

CHAPTER 8

THE USE OF HONEY IN WOUND MANAGEMENT

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Part I: The use of honey in the treatment of meningococcal skin lesions

A preliminary report of this case study was first published in April 2000 in the *Nursing Times* (Dunford, 2000). It had a huge impact and was immediately picked up by the media. The fact that such a natural agent as honey could be responsible for healing such devastating lesions in a young adult so badly affected by meningococcal septicaemia made it newsworthy. There followed numerous national and local newspaper reports as well as TV and radio appearances both nationally and also in New Zealand and Australia. This positive attention no doubt also helped the patient, referred to as Jem, come to terms with his experience. It also introduced honey as a healing agent to the general public and health practitioners alike, and began the significant public and professional interest in honey that has grown in recent years.

Patient history

Jem was fifteen when he contracted meningococcal septicaemia. His was a classic presentation of feeling unwell with myalgia and nausea for a few hours before collapsing. Meningococcal disease is caused by the bacterium *Neisseria meningitidis* that is found in human nasopharyngeal tract. Under certain conditions, such as concomitant viral infection or reduced immune response, penetration of the mucosa leading to

outgrowth in the bloodstream can occur. Further information on this condition is provided in *Figure 8.1*.

- ❖ Bacteraemia manifests first as acute fever, low back pain, generalised aches, and without shock.
- ❖ Fulminant meningococcal sepsis (FMS) with shock and disseminated intravascular coagulation may develop without signs of meningitis.
- ❖ The mortality rate from FMS is 20–80%.
- ❖ Skin haemorrhages are characteristic of meningococcal disease.
- ❖ Skin and limb necrosis requiring amputation or plastic surgery is seen in 10–20% of patients.
- ❖ Meningococcal disease does not consist only of meningitis!

Figure 8.1: Features of meningococcal septicaemia (van Deuren *et al*, 2000)

Jem required immediate resuscitation on admission to hospital and was given full system support and ventilation in intensive care for the next thirty days. Jem developed fulminant meningococcal septicaemia (FMS) as a result of the massive outgrowth of toxins. This resulted in both adult respiratory distress syndrome and acute renal failure. Extensive haemorrhagic skin lesions (ecchymoses) appeared on his lower limbs and peripheral necrosis of both his hands and feet developed. Despite attempts at salvage, bilateral transtibial amputation together with amputation of distal and middle phalanges of both hands was necessary. Multiple skin grafts were harvested and applied to the residual limbs. After two months of treatment Jem was transferred to a regional burns and plastics unit. By this time he had developed a Grade 3 pressure ulcer on his left buttock and he was nutritionally compromised.

The amputation sites on Jem's hands healed without incident but there was to follow six months of skin grafting to his legs, which in most cases proved unsuccessful. This resulted in a number of non-healing donor sites. Pain was a major issue in Jem's care. Despite the administration of Oramorph[®], dihydrocodeine and diazepam for dressing changes, pain was never properly controlled, therefore, the majority of dressing changes (approximately twice weekly) were undertaken under general

anaesthetic. A high-protein, high-calorie diet was started which was supplemented by overnight gastric feeding.

