

# Natural honey as an adjunctive alternative in the management of diabetic foot ulcers

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## Abstract

Diabetic patients have 25% lifetime risk of developing foot ulceration. More than half of these ulcers may eventually become infected, which greatly increases the likelihood of subsequent amputations. And although a multidisciplinary approach is the standard management for treating diabetic foot ulcers (DFUs), in the developing world diabetes is becoming an epidemic and resources are scarce; therefore, other alternatives are urgently sought. This case history demonstrates the feasibility of managing diabetic foot conditions by family physicians with a special interest in the diabetic foot using an alternative, cost-effective and efficacious wound dressing material, mainly natural honey, thereby representing a paradigm shift in the management of DFUs.

*Keywords:* natural honey, diabetic foot ulcer, primary care.

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## Introduction

Diabetic patients have around 25% lifetime risk of developing foot complications<sup>1</sup>, the most common of which is skin ulceration<sup>2</sup>. More than 50% of those wounds end up being infected, which exponentially raises the risk of below-knee amputation<sup>3-7</sup>. Although soft tissue infections represent the majority of diabetic foot infections, bone involvement accounts for 20–60% of cases<sup>7-9</sup>.

Furthermore, osteomyelitis has a worse outcome and often requires surgical resection and prolonged antibiotic therapy<sup>7,9</sup>. Surgical management aims to achieve control of the infection via drainage of any pus and removal of infected and necrotic tissue, thereby creating a healthy wound bed and removal of local pressure points, which hinders healing with the hope of salvaging the limb<sup>10</sup>.

This case study explores the emerging importance of natural honey as an ideal dressing in the management of the diabetic foot ulcer (DFU) and wound closure by secondary intention post-excision of osteomyelitic bone in a patient with type 2 diabetes.

## Patient history

The patient was a 55-year-old male Sudanese patient who presented to Um Gwailinah Primary Health Care Centre, Doha, Qatar, complaining of discoloured toes. The patient reported no history of trauma and after further questioning the patient reported loss of sensation in his feet which he described as being numb, and divulged that he had been diabetic for the last 15 years, and had no history of high blood pressure (BP) or smoking.

He was managed by oral hypoglycemic agents including Metformin 500 mg three times daily, Glicazide 60 mg once daily and 100 mg enteric coated aspirin.

His biochemical profile revealed HbA1c of 8.5% denoting uncontrolled glycaemia, according to the International Diabetes Federation (2005). His lipid profile was normal except he had a high LDL-C level, which showed a value of 3.5 mmol/L. His liver function tests and renal function tests including albumin/creatinine ratio, ESR and CRP were also normal. Vital signs examination revealed a temperature of 36.8° Celsius, a heart rate of 75 beats per minute, which was regular in rate and rhythm, a respiratory rate of 12 per minute and a BP reading of 130/83 mmHg with a BMI of 35 kg/m<sup>2</sup>.

At the time of presentation to the health centre, the patient was taking a BP-lowering therapy, ACE inhibitor (Lisinopril) 10 mg, and Simvastatin 10 mg, a cholesterol lowering drug.

An initial assessment was made by the consultant family physician who has a special interest in diabetic foot conditions. On examination of the left foot there were multiple necrotic skin lesions which were blackish and surrounded by macerated skin margins involving the second, third and fourth toes. Furthermore, his toenails were dystrophic, thickened and discoloured, especially the big hallux (Figure 1) suggesting either onychomycosis and or vascular insufficiency.

### Vascular assessment

Upon palpation, pedal pulses were manually palpable (dorsalis pedis and tibialis posterior), and this was further consolidated by Doppler examination using a hand-held Doppler (Huntleigh Super Dopplex 2; Huntleigh Health Care, UK). This revealed strong triphasic foot pulses (normal sounding) suggesting a lack of peripheral vascular disease.



*Figure 1. Initial presentation showing hyperkeratotic ulcers with necrotic overlying skin.*

The ankle brachial index (ABI; systolic ankle to brachial BP ratio) was also measured using the hand-held Doppler and a BP cuff (Reister Big Ben Round, Germany). The ABI was measured by dividing the systolic ankle pressure at the malleolar level divided by the higher of the two brachial pressures, which gave us a reading of 1.0, denoting normal ABI<sup>11</sup>.

### Neurological assessment

As the patient presented with a painless foot condition, sensory neuropathy was suspected. This was further proven by neurological assessment of light pressure utilising a Semmes-Wienstein 10 g monofilament, which was applied to the head of the big hallux and the bases of the first, third and fifth metatarsals which revealed loss of sensation. This was further consolidated by the use of the 128 HZ tuning fork applied to the big hallux showing loss of sensation. Finally, vibration sense was measured using the Biothesiometer which was applied to the big hallux and showed a recording of 65 volts, denoting diabetic peripheral neuropathy. The Biothesiometer was used as it provides a quick and reliable assessment of vibration threshold, which gives an objective measure of the degree and progress of diabetic peripheral neuropathy<sup>12</sup>.

