

Patrick T. Liu
Adrian C. Moyer
Eric A. Huettl
Richard J. Fowl
William M. Stone

Popliteal vascular entrapment syndrome caused by a rare anomalous slip of the lateral head of the gastrocnemius muscle

Received: 11 May 2004
Revised: 16 July 2004
Accepted: 28 July 2004
Published online: 5 October 2004
© ISS 2004

P. T. Liu (✉) · A. C. Moyer · E. A. Huettl
Department of Radiology,
Mayo Clinic Scottsdale,
13400 East Shea Boulevard, Scottsdale,
AZ 85253, USA
e-mail: liu.patrick@mayo.edu
Tel.: +1-480-3014850
Fax: +1-480-3014303

R. J. Fowl · W. M. Stone
Department of Vascular Surgery,
Mayo Clinic Scottsdale,
13400 East Shea Boulevard, Scottsdale,
AZ 85253, USA

Abstract Popliteal vascular entrapment syndrome can result in calf claudication, aneurysm formation, distal arterial emboli, or popliteal vessel thrombosis. The most commonly reported causes of this syndrome have been anomalies of the medial head of the gastrocnemius muscle as it relates to the course of the popliteal artery. We report two cases of rare anomalous slips of the lateral head of the gastrocnemius muscle causing popliteal vascular entrapment syndrome.

Keywords Popliteal vascular entrapment · Lateral gastrocnemius · MR imaging · Ultrasound · CT scan

Introduction

Popliteal vascular entrapment syndrome is a rare disorder that can result in calf claudication, aneurysm formation, distal arterial emboli, or popliteal vessel thrombosis [1, 2, 3, 4]. The most commonly reported etiologies of this syndrome have been anomalies of the origin of the medial head of the gastrocnemius muscle as it relates to the course of the popliteal artery [5]. The syndrome was originally termed, “popliteal artery entrapment syndrome” [6]; however, several reports have noted that some of these anatomic variants can also cause compression and thrombosis of the adjacent popliteal vein [7, 8, 9, 10, 11]. Thus the term “popliteal vascular entrapment syndrome” has been proposed and may be more appropriate [5].

Anomalies of the lateral head of the gastrocnemius muscle are relatively uncommon causes of popliteal vascular entrapment. In a review of the medical literature, we found only seven reported cases of this rare anomaly [2, 12]. We present two additional cases in which aberrant

slips of the lateral head of the gastrocnemius muscle have caused popliteal vascular entrapment, one demonstrated by CT and the other by MRI.

Case reports

Case 1

A 46-year-old woman presented to the emergency department complaining of a 4-day history of blue discoloration and paresthesias of her right first and fifth toes. Physical examination revealed cyanosis of the right great toe, blotchy discoloration of the right fifth toe, and a diminished dorsalis pedis arterial pulse. Femoral pulses were weak bilaterally, and popliteal pulses could not be well examined due to the patient's obesity.

A conventional arteriogram of the right lower extremity was performed with selective catheterization of the right external iliac artery and digital subtraction technique, demonstrating a thrombus partially occluding the right popliteal artery (Fig. 1A) and emboli in the right dorsalis pedis and posterior tibial arteries (not shown) above the ankle. The anteroposterior images of the popliteal arteries from the angiogram obtained with the knees extended and the an-



Fig. 1A—E Case 1. **A** Selective catheter digital subtraction arteriogram of the right popliteal artery shows medial deviation of the popliteal artery with a notch-like defect indicating partially occluding thrombus in the lateral aspect of the arterial lumen (*arrow*). **B** Axial T1-weighted MR image of both legs at the level of the distal femoral metaphyses shows the aberrant slip of the lateral head of the right gastrocnemius muscle (*long arrow*) curving around the popliteal vessels (*short arrow*). **C**, **D** Sagittal T1-