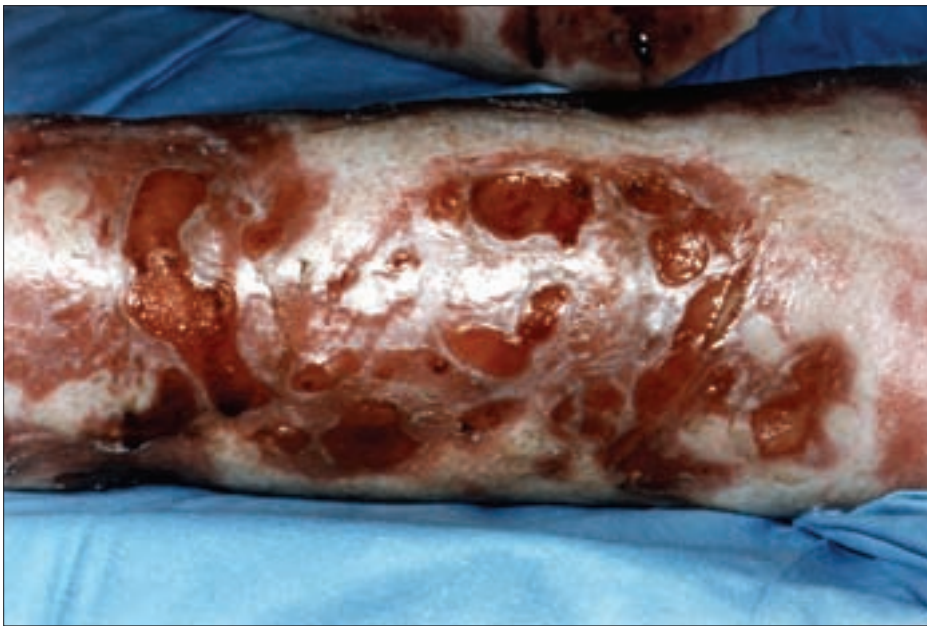


Figure 8.2: Infected meningococcal lesion prior to honey dressings

The skin lesions were initially dressed using paraffin Tulle dressings (Jelonet™, Smith and Nephew) with alginate dressings (Kaltostat™, ConvaTec) to the donor sites. A hydrocolloid dressing (Granuflex™, ConvaTec) was applied to the pressure ulcer. Swabs from his lower legs showed heavy growth of *Pseudomonas spp*, *Staphylococcus aureus* and *Enterococcus spp*. Topical silver sulfadiazine 1% cream was applied as a consequence, but proved ineffective in eliminating the bioburden present in the lesions. Even after copious amounts of analgesia and soaking in a bath, the dressings were still difficult to remove. This meant that dressing and wound debris began to accumulate increasing the risk of infection. All lesions remained static.

It was at this stage that a referral to the tissue viability team was made. Following assessment it was decided to introduce a multi-layer dressing (Tenderwet™, Paul Hartmann AG) with the aim of providing atraumatic dressing removal together with effective wound cleansing. Despite initial favourable results, the frequency of dressing change required for this product proved impracticable.

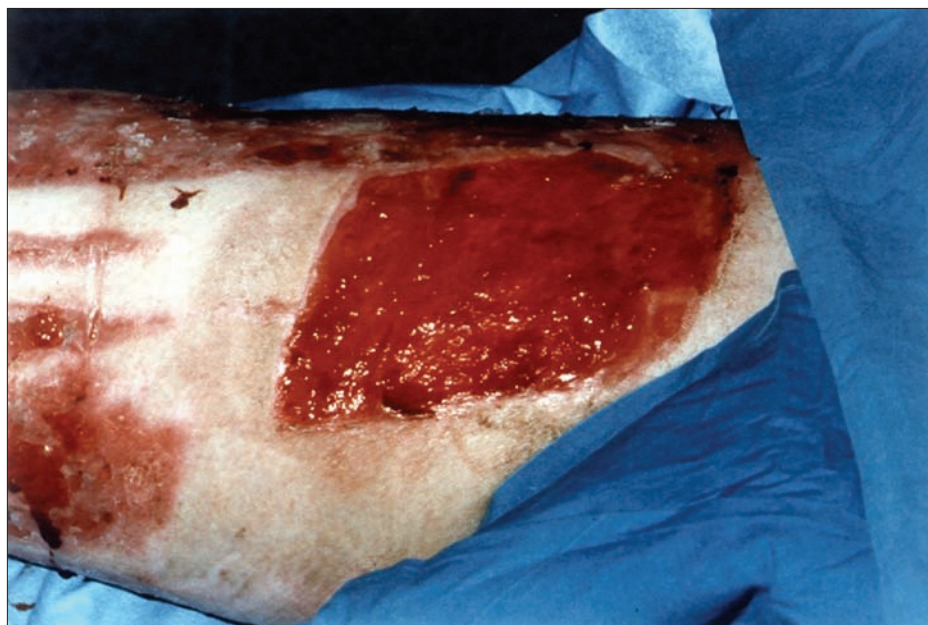


Figure 8.3: A failed donor site prior to treatment

At this point a new approach was necessary. Gamma irradiated absorbent dressing pads impregnated with 25–35g of active Manuka honey (UMF 13) were obtained and initially applied to Jem's right leg only. The left leg continued to be dressed as before as a form of control. Again, a general anaesthetic was used to dress the lesions at three-day intervals. Wound swabs were taken at the start of the honey dressings and at regular intervals throughout the treatment period. The rationale for the use of honey dressings is shown in *Table 8.1*.