Based on the findings of the vascular and neurological assessment, the aetiology of diabetic foot ulceration was felt to be neuropathic in origin.

### Wound management

Following the initial assessment, the consultant family physician used a sharp surgical scalpel to debride the second, third and fourth toes, removing all necrotic and devitalised tissue in accordance with principles of wound debridement, identification and management of infection<sup>13</sup>. Post debridement of the fourth toe revealed a visibly exposed distal phalanx which was necrotic, and surrounded by pus (Figure 2). Sharp debridement not only removes necrotic tissue but also enhances drainage and stimulates new tissue growth<sup>14</sup>. The formation of hard callus around the ulcers on the toes signified repetitive trauma which the patient denied due to diabetic peripheral neuropathy. Therefore removal of hyperkeratosis (callus) at the wound margin helped in leaving a clean ulcer margin and the removal of infected matter in order to help the wound close as reported by other studies<sup>15</sup>.

Following sharp debridement of the fourth toe, a previously invisible bone (distal phalanx) became apparent which was dry and necrotic and had appositional probe to bone test, signifying osteomyelitis until proven otherwise with an 89% positive predictive value<sup>16</sup>. According to Wrobel and



Figure 2. Toes show post-excision of necrotic tissue with visible, exposed distal phalanx of the second toe.

Connolly<sup>17</sup> a visibly exposed or positive probe to bone test was as predictive of osteomyelitis as x-ray, triple-phase bone scan, indicum or concrete (technetium) white blood cell scan, computed tomography (CT) scan and magnetic resonance imaging (MRI)<sup>18</sup>. The exposed bone was excised at the distal interphalangeal joint and sent to the laboratory for analysis along with the nail specimens to rule out onychomycosis. The patient was advised to go to hospital for further management but he refused flatly and asked to be treated at primary health care level.

Due to the localised nature of the visibly exposed distal phalanx, this represented moderate infection posing minimal immediate risk in a well perfused foot<sup>10</sup>.

After excising the distal phalanx and removal of all necrotic tissues the wounds were covered with natural honey and dry gauze was applied and changed on a daily basis. The site where the osteomyelitic bone was excised was left to close by secondary intention after applying honey to the wound. Two weeks later the toes have healed by 70% (Figure 3) where the base of the ulcers showed healthy granulation tissue with reduction of the size of the ulcers and clear ulcer margins free of hyperkeratosis. The ulcer began to fill from the inside out and from the sides of the ulcer with newly formed skin, signifying a rapid healing process facilitated by natural honey. This improvement continued till the end of the two weeks with no complications noted. At the end of the four weeks there was complete healing (Figure 4).

The use of honey as a wound dressing is not confined to ancient records, since recent reports in the medical literature supported the use of honey as a therapeutic agent in many clinical observations and randomised controlled trials including superficial wounds, burns, skin grafts, MRSA-infected wounds, catheter exit sites and pressure ulcers<sup>24</sup>.



Figure 3. Seventy per cent healing demonstrated by formation of granulation tissue and skin covering the digits.

We used natural honey and not "supermarket honey" since this type of honey is heat-treated, which destroys the enzyme responsible for the production of hydrogen peroxide<sup>19</sup>.

Despite the wide array of wound dressing products available in the market, none of these poses the unique features that natural honey has, making honey the ideal wound dressing in almost all wounds. These properties include: the provision of a moist environment, which enhances wound healing; protection of the wound; reduction of pain; and debridement of necrotic and devitalised tissue, which enhances granulation tissue formation<sup>20,21</sup>.

The viscous nature of honey offers a protective barrier, which prevents the wound becoming infected<sup>22</sup>. Furthermore, post-application the honey starts to dilute, preventing the dressing material from adhering to the wound bed, thus enabling the wound to be dressed without compromising the newly, partially healed wound<sup>23</sup>.

Honey continuously releases hydrogen peroxide, which aids in the debridement of the wound without damaging healthy granulation tissue. This is due to the fact that hydrogen peroxide levels produced by honey are approximately 1000 times lower in concentration than a standard rinse solution, killing offending pathogens without damaging the newly growing fibroblasts, that is, physiologically non-toxic<sup>23</sup>.

