CLINICAL VIGNETTE

Popliteal Vasculature Entrapment Syndrome due to Compression by Fluid Collection

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Case Presentation

A 67-year-old male with a past medical history of hypertension, gout, and chronic obstructive pulmonary disease presented to the emergency department with right lower extremity pain and edema for 6 weeks which worsened over the past 4 days. He described the pain as a progressive, waxing and waning stabbing pain that started in the ankle and radiated to the posterior calf. It worsens with ambulation and initially improved with ibuprofen, but is now constant. There was no history of trauma, fevers, chills, nausea, vomiting, abdominal pain, chest pain, or shortness of breath. There was no personal or family history of blood clots or hypercoagulability. His temperature was 98.5 F, pulse 91, respiratory rate 18, blood pressure 153/84, and his oxygen saturation was 100% on room air.

On physical exam, he was grimacing and appeared uncomfortable at rest. The right foot was erythematous and warm with 1+ edema. There was tenderness along posterior calf with a positive Homan's sign. Pain increased with passive dorsiflexion of the R foot. There was a 1+ posterior tibialis pulse. Motor strength and sensation were grossly intact.

His laboratories were significant for a white blood cell count of 12.75 k/uL, C-reactive protein of >8.0 mg/dL, erythrocyte sedimentation rate of 36 mm/hr, uric acid 5.4 mg/dL, procalcitonin of 4.05 ng/mL, and creatine kinase of 815 U/L. Vancomycin and ceftriaxone were started for empiric cellulitis coverage.

One hour after initial presentation, the patient developed numbness and tingling of his right foot and his right toes were cool to the touch. Right dorsalis pedis and posterior tibialis pulses could not be palpated but they were present on the left. Doppler ultrasound did not detect pedal pulses on the right side. Formal arterial ultrasound showed right extremity arterial flow, however with irregular rhythm throughout the arterial vasculature. Computed angiography of the right extremity identified a complex fluid collection measuring 4.8cm transversely, 2.6cm anterior-posterior, and 5.8cm in length around the popliteus tendon and myotendinous area. There was severe stenosis of the right popliteal artery likely secondary to the mass effect from the fluid collection. It was thought that the fluid collection was a complication of a prior subacute popliteus rupture or tenosynovitis. Incidental moderate to severe pre-existing underlying peripheral arterial disease was also identified involving the anterior tibial artery.

The patient was admitted for observation and serial neurovascular exams. Vascular and orthopedic surgery services were consulted and agreed with the diagnosis of popliteal artery entrapment syndrome. There was no clinical indication for urgent surgical intervention given a stable neurovascular exam. In addition, the symptoms were less consistent with critical limb ischemia or compartment syndrome. The right leg was kept elevated and compressed.

Magnetic resonance imaging (MRI) of the right femur and leg were then obtained and demonstrated fluid collection tracking along the popliteus muscle concerning for popliteus muscle rupture or bursitis. Prior medial meniscus injury, osteoarthritis, and a large right knee effusion were also noted. The knee effusion was thought to be a complication of several possible etiologies, including osteoarthritis, pseudogout, or previous medial meniscus injury. The fluid collection was not consistent with an abscess or diabetic myonecrosis. The knee effusion was drained, and the fluid was notable for calcium pyrophosphate crystals consistent with pseudogout.

Throughout the hospital course, the patient remained hemodynamically stable with intermittent paresthesias, stable vascular exam, improving pain, and decreased swelling of the right lower extremity. Vancomycin was continued for four days and ceftriaxone was continued for seven days, as an infectious etiology could not be ruled out. The patient was discharged with colchicine for pseudogout, and aspirin and a statin to prevent progression of peripheral arterial disease. Repeat venous duplex ultrasound at 3 months did not demonstrate a deep vein thrombosis.

