Chronic ulceration following surgical excision of a Morton's neuroma due to an underlying arteriovenous malformation of the foot

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An arteriovenous malformation (AVM) of the foot is a rarely encountered clinical pathology. Arteriovenous malformations are most commonly found in the brain and lungs, and when found in the lower limbs, are usually in the pelvis or thigh and not the foot or ankle. We describe a rare malformation that complicated surgical removal of a Morton’s neuroma, with chronic ulceration and failed wound healing. Successful treatment with conservative measures ensured full recovery. We believe all health care professionals should be aware of this pathology and consider it as a cause of delayed healing.

Keywords: arteriovenous malformation, foot, chronic ulceration, delayed wound healing

We present the case of a 38-year-old bus driver who presented with a non-healing ulcer of the left foot following the removal of a Morton’s neuroma 10 months prior at a community allied medical institution unrelated to ours. Initially, he presented in the community with ongoing focal pain in the 2nd and 3rd metatarsophalangeal joint web-space and was treated conservatively. With no resolution in pain and a slow growing mass on the dorsum of his left foot, an ultrasound scan was undertaken which confirmed a 10mm neuroma in the second web space. Two injections of steroid/local anaesthesia provided temporary relief but were subsequently followed by surgical removal of the neuroma. The wound never healed with constant ooze and further surgical debridement was undertaken in the community, accompanied by multiple courses of oral antibiotics. He had been unable to work since the neuroma removal and at 10 months post-removal he was referred for review at our institution.

He now revealed that he had had a right hip replacement at age 34 for traumatic osteoarthritis, and left testicular vein embolisation for varicocele. Physical examination of the left foot revealed a bleeding 0.5x0.5cm ulcer on the dorsum between the 2nd and 3rd web spaces (Figure 1). A dusky, pulsatile mass of 3cmx3cm was noted underlying the ulcer with surrounding hemosiderin deposition and a palpable thrill was present. Neurovascular and musculoskeletal examination were unremarkable and there were no abnormalities to the right foot.

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Figure 1 Photographs of skin appearances of the left foot 1 week after presentation. Dorsum – visible features are a healing ulcer between the 2nd and 3rd digits, hemosiderin deposition and engorged veins. Sole - ulceration and a dusky swelling are evident.

Figure 2 CT angiogram of lower limbs demonstrating distended veins throughout the left lower limb with a leash of abnormal vessels from the knee to the foot.

Figure 3 T1-weighted MR sagittal image of the left foot demonstrating the nidus of the AVM with an engorged draining vein.

A Doppler scan was performed which detected fully patent arteries throughout the left limb with triphasic flow with a prolonged diastolic component, suggestive of hyperaemia. A computed tomography (CT) with contrast and magnetic resonance angiogram (MRA) confirmed a low flow vascular malformation (Figures 2-4). These images revealed an enhancing soft tissue mass lesion with feeding vessels overlying the left second and third metatarsals, extending into the plantar soft tissues and a leash of abnormal vessels from the knee (Figure 2).

Angiography was undertaken demonstrating a large arteriovenous malformation (AVM) around the second toe with multiple feeding vessels (Figure 5). This was deemed unsuitable for selective endovascular embolisation due to congestion. The patient was successfully treated with limb elevation and Class 1 compression stockings: three months later the patient reported great clinical improvement and had returned to work.
An AVM is an abnormal collection of blood vessels allowing direct communication between arteries and veins. The incidence is between 1-10/100000 and they are commonly found in the brain & trunk [1] and arise from congenital malformations, trauma and degenerative vascular diseases. Their growth is exacerbated by local trauma and hormonal changes, such as puberty and pregnancy [2]. Most are isolated pathologies, but multiple malformations are associated with syndromes such as Osler-Weber-Rendu, Sturge-Weber and Klippel-Trenaunay [3].

The symptoms are dependent on locality and size and most remain small and clinically silent [3]. Symptoms occur due to effects on surrounding structures, such as bone, muscle and soft tissue and include pain, swelling, ulceration and deformity, and can be debilitating. If left untreated, chronic ulceration may cause haemorrhage or necrosis.

Clinical presentation can include a pigmented lesion, or birthmark, visible pulsation, or palpable thrill and engorged veins (varicosities). Due to the local skin appearances, and rarity, the diagnosis can be mistaken for dermatological pathologies such as angiodermatitis or Kaposi’s sarcoma, or vascular tumours and chronic infection [3,4]. In this case the prolonged time to heal, ongoing ooze and bleeding from the surgical site were the findings on presentation. It is unclear whether the AVM preceded surgery but certainly was recognisable in the form of a pulsatile mass at presentation to our clinic. Surgical intervention for neroma excision may have exacerbated the AVM, given its extensive nature.

Initial investigation includes radiography to evaluate surrounding bony structures for erosion [3]. Color Doppler ultrasonography is a useful initial non-invasive investigation for evaluating location, size and flow of the AVM. CT with angiography will visualise bone and tissue involvement, calcification, the presence of thrombi and size. However, magnetic resonance imaging is particularly important in providing imaging of adjacent soft tissue structures that can be involved. Using dynamic gradient pulse sequences in MR is useful in evaluating whether the lesion is high flow or low flow [5].

Discussion

We demonstrate the presentation of an incidental rare underlying pathology complicating a simple procedure. A high degree of clinical suspicion is needed to avoid missing this diagnosis. Although AVMs do occur in the lower limbs, there are very few case reports in the literature of malformations arising so distally, in the foot. Therefore this is an unusual pathology that all foot and ankle practitioners should be aware of and should understand its management, as conservative treatment can provide a low cost and highly efficacious treatment.
Interventional therapies include ligation of feeding vessels, surgical excision, sclerotherapy or embolisation with coils/particles [6-8]. However, as demonstrated here, low-flow AVMs respond well to conservative measures, such as limb elevation and graded compression stockings.

This case is unusual as to why an extensive AVM should arise. It is quite possible the surgical intervention for neuroma excision gave rise to the AVM or exacerbated the AVM, but given its extensive nature, it would most likely have been present prior to surgery. It demonstrates the importance of appropriate clinical examination and high suspicion when delayed healing has complicated a simple wound. It highlights the importance of understanding wound pathology and the role of simple clinical management to healing.

References