

Treating difficult-to-debride wounds using a manuka honey dressing: a case study evaluation

KEY WORDS

- ▶ Antimicrobial activity
- ▶ Debridement
- ▶ Manuka honey dressing
- ▶ Necrosis
- ▶ Slough

This series of four case studies focuses on patients with difficult-to-debride wounds who were treated with ManukaDress IG (Medicareplus International), which is impregnated with 100% pure, medical-grade manuka honey. ManukaDress IG is indicated for autolytic debridement of necrosis and slough, to encourage granulation and epithelialisation, and can be used on wounds with any level of exudate when used with an appropriate secondary dressing.

The three main considerations for optimal wound bed preparation are regular debridement to reduce devitalised tissue and encourage formation of healthy granulation tissue, restoration of bacterial balance and promotion of a moist wound-healing environment through exudate management (Schultz et al, 2003). Honey provides a moist wound-healing environment, and is antimicrobial, de-odourising, anti-inflammatory/antioxidant and debriding — all of which address the key issues in wound bed preparation and bioburden management to promote healing in chronic wounds (Acton and Dunwoody, 2008; Seckam and Cooper, 2013). Honey's multimodal action justifies its use in a variety of wounds, addresses multiple clinical factors and eliminates the need for multiple products, making it a safe and easy treatment option.

The biochemical and antimicrobial components of various honeys differ depending on factors such as floral source, climate and harvesting conditions (Kwakman et al, 2011; Cooper and Gray, 2012). Medical-grade honeys designed for clinical use are specially treated to destroy bacteria and contaminants, are from specific, traceable sources and have proven antimicrobial activity (Cooper and Gray, 2012). Clinicians should be aware that the antimicrobial potency of honey products varies (Molan, 2002; Acton and Dunwoody, 2008).

Honey facilitates autolytic debridement by creating moist conditions, although it has also been suggested that the sugar content of honey is involved in activating proteases, which aid the debridement process (Molan, 2009). Honey may also promote autolytic debridement by causing an enzymatic

action that activates plasmin, which disintegrates blood clots that bind necrotic tissue to the wound bed (Molan, 2005).

A particular kind of honey made from the nectar of the manuka plant (*Leptospermum scoparium*) found in New Zealand has been shown to be particularly effective in wound bed preparation. Manuka honey is particularly useful in facilitating wound cleansing and autolytic debridement due to its high osmolarity, promoting a moist wound environment and creating an acidic environment conducive to healing (Montoya, 2013; Wahab, 2013). One review also found that manuka honey is more effective at removing attached slough, necrotic tissue and eschar than other types of honey (Molan, 2009).

MANUKADRESS IG

ManukaDress (Medicareplus International) is available as a gel (ManukaT) and a contact layer dressing (impregnated gauze: ManukaDress IG and ManukaDress IG Max).

The gel formulation is indicated for use in traumatic and surgical wounds, chronic ulcers (e.g. venous, diabetic foot and pressure ulcers), first- and second-degree burns, and lacerations and abrasions. The gel may also be used on sinus or cavity wounds with low exudate levels. It can be used to debride necrotic tissue or top-up dressings where the honey has been diluted by exudate.

The dressings are indicated for shallow or granulating wounds with low to moderate exudate levels and can be used to debride small areas of necrotic or sloughy tissue. For deeper wounds, the dressings may be used in combination with the gel.

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Medical honey should not be used in patients with known sensitivity to honey. Although concerns have been raised about its use in patients with diabetes, there is no evidence of increased blood sugar levels. However, it may be advisable to monitor blood sugar levels during use. Some patients may complain of a temporary increase in pain due to the osmotic action and/or low pH of the honey. If pain persists, discontinue use and gently irrigate the wound with sterile saline solution.

Due to the lack of toxicity, manuka honey may be used for longer than 2 weeks. However, regular review should be performed and if there is no improvement at 2 weeks, consider an alternative approach. ManukaDress should be discontinued when treatment goals have been achieved.

CASE SERIES

Because of manuka honey’s many modes of action in wound bed preparation — in particular, debridement — ManukaDress IG was investigated in a series of wounds that presented with high levels of necrosis and/or slough but were unsuitable for sharp debridement.