weighted MR images from the lateral (**C**) and midline aspects (**D**) of the knee show the normal origin (*long arrow*) and the aberrant slip of the lateral head (*short arrow*) of the gastrocnemius muscle. **E** Coronal thick slab maximum intensity projection from the arterial phase of the MR angiogram shows partially occlusive thrombus in the right popliteal artery (*arrows*). The *long arrow* also marks the location of the T1-weighted axial image displayed in **B**

kles in the plantar flexed and neutral positions both showed slight medial arterial deviation at the level of the thrombus but no variation with ankle positioning. The remainder of the arteries in the legs, as well as the abdominal aorta, contained no atherosclerotic plaques. Thus, the popliteal artery thrombus was felt to be the source of the distal calf emboli.

Because this was an unusual location for an acute thrombus in a patient without atherosclerotic disease, a combined MRI and MR angiography (MRA) study of the right leg was performed to exclude an anatomic cause for the isolated popliteal artery thrombus. MRI (Fig. 1B—D) showed an aberrant muscle slip originating from the posterior cortex of the distal femoral metaphysis, passing lateral to the popliteal vascular bundle, and then joining with the lateral head of the gastrocnemius muscle in the proximal calf. MRA of both lower extremities, performed with precontrast, arterial and venous phase three-dimensional fast gradient echo gadolinium-enhanced technique (Fig. 1E), revealed no obvious displacement of the popliteal vessels by this aberrant muscle slip; however, the popliteal artery thrombus was found to begin at the level where the aberrant gastrocnemius muscle slip coursed immediately adjacent to the popliteal vessels and extended distally. There was no thrombosis of the popliteal vein and no corresponding muscle anomaly in the opposite leg.

The patient was treated with mobilization of the aberrant slip of the lateral head of the gastrocnemius muscle, endarterectomy of the popliteal artery, placement of a saphenous vein graft patch, and thrombectomy with intra-arterial instillation of tissue plasminogen activator (TPA) for the distal emboli. She was doing well at her 5-week postoperative appointment but neglected to pursue further follow-up.

