectomy and aneurysmorrhaphy owing to a partially thrombosed 24 mm popliteal pseudoaneurysm. He then needed multiple re-interventions (Fig. 1), including (1) an expanded polytetrafluoroethylene covered stent graft (GORE® VIABAHN®; W. L. Gore & Associates, Newark, DE, USA), owing to bypass stenosis and a recurrent 25 mm pseudoaneurysm (2015; Fig. 1A); (2) a redo femoropopliteal bypass, using the ipsilateral remnant great saphenous vein, owing to stent thrombosis (2016; Fig. 1B); and (3) bypass revision after thrombolysis with a 2 cm resection and end to end anastomosis (2018; Fig. 1C, “bypass kinking”). Of note, all of the post-operative DUS results were normal at rest. Moreover, the patient was asymptomatic between thrombotic episodes, which occurred under well conducted oral anti-coagulation (vitamin K antagonist). Acquired and congenital thrombophilia were also excluded. Thus, all of these late complications were interpreted as late vein graft degenerations, and graft material inadequacies (placement of a stent graft across the knee flexion zone, or use of a varicose remnant saphenous vein).

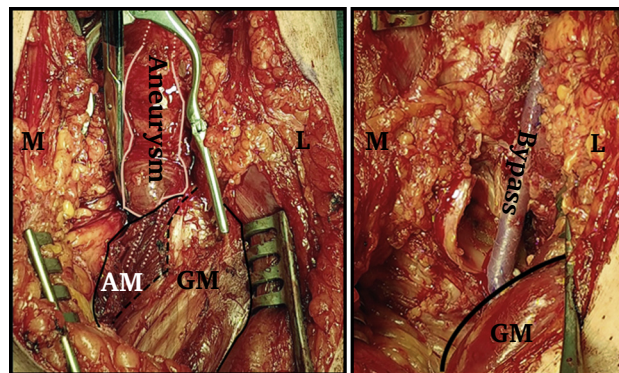


Figure 2. Popliteal artery entrapment *in situ* before (left) and after myotomy and bypass (right), demonstrating the lateral insertion of the gastrocnemius muscle medial head (GM) and its accessory slip/muscle (AM). M = medial; L = lateral.

Finally, in October 2019, the patient presented with a 52 mm popliteal pseudoaneurysm, causing significant knee pain during flexion (Fig. 1D). PAES was suspected on CT scan, but artefacts made the interpretation difficult. No “baseline” imaging study was available. Thus, the patient underwent surgery, which allowed the definitive diagnosis. Using a retrogeniculate approach, the popliteal artery entrapment was identified for the first time. An abnormal lateral insertion of the gastrocnemius muscle medial head, and its accessory slip, constricted the artery and also involved the popliteal vein (Type V; Figs. 2 and 3). Surgery consisted of resecting the accessory slip, as well as the aneurysmal bypass. The complete fusion of the medial with the lateral head of the gastrocnemius muscle insertion precluded its transposition, which was partially resected (Fig. 2). The artery was reconstructed with the cephalic vein. Histopathological analysis indicated non-specific fibrotic

