Case study Holistic assessment and management of a complex wound

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This case study was a winning entry in the inaugural Paul Hartmann/AWMA Advanced Wound Care Course Scholarships in 2006. Winners were chosen following a rigorous assessment process by members of the AWMA Executive Committee and AWMA Education and Professional Development Subcommittee.

Presentation

Mrs F is an 86 year old widow who, until 2004, lived alone in her own home with good family support. She was alert but was prone to confusion at times. Mrs F had been a regular patient of a large tertiary teaching hospital in NSW since 1995. In 2000, cellulitis of the right lower leg developed into three sloughy ulcers which in time joined to form one large ulcer on the gaiter area above the medial malleolus (Figure 1).

Medical history

Mrs F had a complex medical history of non-insulin dependent diabetes mellitus (NIDDM), atrial fibrillation, recurrent urinary tract infections, extensive osteoarthritis, peripheral vascular disease (PVD), post-operative pulmonary embolism and a chronic leg ulcer. Mrs F was allergic to prawns and Micropore tape.

Surgical history

Her surgical history involved an amputation of (L) great toe in 1995, R/O (R) foot bunion in 1999, a partial foot amputation in 2002 and, in 2004, amputation of the third metatarsal (L) foot and debridement of the (L) forefoot.

Medications

On presentation, Mrs F was taking the following medications:

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Warfarin 3mg nocte, Mobic 15mg mane, Losec 20mg BD, Capoten 50mg tds, Glucophage 250mg tds, Lanoxin PG mane, Tramal SR 150mg BD, Normison 10mg nocte, FGF mane, Lasix 20mg BD, Prothiaden 50mg nocte, Digesic x2 qid prn.

Wound profile

Mrs F's right lower leg ulcer was located just above the medial malleolus and occurred secondary to cellulitis. Dressings were attended three times a week by district nurses, sometimes daily depending on exudate levels. Previous dressing regimens used over the past 5 years included Jelonet, Adaptic, Kaltostat, Intrasite gel, Stomahesive powder, Allevyn, Lyofoam Extra, Duoderm Thick, Gaviscon liquid on excoriated wound edges, protective barrier wipes, Duoderm stoma paste, Eleuphrat Ung, Medihoney, SSD cream, Intrasite/SSD soaked gauze, Biotain, as well as patient self-treatments with over the counter preparations.

Numerous courses of antibiotics for *Staphylococcus* and *Pseudomonas aeruginosa* infections had been prescribed over this period. The vacuum assisted closure dressing (VAC) was used in February 2004 when the ulcer deteriorated, with exposure of tendon and lymphatic leakage. During this period, her blood sugar levels (BSLs) ranged between 10.9 to 21.2 mmol.

Mrs F was admitted into an aged care facility in September 2004. On admission, a comprehensive holistic assessment identified multiple underlying factors which were having a negative impact on the healing of Mrs F's wound such as PVD, diabetes with high BSLs, age, obesity and inactivity, anaemia, osteoarthritis and drug therapy.

Aetiology of the wound

Classical clinical signs of venous disease were present. Pulses were palpable but capillary return was delayed. Variable non-dependent pain was reported, exacerbated by cellulitis or oedema and described as burning or stinging, indicating a neuropathic origin due to persistent nerve

injury. An ankle brachial pressure index (ABPI) of 6.0 from a previous consultation with a specialist in venous disease concluded arterial calcification compounded by diabetes. Assessment findings indicated an ulcer of mixed aetiology with predominantly venous characteristics.

Clinical characteristics of the wound

Location of the wound

The wound was located over the gaiter area immediately above the medial malleolus

Wound bed status

The wound exhibited deep red coloured friable granulation tissue over an ulcer that bled on contact. No necrotic tissue was present but about 10% of the wound was covered in slough.

Wound edge

Irregular margin with a gently sloping border.

Wound measurements

The wound measured $12.5 \text{cm} \times 5 \text{cm}$ with a depth of 0.8 cm with no undermining or tracking present (Figure 2).

Wound odour

The wound was slightly offensive.

Peri-wound skin

The skin around the wound was dry, scaly, tight, shiny and oedematous. Ankle flare was present with distended venules below the malleolus.

Wound exudate

Copious amounts of serous cloudy fluid were exuding from the ulcer,

Laboratory tests

A wound swab identified light staphylococcus infection. Blood tests for serum albumin (38g/L) showed Mrs F had a serum albumin of 35g/l, indicating adequate delivery of nutrients to the wound ¹. A low haemoglobin was treated with FGF (Fergon ferrous gluconate).

