Venous ulceration: understanding the commonest lower limb wound

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This case demonstrates the importance of treating the underlying pathophysiology and discusses the imaging in the treatment of venous ulcers. The patient presented with a history of a non-healing ulcer for over five months. The patient attended a wound clinic for dressings and a negative-pressure wound therapy (NPWT) dressing with no decrease in ulcer size. When the patient presented to our clinic, a duplex ultrasound (DUS) imaging was done to understand the underlying pathophysiology. The findings were deep and superficial venous reflux and obstructive disease (May–Thurner syndrome). Combined procedures to treat obstructive and superficial venous reflux were performed. Six weeks after the treatment the ulcer had completely healed with the use of compression bandaging.

Keywords: venous ulceration, lower limb wound, May–Thurner syndrome

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Introduction

Venous leg ulcers (VLU) are a common chronic problem in many countries especially in Northern Europe and America. The overall prevalence of this condition is 1% rising to 3% in the over 65 years age group. Over the last 25 years, there have been many developments applicable to its diagnosis and treatment. These advances, notwithstanding healing response and recurrence, are variable, and the venous ulcer continues to be a clinical challenge. Chronic VLUs result in reduced mobility, significant financial implications and poor quality of life.[1]

The pathogenesis of venous ulcers is unrelieved ambulatory venous hypertension resulting mostly from deep venous thrombosis leading to venous incompetence, lipodermatosclerosis, leucocyte plugging of the capillaries, tissue hypoxia and microvascular dysfunction.[1] It is therefore important to treat the underlying pathogenesis to accelerate ulcer healing and prevent recurrence. Venous pressure in the lower limb should decrease with walking but with venous hypertension, the pressure does not decrease by much and quickly rises again. May–Thurner syndrome (MTS) and chronic obstructive disease resulting from the post-thrombotic syndrome are examples of where the pressure increases with walking.

Post-thrombotic syndrome (PTS) is a condition that may occur after one experiences a deep vein thrombosis (DVT). Pulmonary embolus (PE) is an acute and life-threatening event which is a complication of DVT, while PTS is the major chronic condition that can affect quality of life. PTS can be debilitating and is often unrecognised and inadequately managed in patients with DVT.

After a DVT has occurred, one may experience different outcomes in the leg within the following six months – the thrombus may completely resolve; the thrombus may chronically obstruct the deep vein or stenose it (*obstructive deep disease)*; or it may damage the valves within the vein causing venous incompetence *(reflux deep disease)*.

Thus, deep venous pathology may be obstructive, refluxing or a combination. Extrinsic compression of veins within the pelvis – such as iliac compression/MTS will cause outflow disease/obstructive disease.

Superficial venous disease resulting from incompetent valves may contribute to complications such as skin damage and wounds.

All the venous pathologies may result in increased venous hypertension with the pattern described above.

Diagnosis of VLUs is based on a clinical examination followed by ultrasound Doppler measurement of ankle-brachial systolic pressure index (ABI or ABPI) to exclude arterial disease.[2] With the improvement in DUS technology and sonographer training, it is now possible to correctly diagnose MTS syndrome with DUS. This is important as DUS is relatively cheap and readily available in many medical institutions.[3,4]

In the treatment of venous ulcers, it is imperative to understand the underlying pathophysiology, which areas are affected and which should be surgically treated:

Suprainguinal refers to the deep pelvic and abdominal veins – iliac veins, inferior vena cava (IVC), renal and ovarian veins.

Infrainguinal can be divided into:

- Superficial – great saphenous vein and its branches. The small saphenous vein and its branches.
- Deep – deep veins follow the leg arteries.

It has been shown that treating superficial venous disease and compression can expedite ulcer healing and prevent ulcer recurrence.[5] The treatment of this includes radiofrequency ablation (RFA), ultrasound-guided foam sclerotherapy (USGFS), mechanochemical ablation, thrombectomy, endovenous stent placement and venoplasty.[5]

The authors describe a case that illustrates the importance of the investigations mentioned above and the favourable outcome that can be achieved.

