

Popliteal artery entrapment syndrome as a cause of failed treatment of a false popliteal aneurysm

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Abstract

Objective: Popliteal artery entrapment syndrome is a rare cause of popliteal artery aneurysms. We present a rare case of a false aneurysm associated with popliteal artery entrapment syndrome that was treated with endovascular repair that initially failed.

Case report: A 60-year-old man with a false popliteal artery aneurysm and limb ischemia was treated with endovascular repair that initially failed. The popliteal artery was suspected to be compressed by an abnormal bundle of muscle according to the findings of a subsequent magnetic resonance imaging examination. The popliteal artery was entrapped by an abnormal slip of the medial gastrocnemius muscle head. Parts of the popliteus muscle were also involved in compression of the popliteal artery, which was not distinguished on preoperative magnetic resonance imaging. Thus, the patient was diagnosed with a mixed type of popliteal artery entrapment syndrome (types III and IV). Bypass with the small saphenous vein was then performed. The patient was finally discharged with satisfactory relief of his ischemic symptoms.

Conclusion: Popliteal artery entrapment syndrome should be considered before treating popliteal artery aneurysms, especially atypical pseudoaneurysms without significant atherosclerosis. Definitive surgical management rather than endoluminal treatment is required unless combined with open decompressive surgery to correct the musculotendinous anatomy.

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Keywords

Popliteal artery, popliteal artery entrapment syndrome, aneurysm, stents, bypass surgery, limb ischemia

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Introduction

The popliteal artery is the most common site for peripheral artery aneurysms. Approximately 90% of popliteal artery aneurysms (PAAs) are caused by atherosclerosis.¹ Popliteal artery entrapment syndrome (PAES) is a rare cause of PAAs. In this condition, the popliteal artery is compressed by aberrant surrounding musculo-tendinous structures.² Pathological changes of the popliteal artery, such as stenosis, occlusion, or thrombosis, can develop secondary to persistent microtrauma from these structures. Aneurysms can also develop and manifest as a pulsatile mass and chronic or acute limb ischemia.^{3,4} False popliteal aneurysms are rare but can also be seen in patients with PAES.^{5,6} We herein present a rare case of a false PAA associated with PAES. Written informed consent for publication was obtained from the patient. Ethics committee approval was not required because this is a case report, not a clinical study.

Case report

A 60-year-old man with a sudden onset of claudication was referred to the vascular surgery department of our tertiary medical center. Two months earlier, he had experienced a sudden decline of his walking distance to about 20 m because of pain in the right calf. The man developed pallor and coolness of the right foot soon thereafter. He had a 10-year history of hypertension and a 4-year history of type 2 diabetes mellitus, both of which were well controlled.

He had no history of trauma or surgery involving the popliteal fossa. On presentation, the patient's temperature was normal. A pulsatile mass (5 × 10 cm) was palpated in the right popliteal fossa with no dermal redness, edema, or heat. The pulses of both the distal posterior tibial artery and the dorsal pedis artery were very weak and difficult to find. Computed tomographic angiography (CTA) revealed an irregular mass with a diameter of approximately 3.8 cm and communication with the right popliteal artery (Figure 1(a), (b)). The patient's total white blood cell count, erythrocyte sedimentation rate, and high-sensitivity C-reactive protein concentration were all within the reference ranges. Digital subtraction angiography was then performed. A false PAA was confirmed, and slight medial deviation of the popliteal artery was also noted (Figure 1(c)). The diameter of the proximal popliteal artery was approximately 6 mm. The false aneurysm was successfully repaired with a 6- × 100-mm VIABAHN endoprosthesis (W. L. Gore, Flagstaff, AZ, USA) with post-dilatation (Figure 1(d)). After the procedure, the patient was prescribed rivaroxaban at 10 mg/day, aspirin at 100 mg/day, and atorvastatin at 20 mg/day. His symptoms were significantly relieved. He was discharged with no complications.

Approximately 2 weeks after discharge, the patient was re-admitted because of recurrence after a long-distance bicycle ride. Stent thrombosis was diagnosed by CTA (Figure 1(e)). The peroneal artery seemed to be occluded, and thrombosis in

