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# Treatment of exposed tibial bone by old school burr holes – a case report

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## Summary

**Background:** We report a successful wound treatment of a chronic ulcer with bone exposure using a somehow forgotten technique of creating burr holes into the bone. Most clinics would promote flap surgery to cover wounds with bone exposure, however, in some cases invasive surgery is not mandatory. We bring up an alternative treatment for such cases. **Case:** We report a case of chronic ulcers on both lower extremities in a 43-year-old Caucasian male. He suffers from a leukocytoclastic vasculitis and sarcoidosis which is medicated by immunosuppressive medication. The patient's wounds were initially treated with mechanical debridement and split-thickness skin grafts, however, his wounds tended to worsen the more they were manipulated and finally resulted in tibial bone exposure. After levelling up his immune suppressive drugs, the wounds finally stabilized but didn't heal after several weeks of follow-up. The wound was ultimately treated by placing burr holes in the underlying cortical bone. **Conclusion:** Chronic ulcers with bone exposure at the lower leg are challenging to treat. They often require local or free flap surgery. In some cases, because of underlying systemic disease, it is mandatory to stay away from invasive flap surgery. With this case, we like to put under attention an old technique of decorticating the exposed bone to promote secondary wound healing. It has been described mainly for scalp injuries, however, we have proven the viability of this technique for pretibial wounds as well.

**Key words**

lower limb reconstruction – burr holes – chronic wounds – limb salvage

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## Introduction

Pretibial wounds are a common injury and are frequently presented to plastic surgery department. This pathology may evolve into a chronic ulcer mainly in the elderly or individuals with substantial comorbidities, as their fragile skin makes them susceptible to wounds from even minor trauma [1]. The healing of wounds involves a sequential progression through four phases – hemostasis, inflammatory, proliferative, and remodeling. Various factors, such as infection, necrosis, poor perfusion, inadequate angiogenesis, smoking, and underlying conditions (e. g., diabetes, vasculitis, vascular insufficiency), can lead to delayed healing [2–4]. Full-thickness wounds heal through formation of granulation tissue, wound contraction and ultimately, re-epithelialization from the wound margins [5]. Healing time depends on various factors, such as age, location, depth, and whether the periosteum has remained intact [6]. Wounds with exposed bone, especially when devoid of periosteum, often exert delayed healing or do not heal at all due to insufficient granulation of the wound bed [7]. Furthermore, prolonged exposure of cortical bone without its periosteum may lead to desiccation and devitalization of the outer cell layers of the bone, thereby causing additional delays in wound healing [8]. In the past, placement of burr holes or fenestration of the cortical bone has proven to be a valuable method for treating post-traumatic scalp injuries [9] lesions after Mohs surgery on the scalp [8,10]. Afterwards, this bed of granulation tissue can re-epithelize from the wound margins or may be skin grafted [11].

Fenestration of cortical bone should be considered in poorly healing wounds with exposed bone [5]. If the exposed bone is deperiostated and the outer lining of the cortex has dried out and shows poor viability, the growth of additional granulation tissue can be encouraged using a power drill to create burr holes, distant about 5–10 mm from each other. Alternatively, the exposed cortex can be completely removed up to the spongy bone or can be thinned out until bleeding occurs out of the deep cortical layer, reaching the haversian system [8].