Treatment aims

The objective of wound management in Mrs F's case was to improve circulation to the limb, reduce the level of pain, control the amount of exudate, protect the surrounding skin, stabilise her BSLs, ensure adequate nutrition and, ultimately, heal the ulcer.

Wound management plan

The treatment of choice for clinical venous ulceration is graduated compression bandaging ². However, in this case, treatment options were governed by available resources and the aetiology of the ulcer. The management plan considered all assessment findings and tried to address as many factors as possible that impacted on wound healing ³. PVD deprives extremities of vital nutrients, making infection difficult to eradicate. Furthermore, the wound was unlikely to heal until the level of oedema had been reduced or had resolved.

The management plan included:

- Addressing the wound bio-burden and ensuring less frequent dressing changes.
- Managing exudate levels to promote a moist wound environment.
- Controlling the patient's dietary intake with a diabetic diet to reduce BSLs and ensure appropriate nutritional intake.
- Encouraging a regimen of exercises to promote circulation and aid venous return. Elevation of the limb was used to reduce swelling and pain.
- Providing pain relief titrated to the patient's needs and improving sleeping patterns.

Initially the ulcer was cleansed under the shower. Conveen barrier cream was applied to protect healthy skin around ulcer. The ulcer itself was dressed with SSD/gel, Telfa, combines and crepe bandage. Glycerine 10% in white soft paraffin was applied to any dry scaly skin. Unfortunately, the leg became cellulitic and the ulcer deteriorated, oozing offensive green exudate indicative of pseudomonas. Oral antibiotics were commenced and the dressing changed to Atraumann Ag, a non-adherent triglyceride impregnated silver tulle used to prepare the wound bed and restore bacterial balance ⁴. The wound at this point (9 October 2005) was cleansed with water to prevent the binding of silver molecules. A secondary dressing of Telfa, combine and crepe bandage was applied and left intact for 4 days.

It was essential at the outset that collaboration between the staff of the nursing home, the patient's GP, the patient and the patient's family was established in order that the management plan would be adhered to.

Progress and follow up

Assessment 1

After 4 days, the infection had abated. Epithelial tissue was noted and the size of the wound had decreased markedly.

Assessment 2

Two weeks later, 50% of the wound was covered with epithelial tissue and there was clear evidence of wound contraction.

Assessment 3

By 21 November 2005 the leg ulcer had healed after a total period of 44 days. The wound was completely epithelialised and the scar was beginning to mature (Figure 3). Several crusty areas remained which were improving with daily moisturising and application of leg protectors.

Discussion

Wound management is an interdisciplinary endeavour and should be practised according to the best available evidence for optimising healing in chronic wounds ⁴. It is also acknowledged that the individual competency of nurses varies, and that therefore, treatment management guidelines need to be implemented based on current evidence to promote best nursing practice.

When there is no evidence of healing, a comprehensive assessment should be carried out at 3-monthly intervals, or sooner if a clinical condition deteriorates ². With recurrence rates in leg ulcers as high as 76% within 1 year ², educational programmes should reflect a continuum of care which culminates in effective treatment protocols that promote healing as well as prevent recurrence. Collaborative assessment and treatment planning with the client and family ², and establishing the underlying causes of any wounds is essential. Failing to do so will most likely result in delayed wound healing.

Summary

This case study illustrates the need for holistic assessments to address systemic and local factors which can impact on wound healing. PVD is a major problem, particularly among diabetics, and may impair wound healing in leg ulcers should they develop. Anti-microbial dressings are valuable adjuncts to wound management regimens where the wound is either heavily contaminated or infected. Using efficacious treatments may lead to improvements in both healing rates and quality of life for patients with leg ulcers².

Recommendations

 Guidelines for the management and treatment of leg ulcers should be used to guide the clinical practice of doctors and nurses when managing patients with leg ulcers to ensure treatment concords with the best available evidence. Educational programmes on leg ulcers should reflect the continuum of care, inclusive of assessment, diagnosis, treatment protocols, healing times, and prevention of recurrent episodes of leg ulceration.

References

- Templeton S (Ed). Wound Care Nursing A Guide to Practice. Melbourne: Ausmed Publications, 2005.
- RNOA. Assessment and Management of Venous Leg Ulcers. Nursing: Best Practice Guidelines 2004. Available http://www.rnao.org/bestpractices.
- Brown P & Phelps Maloy J. Quick Reference to Wound Care (2nd ed). Canada: Jones and Bartlet Publishers Inc, 2005.
- 4. Carville K. Wound Care Manual (5th ed). Perth WA: Silver Chain Foundation, 2005.

Further reading

Benbow M. Evidence-Based Wound Management. London: Whurr Publishers Limited, 2005.



Figure 1.



Figure 2.



Figure 3.