Popliteal Vein Aneurysms:
The Diagnostic and Surgical Dilemma

Roger L. Flekser, BSc, MBBS; Walid Mohabbat, MB BS (Syd) FRACS (Vasc) From North Shore Vein Clinic, Sydney, Australia, and Specialist Vascular Clinic, Sydney, Australia.

ABSTRACT: Primary venous aneurysms are exceedingly rare. Despite their paucity, popliteal venous aneurysms (PVAs) represent a potential source of thromboemboli and can therefore be associated with fatal or near-fatal pulmonary emboli. Here we report a case of a large popliteal aneurysm that resulted in a pulmonary embolus. The diagnosis, and more importantly surgical management, of PVAs remain uncertain. This case highlights the diagnosis and surgical approach to such aneurysms.

VASCULAR DISEASE MANAGEMENT 2015;12(2):E26-E32
Key words: Popliteal venous aneurysm, pulmonary embolism, thrombolysis, aneurysm repair, lateral venorrhaphy

Primary venous aneurysms are exceedingly rare. While the overall incidence of popliteal venous aneurysms (PVA) are unknown, the male to female ratio is equal and they are usually found in patients between the ages of 50 and 59. Most PVAs are asymptomatic and detected on routine investigation for varicose veins. However, they are a potential source of thromboemboli and can therefore be associated with fatal or near-fatal pulmonary embolism (PE). Here we report a case of a PE in a patient who was found to have a large popliteal vein aneurysm.

The Case

A 65-year-old woman who was on vacation was found collapsed but conscious on the floor by her neighbor. Emergency services noted on arrival that she was diaphoretic with a respiratory rate of 23 breaths per minute and oxygen saturation of 86%. She was afebrile with a heart rate of 78 beats per minute and a blood pressure of 140/86 mmHg. She had a history of hypertension. Three weeks prior to her collapse she developed severe plantar fasciitis of the left leg requiring complete immobilization with a Controlled Ankle Movement Walker boot.

On arrival in the emergency department her oxygen saturation was 96% on 6 liters of oxygen delivered by face mask. Her other vitals were normal. She had decreased air entry at both her lung bases. The remainder of her exam, including examination of her lower limbs, was normal.

Given her dyspnea, recent air travel and immobile foot, a computed tomography (CT) pulmonary angiogram was performed. Bilateral acute pulmonary embolism were noted involving segmental arterial branches of the right
and left lower lobes (Figure 1). A venous duplex scan of her legs, however, showed no evidence of deep vein thrombosis or PVA.

The patient was transferred to the coronary care unit and started on an intravenous unfractionated heparin infusion. During her hospitalization, she had a battery of tests including a transthoracic echocardiogram, which was unremarkable. She was also screened for inherited and acquired coagulation disorders (factor V Leiden and prothrombin G20210A mutations, deficiencies of protein C, protein S and antithrombin, hyperhomocysteinemia, the antiphospholipid syndrome and increased levels of fibrinogen) without detection of any thrombophilia.

One week after the patient presented to the hospital, she was discharged on warfarin (international normalized ratio [INR] 2-3) and the etiology of her pulmonary emboli was unknown.

Four months after the patient was discharged from the hospital, she presented to our institution with a history of pain in her right calf. A routine venous duplex study revealed an unexpected partially thrombosed 3-cm right popliteal vein aneurysm. To further elucidate this unexpected finding, the patient went on to have a CT scan of her abdomen and lower limbs to confirm the presence of the aneurysm and to rule out other potential pathologies (Figure 2).

On further examination, we determined that the etiology of the pulmonary emboli was most likely the popliteal vein aneurysm. To reduce the risk of further pulmonary emboli it was decided to proceed with surgical repair of the aneurysm. With the patient’s consent, she preoperatively had a Celect inferior vena cava (IVC) filter (Cook Medical) implanted.

The patient received catheter-directed thrombolysis before proceeding to open surgical repair of the aneurysm. In the prone position, the ipsilateral short saphenous vein at the ankle was cannulated using a 21G micropuncture needle and sheath (Cook Medical). The micropuncture sheath was exchanged for a 5 Fr 11-cm Brite Tip sheath (Cordis Corporation). With the aid of a 0.35” Glidewire (Terumo) and Glidecath (Terumo), the saphenopopliteal junction was traversed. The support-
ing glide catheter was removed and a Cragg-McNamara Valved Infusion Catheter (Covidien) was placed in the distal superficial femoral vein. A venogram confirmed the correct placement of the catheter. This was followed by 100,000 units of Urokinase as a pulse spray into the partially thrombosed popliteal vein. Completion venography post Urokinase demonstrated a large 3-cm saccular aneurysm without evidence of thrombosis (Figures 3A and 3B).