Case 1: Dehiscd surgical wound

Ms B, a 70-year-old woman, presented to accident and emergency following a prior mass bowel resection and colostomy formation that had resulted in a dehiscd abdominal wound.

The wound was 3 weeks old and measured 18cm × 10cm, and the colostomy was at risk of leaking into the wound. Wound pain was rated 5 on a visual analogue scale (VAS; 1–10). Slough covered 75% of the wound bed, with some necrotic tissue and granulation tissue also present. Surgical sutures were visible. Exudate levels were moderate. Antibiotics were initiated; however, they loosen the colostomy discharge, which then poses greater risk of leakage and infection.

To lift the slough and ready the wound for negative pressure wound therapy, ManukaDress IG was initiated along with a gel adhesive hydrocellular foam dressing (ALLEVYN Gentle Boarder, Smith & Nephew) to protect the periwound area and achieve a protective seal from potential colostomy leaks. Dressing changes were scheduled every 2 days, or more frequently if colostomy leakage occurred.

After 1 week, wound size remained the same, however the slough had reduced and was beginning

to lift from the wound and necrotic tissue now comprised 50–75% (Figure 1a). Wound-related pain was rated as 3 on the VAS. The dressing regimen was deemed effective and continued unchanged. In addition, a colostomy nurse was scheduled to address the leakage before the next wound assessment.

At week 2, the wound appeared larger due to the debridement of all necrotic tissue. Slough covered just over 50% of the wound, and the rest was granulation tissue. The patient reported no pain, and said that she was feeling much happier due to the improvement in the wound and the resolution of colostomy leakage. The dressing regimen was deemed effective and continued unchanged, with dressing changes extended to every 3 days.

After 3 weeks, wound size reduced to 12cm × 8cm (47% reduction since presentation), exudate levels were low, there was no wound- or dressing-related pain and the wound remained infection-free despite occasional leakage from the colostomy. Granulation tissue covered two-thirds of the wound bed and slough the remaining third (Figure 1b). The dressing regimen continued unchanged.

After 4 weeks, the wound measured 9cm × 5cm (75% reduction since presentation), with >75% granulation tissue and minimal slough (Figure 1c). As the goals for its use had been achieved, ManukaDress IG was discontinued and alignate fibre dressing (Sorbsan, Aspen Medical) was initiated to manage the low exudate level and promote further granulation and wound closure.

Case 2: Category IV sacral pressure ulcer

Ms W is a 74-year-old woman who was immobile due to a stroke. She had well-controlled diabetes, and her diet was good. She developed a sacral pressure ulcer (PU) from a wheelchair cushion that was not fit for purpose during the course of a day out.

On presentation, the PU was ungradable due to necrotic tissue obscuring the extent of the ulcer (ultimately judged to be category IV). The PU was 2 weeks old and measured 6cm × 7cm. There was no pain associated with the wound, which had previously been treated with a hydrocolloid dressing (Granuflex). The wound area comprised >75% necrotic tissue that was lifting at the edges and the rest was slough. It was determined that ManukaDress IG should be initiated to debride

Figure 1. Ms B’s dehiscd abdominal wound at (a) 1, (b) 3 and (c) 4 weeks after presentation.

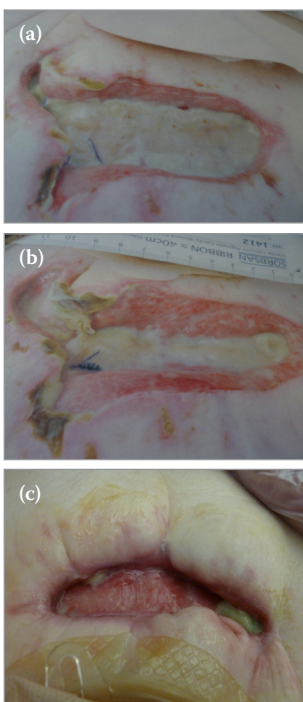


Figure 2. Ms W's sacral pressure ulcer at (a) 2 and (b) 4 weeks after presentation.