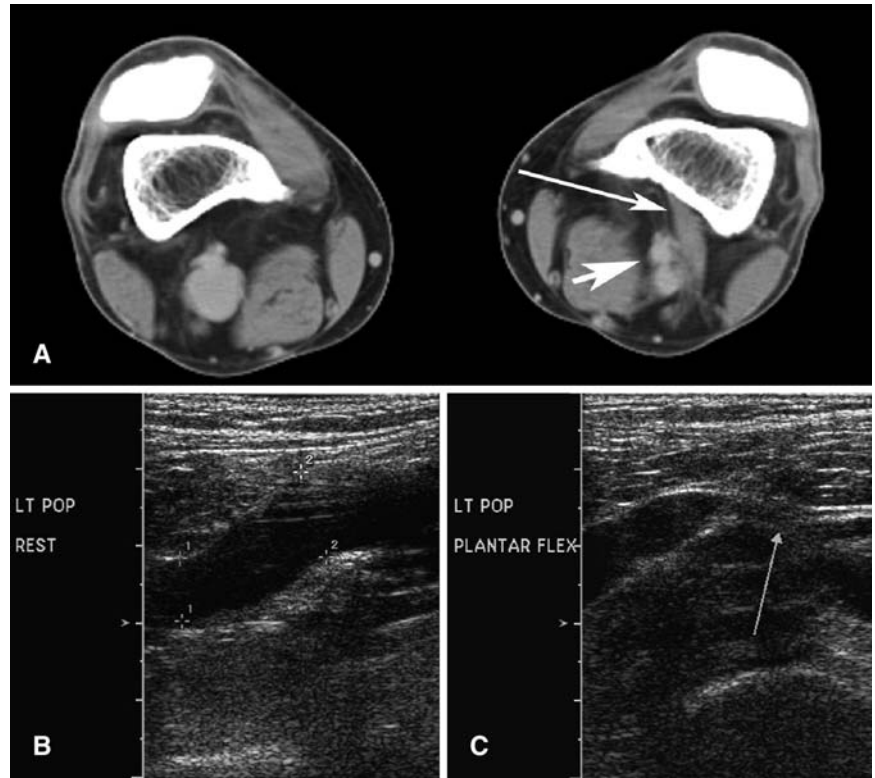
Case 2

A 58-year-old man presented to an internist for management of recurrent pulmonary emboli and left deep vein thrombus. Eleven months previously, he had initially presented to an outside medical institution with new-onset dyspnea and was found to have multiple pulmonary emboli and a deep venous thrombosis of the left leg (no further details available). After 6 months of anticoagulation treatment using lovenox and coumadin, his symptoms had completely resolved and his anticoagulation therapy was discontinued. One month later, however, his dyspnea recurred, and he was found to have multiple new pulmonary emboli. He was again placed on a 6-month course of coumadin therapy, and came to our institution for additional investigation of a possible hypercoagulable syndrome.

Prior to suffering these pulmonary emboli, the patient had been running 5 miles (8 km) a day regularly and had no significant medical history. Both these emboli occurred while the patient was taking low-dose aspirin for myocardial infarction prophylaxis. Medical investigation revealed no evidence for hypercoagulable syndrome. In order to assess the status of the patient's pulmonary vasculature, a contrast-enhanced CT examination of the chest was performed at our institution. This included routine axial CT images of the thighs and lower legs to examine for deep venous thrombosis.

The chest CT examination showed no new or residual pulmonary emboli. The scans of the thighs and legs revealed no deep venous thrombosis but did show an aberrant slip of the lateral head of the left gastrocnemius muscle impinging upon the popliteal vein and artery after arising from the posterior midline cortex of the distal femoral metaphysis (Fig. 2A).

Fig. 2A—C Case 2. **A** Axial CT scan of the distal thighs at the level of the distal femoral metaphyses shows the aberrant slip of the lateral head of the gastrocnemius muscle (*long arrow*) impinging upon the popliteal vessels (*short arrow*) in the left knee. An incidental finding is ectasia of the popliteal vein in the contralateral right knee. **B, C** Longitudinal ultrasound scans of the left popliteal fossa shows **B** patency of the popliteal vein with the knee extended and the ankle in the resting position and **C** occlusion (*arrow*) of the popliteal vein with the knee extended and the ankle plantar flexed



Ultrasound examination of the left popliteal vein with color Doppler imaging in transverse and longitudinal orientations with the knee extended showed normal flow when the patient's ankle was in the resting (neutral) position. However, complete occlusion of flow in the vein was seen when the patient performed plantar flexion of the ankle resisted by an elastic band (Fig. 2B, C). This occlusion of flow was felt to be due to compression by the anomalous slip of the lateral head of the gastrocnemius muscle. Ultrasound of the opposite leg using the same maneuvers did not show any similar compression or occlusion of the popliteal vein.

The patient was advised of these findings but he elected not to pursue surgical treatment, choosing instead to restart anticoagulation therapy with coumadin.

Discussion

In 1879 a medical student provided the initial anatomic description of variant calf muscle anatomy causing compression of the popliteal artery [13]; however, this complex went essentially unrecognized as a clinical entity until a 1959 report [14]. The term "popliteal artery entrapment" syndrome was then introduced in 1965 [6]. In 1967 it was reported that variant gastrocnemius muscular anatomy could entrap the popliteal vein as well as the artery [10].

Normally in adulthood, the medial and lateral heads of the gastrocnemius originate from the posterior cortices of the distal femoral metaphyses, just cephalad to the medial and lateral femoral condyles. The popliteal artery and

vein normally course between these two muscle heads in the midline of the popliteal fossa.

Rich et al. classified the causes of popliteal vascular compression into five anatomic types [5]. In type I the popliteal artery courses in an aberrant location, medial to the medial head of the gastrocnemius muscle. In types II through IV, accessory slips of the medial head of the gastrocnemius muscle originate from an anomalous midline location on the posterior cortex of the distal femur, instead of from the medial femoral condyle, and compress the popliteal artery. Type V refers to a case having any of the features of types I—IV but with compression of the popliteal vein as well.