After the catheter thrombolysis, while in the prone position, the aneurysm was exposed through a lazy “S” posterior knee incision. A surgical plane was carefully developed between the popliteal artery and vein. Once the vein was isolated and looped, 5,000 IU of intravenous unfractionated heparin was administered. The venous sac was subsequently opened via longitudinal venotomy. This was followed by a thrombectomy of the popliteal vein. Having removed the majority of the thrombus from the sac and ascertained that inflow and outflow of the vessel were adequate, a tangential aneurysmectomy and lateral venorrhaphy were performed to reduce the size of the aneurysmal sac. Vessel closure was carried out by a 6/0 polypropylene direct suture of the residual venous sac. A 10 belovac drain was placed in the popliteal fossa.

Six hours postoperatively, an unfractionated heparin infusion was started. Subsequently the patient was started on warfarin again. Her postoperative course was uneventful. She was discharged home on warfarin (INR 2.5) and class 2 compression stockings. Three-, 6-, and 12-month follow-up reviews with arterial and venous duplex scans have been normal with no discernible DVT or aneurysmal dilatation of the left popliteal vein. The IVC filter was removed 4 months postoperatively and the warfarin was subsequently ceased at 5 months.

**DISCUSSION**

Popliteal aneurysms are rare. The first case of a popliteal vein aneurysm was reported in the literature in 1968 by May and Nissel. By 2006 there had been a total of 105 cases reported in the world literature.
Perhaps because of their rarity, there is some conjecture as to what constitutes a venous aneurysm. McDevitt et al3 suggested an isolated venous dilatation twice the size of a normal vein as aneurysmal, while Maleti et al defined venous aneurysms as three times the size of normal.4 Approximately 75% of these aneurysms are saccular and 25% are fusiform.5

Various etiologies associated with popliteal venous pathology have been suggested including congenital, trauma (e.g. arteriovenous fistula), varicose veins, localized degenerative changes, and inflammation. In our patient, however, none of these pathologies were identified. However, one mechanism described by Aldridge et al6 best reflects the underlying anatomical pathology and histological findings of our patient – endophlebohypertrophy, progressive intimal proliferation of elastic, muscle, and connective tissue. This is a process not dissimilar to atherosclerosis. Endophlebosclerosis is a loss of elastic fibers and medial muscle cells.

While the vast majority of popliteal venous aneurysms are mostly asymptomatic, their prevalence is unknown as there are no screening studies for patients with healthy veins. Winchester et al found a 70% to 80% pulmonary embolism rate in those patients initially diagnosed with PVA.7 Other symptoms include a popliteal mass, pain and swelling in the popliteal fossa, pain secondary to tibial nerve compression or superficial thrombophlebitis. Less common presentations include CVA (paradoxical PE) claudication or even sudden death.8

The diagnosis of popliteal venous aneurysms can be challenging. Physical examination is not helpful unless the aneurysm is large enough to be palpated. Plethysmography and hand-held Doppler examination are unhelpful as there is usually no venous outflow obstruction. Duplex sonography is the most frequently used noninvasive imaging modality because it can define an aneurysm topographically, giving details of its shape, its diameter, and the presence or absence of thrombus.9 It is imperative that the duplex be performed by an experienced vascular ultrasonographer. Unfortunately as is evident in this case, the initial ultrasound was undertaken in a small regional hospital with a less experienced vascular ultrasonographer and as a result the aneurysm was not diagnosed. Multiple longitudinal images were acquired of the popliteal vein with very few images acquired in cross section.

