Advanced wound management of squamous cell carcinoma and systemic lupus erythematosus: Case report

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This case report demonstrates the management of a wound in the forehead with exposed underlying bone caused by a squamous cell carcinoma (SCC) wound, in a patient with systemic lupus erythematosus (SLE). An understanding of the relationship between inflammation, autoimmune conditions and SCC is discussed. Furthermore, the importance of a multidisciplinary team approach to achieving wound healing using fenestration and good wound management is demonstrated. In this patient, the fenestration technique showed to be effective and led to a 70% decrease in wound size by week eight.

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Introduction

Normal wound healing is a complex event occurring over four overlapping stages, namely haemostasis, inflammation, proliferation and maturation, to achieve complete wound closure. However, the relationship between rare autoimmune diseases and the effect thereof on delayed wound healing is under-recognised.[1] Autoimmune diseases activate chronic inflammation that contributes to delayed wound healing and the development of various types of cancer such as squamous cell carcinoma (SCC).[1] The relationship of cancer-related inflammation and delayed healing associated with chronic inflammation, includes the presence of inflammatory cells and mediators in tumour tissue, angiogenesis and tissue remodelling occurring during wound healing.[2] Therefore, the presence of chronic inflammation and non-healing wounds may contribute to the formation of SCC. This case study demonstrates the management of a wound on the forehead with exposed underlying bone caused by an SCC in a patient with systemic lupus erythematosus (SLE).

Case report

An 81-year-old female presented at a private wound care clinic in Pretoria, South Africa, with multiple infected ischaemic skin lesions to her right lower leg, the residual phalanx of amputated digits 1, 2, 4 and 5 of the right hand and residual phalanx of digits 2 and 5 of the left hand. The patient was known to have been diagnosed with SLE at the age of 23 and had developed a vascular condition associated with systemic scleroderma (CREST syndrome),[3] which led to the spontaneous amputation of distal phalangeal joints on both hands. The patient also had rheumatoid arthritis, osteoporosis and diverticulitis. The patient was allergic to citrus fruits and presented with mouth ulcers extending to the oesophagus. The patient also appeared skinny and confirmed weight loss over the past few months.

The patient was treated for multiple infected ischaemic lesions using Acticoat® (Smith & Nephew, UK) dressings as from March 2020, leading to the decrease of wound size and decrease of odour and exudate. Due to SLE delayed wound healing was expected. However, a delay in wound progression occurred early in June 2020, as no further decrease in wound size could be observed. After a thorough holistic assessment of the patient, a new ulcer was noted on the forehead. The ulcer first presented as an elevated lesion measured at 1 x 0.5 cm, that rapidly increased to 3.5 x 2 cm over the following two weeks (Figure1).

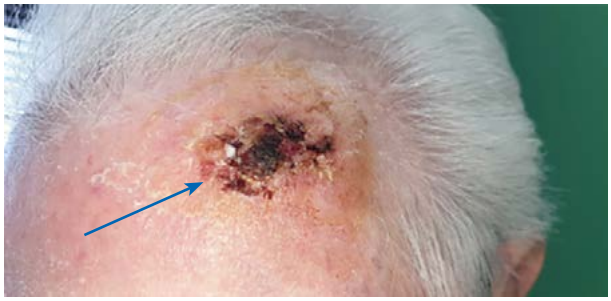

Figure 1: SSC lesion with 3.5 x 2cm dimensions after biopsy

The patient was referred to a plastic surgeon who performed a biopsy of the wound on the forehead and the right lower leg. The biopsy confirmed the diagnosis of a moderately differentiated, keratinising SCC on the forehead, but found no signs of neoplasia in the right lower leg.

The patient underwent surgical excision of SCC with a skin graft in July 2020 and the intraoperative frozen section confirmed clear surgical margins. Wound management was continued three times per week using a single-layer Jelonet (Smith & Nephew, Canada) covered with Inadine™ (Systagenix, USA) and crepe bandage. Despite regular wound care and nutritional supplementation, the skin graft failed (Figures 2A and 2B).