Several other authors have proposed alternative classification schemes for these anatomic variants, and it is unclear which scheme is the most widely accepted [15, 16, 17]. Therefore, detailed descriptions of these specific anatomic variations remain important when reporting on cases of popliteal vascular entrapment in order to avoid potential confusion.

Popliteal venous and arterial compression has also been shown to occur in two positions that contract and bring the gastrocnemius muscle in closer proximity to these vessels: (1) with the knee hyperextended and (2) with the knee extended and the ankle plantar flexed. This position-dependent vascular compression has been termed "functional entrapment." While functional entrapment can be seen in some asymptomatic patients without thrombosis, it can

result in venous insufficiency, venous thrombosis, and arterial insufficiency in others [2, 18, 19, 21]. Postulated etiologies for functional entrapment include hypertrophy of the gastrocnemius muscle and a soleus muscle sling [18] causing vascular impingement.

The presence of functional entrapment of the popliteal vein was found in 27% of asymptomatic individuals by Doppler ultrasonography in one study [22], raising the question of whether this finding is clinically significant. Therefore the presence of position-dependent popliteal arterial occlusion in case 2, involving only the knee with the aberrant slip of the lateral head of the gastrocnemius, provides supportive but not definitive evidence that the muscle anomaly was the cause of the patient's previous deep venous thrombosis.

In a discussion of the predominant involvement of the medial head of the gastrocnemius muscle in this syndrome, Gibson et al. [23] postulated an anomaly of embryologic migration of the medial head origin from the posterior cortex of the lateral tibial plateau to the lateral femoral condyle. This hypothesis does not, however, account for the aberration of the lateral head of the gastrocnemius that we have observed in these two patients. The lateral head has been reported to be a cause of popliteal vessel entrapment in only two cases of venous narrowing (without thrombosis) reported by Nelson et al. [12] and in five cases of venous compression and lower extremity congestive symptoms by Raju and Neglen [2], also without thrombosis.

Our case 2 had findings similar to these seven previously reported cases, with popliteal venous (but not arterial) compression caused by an anomalous slip of the lateral head of the gastrocnemius muscle. Even though no thrombus was found in this patient's popliteal vein at the time of presentation to our institution, we did observe

functional entrapment of the popliteal vein by the aberrant slip of the lateral head of the gastrocnemius muscle during ankle plantar flexion. Compression of the popliteal vein without arterial compression can be explained by the more lateral location of the popliteal vein relative to the artery, and its proximity to the lateral head of the gastrocnemius.

Case 1 is the first reported case, to the best of our knowledge, in which an anomalous slip of the lateral head of the gastrocnemius muscle has caused thrombosis of the popliteal artery. Cross-correlation of the axial MR images through the distal thighs with the MR angiogram series, and the lack of any other atherosclerotic disease in the abdominal aorta and lower extremities, provided convincing evidence that the aberrant muscle slip was the cause of the thrombosis. The inability of the MRI/MRA study, but not arteriography, to demonstrate actual deviation of the popliteal vessels in this case indicates that the impingement was likely positional in nature. The degree of ankle plantar flexion on the MR studies might have been limited by the constrictive nature of the peripheral vascular receiver coil wrapped around the patient's legs.

Conclusion

An anomalous slip of the lateral head of the gastrocnemius originating from the midline posterior cortex of the distal femoral metaphysis can cause popliteal vascular entrapment. When a patient presents with occlusion of the popliteal artery or vein, especially if the clinical presentation is atypical, radiologists should evaluate the anatomy of the popliteal fossa to exclude the presence of anomalies of either the medial and lateral head of the gastrocnemius muscle.