Honey also deodorises infected wounds<sup>24,25</sup> and offers an ideal, mildly acidic pH medium, thereby aiding fibroblast migration, proliferation and organisation of collagen<sup>26</sup>. Finally, natural honey has anti-inflammatory properties<sup>25</sup> and offers nutrients such as laevulose and fructose, which improves local nutrition in the wound, thus enhancing rapid epithelialisation<sup>20</sup>.



Figure 4. Complete healing seen with the formation of normal skin.

In this case we applied honey directly on the wound and covered it with sterile cotton gauze, which was changed on a daily basis coupled with offloading via a change in footwear in order to prevent further damage and facilitate healing. This is a pivotal aspect of DFU management since repetitive pressure as well as tight footwear places the foot at high risk of re-ulceration<sup>27</sup>. As a consequence, custom-made protective footwear "Pedors" were prescribed for the patient. These are made of canvas material and have a seamless forefoot to reduce rubbing and abrasion of the foot during walking.

During the initial treatment phase the nail culture showed *Trichophyton Rubrum* sensitive to Terbinafine, which was prescribed to the patient 250 mg daily for three months. The three-month regimen is as effective as a 12-month course according to Goodfield *et al.*<sup>28</sup> after conducting a randomised, double-blind, controlled trial.

The dressing change was facilitated by pouring sterile, normal saline on top of the gauze for two minutes prior to changing the dressing to prevent unnecessary damage to the newly formed granulation tissue, fibroblasts and capillaries. At week four the toes have healed completely.

### Long-term management

Although this case showed complete remission, the risk of future re-ulceration is as high as 25–80% even in specialised clinics<sup>2</sup>. As a result the patient was counselled by the consultant family physician to wear protective footwear "Pedors" continuously for the rest of his life to prevent re-ulceration. Most patients only wear protective footwear when they come for follow-up and neglect to wear them at home where they go back to wearing sandals or walk barefooted due to the hot weather in this part of the world. Wearing custom-made, protective footwear reduces

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foot pressure, especially when padded, thereby reducing both static and shear pressure. Furthermore, the effect of therapeutic footwear was studied by Edmonds *et al.*<sup>29</sup>, who found that ulceration reoccurred in 83% more cases in those wearing their own footwear, compared to those wearing therapeutic footwear.

Hence foot care and footwear instructions must be specific, targeted activities both outside and inside the house. Due to the high-risk nature of this case (peripheral neuropathy) we opted for a regular follow-up of this patient, with monthly scheduling<sup>30</sup>.

Health education is not enough to treat nor prevent or reduce recurrence of re-ulceration. A combination of tight glycaemic control, cholesterol-lowering therapy, anti-platelet therapy, BP control, psychological adjustment, family support, protective therapeutic footwear, regular screening, nutritional support, smoking cessation and an integrated health system will help to minimise the risk of ulceration and re-ulceration, especially among high-risk patients.

## Conclusion

This case demonstrated the feasibility and cost-effectiveness of managing diabetic foot complications at primary care level provided that the attending physician is competent and trained to handle such cases. The cost of the entire course of treatment using honey is US\$30. In comparison, using other products or dressings that contain silver or alginate cost between US\$40 and US\$588 per product unit. It has been estimated that in the United States the cost of treating a single DFU costs US\$8000, US\$17000 if it was infected and US\$45000 if it required amputation<sup>31</sup>. Hence, it is evident that using natural honey is very cost-effective.

The family physician with special training in managing diabetic foot conditions is at an added advantage compared to his counterparts including podiatrists and ankle and foot surgeons. This is because family physicians can provide other services to the patient, including metabolic control, and BP and cardiovascular risk management. They can carry out counselling and prescribe anti-depressants, since one-third of diabetic patients with foot ulceration are already depressed<sup>32</sup> and offer dietary advice. As a result, the involvement of family physicians with a special interest in diabetic foot management may represent a paradigm shift in the management of diabetic foot pathology.