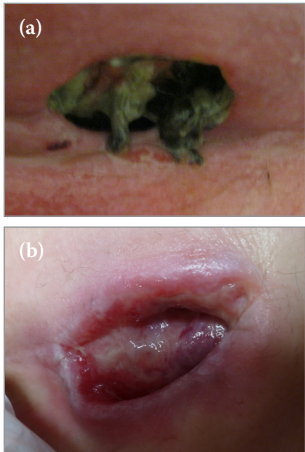
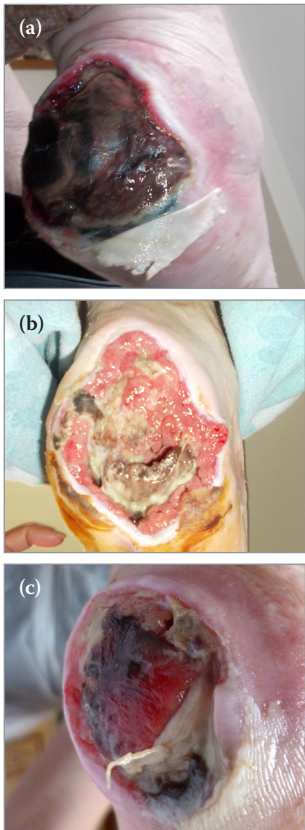


Figure 3. Ms B's necrotic heel ulcer at (a) 1, (b) 2 and (c) 4 weeks after presentation.



the wound. A gel adhesive hydrocellular foam dressing (ALLEVYN Gentle, Smith & Nephew) was used as a secondary dressing to manage exudate. A barrier film (Sorbaderm, Aspen Medical) was applied to protect the periwound area. Dressing changes were scheduled for every 3 days, or more frequently depending on exudate levels. Ms W's blood sugar levels were closely monitored.

After 1 week, the wound size had not changed and necrotic tissue remained at >75% of the wound bed but was lifting. The dressing regimen continued unchanged. The clinician reported that the dressing was easy to apply after it had been wetted, although it was sticky, meaning that gloves may have needed changing before applying the secondary dressing.

At week 2, necrosis made up 25% of the wound bed and was lifting, with approximately 50% slough and the remaining quarter granulation tissue (Figure 2a). The wound size remained the same. The dressing regimen was maintained, but the secondary dressing was changed to an absorbent foam adhesive dressing (ALLEVYN) to achieve a better seal.

After 3 weeks, the wound had increased in size due to the lifting of almost all the necrotic tissue, with granulation tissue in 50% of the wound bed and <25% slough. To complete debridement, the dressing regimen was maintained, but with dressing changes extended to every 4 days.

At week 4, the wound had not changed in size but granulation tissue was present in >75% of the wound bed, with some remaining necrosis and no slough (Figure 2b). The dressing regimen was maintained as it was expected that the wound would reduce in size as debridement was completed and granulation progressed to epithelisation.

Case 3: Heel ulcer with 100% necrotic tissue

Ms B is a 96-year-old woman who had fractured the neck of her femur and required a hip replacement. She had chronic obstructive pulmonary disease, but had been independent and mobile before the fracture and surgery. After the procedure, she took up residence in a nursing home and a necrotic heel ulcer developed.

On presentation, the wound was ungradable due to necrotic tissue obscuring the extent of the ulcer. The 4-week-old wound covered the entire heel and had received no prior treatment. The heel was warm and erythema was present around the

wound edges. There was no exudate. The patient reported wound-related pain as 9 on a VAS.

To debride the necrosis and address the local infection, ManukaT gel was initiated. A pad and bandage were used as secondary dressings to absorb exudate that was expected to arise as the necrosis began to lift with gel treatment. Dressing changes were scheduled daily.

After 1 week, the wound size had not changed, and the wound bed was still covered by necrotic tissue (Figure 3a). Wound-related pain and pain at dressing change were both rated 5 on the VAS; the patient was taking paracetamol 4-hourly and Tramadol for analgesia. To accelerate debridement, ManukaDress IG was initiated in favour of the ManukaT. The secondary dressing regimen was continued. To allow the honey more contact time with the necrotic tissue, dressing changes were decreased to every 2 days.

