**Case Study: Tophaceous gout lesions: problem wounds with complex management needs**

**Introduction**

Hyperuricaemia is the underlying cause of a condition commonly known as gout. Patients with hyperuricaemia may be asymptomatic for life or may experience frequent attacks of painful gouty arthritis.2 Gout is, therefore, a condition with both acute and chronic characteristics.3

It is a priority of the management of chronic gout to lower the serum urate (SU) level to below 6 mg/dL, which is the goal range.3 Failure to do so allows the SU level to cross the limit of solubility in the body. Once that threshold is crossed, it ultimately results in the laying down of uric acid deposits in joints and soft tissue,4 as the ability of the kidneys to clear the body of uric acid is reduced. The long term complications of poorly controlled uric acid levels include recurrent acute incidences of flare-up and the development of tophaceous gout.1

**Pathophysiology**

Gouty arthritis2 is caused by articular deposits of monosodium urate (MSU) crystals, either in soft tissue or in joints and ligaments, leading to an acute inflammatory reaction5 that is usually very painful.2 Adult men are often the victims of this condition,6 the severity of which is directly related to dietary habits and the intake of alcohol.2 Although literature suggests that only 10% of all gout sufferers may develop tophaceous gout lesions,4 it should not be underestimated, as the condition can destroy not only the joint or tissue affected, but also the skin overlying the lesion.7

Pressure on the skin, from the inside out as the tophaceous lesion develops and grows bigger, will subsequently lead to skin ulceration with small leaky areas and resultant wound infection.7 Surgery then becomes necessary, as debridement of the tophus8 becomes the only intervention that will spare some of the joint, underlying structures and skin stretched to the point of bursting.

**Postoperative tissue necrosis**

Due to the very poor condition of the overlying skin, tissue necrosis postoperatively2 is a huge risk. An added risk factor for postoperative wound complications, is the fact that this patient is most probably taking non steroidal anti-inflammatory drugs (NSAIDs) as chronic management regimes,9 which all have an effect on the inflammatory response needed to achieve surgical skin repair.

Another factor to keep in mind is that the chemical destruction of the joint also affects the integrity of the skin in all its layers. This process involves complement membrane attack complex (C5b-9)5 with the increased secretion of cytokines, chemokines and more inflammatory mediators, and the resultant migration of neutrophils into the area. In our department, that skin condition is informally classified as “chemical cellulitis”. Once the damage is done locally, it is very hard to reverse with systemic treatment only.7,8

**Case study**

This patient is a 53-year-old-male with a history of chronic gout attacks from the age of 25 years. He has been treated with colchicine, on and off, since then. His body mass index is 21 and he uses no alcohol, but he smokes 30 cigarettes a day.

His first tophaceous lesions appeared at around the age of 40. Most of the lesions were removed surgically when their size or the acuteness of the pain made it intolerable for him. In the last five years, he has had surgical interventions to his fingers, hands, forearms, elbow joints, ankles and toes.

At the time he presented with this foot lesion (figure 1), he was still recovering after tophus removal surgery to his left elbow and had a pencil drain in situ that was shortened and removed after a week. Surgery to the foot was withheld until full closure of the elbow was

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achieved. He was taken for surgical excision of the tophus mass on 11 March 2010. Unfortunately, the tophus mass penetrated through the skin a day before surgery was done.

Postoperatively, on opening the dressing, the initial stages of necrotic sloughing of the skin was visible, although it was not yet demarcated (figure 2). There was also a haematoma in the incision. The still continuous draining of gouty crystals from the joint was also clearly visible in the macerated area in the middle of the incision. The patient was complaining of procedural pain in the range of 8 on the Visual Analogue Pain Scale (VAS) on this particular day. Wound bed culture was sent off to the laboratory, as the wound had a strange putrid smell. The patient was taken back to theatre for a debridement and removal of the haematoma. The wound bed was not sutured again, and was covered with a honey based preparation.