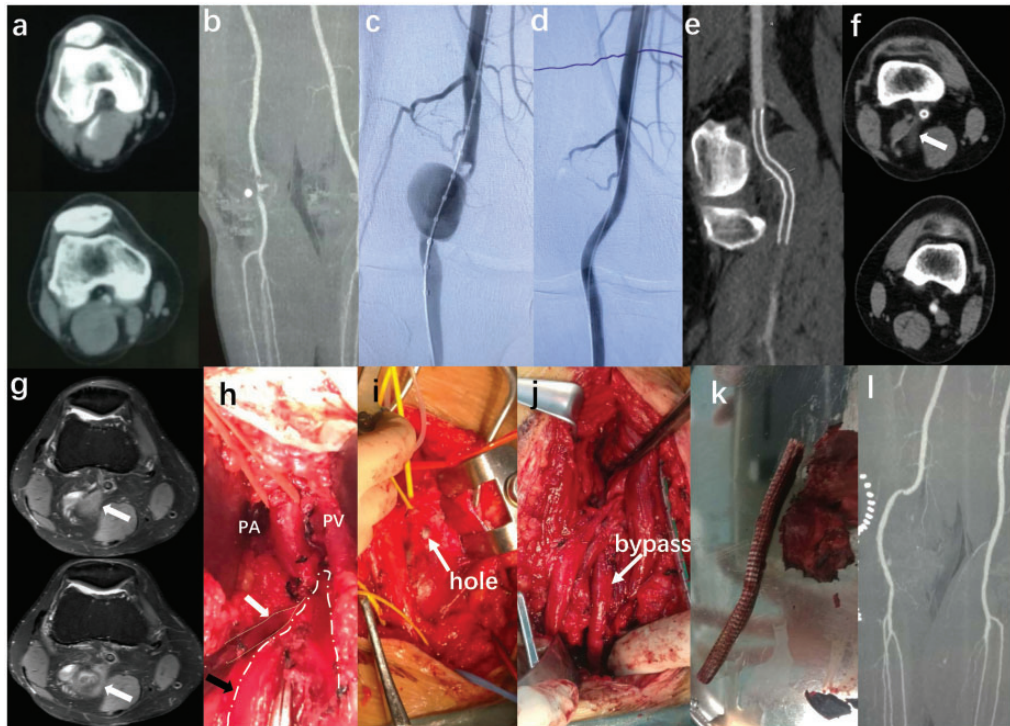


Figure 1. Images throughout the treatment process. (a, b) False aneurysm of the right popliteal artery on computed tomography angiography (CTA). (c, d) Digital subtraction angiography before and after endovascular repair. (e) Stent thrombosis found by CTA. (f, g) Abnormal slip of the medial head of the gastrocnemius muscle shown on CTA and magnetic resonance imaging (white arrow). (h) Intraoperative photograph showing the popliteus muscle (white arrow) and the abnormal slip of the medial head of the gastrocnemius muscle (black arrow). (i) A hole was found on the popliteal artery wall. (j) Bypass with the small saphenous vein. (k) Stent taken out by surgery. (l) Postoperative CTA.

the distal part of the posterior and anterior arteries could not be excluded. However, the patient refused to undergo catheter-directed thrombolysis or any other endovascular intervention for fear of further failure and additional cost. The patient was treated conservatively with anticoagulation using low-molecular-weight heparin. The CTA images were carefully re-evaluated, and an aberrant musculotendinous structure was suspected to be present immediately above the pseudoaneurysm (Figure 1(f)). This structure was further confirmed by magnetic resonance imaging (MRI) (Figure 1(g)). The CTA image of

the contralateral popliteal fossa was also carefully re-evaluated. No sign of symmetric PAES was found. The popliteal fossa was surgically explored using a dorsal S-shaped incision. The proximal attachment of the medial head of the gastrocnemius (MHG) arose slightly higher than its normal position. An abnormal slip of the MHG was inserted between the popliteal artery and vein, resulting in entrapment and medial deviation of the popliteal artery at this site. The distal attachment of the popliteus muscle was at the posterior surface of the proximal tibia extremity, which was higher than usual and led to

medial deviation of the popliteal artery. Parts of the popliteus muscle were also involved in the entrapment of the popliteal artery but could not be clearly recognized on CTA or MRI (Figure 1(h)). The false aneurysm was ligated, opened, and partially resected. A hole was found on the primary popliteal artery wall within the aneurysmal sac (Figure 1(i)). A bypass with the reversed small saphenous vein was performed with end-to-side anastomosis to the healthy artery at both ends; this allowed for resection of the false aneurysm, removal of the stent, and avoidance of an intervention involving the unhealthy arterial wall. The abnormal muscular structures were not resected because the graft could be placed posteriorly to these structures without compression (Figure 1(j)). The removed stent is shown in Figure 1(k). Patency of the graft was demonstrated by CTA (Figure 1(l)). The ankle-brachial index increased from 0.2 before surgery to 0.7 after surgery. Rivaroxaban at 10 mg/day was prescribed because of suspected thrombosis and embolism in the distal runoff vessels. The patient was discharged and followed up in the outpatient department. In the early postoperative period, the patient could walk with a claudication distance of about 1000 m. He became asymptomatic 3 months after surgery and continued to do well thereafter. Notably, the runoff status also recovered as shown on follow-up CTA 12 months after surgery.

Discussion

We have herein presented a rare case of a false PAA caused by PAES. The popliteal artery was compressed by both an abnormal slip of the MHG and part of the popliteus muscle. This case could not be classified as any of the single traditional types of PAES.⁷ Instead, it was more likely a combination of type III and type IV entrapment. In addition, the aneurysm

was a false aneurysm, which has rarely been reported before now.⁵ The patient was first treated by endovascular repair, which failed soon after the intervention, probably because of external compression of the abnormal structures. Open surgery with bypass was finally performed with a satisfactory result.