Surgical debridement with a scraping of the exposed underlying bone was performed in theatre on day 14 post first operation (Figure 3A).

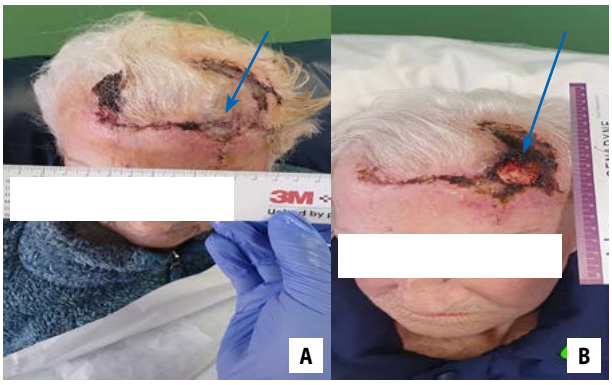

Figure 2: Postoperative pictures of the patient. A: Four days after surgical procedure, graft still in place; B: 11 days after the operation, graft failed.

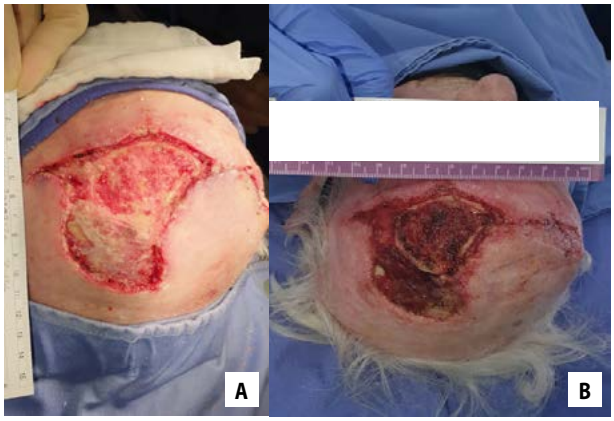

Figure 3: A: Intraoperative picture of the first surgical debridement, leaving a clean surgical wound with a viable wound bed; B: Deterioration of the wound 24 hours after the operation.

Negative pressure wound therapy (NPWT, Clinigen, SA) was applied using white foam to protect the exposed bone, and a -50 mmhg setting of pressure. A follow-up after 24 hours showed that the wound had deteriorated as shown in Figure 3B.

The NPWT was discontinued and autolytic debridement commenced using a honey-based ointment (L-Mesitran®, Safarmex, Europe) and absorbent adhesive dressings (Allevyn, Smith and Nephew™, UK) on alternating days. A third surgical wound debridement was done early August 2020 (Figure 4). During the procedure, the plastic surgeon drilled several 2 mm ectocranial holes into the exposed skull, a procedure referred to as fenestration.[4] The wound was treated using honey-based soft wound gel (L-Mesitran®, Safarmex, Europe) and single-layer Jelonet™ (Smith & Nephew, Canada), covered with sterile absorbent

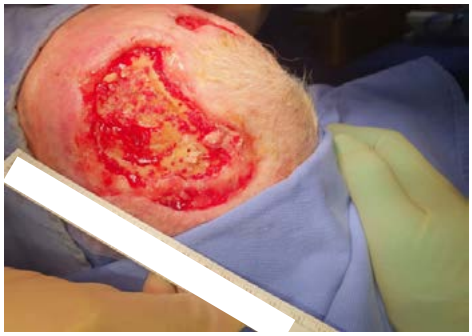

Figure 4: An intraoperative picture of the third surgical debridement with the fenestration of the exposed bone. Wound: 8.5 x 7 x 0.8 cm.