It is possible that because of the anatomical nature of the aneurysm with thrombus lining the dilated area of the vein, in the longitudinal view the diameter of the vessel may have appeared normal as one scanned from the femoral vein through to the distal popliteal vein. This can be overcome by scanning the vessel in cross section with and without compression. Also, color duplex can be very useful when trying to rule out popliteal aneurysms. The unusual appearance of “blood swirling” or “aliasing” within a vein during augmentation may be a subtle clue of aneurysmal pathology. It is essential that patients be scanned standing (if possible) as this can accentuate abnormal vessel hemodynamics as the vein becomes distended. Initially the patient in this case was scanned in the supine position, with the vein “collapsed,” which also may explain why the aneurysm was not diagnosed. It is our preference to obtain a second imaging modality particularly when the presentation is unusual. Although computed tomography (CT) scanning and magnetic resonance imaging (MRI) are important noninvasive radiologic options, we and others10 believe that venography is mandatory to precisely define
the venous anatomy before surgical repair, particularly in patients with deep vein thrombosis.

The management of asymptomatic popliteal venous aneurysms, including anticoagulation therapy and close surveillance, is controversial. Anticoagulation alone has been shown to be ineffective in preventing PE, with up to 80% recurrence rate and death from recurrent PE despite anticoagulation. It is for this reason that some institutions advocate surgery. Other institutions, particularly in cases of fusiform aneurysms less than 20 mm in diameter, will only offer surgical intervention when patients present with thromboembolic symptoms.

There seems to be consensus that in symptomatic patients with PE and concomitant PVA, particularly those patients with saccular aneurysms greater than 2 cm in size, that surgery is the treatment of choice. It is unclear from the literature whether the insertion of an IVC filter preoperatively reduces the morbidity and mortality of patients undergoing surgery. However, it is the authors’ opinion and the opinion of others that prophylactic IVC filter placement can reduce the risk of PE during deep vein aneurysm surgical repair particularly in cases such as the one described here where there is evidence of a free-floating “tongue” of thrombus that could potentially embolize during manipulation of the popliteal vein (Figure 3A).

Our decision to proceed with catheter-directed thrombolysis before open surgical repair was made based on several factors. One, an attempt was made to reduce the large, precarious thrombus load within the aneurysm thereby reducing the associated risk of embolization during open repair while manipulating the popliteal vein. Two, having adequately cleared the majority of thrombus from the aneurysmal sac, a clearer insight can be gained into the anatomic pathology of the vein in question, thereby allowing a safer approach to open repair. Three, the venogram also afforded the authors an opportunity to image the entire venous vasculature, not only of the affected leg but also both common iliac veins, the IVC, and the left leg. In so doing we were able to rule out other potential venous aneurysms or pathologies that may have led to the patient’s initial presentation that may not have been evident on the ultrasound or CT scan.

Several surgical procedures have been reported. A posterior approach to the popliteal fossa is feasible in most cases. However, one limitation is proximal control of the vessel, particularly in larger aneurysms. While most aneurysms can be resected and the residual sac closed with lateral venorrhaphy, the large, more fusiform aneurysms may require reconstruction. In this instance a vein patch or an anatomical or preferable an extra anatomical vein bypass may be required. Ligation or resection of the aneurysm is not an attractive option as this may result in significant deep vein insufficiency and swelling. Endovascular treatment of popliteal vein aneurysms has not yet been described.

Postsurgical complications have been described and include hematomas, thrombosis of the popliteal vein repair, common peroneal or tibial nerve palsies, wound infection, and fusiform dilation of the venous anastomoses.

While most patients in the literature receive some form of anticoagulation postoperatively, the anticoagulant used and its duration varies. In the literature, most patients received oral anticoagulation for 3 months to 6 months. Sessa et al recommended compression stockings and low molecular weight heparin for 3 weeks after
surgery. We opted for oral anticoagulation therapy for 5 months combined with class 2 graduated compression stockings (23–32 mmHg).

In conclusion, popliteal venous aneurysms are rare. The diagnosis should be considered in patients without thrombophilia and where deep vein thrombosis is not immediately obvious. Symptomatic aneurysms greater than 2 cm, particularly saccular aneurysms, should be treated operatively as thrombotic material in the aneurysm can embolize with fatal outcomes.

Editor’s note: Disclosure: The authors have completed and returned the ICMJE Form for Disclosure of Potential Conflicts of Interest. The authors report no financial relationships or conflicts of interest regarding the content herein.

Manuscript submitted August 22, 2014; provisional acceptance given October 17, 2014; final version accepted November 7, 2014.

Address for correspondence: Dr Roger L. Flekser, BSc, MBBS, North Shore Vein Clinic, 303/156 Pacific Highway, St. Leonards, Sydney, NSW 2065, Australia. Email: rflekser@optusnet.com.au

REFERENCES