PAAAs might result from post-stenosis dilatations and long-term repeated injuries from the surrounding musculotendinous structures in patients with PAES. Rosset et al.⁸ reported that aneurysms or ectasia were seen in 9.1% (34/374) of PAES-affected limbs, and Farina et al.⁹ reported that 6% (3/50) of PAAAs were related to PAES. PAES should be suspected as a potential cause of PAAAs in patients who have no definitive cause. PAAAs are relatively more common in older patients with PAES.^{10,11} PAES should not be arbitrarily excluded in the presence of traditional atherosclerotic risk factors.

The stress test combined with Doppler, ultrasonography, or angiography is helpful for the diagnosis of PAES. However, because of complicated anatomical changes of both the PAAAs and the compressing structures, the diagnosis of PAES might be difficult when complicated with PAAAs. Some diagnoses might not be reached until aberrant anatomical structures are directly inspected during surgery. CTA might be helpful for the recognition of abnormal structures and deviation of the popliteal artery. However, PAES might still be misdiagnosed, as shown in our case and other reports.^{3,12} MRI provides better resolution of soft tissues, and it can be helpful for the recognition of abnormal musculotendinous structures and the diagnosis of PAES.¹³ Nevertheless, the diagnosis can still be missed by MRI, especially the diagnosis of type IV PAES. This is probably because the involved structures are deep and tiny, making them difficult to distinguish, as in the present case.

Both surgical and endovascular repair can be used for the treatment of PAAs.¹⁴ However, endovascular repair without decompression will lead to graft failure, as in this case. di Marzo et al.¹⁵ also reported failure of endovascular stenting in a patient with PAES that manifested as artery occlusion. Additionally, the distal runoff status might be compromised by thrombosis or embolism after failed endovascular treatment, as shown in this case. Decisions regarding endoluminal repair should be made prudently once the diagnosis is conclusive, especially in the present so-called “endoluminal era.” Open decompression and reconstruction of the popliteal artery is still recommended for PAAs caused by PAES. An autologous vein is the graft of choice. Because thrombosis or embolism of distal outflow arteries is common in PAAs, postoperative anticoagulation with or without antiplatelet therapy might be helpful for long-term patency.

Conclusion

PAES should be kept in mind before treating PAAs, especially atypical pseudoaneurysms without significant atherosclerosis. Definitive surgical management rather than endoluminal treatment is required unless combined with open decompressive surgery to correct the musculotendinous anatomy.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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References

1. Davidovic LB, Lotina SI, Kostic DM, et al. Popliteal artery aneurysms. *World J Surg* 1998; 22: 812–817.
2. Sinha S, Houghton J, Holt PJ, et al. Popliteal entrapment syndrome. *J Vasc Surg* 2012; 55: 252–262.e30.
3. Lopez Garcia D, Arranz MA, Tagarro S, et al. Bilateral popliteal aneurysm as a result of vascular type IV entrapment in a young patient: a report of an exceptional case. *J Vasc Surg* 2007; 46: 1047–1050.
4. Levien LJ and Veller MG. Popliteal artery entrapment syndrome: more common than previously recognized. *J Vasc Surg* 1999; 30: 587–598.
5. Chen CH, Wen JS and Wang CK. Huge pseudoaneurysm and cystic adventitial disease from popliteal artery entrapment. *Circulation* 2015; 132: 1284–1285.
6. Stuart TP. Note on a variation in the course of the popliteal artery. *J Anat Physiol* 1879; 13: 162.
7. di Marzo L and Cavallaro A. Popliteal vascular entrapment. *World J Surg* 2005; 29: S43–S45.
8. Rosset E, Hartung O, Brunet C, et al. Popliteal artery entrapment syndrome. Anatomic and embryologic bases, diagnostic and therapeutic considerations following a series of 15 cases with a review of the literature. *Surg Radiol Anat* 1995; 17: 161–169, 23–7.
9. Farina C, Cavallaro A, Schultz RD, et al. Popliteal aneurysms. *Surg Gynecol Obstet* 1989; 169: 7–13.
10. Ferreira J, Canedo A, Graca S, et al. Thrombosed popliteal aneurysm - first manifestation of bilateral popliteal entrapment syndrome. *Int Angiol* 2010; 29: 83–86.
11. Iwasaki H, Shibuya T, Ishizaka T, et al. Unilateral acute lower extremity ischemia with popliteal artery aneurysm as a result of vascular type III entrapment in an elderly patient. *Vascular* 2010; 18: 116–120.
12. Paraskevas N, Castier Y, Fukui S, et al. Superficial femoral artery autograft reconstruction for complicated popliteal artery entrapment syndrome. *Vasc Endovascular Surg* 2009; 43: 165–169.

13. Ozkan U, Oguzkurt L, Tercan F, et al. MRI and DSA findings in popliteal artery entrapment syndrome. *Diagn Interv Radiol* 2008; 14: 106–110.
14. Leake AE, Segal MA, Chaer RA, et al. Meta-analysis of open and endovascular repair of popliteal artery aneurysms. *J Vasc Surg* 2017; 65: 246–256.e2.
15. di Marzo L, Cavallaro A, O'Donnell SD, et al. Endovascular stenting for popliteal vascular entrapment is not recommended. *Ann Vasc Surg* 2010; 24: 1135.e1-3.