

The application of TIME (wound bed preparation principles) in the management of a chronic heel ulcer

Foley L

A review of the treatment of a difficult to heal chronic heel ulcer using TIME (wound bed preparation principles).

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Introduction

A female patient (Mrs J) was referred to the podiatry department for a review of a chronic heel ulcer of unknown cause. This had been present since 1996 and various forms of treatment had been unsuccessful in achieving healing. TIME (wound bed preparation principles) were applied to encourage healing.

Case report

Medical history

The patient's medical history was unremarkable apart from radiotherapy of a heel spur in 1967 and surgical removal of a left "heel bump" in 1996. This "had not healed since" reported the patient. There was no information available on what treatment or care was delivered in the period 1996 – March 1999.

In March 1999, the patient was referred to the Fremantle Hospital plastics clinic for a review of the problem. A biopsy was performed; differential diagnoses included tuberculosis and actinomycosis. The following month, Mrs J was referred to the infectious diseases department (IDD) of Fremantle Hospital Health Service (FHHS) for ongoing management. A report at that time stated "... most likely Mycobacterium,

possibly Spirotrichosis... started as small area of redness over the heel and progressed to a large indurated area with several non-healing ulcerative areas, with possible fistula formation... no obvious cause... several courses of antibiotics". The patient was commenced on Clarithromycin 55mg bd, Ciprofloxacin 500mg bd and Rifampicin 600mg daily.

At a review 1 month later, "remarkable improvement" was noted; this led to a diagnosis of Mycobacterium. Within a short time the patient was complaining of nausea and lethargy – abnormal liver function tests (LFTs) were recorded. The patient was directed to cease the Rifampicin. A lower dose was tried when the LFTs were normal but the nausea and lethargy returned. Alternatively, Ethambutol was added to the treatment regime.

At June 1999 no further improvement had taken place, LFTs were normal and Rifampicin 300mg nocte was resumed. Surgical debridement was mooted if healing did not improve. By the end of September 1999, IDD recorded a "significant deterioration" in the heel. Resistance to the antibiotics or possible osteomyelitis were considered as possible causes. Multiple punch biopsies of the heel and a CT scan were conducted. Differential diagnosis included neoplasm, tuberculosis or actinomycosis. The histopathology report stated:

Multiple granulomata. Small amount of Staph. aureus as anticipated (given the superficial nature of the lesions). Mycobacteria not seen but difficult to isolate. From five specimens, ulceration with extensive necrotising granulomata within dermis to epidermis. Diagnosis: necrotising granuloma(ta) ulceration.

The CT scan was negative for osteomyelitis.

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By February 2000, IDD notes reported that the heel had improved, and the lesions were more superficial. In the intervening period, the patient was managed by her GP until she was referred to the podiatry department. By January 2004, the problem was still presumed to be a Mycobacterium infection and the patient was now also prescribed Kenacomb ointment.

Podiatry review

The patient presented at the podiatry clinic with a chronically painful left heel of approximately 8 years’ duration. She had been unable to wear shoes with closed heels for that same period due to the chronic swelling of the heel and the pain caused by a shoe heel counter. The posterior aspect as well as the immediate lateral and medial aspects were affected (Figures 1-3).

The lesions were assessed using the TIME principles of wound bed preparation:

- *T: Tissue* – removal of non-viable or deficient tissue.
- *I: Infection* – control of infection or inflammation.
- *M: Moisture* – imbalance correction of excessive moisture or prevention of dessication.
- *E: Edge of wound* – revision of the edge of the wound ¹.

Tissue removal

The heel was covered with many lesions of varying sizes (Table 1). The wound beds were a mix of yellow non-viable tissue and granulation tissue. As the lesions were superficial, there was little debris to debride. Previous swabs had shown *Staphylococcus aureus* to be confined to the ulcerated surface so further microbiology was not ordered. An x-ray was negative for osteomyelitis.

Control of infection or inflammation

Chronic inflammation was evident given the amount of swollen peri-wound tissue and the general erythema of the posterior aspect of the heel. The patient was using open heeled shoes to limit heel counter pressure but heel pain, whilst in bed, was significant. All the microbiological investigations indicated the presence of *S. aureus* on the wound surface.

Moisture imbalance

Minimal exudate was noted on the dressings; the rest of the heel surface was noticeably anhidrotic. Close examination of the wound surfaces indicated dry inflamed areas of granulation and slough.

Edge of wound

The wound edges were noticeably raised and firmly adhered, with no undermining (Figures 1-6).



Figure 1. Left heel: medial.



Figure 2. Left heel: posterior.



Figure 3. Left heel: lateral.

Table 1. Ulcer dimensions (mm).

	23 Feb 2004	8 Mar 2004	7 Apr 2004	19 May 2004	7 Jun 2004
Posterior	38x21	35x18	33x19	10x5	Healed
Lateral	20x9	14x11	9x6	Healed	Healed
Medial	24x18;9x8	21x17;9x7	20x12;10x5	Healed	Healed



Figure 4. Left heel: medial.



Figure 5. Left heel: lateral.



Figure 6. Left heel: posterior.



Figure 7. Left heel: posterior.



Figure 8. Left heel: posterior.

Treatment

Treatment was commenced to reduce the amount of nonviable tissue and to reduce or remove the suspected bacterial load. The treatment plan consisted of second daily cleansing with saline solution and applications of Iodosorb paste with Biatain foam. The patient was issued with a foam collar to raise her heel off the bed to assist with her sleeping.

A noticeable improvement was recorded at 4 weeks (Figures 4-6). Healing of two of the three lesions was noted by 17 May (Figure 7), with complete healing taking place by 7 June. At her final review on 4 August 2004 (Figure 8), the healing was still complete and not painful to palpation. A Silipos heel sock was issued to assist with shoe wearing.

Discussion

Granuloma is defined as "A tumour like mass or nodule of granulation tissue, with actively growing fibroblasts and

capillary buds... It is due to chronic a chronic inflammatory process associated with infectious disease e.g. tuberculosis, syphilis, sarcoidosis, leprosy... or invasion by a foreign body"². This term is used widely to describe many different chronically inflamed tissues.

Apart from the patient's intermittent use of tea tree oil and Savlon cream, no topical treatment had been prescribed to treat the lesions. Given the chronic nature of the problem (8 years approximately), it was felt that the treatment plan should reflect a reduction in the chronic pain and the inflammation in the heel. The patient was not pregnant and was not experiencing any thyroid problems, so a second daily application of Cadexomer Iodine was recommended.

As the lesions had always been deemed to be superficial and the biopsies had reflected this, Cadexomer iodine in the form of Iodosorb paste was prescribed. The Iodosorb paste was to be applied second daily and covered with Biatain foam and fixed with Fixomull adhesive. At the first review 4 weeks later, some encouraging progress had been made and the treatment regime was continued for another 6 weeks. After two reviews and 13 weeks of the Cadexomer Iodine, complete healing had taken place.

Summary

A chronic foot wound was assessed and treated according to the TIME principles of wound management.

References

1. Chin C, Schultz G, Stacey M & Contributions from the Wound Bed Advisory Board. Principles of wound bed preparation and their application to the treatment of chronic wounds. *Primary Intention* 2003; **11**(4):171-182.
2. Dorland's Illustrated Medical Dictionary (26th ed). Philadelphia: WB Saunders, 1981. ■