Case report

A 25-year-old female presented to our vascular clinic in July 2019 with a history of recurrent DVT of the left lower limb which started in March 2012 and had been treated with warfarin for five years. The patient stopped taking warfarin in 2017 and subsequent bilateral DVTs of the common femoral vein. Of note is that she has a family history of thrombophilia and has Factor V Leiden and prothrombin 20210A, which are both genetic conditions that increase a patient's risk of developing blood clots. These were diagnosed in 2017.

The patient presented with an ulcer on the left lower limb which had been present for more than ten months and was associated with left lower limb swelling (Figure 1). The patient had also been diagnosed with erythema nodosum and fibromyalgia. She had been attending wound care and eventually had a NPWT dressing applied which did not help in ulcer healing, and she was referred to the vascular clinic.

Clinical examination

Clinically, the patient had oedema of the left lower limb, posterior calf varicosities and an ulcer with a diameter of 2.5 cm x 2 cm. The ulcer was not infected and did not look ischaemic. Pedal pulses were present and normal. The ulcer was located on the left medial mid-calf area. According to the venous classification system of 'Class, Etiology, Anatomy and Pathophysiology' (CEAP), the patient had CEAP Class 2, 3, 6 indicating varicosities, oedema and an open ulcer.

Investigations

A duplex Doppler ultrasound (DUS) was performed and showed compression of the left common iliac vein between the spinal column and right common iliac artery (suggestive of MTS), synechiae in the left common iliac vein (CIV) and left external iliac vein indicating previous thrombosis. The compression was significant. The DUS also showed bilateral lower limb superficial venous reflux and left lower limb deep venous reflux. A computed tomography (CT) venogram was also performed and confirmed the left common iliac vein compression.

An intra-vascular ultrasound (IVUS) and venogram confirmed a very tight compression of the left CIV.

Diagnosis

The patient was diagnosed with PTS as a result of MTS, with underlying severe venous hypertension from a combination of deep venous obstruction, deep venous reflux and superficial venous reflux.

Treatment

The patient was planned for iliac vein stenting and bilateral RFA and bilateral USGFS as well as some balloon venoplasty for some areas.

A stent was placed in the left CIV and the superficial venous disease was treated with RFA and USGFS. Co-flex zinc compression bandage was applied to left lower limb. The patient was also started on a direct oral anticoagulant.

The aim of the treatment was to relieve lower limb venous hypertension (caused by venous obstruction) and venous reflux, which would help expedite the healing of the ulcer.

Follow-up

The patient was followed up a week after surgery. A DUS was performed and showed that the stent was patent, all the treated superficial veins were sealed, and the deep veins were patent with no DVT. The ulceration was clean and granulating. A new co-flex zinc compression bandage was applied. The patient was then followed up weekly for four weeks and the compression bandage was re-applied every week. There was good progress (Figure 2). By the sixth week, the wound had healed (Figure 3).

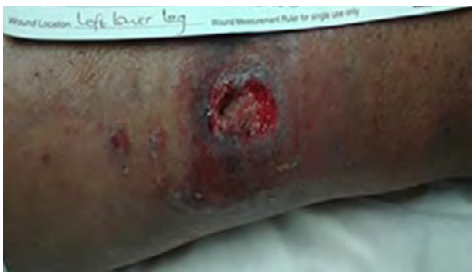

Figure 1: Ulcer on presentation

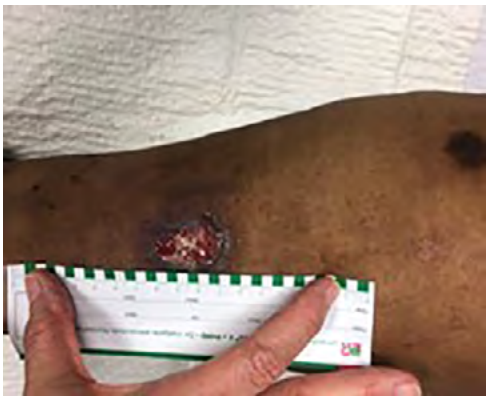

Figure 2: Ulcer one week post-op indicating increased granulation tissue formation