Within a few days the right leg showed signs of epithelialisation with a corresponding reduction in wound bacteria. Following this, all lesions were dressed using honey, including the pressure ulcer. The *Pseudomonas spp* and *Enterococcus spp* were both eliminated within a few weeks of honey dressings. Interestingly enough, *Staphylococcus aureus* remained throughout the healing process without appearing to hinder it. It was also noticed that all traces of odour from the lesions were eliminated within the first dressing change. However, it was not fully appreciated by anyone at this stage how much of an impact the odour and its associations were having on Jem. The rate of healing and reduction in odour allowed Jem to gain more control over his situation and within a few weeks he

was able to undertake dressing changes using a mixture of Entonox[®] and oxygen. The honey dressings did not adhere and were easily removed with use of a shower trolley. The epithelialisation continued and further skin grafts were applied six weeks after starting the honey treatment that this time proved successful after many previous failed attempts.



Figure 8.4: Signs of improvement following one week of honey dressings

Within ten weeks all lesions, including the pressure ulcer, were completely healed and Jem was able to be discharged and to begin his very successful rehabilitation programme. Not only did the lesions finally heal within a relatively short period of time, but also the resultant scar tissue was of good quality with no evidence of hypertrophic scarring. As with burn injuries, contracture and hypertrophic scarring can be a long-term problem in meningococcal lesions.



Figure 8.5: Decrease in bacterial loading evident following four weeks of treatment



Figure 8.6 a and b: Signs of significant improvement with epithelialisation present at week six

Discussion

Jem's was the first reported case of using honey for infected skin lesions on multiple meningococcal lesions. An excellent clinical outcome was achieved, highlighting the effectiveness of honey in the various stages of wound healing as described in *Table 8.1*. The honey was able to eliminate the *Pseudomonas spp* that had proved so problematic for many months. An additional outcome from using honey dressings was the thorough wound bed preparation that facilitated successful skin grafting after many failures. Although the antimicrobial and anti-inflammatory properties of honey did play a significant part in 'kick starting' the healing process, they were probably not the factors that helped to turn the whole situation around for Jem. Prior to the application of honey, dressing changes had been a time of major anxiety and distress for Jem. What we had failed to realise was the impact that the smell of his wounds was having on him, as the smell was not considered particularly malodorous by those involved in his care. The original case study included an account by Jem in which he states the following, 'one of the first things I noticed [after using the honey dressing] was the smell wasn't nearly so bad. I'm not saying that it was worse than the pain, because the pain was bad, but the smell was one of the things that bothered me most'. Jem had had the traumatic experience of having to live with his decaying limbs and their smell before the decision was made to amputate them. The association between smell and emotion is well recognised. The smell of hospitals can cause major anxiety in some people and the smell of cabbage, associated with school dinners, can spoil the enjoyment of a meal. By eliminating the smell, Jem did not have such a strong trigger for his anxiety and was able to gain further control of his situation. This also allowed him to gain better control of his own pain relief. For many patients, particularly with non-healing wounds, the elimination of odour is of major importance. The ability of honey to do this effectively and quickly should not be underestimated.

There may be a place for using honey earlier on in the treatment of meningococcal lesions. Oedema increases the risk of purpuric skin lesions deteriorating into necrotic areas. The ability of honey to reduce inflammation and oedema could prove advantageous at this stage, and as demonstrated so well in this case, could reduce any associated malodour.