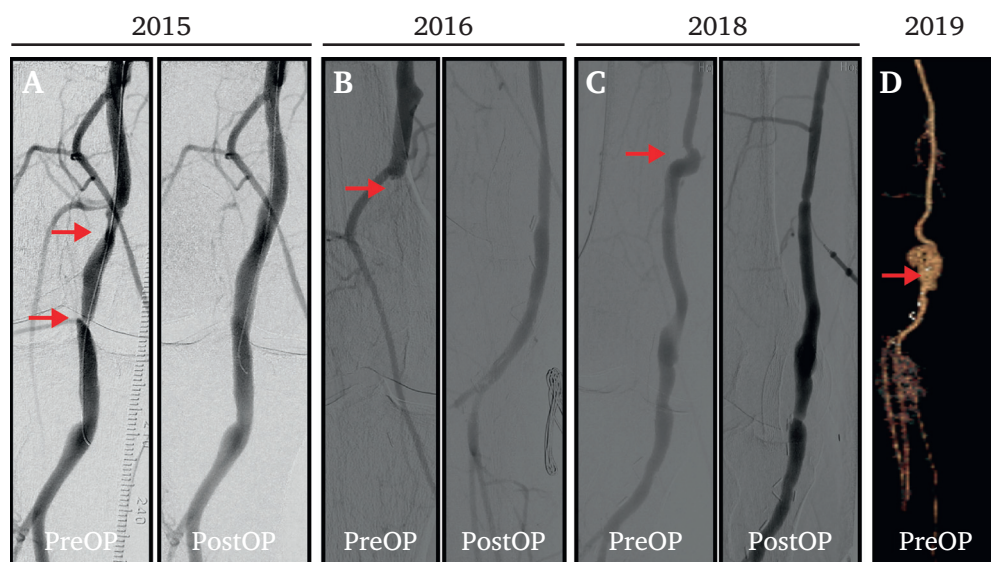


Figure 1. Timeline of vascular reconstruction. (A–C) Angiography and (D) three dimensional computed tomography arteriography obtained before (PreOP) and after (PostOP) surgery. Arrow indicates vascular lesions.

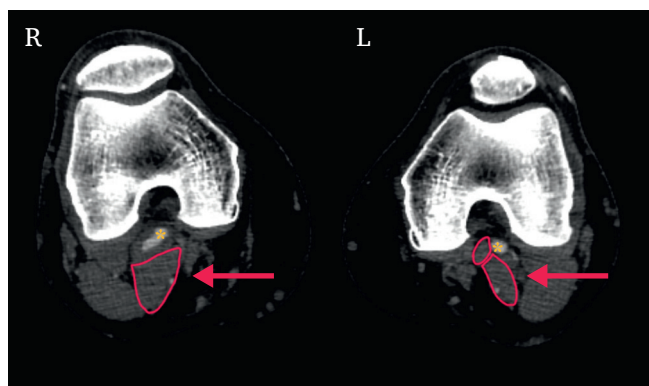


Figure 3. Computed tomography scan of the left (after the first redo surgery, 2015) and right limb (contralateral, unoperated) demonstrating the lateral insertion of the gastrocnemius muscle medial head and its accessory slip. Red circles and arrows indicate the aberrant muscle. Yellow star indicates the popliteal artery.

vein remodelling, in direct contact with skeletal muscle. Post-operative clinical examination revealed palpable pedal, and posterior tibial pulses. The ankle brachial index was 1.22 (vs. 1.18 pre-operatively). DUS showed a patent bypass, with peak systolic velocity of 75 cm/second. All three distal crural vessels were patent (DUS and MRI). On the contralateral side, DUS and MRI during forced bilateral plantarflexion, and dorsiflexion, showed a type V PAES. As of July 2020, the patient still declines surgery on the contralateral limb.

DISCUSSION

Here, a case of recurrent popliteal artery reconstruction failure due to PAES is presented. The patient underwent multiple imaging and surgical procedures before receiving the appropriate diagnosis and treatment 19 years later. This is the longest reported misdiagnosed PAES.^{5,6} In this case, the acute presentations, without prior symptoms of arterial stenosis, or compression, precluded adequate pre-operative testing. In addition, the patient's history of a traumatic arterial injury and subsequent thrombosis did not immediately lead to the correct diagnosis (PAES). All of the arterial reconstruction failures were thought to be related to inadequate materials (stents and varicose veins). Interestingly, cases of recurrent PAES after femoropopliteal bypass have been described.⁸ As seen in this case, if not actively suspected after popliteal artery thrombosis, and investigated with dedicated functional imaging, endovascular, or surgical revascularisation using a medial approach might miss the diagnosis. Diagnosis can be further delayed in unusual presentations of PAES, such as a pulsatile mass without ischaemia.^{5,9} In some cases, patients even underwent unnecessary surgery for presumed chronic compartment syndrome, later diagnosed as PAES.⁶

In the present case, the patient remains asymptomatic on the contralateral side. Although a clear relationship exists

between the development of a local occlusive, or aneurysmal changes of the popliteal artery in a patient with symptomatic impingement,¹⁰ its natural history in asymptomatic patients has not been studied. It seems reasonable that in anatomical type I – V entrapment, the artery should undergo surgical release if symptomatic, before deterioration of the popliteal artery by repeated compression. Importantly, the long term patency rate is inversely correlated to the extent of injury to the popliteal artery, highlighting the importance of early treatment.⁷

Thus, an individual with no cardiovascular risk factors, an isolated popliteal artery lesion, or recurrent femoropopliteal reconstruction failures, should be considered to have PAES, unless proven otherwise.

CONFLICTS OF INTEREST

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