Discussion

Popliteal artery entrapment syndrome (PAES) is a vascular disease that occurs when the popliteal artery is compressed in the popliteal fossa by adjacent musculotendinous structures.[1] Arterial compression decreases blood supply to the leg, leading to intermittent claudication.[1,2] PAES is rare, with an estimated prevalence between 0.17% to 3.5% of the population.[3] It is also an underdiagnosed disease,[4] with reported average symptoms of 34 months until diagnosis.[5]

PAES most often occurs in young male athletes without atherosclerotic risk factors. It has an 83% male and a 40-74% bilateral predominance.[1,5] PAES can be divided into two groups

anatomical and functional.[4] Common causes of PAES include muscular hypertrophy and congenital anatomical anomalies. Muscular hypertrophy, seen in athletes who perform weight resistance training can compress the popliteal artery, leading to PAES.[2] Congenital PAES often occurs due to embryonic anatomic variations of the popliteal artery or leg musculature. Common anomalies involve a more medial course of the popliteal artery or the attachment of the gastrocnemius medial head to the intercondylar notch.[6] PAES is rarely seen in people over 40 years old,[2] with the average age on presentation of 30.5 years.[5] There have been several case reports of older individuals with PAES due to anatomic anomalies,[6-8] complications of osteoarthritis,[9] and muscular hypertrophy.[8]

The characteristic presentation for PAES is subacute to chronic exertional cramping in the calf which usually resolves at rest. If the surrounding popliteal vein or nerve are also compressed, lower extremity swelling, numbness, and skin discoloration may also occur.[10] On exam, a bruit may be auscultated in the popliteal fossa if the popliteal artery has > 50% occlusion. Ischemic signs including pallor, coldness, and decreased pulses may be identified. Additionally, repetitive plantarflexion may elicit calf pain.[4]

Diagnosis of PAES can be challenging because it is an underrecognized cause of exertional leg pain and initial physical exam is often normal. Diagnostic testing for PAES includes ultrasound, Magnetic resonance angiography (MRA) or computed angiography (CTA) and angiograms. Duplex arterial ultrasound may demonstrate decreased systolic flow, especially when the foot is plantarflexed and the knee is flexed.[10] Doppler ultrasound, MRA and CTA without a use of provocative maneuvers can result in false negative.[1] The ankle-brachial index, although somewhat unreliable,[4] may sometimes show decreased ratio to below 1.0 during exercise.[2] MRA and CTA may demonstrate evidence of compression, thrombosis, or aneurysm of the popliteal artery oftentimes with an accompanying muscular anomaly. If diagnosis of PAES remains unclear after imaging, angiography, can demonstrate focal narrowing of the popliteal artery and is highly diagnostic in 97% to 100% of cases.[11]

Treatment of PAES for most symptomatic patients is often operative.[4] Popliteal vasculature compression can be dangerous and ultimately lead to aneurysms, stenosis, thrombosis, or embolic events and can lead to limb ischemia.[10] These complications may result in permanent damage of the affected limb.[2] Operative treatment involves decompression of the popliteal fossa through partial fasciotomy, myotomy, excision of fibrous bands, or partial removal of the gastrocnemius medial head.[4,10] Bypass grafting with vascular surgery can be performed if there is irreversible vessel wall damage.[10] Anatomical causes of PAES generally respond well to surgery, while muscular hypertrophy has less consistent results.[4]

Conclusion

This case highlights the presentation and management of acquired PAES due to extrinsic fluid compression in an older male. Interestingly, our patient did not have anatomic anomalies or muscular hypertrophy, but rather a fluid collection around the popliteus muscle that was thought to cause vasculature compression. The etiology of the fluid collection remained unclear. It is possible that any of the patient's knee pathology, including medial meniscus injury, osteoarthritis of the knee, or pseudogout could have led to a popliteal cyst complicated by possible popliteus rupture. The patient did not require surgery, with symptoms improving throughout hospitalization. Improvement of symptoms could have been due to compression of the leg or possibly decreasing size of the fluid collection as it was naturally resorbed. While rare, this patient's presentation exemplifies the importance of considering PAES and maintaining a broad differential when a patient presents with leg swelling and pain.

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