At week 2, signs of local infection had not reduced, but necrosis had reduced to 75% of the wound bed, with the rest covered by slough (Figure 3b). Wound size, wound-related pain and pain at dressing change were unchanged. The ManukaDress IG dressing was judged to be easier to keep on the wound and more effective in penetrating the necrotic tissue than the gel had been, so the dressing regimen was maintained, with dressing changes decreased to every 3 days and analgesia increased.

After 3 weeks, the wound size remained static but granulation tissue had appeared at the wound margins. Necrotic tissue had reduced to approximately 50% of the wound bed, with slough comprising the rest (Figure 3c). Wound- and dressing removal-related pain had both reduced to 3 on the VAS, and the patient's analgesia was reduced. The dressing regimen continued unchanged.

At week 4, wound size remained static but approximately 50% of the wound bed was comprised of granulation tissue and 50% slough. Wound- and dressing change-related pain had not reduced, but the patient no longer required analgesia. The dressing regimen was deemed effective and continued unchanged.

Case 4: Sacral pressure ulcer with 100% necrotic tissue

Ms H, an 82-year-old woman, is a nursing home resident with multiple sclerosis who developed an ungradable PU on her sacrum. Her diet and general

state of health were poor.

On presentation, the PU was 3 weeks old with 100% necrotic tissue coverage. To-date, a hydrogel dressing (INTRASITE gel, Smith & Nephew) had been used to treat the wound, exudate levels were moderate and there were no signs of local infection. There was no reported wound-related pain.

ManukaDress IG was initiated, along with a foam secondary dressing (ALLEVYN) to manage exudate, with dressing changes scheduled every 4 days.

After 1 week, wound size had not changed but the necrosis had softened and was moving inward from the wound edges. Although malodour had developed, there were no clinical signs of infection. As a precaution, a swab was taken. The dressing regimen was maintained, with changes scheduled every 3 days, to give the honey more time to exert its effect.

At week 2, the necrosis covered approximately 25% of the wound bed, the rest was slough or exposed wound bed (Figure 4a). Wound size appeared to be increasing due to a reduction in necrosis, exposing its true extent. No culturable organisms were found, so it was determined that the malodour reported could be attributable to the smell of softened necrotic tissue. ManukaDress IG dressing and change frequency was maintained, but the secondary dressing was changed to an absorbent foam (ALLEVYN) to manage exudate.

After 3 weeks, the malodour resolved, necrotic tissue was no longer present, and slough covered 25–50% of the wound bed. Granulation tissue was not present, but a clean, healthy wound bed was exposed (Figure 4b). The dressing regimen was continued unchanged, but the secondary dressing was changed to a foam (ALLEVYN) as there was a reduction in the amount exudate.

At week 4, no slough was present and granulation had commenced (Figure 4c). ManukaDress IG had achieved its purpose and negative pressure wound therapy was commenced to close the wound.

DISCUSSION

Debridement was successful in all four cases, including in wounds that initially presented with 100% necrosis. Ms B ended the 4-week trial not only fully debrided, but with wound size reduced by 75% from initial presentation. In Ms W's case, debridement was effective and the antimicrobial

properties of ManukaDress IG may have helped prevent infection in an anatomical area prone to colonisation. The 100% necrosis cover in Ms B was fully debrided by the end of 4 weeks' treatment. In Ms H, despite a slow start, the wound was fully debrided and ready to move on to negative pressure wound therapy by the end of the trial.

In all cases ManukaDress was found to be easy to use and apply, and effective once given time to soften necrotic tissue. Although the dressing is inherently sticky, it did not leave debris in any of the wounds. Furthermore, pain decreased with the use of ManukaDress IG in the patients who reported wound-related pain.

CONCLUSION

Honey is an effective and viable option for debridement and the maintenance of an optimal environment for wound healing. ManukaDress in particular has been shown to safely and effectively debride wounds that are not appropriate for sharp debridement, along with promoting healing in a multimodal fashion.

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DISCLAIMER

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“Debridement was successful [with ManukaDress (Medicareplus International)] in all four cases, including in wounds that initially presented with 100% necrosis.”

Figure 4. Ms H's sacral pressure ulcer at (a) 2, (b) 3 and (c) 4 weeks after presentation.