## References

1. Singh N, Armstrong DG & Lipsky BA. Preventing foot ulcers in patients with diabetes. *JAMA* 2005; 293:217–228.
2. Lavery LA, Higgins KR & Lanctot DR. Preventing diabetic foot ulcer recurrence in high risk patients. *Diabetes Care* 2007; 30(1):13–20.
3. Lavery LA, Armstrong DG, Wunderlich RP, Boulton AJM & Tredwel JL. Diabetic foot syndrome; evaluating the prevalence and incidence of foot pathology in Mexican Americans and Hispanic Whites from a diabetes disease management cohort. *Diabetes Care* 2003; 26:1435–1438.
4. Armstrong DG & Lipsky BA. Advances in the treatment of diabetic foot infections. *Diabetes Technol Ther* 2004; 6:167–177.
5. Armstrong DG & Lipsky BA. Diabetic foot infections; stepwise medical and surgical management. *Int Wound J* 2004; 1:123–132.
6. Lavery LA, Armstrong DG, Wunderlich RP, Mohler MJ, Wendel CS & Lipsky BA. Risk factors for foot infections in persons with diabetes mellitus. *Diabetes Care* 2006; 29:1288–1293.
7. Lipsky BA, Berendt AR, Deery HG *et al.* Diagnosis and treatment of diabetic foot infections. *Clin Infect Dis* 2004; 39:885–910.
8. Norden C. Bone and joint infection. *Curr Opin Infect Dis* 1996; 9:109–114.
9. Lipsky BA. Osteomyelitis of the foot in diabetic patients. *Clin Infect Dis* 1997; 25:1318–1326.
10. Van Baal JG. Surgical Treatment of the Infected Diabetic Foot. *Clin Infect Dis* 2004; 39:S123–8.
11. Ouriel K. *The Lancet* 2001 13 Oct; 358:1257–1264.
12. Bloom S, Till S, Sonksen P & Smith S. Use of Biothesiometer to measure vibration threshold and their vibration in 519 non diabetic subjects. *Br Med J (Clin Res edn)* 1984 16 June; 288(6433):1793–1795.
13. Hinchcliffe RJ, Valk GD, Apelqvist J *et al.* A systemic review of the effectiveness of the interventions to enhance the healing of chronic ulcers of the foot in diabetes. *Diabetes/Metab Res Rev* 2008; 24(Suppl):S119–S144.
14. Edmonds M, Foster AVM, Vowden P (2004) Wound bed preparation for diabetic foot ulcers. In: European Wound Management Association (EWMA) position document: Wound bed preparation in practice. e.London: MEP Ltd, pp. 6–11.
15. Edmonds ME, Foster AVM. Diabetic foot ulcers: ABC of wound healing. *Br Med J* 2006; 332:407–10.
16. Grayson ML, Gibbons GW, Balough K *et al.* Probing to bone in infected pedal ulcers; a clinical sign of osteomyelitis in diabetic patients. *JAMA* 1995; 273:721–723.
17. Wrobel JS & Connolly JE. Making the diagnosis of osteomyelitis; the role of prevalence. *J Am Podiatr Med Assoc* 1995; 273:721–723.
18. Wagner FW. The diabetic foot. *Orthopedics* 1987; 10:163–172.
19. Molan P & Allen KL. Effect of gamma irradiation on the antibacterial activity of honey. *J Pharm Pharmacol* 1996; 48:1206–9.
20. Subrahmanyam M. Honey dressing versus boiled potato peel in the treatment of burns. A prospective randomized study. *Burns* 1996; 22:491–3.
21. Bello YM & Phillips TJ. Recent advances in wound healing. *JAMA* 2000; 283:716–8.
22. Medihoney. Honey and wound care. Brisbane, Australia: Capilano Honey Ltd, 2001.
23. Lusby PE, Coombes AL & Wilkinson JM. Honey: A potent agent for wound healing. *Wound Care* 2002; 29:295–300.
24. Molan P. The role of honey in the management of wounds. *J Wound Care* 1999; 8:415–8.
25. Molan P. A brief review of the use of honey as a clinical dressing. *Aust J Wound Manage* 1998; 6:148–58.
26. Calvin M. Cutaneous wound repair. *Wounds* 1998; 10:12–32.
27. Armstrong DG, Nguuyen HC & Lavery LA. Off-loading the diabetic foot wound. *Diabetes Care* 2001; 24(6):1019–22.
28. Goodfield MJD, Andrew L & Evans EGV. Short-term treatment of dermatophyte onychomycosis with Terbinafine. *BMJ* 1992; 304:1151–4.
29. Edmonds M, Blundell M & Morris M. Improved survival of the diabetic foot: the role of specialized foot clinic. *Q J Med* 1986; 60:763–771.
30. Fykberg RG, Zgonis T, Armstrong DG, Driver VR, Giurini JM, Kravitz SR & Landsman AS. Diabetic foot disorders. A clinical practice guideline (2006 revision). *J Foot Ankle Surg* 2006 Sept–Oct; 45(5):S2–S66.
31. Kruse I & Edelman S. Evaluation and Treatment of Diabetic Foot Ulcer. American Diabetes Association. *Clinical Diabetes* 2006 Apr; 24(2):91–93.
32. Ribu L, Hanestad BR, Moum T, Birkeland K & Rustoen T. A comparison of health-related quality of life in patients with diabetic foot ulcers with diabetes group and non diabetes group from the general population. *Qual Life Res* 2007; 16:178–189.