References

1. Ring DH Jr, Haines GA, Miller DL. Popliteal artery entrapment syndrome: arteriographic findings and thrombolytic therapy. *J Vasc Interv Radiol* 1999; 10:713–721.
2. Raju S, Neglen P. Popliteal vein entrapment: a benign venographic feature or a pathologic entity? *J Vasc Surg* 2000; 31:631–641.
3. Elias DA, White LM, Rubenstein JD, Christakis M, Merchant N. Clinical evaluation and MR imaging features of popliteal artery entrapment and cystic adventitial disease. *AJR Am J Roentgenol* 2003; 180:627–632.
4. Macedo TA, Johnson CM, Hallett JW Jr, Breen JF. Popliteal artery entrapment syndrome: role of imaging in the diagnosis. *AJR Am J Roentgenol* 2003; 181:1259–1265.
5. Rich NM, Collins GJ Jr, McDonald PT, Kozloff L, Clagett GP, Collins JT. Popliteal vascular entrapment. Its increasing interest. *Arch Surg* 1979; 114:1377–1384.
6. Love JW, Whelan TJ. Popliteal artery entrapment syndrome. *Am J Surg* 1965; 109:620–624.
7. Gerkin TM, Beebe HG, Williams DM, Bloom JR, Wakefield TW. Popliteal vein entrapment presenting as deep venous thrombosis and chronic venous insufficiency. *J Vasc Surg* 1993; 18:760–766.
8. Iwai T, Sato S, Yamada T, Muraoka Y, Sakurazawa K, Kinoshita H, et al. Popliteal vein entrapment caused by the third head of the gastrocnemius muscle. *Br J Surg* 1987; 74:1006–1008.
9. Connell J. Popliteal vein entrapment. *Br J Surg* 1978; 65:351.
10. Rich NM, Hughes CW. Popliteal artery and vein entrapment. *Am J Surg* 1967; 113:696–698.
11. Edmondson HT, Crowe JA Jr. Popliteal arterial and venous entrapment. *Am Surg* 1972; 38:657–659.
12. Nelson MC, Teitelbaum GP, Matsumoto AH, Stull MA. Isolated popliteal vein entrapment. *Cardiovasc Intervent Radiol* 1989; 12:301–303.
13. Stuart T. Note on a variation in the course of the popliteal artery. *J Anat Physiol* 1879; 13:162–165.
14. Hamming JJ. Intermittent claudication at an early age, due to an anomalous course of the popliteal artery. *Angiology* 1959; 10:369–371.

15. Levien LJ. Popliteal artery entrapment syndrome. *Semin Vasc Surg* 2003; 16:223–231.
16. Hoelting T, Schuermann G, Allenberg JR. Entrapment of the popliteal artery and its surgical management in a 20-year period. *Br J Surg* 1997; 84:338–341.
17. Delaney TA, Gonzalez LL. Occlusion of popliteal artery due to muscular entrapment. *Surgery* 1971; 69:97–101.
18. Erdoes LS, Devine JJ, Bernhard VM, Baker MR, Berman SS, Hunter GC. Popliteal vascular compression in a normal population. *J Vasc Surg* 1994; 20:978–986.
19. Levien LJ, Veller MG. Popliteal artery entrapment syndrome: more common than previously recognized. *J Vasc Surg* 1999; 30:587–598.
20. Chernoff DM, Walker AT, Khorasani R, Polak JF, Jolesz FA. Asymptomatic functional popliteal artery entrapment: demonstration at MR imaging. *Radiology* 1995; 195:176–180.
21. Di Cesare E, Marsili L, Marino G, Masciocchi C, Morettini G, Spartera C, et al. Stress MR imaging for evaluation of popliteal artery entrapment. *J Magn Reson Imaging* 1994; 4:617–622.
22. Leon M, Volteas N, Labropoulos N, Hajj H, Kalodiki E, Fisher C, et al. Popliteal vein entrapment in the normal population. *Eur J Vasc Surg* 1992; 6:623–627.
23. Gibson MH, Mills JG, Johnson GE, Downs AR. Popliteal entrapment syndrome. *Ann Surg* 1977; 185:341–348.