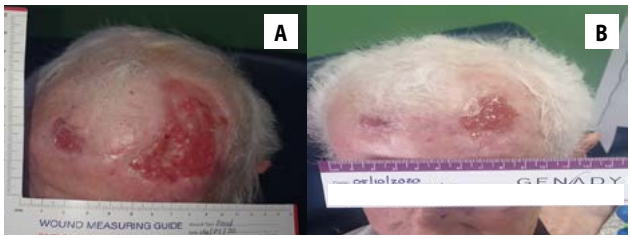

Figure 5: A: After four weeks of last surgical procedure, wound: 5 x 4 cm; B: After 60 days, wound: 3 x 2.5 cm.

adhesive dressings (Allevyn™, Smith & Nephew, UK). Dressings were changed on alternating days.

The wound progressed forming viable granulation tissue covering the exposed bone within 30 days of the last procedure. Once the exposed bone was fully covered, dressing materials were changed to Cutimed® Sorbact® (DACC, BSN Medical, Germany) antimicrobial dressings twice a week. By week eight, the wound bed had achieved 70% epithelial advancement, with no indication of wound breakdown or stagnation of wound growth as previously noted (Figure 5A and 5B).

Discussion

During the excision of SCC of the scalp, the patient is often left with a wound with underneath exposed skull which can be covered by means of a local cutaneous flap (ideally) or a skin graft. When flap or graft coverage is not possible, the wound is left to heal by secondary intention. During the excision procedure, the exposed bone may be stripped from the periosteum, leading to a delay in the formation of new connective tissue and wound healing.[5] Furthermore, autoimmune disorders such as SLE impair wound healing due to its role in the inflammatory phase of healing. The mechanism of chronic inflammation in patients presenting with SLE is contributed by leukocyte adhesion deficiencies (LADs). Three types of LAD are present namely: LAD-I caused by a gene mutation that encrypts the integrin's β2 chain; LAD II caused by a genetic defect resulting in the absence of viable selectin ligands in the synthesis of fucosylated glycostructures; and LAD III caused by a leukocyte integrin genetic defect.[6] The occurrence of all the LADs results in reduced neutrophil migration to the injured tissue and a loss of neutrophil phagocytosis in the wound bed, leading to prolonged inflammation and a delay in wound healing.[6]

By making use of fenestration, the surgeon uses a drill to burr several small holes (2 mm deep and 5 mm apart) in the skull, allowing access to the cortical bone circulation.[4] Superficial bleeding occurs, initiating the phases of wound healing. During the proliferative phase, the granulation tissue is formed from within the fenestra canals and from the wound edges, thereby attaining cover of the exposed skull. Although fenestration has the risk of thermal injury to the bone that may cause delayed healing, it also facilitates granulation formation by up to 0.5 mm granulation migration per day.[4] Due to the poor elasticity of the scalp, wound contraction will be minimal; more likely, the migration of epithelial cells from the wound edges will occur to achieve complete wound closure.

A moist wound environment is required to promote optimal wound healing. In this case, paraffin tulle was impregnated with a honey-based soft wound gel and placed directly to the wound bed and exposed

skull, covered with an absorbing secondary dressing. The dressing selection has shown to be cost-effective, as the 50 g honey-based soft gel was used throughout the first four weeks of wound healing until full granulation formation was achieved.

Honey-based gel (L-Mesitran®, Safarmex, Europe) has anti-microbial properties due to its strong osmotic activity, decreasing bioburden and inflammation, and also contains vitamins C, E and Zinc oxide which promote cell migration.[7] The primary dressing was covered with an absorbing adhesive dressing. Due to the formation of hyper-granulation tissue on the wound bed, the product was changed to Cutimed® Sorbact® (DACC, BSN Medical, Germany) anti-microbial dressings twice a week; this aided in the decrease of hyper-granulation formation and promoted epithelial advancement which was continued until complete wound closure was achieved. The total product cost of conventional wound care over eight weeks was valued at R3 627.80, which was approved and covered in full by the medical funder.

Conclusion

This case study describes the use of fenestration on the exposed bone of the forehead, to promote the formation of new connective tissue and wound healing by secondary intention in a patient with SLE. In this patient, the fenestration technique showed to be effective and led to a 70% decrease in wound size by week eight.

Conflict of interest

The author declares no conflict of interest.

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Ethical approval

Patient consent was obtained.

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