Table 8.1: Rationale for use of honey dressings

Clinical action	Mode of action
Odour elimination	Glucose metabolism by the infecting bacteria to lactic acid instead of amino acids from serum and dead cells resulting in ammonia and sulphur compounds
Antimicrobial activity	<ul style="list-style-type: none"> ❖ Production of hydrogen peroxide, action of non-peroxide components (phyto-chemicals) and acidity ❖ Stimulation of immune response including β-lymphocytes, neutrophils and cytokines. Supply of glucose for respiratory burst and for energy production in macrophages
Anti-inflammatory activity	<ul style="list-style-type: none"> ❖ Decrease in leukocytes ❖ Inhibition of reactive oxygen species (ROI) production as a result of antioxidant activity ❖ Suppression of inflammatory process through scavenging of free radicals by antioxidants
Reduction in pain	<ul style="list-style-type: none"> ❖ Anti-inflammatory properties, as above ❖ Moist wound healing environment ❖ Non-adherence, resulting in atraumatic removal
Stimulation of healing	<ul style="list-style-type: none"> ❖ Increased phagocytosis due to stimulatory effect of honey on macrophages ❖ Increased autolytic debridement due to osmolarity effects ❖ Increased angiogenesis, collagen synthesis, granulation and epithelialisation as a result of honey components and low hydrogen peroxide production with antioxidant protection that modifies proteins important to cell growth
Reduction in scarring	Suppression of inflammatory process, as above

Reference: American National Honey Board, 2003

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Part II: The use of honey in venous leg ulcers

This section illustrates the effectiveness of honey by use of a single case study examining an unusual leg ulceration presentation.

A summary of the reported wound healing properties of honey is shown in *Table 8.2*. The most important of these being its antimicrobial properties.

Table 8.2: Reported wound healing properties of honey		
Property	Anticipated clinical	Mode of action
Antimicrobial activity	<ul style="list-style-type: none"> ❖ Inhibits wide range of Gram negative and Gram positive bacteria and fungi ❖ Deodorises wounds ❖ Prevention of cross-contamination through protective barrier 	<ul style="list-style-type: none"> ❖ Production of hydrogen peroxide and action of phytochemicals ❖ Stimulation of immune system ❖ Glucose metabolism by bacteria to produce non-odorous lactic acid (instead of malodorous ammonia and sulphur compounds) ❖ High viscosity creates physical barrier
Anti-inflammatory activity	<ul style="list-style-type: none"> ❖ Reduction in oedema ❖ Reduction in pain ❖ Reduction in scarring 	<ul style="list-style-type: none"> ❖ High osmolarity leading to fluid outflow and creation of moist healing environment ❖ Decrease in leukocytes associated with inflammation ❖ Inhibition of reactive oxygen intermediates (ROI) production as a result of antioxidant production
Stimulation of wound healing	<ul style="list-style-type: none"> ❖ Increased autolytic debridement and phagocytosis ❖ Increased angiogenesis and formation of granulation tissue ❖ Cell proliferation ❖ Collagen synthesis ❖ Re-epithelialisation 	<ul style="list-style-type: none"> ❖ Stimulatory effect of honey proteins on macrophages ❖ Increased debriding by moist environment and outflow of lymph and nutrients ❖ Increased oxygen supply secondary to the outflow of lymph and acidity of honey ❖ Controlled low hydrogen peroxide production stimulates cell production

Adapted from American National Honey Board, 2003

Case study

The patient was an eighty-five-year-old lady who presented with a twenty-month history of venous leg ulceration, presenting as numerous small ulcers present on her right leg. The largest of these ulcers measured 1.8 cm in length and 1.1 cm in breadth (*Figure 8.7*). She was otherwise fit and healthy with no physical problems of note. Her only medication being paracetamol for general aches and pains. Her ankle/brachial pressure index was 0.98 in each leg and she was treated using a three-layer compression system in conjunction with a simple non-adherent dressing. Investigations, including full blood count, liver function, thyroid function and calcium and parathyroid hormone levels were all within normal range. Community nursing staff at a local leg ulcer clinic, which she attended on a weekly basis, managed her leg ulceration.

The patient also presented with subcutaneous calcium deposits that appeared as small, hard lumps palpable beneath the skin of both lower legs. The calcium deposits also presented within the ulcer beds where they appeared as hard, irregular shaped lumps, which were attached to the wound bed (*Figure 8.7*). There was evidence of chronic inflammation in each of the ulcers, possibly as a consequence of the foreign body reaction to the deposits. The wound margins were poorly defined and the ulcer beds were also superficially sloughy. She experienced mild to discomforting pain as a result of the ulcers, which was effectively controlled with paracetamol. There had been no evidence of any reduction in size of the ulcers during her visits to the leg ulcer clinic.

