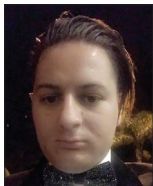


A 3-year leg ulcer challenge solved in 3 months



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A complex patient with multiple disease states knocks at my door looking for solutions to a troubling medical issue. “Am I up to the task?” you may ask. “Can I help her attain a satisfactory outcome and improve her quality of life?” This case study outlines the clinical approach and successful management of a woman with rheumatoid arthritis who presented with a leg ulcer of 3 years’ duration.

Venous leg ulcers are the most common cause of leg ulcers, with a high risk of becoming chronic, significantly impacting mobility and quality of life. The primary pathology is venous hypertension, which can be caused by previous thrombosis, perforator, superficial or deep vein insufficiency or failure of the calf muscle pump. The development of venous hypertension leads to fibrin deposition, uncontrolled inflammation and dysregulation of cytokines, causing poor capillary function and tissue hypoxia.

The nurse clinician needs to recognise the poor healing tendency of these wounds and think beyond dressings, because dressings alone will not heal the wound (World Union of Wound Healing Societies [WUWHS], 2016). Wound healing needs to be augmented with a systematic approach which recognises the complexity of the pathophysiology.

This report summarises the clinical approach and successful management of a patient who presented with a leg ulcer of 3 years’ duration.

Case report

Mrs A presented as a new patient to a wound care nurse working in general practice with a leg wound that had been present for 3 years and that had deteriorated in the previous 6 months. Her previous GP had retired.

Mrs A was 72 years of age and had long-standing rheumatoid arthritis complicated by significant joint deformities and rheumatoid nodules. A large rheumatoid nodule has been resected from around the humeral part of her radial nerve. A left hip replacement where an acetabular protrusion required disimpaction

was complicated by inadvertent compression of the left common peroneal nerve, with resultant neuropathic pain, for which she was taking tramadol (300 mg per day).

Mrs A was taking disease modifying anti-rheumatic drugs. She had developed steroid-induced type 2 diabetes of 4 years’ duration and was on insulin. Her lifestyle has been greatly affected by the presence of the ulcer and she was no longer driving or managing her usual activities of daily living, such as tying shoe laces. She was a non-smoker and did not consume alcohol.

Initial assessment and examination

A comprehensive lower limb assessment was undertaken, addressing limb structure, skin colour and warmth, circulation and capillary refill, range of movement, oedema and sensation.

The structure of both lower limbs was intact. The skin on the right leg appeared fragile and dry with normal tone. The skin on the left leg appeared to be dry, with scars and a wound present. The left leg skin showed brown pigmentation consistent with haemosiderin staining, from knee to metatarsal heads. Neither limb showed any colour changes on elevation. Both limbs were warm to touch, with no differentiation between the limbs.

No visible oedema was apparent on the right leg. Oedema was present on the left from the ankle to the knee, while the lower leg demonstrated significant calf muscle wasting. The dorsalis pedis and posterior tibial pulses were present bilaterally. The range of motion in the right knee, ankle and great toe were



Figure 1. The wound before treatment commenced.

decreased, but markedly reduced in the left knee, ankle and great toe due to joint deformity and arthritis.

Sensation tested using a 10 g monofilament found no sensory deficits (WUWHS, 2016). However, Mrs A described patchy burning and tingling dysaesthesia intermittently in the left leg. A pain assessment was completed using a validated pain tool (Gregory, 2019). Pain assessment identified deep, dull pain on both legs rated 7/10, reducing to 3/10 with medication. Capillary refill on the dorsum of the foot and toes was less than 3 seconds on both feet.

The ankle-brachial pressure index (ABPI) was 1.16 on the left leg and 1.02 on the right leg. No wound biopsy was undertaken as it was felt unnecessary in view of ABPI. Pedal pulses were present and biphasic bilaterally via Doppler.

Ulcer examination

The ulcer was located on the left lower gaiter region near the lateral malleolus [Figure 1]. The wound measured 7 cm x 6 cm, with a depth of 2.5 cm

The wound bed contained a large amount of non-viable tissue (90%) with pale red granulation/epithelial tissue (10%) around the wound margins. The wound bed was uneven with deep indentations. The edge of the wound was irregular but not overly raised. There was no tunnelling or undermining and no bone, tendon or ligament was exposed. The wound was exuding a large amount of pale yellow exudate, which was malodorous and indicative of possible localised infection or critical colonisation (WUWHS, 2019). A

region of 2–3cm around the wound showed erythema, but there was no clear evidence of cellulitis. The surrounding skin was macerated with skin shearing away from macerated tissue. There was evidence of some hyperkeratosis and haemosiderin staining.