Four days later, there was still some necrotic tissue visible that demarcated after surgery. All the loose necrotic skin was removed at the bed side in a sharp debridement procedure performed by the surgeon himself. The destruction of the joint and tissue structures are clearly visible in figures 3 and 4.

Every wound dressing procedure was extremely painful for the patient, despite the administration of analgesics, distraction of the patient, and very gentle management of the wound bed. Later, he could only tolerate lukewarm fluid irrigation of the wound bed. Pain, as indicated on the VAS, remained between 8 and 10.

The wound bed culture revealed a *Clostridium* spp. anaerobic infection, together with coagulase-negative *Staphylococcus aureus*. Clinical signs of *Clostridium* spp. anaerobic soft tissue infection include pain out of proportion to the lesion, a putrid smell and a bronze discolouration of still intact skin. Given the extent of destruction to underlying structures and the impaired local inflammatory response, infection risk is not to be underestimated in this type of tophus surgery.

The patient was treated with oral antibiotics and wound dressings with a honey base were still applied which, unexpectedly, did not add to his pain. The application of the honey based ointment resulted in granulation formation and some reduction in wound bed size. The effect of the osmolarity of the ointment is clearly visible in the surrounding skin (figure 5), which now appears free of swelling, slough or necrosis and is starting to adhere to the wound bed. The smell had improved. Progress, though, was slow, and the VAS pain levels remained at 8-10 during procedures, particularly on the dorsal side of the foot sole.

Due to the slow progress and the severity of the pain during procedures, the surgeon decided to readmit the patient to hospital on 30 March for aggressive treatment with intravenous antibiotics. It was also decided to set up a continuous wound irrigation system with saline and Betadine® solution, in equal proportions, directly delivered to the wound bed via a separate tubing system. Although this procedure is thought of as old fashioned, it enabled the flushing of the deeper side of the wound bed to get rid of the bio-burden and enhanced granulation formation at the same time. Continuous irrigation also cleaned the wound without the application of fingers or forceps, resulting in a reduction of pain to the level of 5 on the VAS scale.
Continuous irrigation was undertaken for six days, with 6 litres of fluid altogether in total. The wound bed was so well granulated that a skin graft to close the huge tissue loss area was performed on 5 April.

The patient was followed up meticulously and dressings were renewed, at first, every 48 hours and, after a week, twice a week. Pain reduction was complete, with VAS level at 0 without any need for analgesia at any given time after the skin graft was done. (See figures 6 – 9.)

Discussion

In the literature on surgical intervention for the management of tophaceous gout, it is clear that the complexity that accompanied this case is the norm rather than the exception. Surgical intervention (in this case, simple open debridement) is an option for these patients, although techniques may vary. Minimally invasive surgery, with small incisions and utilising a shaving technique, may curb some of the tissue loss postoperatively. There has been criticism in cases where tendons, ligaments and small nerve fibres are involved, because the clinician cannot control which functional structures are lost into the shaver, as it is a partially blind procedure.

Closure of the tissue deficit after surgery cannot be achieved by depending on the patient's own ability to heal. Not only will healing be slow, but it will also result in the deposition of poor quality collagen due to the impaired and halted local inflammatory response resulting from the gout treatment regimen.

This case involved complicated surgery and wound care alike. The outcome was determined by a team that was formed around this patient, comprising an orthopaedic surgeon, a clinical nurse specialist in wound care, a dietitian, the hospital ward personnel and the patient himself, who had to stop smoking within a day to prevent further tissue loss around the incision.

The patient is fully mobile again and the lesion has not flared to date. He has no pain at all in the foot and can wear closed, soft shoes. He started smoking again after three weeks, though a limited number of cigarettes per day, and has had no problems with the graft area. The challenge is now to keep his SU levels in such a tight range that flare can be prevented. He has been referred to a physician who will now take over the management of this patient with this rare extensive tophaceous gout condition.

References