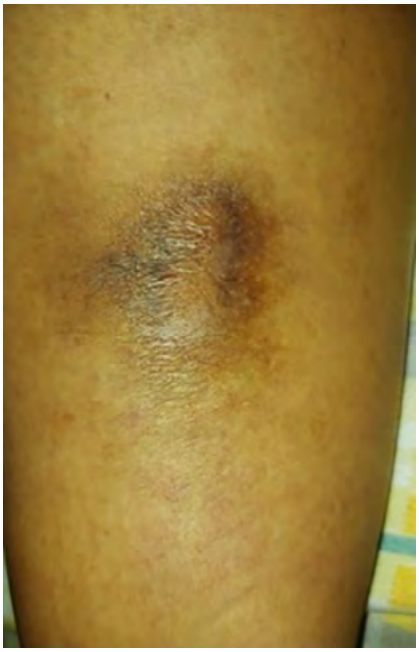

Figure 3: Ulcer six weeks post-op

Discussion

May–Thurner syndrome is a rarely diagnosed condition. It is estimated that in two out of five individuals with symptomatic lower extremity venous disorders, MTS is the underlying cause.[4] MTS is an anatomical variant leading to venous outflow obstruction due to extrinsic venous compression in the iliocaval territory.[4] The compression is usually caused by extrinsic compression of the venous system by the arterial system against a bony structure in the iliocaval territory. As was the case in this patient, the most common variant of MTS is the compression of the left CIV by the overlying right common iliac artery (CIA) against the fifth lumbar vertebrae.[4]

Obstruction may be asymptomatic but can progress to result in symptoms related to chronic venous hypertension or result in the development of venous occlusion with or without DVT.

The risk factors for developing MTS are the female gender, scoliosis, dehydration and hypercoagulable disorders.[4]

Our patient was at high risk for developing MTS as she was a young, obese female and had hypercoagulable disorders. She progressed to develop every combination of venous pathology causing venous hypertension:

- Deep obstructive and reflux disease.
- Extrinsic compression and venous outflow obstruction.
- Superficial vein disease.

The extrinsic compression and superficial venous reflux can be treated as described above. Elements of PTS in the deep system in the leg, such as deep reflux and deep obstruction, cannot be sufficiently treated once problematic. Some newer surgical techniques are being attempted but nothing will cure the deep venous disease and a patient will need lifelong compression.[6] In this case, we treated the extrinsic compression with a stent, the superficial disease with radiofrequency and some of the deep obstructive disease was treated with balloon venoplasty. The aim of doing this is to optimise venous physiology as much as possible.

The importance of investigating the suprainguinal veins in addition to infrainguinal superficial and deep veins in all patients with advanced disease cannot be disputed. CEAP Class 3 to 6 – referring to oedema, skin changes (hyperpigmentation, venous eczema and lipodermatosclerosis) and wound formation – and previous DVT fall into the advanced category.

Early diagnosis and management of DVT to try and prevent PTS is vital. This includes the use of compression and if swelling is marked, lymph drainage for the phlebolymphoedema.

Venous ulcers can take anywhere from three months to over a year to heal. It is important to accurately diagnose MTS and offer treatment in order to expedite the healing of the ulcer and to prevent further recurrence.

This patient's ulcer was healed six weeks after treatment with only co-flex zinc compression bandage used even though the patient still had deep vein reflux. The patient will still need to wear a lifelong compression stocking as she has deep venous reflux and valve surgery of the deep veins is still in its infancy.[6]

Lymph drainage may have expedited the healing even more so but this was not done at the time as the ulcer was responding well to the zinc bandaging on weekly visits. Compression will reduce the rate of recurrence and the patient will need to remain on anticoagulation and continued lifelong surveillance.

Conclusion

An understanding of the venous system and its pathology, as well as methods of intervention, has advanced exponentially in recent years. As demonstrated in the reported case, intervention of obstructive or refluxing veins is beneficial in advanced disease to normalise the physiology as much as possible. By achieving this, oedema can be reversed, skin changes can be halted, wounds healed and overall quality of life vastly improved.

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