Investigations

Mrs A's albumin levels were within normal limits. Her HbA_{1c} was 7.5% and haemoglobin was 128 g/l with a ferritin of 60 ug/l .

ABPIs were performed, showing 1.16 left, and 1.02 right. A normal range is 1.0–1.4 and values above 1.4 suggest noncompressible arterial calcification (Wounds UK, 2019).

Clinical impression and provisional diagnosis

As venous ulcers are more commonly found on the medial leg, the lateral placement of this ulcer required careful clinical evaluation. The wound was shallow with irregular sloping edges with haemosiderin staining, suggestive of a venous aetiology. The normal ABPI measurements supported normal peripheral arterial perfusion.

A provisional diagnosis of venous insufficiency was made. Short-term management goals were formulated to improve wound healing by aiming to:

- Reduce the bacterial burden in the wound within 2 weeks
- Reduce the exudate and oedema
- Decrease associated malodour and pain.

Long-term goals were to:

- Obtain closure of the wound
- Gain compression therapy concordance
- Prevent recurrent ulceration.

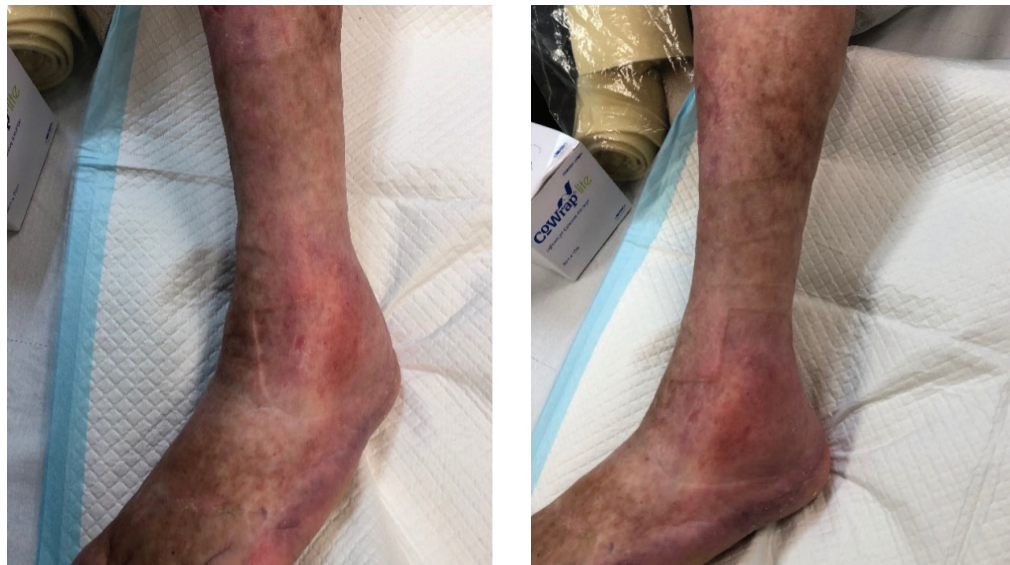


Figure 2. The wound 3 months after treatment commenced.

Wound management

The previous management plan was Betadine application covered with Melolin and changed every 2 days.

The proposed new management plan was as follows: the wound was soaked for 15 minutes with Prontosan, which is a unique combination of betaine surfactant and polyhexamethylene biguanide (PHMB) antimicrobial solution (Bradbury and Fletcher, 2011). This was followed by mechanical cleaning (Wounds UK, 2013).

Wound bed preparation used a single use curette to disrupt biofilm, followed by cleansing with moistened gauze to remove residual devitalised tissue and skin (Guthrie and Clasper, 2011; Leak, 2012; Moore, 2012; Harris, 2013; Rodd-Nielsen and Harris, 2013; Wounds UK, 2013; Milne, 2015; Percival and Suleman 2015). Cleansing was completed by irrigation with normal saline.

The primary dressing used was Zorflex (imported by Sentry Medical Pty Ltd), an activated carbon cloth dressing. This dressing is able to sequester and retain matrix metalloproteinases (MMP), including MMP-1, MMP-2 and MMP-9. Exudate in chronic wounds contains high levels of proteolytic enzymes, such as elastase from human polymorphonuclear granulocyte. Activated carbon cloth dressings reduce protease levels within a wound, are conductive, manage odour and bacterial load and prevent increased microbial activity.

The secondary dressing was a super absorbent pad to manage the high level of exudate. The dressing was lightly taped with silicone fixation tape.

Short-stretch reusable compression therapy

was applied to reduce oedema and, over time, reduce exudate production. Lymphlex REDUCE (Sentry Medical Pty Ltd) was chosen, which delivers sub-bandage pressures of 30–40 mmHg at the ankle and at rest. The stretch of this product was modified on initiation of treatment to enable the patient to adjust, thereby allowing for optimal application over time. It was initially applied with no stretch and then reviewed at 72 hours, applied again with no stretch for a number of visits.