Subcutaneous calcification or calcinosis cutis is the abnormal deposition of calcium and phosphate in the skin (Chave *et al*, 2001). The presence of these calcium deposits in soft tissues may lead to non-healing chronic ulcers (Brietstein *et al*, 2002). There are a number of causes for this condition, which have been categorised into dystrophic, metastatic, idiopathic or iatrogenic (Walsh and Fairley, 1995). Metastatic calciphylaxis involves abnormalities of calcium and/or phosphate metabolism, which leads to microvascular calcification and is a serious but rare complication of end-stage renal disease (Burkhart *et al*, 1999). Dystrophic calcinosis cutis can be seen in patients with venous hypertension and venous ulceration where it is confined to the soft tissues and does not involve the vessels. Chave *et al* (2001) suggest that calcification in this instance may be a result of the inflammation from venous leakage. This condition is rarely reported in the literature. Treatment measures are confined to

surgical excision on the deposits and treatment of the underlying venous hypertension.

Prior to commencement of honey dressings, the patient had had to attend a vascular clinic every three months to have the deposits removed from the ulcer beds using sharp debridement. Unless removed on a regular basis, the deposits tended to merge together and so increase in size. The debridement process often resulted in pain and bleeding and the patient disliked the experience.

The patient was recruited into a small clinical trial designed to determine the acceptability of honey for non-healing leg ulcers (Dunford and Hanano, 2004). Honey dressings were applied on a weekly basis under the existing compression system. Assessment was undertaken every two weeks for a total of twelve weeks, in which perceived levels of pain, odour and patient satisfaction with the dressings were monitored.

It was noted on the first assessment that pieces of calcium were evident on the honey-dressing pad on removal. This finding was not anticipated. This atraumatic removal of deposits continued with further use of honey dressings. In addition, the condition of the ulcer beds improved with a reduction in size, local surrounding inflammation and slough. There was no reported increase in pain as a result of using the honey dressings and the ulcers remained odour free. Patient satisfaction with the dressings was understandably high, as the traumatic experience of surgical debridement of the deposits was no longer required.



Figure 8.7: The limb on presentation showing multiple ulcers with calcium deposits

- ❖ Only use wound care honey which has been sterilised using gamma irradiation.
- ❖ Use sufficient honey for the wound in question. More may be required if the wound is large, heavily exudating or is sloughy or necrotic.
- ❖ Expect wound exudate to increase initially, so prepare patient for this and ensure that resources are available for dressing changes.
- ❖ Choose a honey preparation that is suitable for the wound, eg. gel or impregnated dressing.
- ❖ If wound is heavily exudating use a suitable secondary dressing to prevent maceration of surrounding skin.
- ❖ If packing a wound choose an appropriate dressing (eg. mannuronic acid alginate) to use in conjunction with the honey.
- ❖ Honey can cause a transient stinging and drawing sensation — make patients aware and ensure effective pain control prior to application.
- ❖ Honey staining can occur to skin but this is easily removed using soap and water.

Figure 8.8: Factors to consider when using honey dressings

Discussion

Not all patients with leg ulcers respond to recognised treatment regimes (eg. compression therapy). Unfortunately, for some leg ulcer patients the end point objective of total wound healing is not always achieved. This is a patient group where quality of life issues, such as pain and odour, are well documented (Douglas, 2001). This case study has focused on one such patient where total wound healing was an unrealistic outcome. The use of honey, not only resulted in a reduction in local inflammation and improved wound bed, but also the atraumatic removal of calcified deposits. This was an unexpected but very positive outcome. It is felt that the osmotic potential and viscosity of the honey helped to remove the deposits. This may account for the ability of honey to debride sloughy tissue and to assist in the removal of dirt.

Clinical observations suggest honey holds significant promise as an effective treatment in the management of wounds and that this is an area worthy of further research.

The case study presented in Part II of this chapter, is reproduced by kind permission of *Professional Nurse* 20(8).

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