After several weeks, the compression therapy was applied with half stretch and at this stage the wound healing started to progress.

Mrs A did report some pain from her pre-existing common peroneal nerve injury following application with half stretch, but we were able to address this with GP input and advised Mrs A to take paracetamol in the daytime after the compression was applied. Her pain was worse the night after dressing procedures, so the GP suggested Mrs A have an additional 50 mg tramadol on the evening after dressing changes. Her need for extra tramadol diminished 6 weeks into treatment and was not reported in the final month of healing of her wound.

The dressing change frequency three times a week for the first 4 weeks was reduced to twice a week thereafter. Within the first 2 weeks the exudate significantly reduced. At 3 weeks, wound odour had resolved, peri-wound erythema was significantly less and the wound bed was shallower, with slough replaced by about 50% granulation tissue.

This plan was followed consistently for 3 months. Mrs A was advised to elevate her leg



Figure 3. Time line photos of wound from initial presentation to healed wound.

when possible. Pain assessment was performed at each visit, by lightly touching the wound and leg. Consistent reduction in the pain levels occurred over the course of treatment.

Zorflex was used throughout the 3-month period as the primary dressing. Initially, the secondary dressing used was a super absorbent pad to manage the high level of exudate and this was required for the first 3 weeks. Following this, as exudate and wound size reduced, the secondary dressing was swapped out for a foam dressing to maintain moisture level.

In the last few weeks, once the wound had healed, this dressing regime was ceased and a silicone foam dressing was applied with compression continuing for the 2 weeks post healing. In this final maturation phase, the silicone dressing helps with increasing the tensile strength of the wound by facilitating remodelling of collagen: lack of tensile strength in wounds can play a role in increased risk of wound breakdown.

Patient education was provided by the treating nurse, informing the patient of the wound aetiology and wound management strategies to gain patient cooperation and confidence ensuring good adherence with the treatment plan.

One month from presentation, the wound was showing signs of healing. At this point, some skin dryness was noted due to the compression therapy. Moisturiser application was started on the lower leg to prevent dryness of the

surrounding skin and potential new wound formation. Moisturising was required at each visit.

Mrs A's case has significantly changed my clinical management of leg ulcers; I now routinely apply moisturiser as a good additional holistic measure.

By the end of the third month, the wound was completely healed [Figure 2]. Mrs A and her family were happy with the outcome.

Discussion

Venous leg ulcers are a chronic condition with a poor healing tendency (WUWHS, 2016). Reducing oedema can improve capillary function, facilitating nutrient delivery and oxygenation to the wound site and improving waste clearance, thus combating infection and accelerating the rate of healing (Pascarella and Shortell, 2015). The control of oedema is achieved by the application of graduated pressure improving both deep and superficial venous systems. Prior to the application of compression therapy, any suspicion of arterial insufficiency needs to be excluded. Further caution was needed in this case to avoid any increase in neuropathic pain or wound pain (Pascarella and Shortell, 2015).

The wound was continually monitored for signs of infection or wound deterioration. The frequency of dressing changes was largely determined by exudate level and the use of the super absorbent pad assisted with exudate

management. This dressing regimen proved effective, with significant reduction in size of the wound bed and exudate level and absence of wound odour after the third week.

By week 4, with significant improvements in wound pain and debility, Mrs A was able to be encouraged to have regular physiotherapy and do physical exercise to improve lower limb circulation and function (Singh et al, 2005). Mrs A was confident with the prescribed plan and attended appointments as required, indicating good adherence with therapy. The need for ongoing compression stockings after wound closure to prevent recurrence was also discussed and promoted.

The result overall was extremely pleasing for both patient and clinician, with the wound completely healed at the end of the third month.

Conclusion

The short-term goals of reducing the bacterial burden in the wound, reducing exudate and associated malodour and decreasing oedema and pain were achieved. Following this, consistent reduction in the size of the wound and continued improvement in the condition of the periwound skin lead to complete healing of this chronic leg ulcer.

As the patient's symptoms and comfort improved, she became more engaged in her management and has continued to use compression stockings to prevent recurrence. She is now self-sufficient in application and removal of her compression stockings with consistent support and help from her husband.

The keys to successful healing in this case were the careful clinical evaluation and planning prior to commencement of dressing, achieving results in a relatively short period of time. Involving the patient in treatment will reduce the risk of recurrence. As satisfying as it is to heal a wound completely, the most rewarding aspect of the case was the

improvement quality of life and confidence in Mrs A.

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