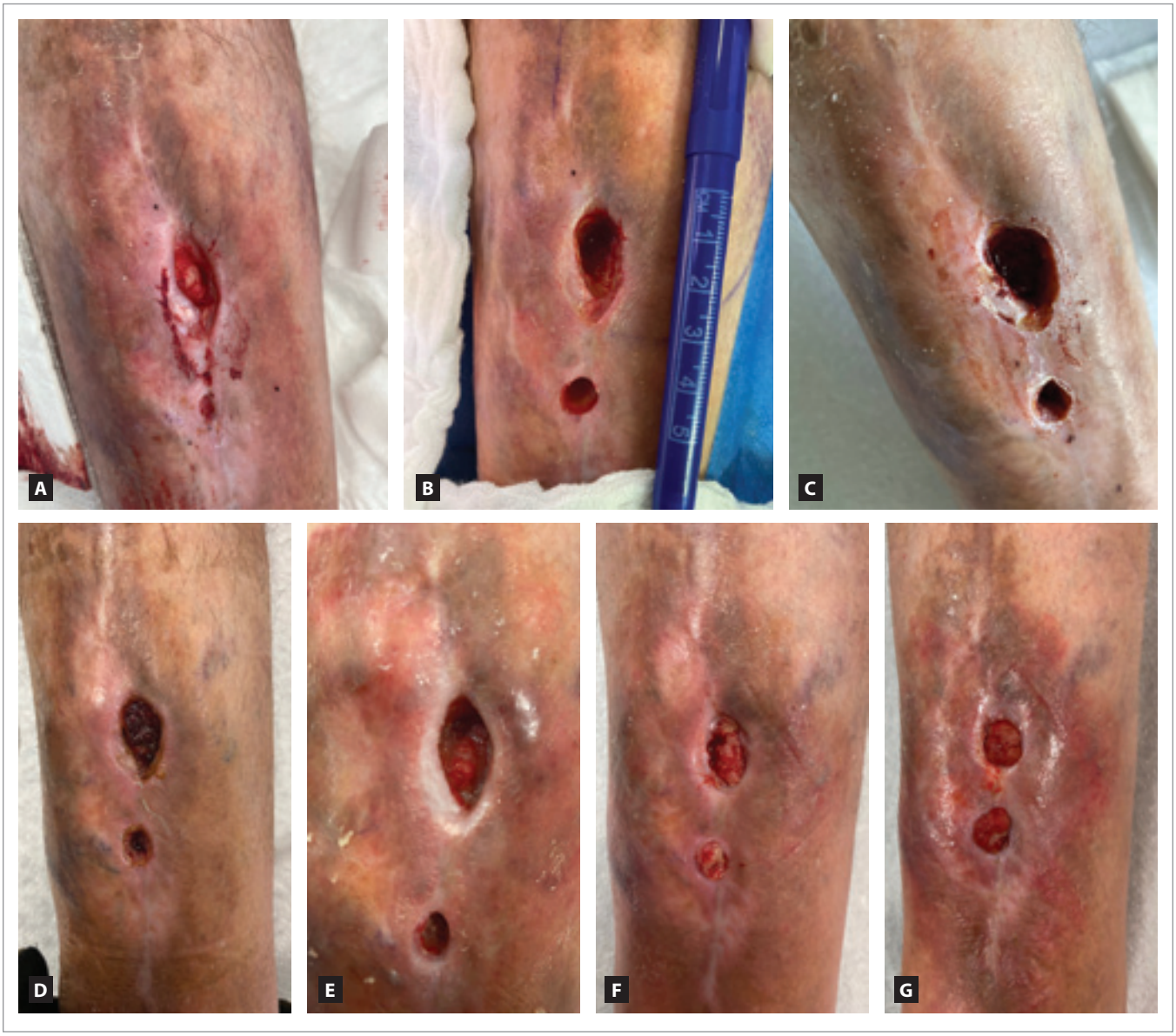


**Fig. 1. A) Pretibial chronic wound at first presentation. B) Peroperative state of the chronic wound after debridement. The denuded area measured 2 × 1 cm in size. C) State of the wound at 2 weeks after burr hole placement. D) 3 weeks after burr hole placement. At this point, the entire wound bed was covered with granulation tissue and no exposed bone was visible. E) 7 weeks after burr hole placement. F) 12 weeks after burr hole placement. G, H) 16 weeks post burr hole placement. Formation of hypergranulation tissue above skin level. At this point, treatment with silver nitrate was started.**

Next to power driven instruments, classical chisels or rongeur can be used. The objective is to reach a tissue level with superior vascularity which will induce wound healing, as mentioned above.

## Case report

A 43-year-old male suffers from chronic ulcers on both lower extremities. A small injury led to a chronic ulcer due to a complex medical history of type 2 diabetes mellitus, chronic obstructive pulmonary disease, sarcoidosis, and leukocytoclastic vasculitis (LCV) with erythema elevatum diutinum (EED). The patient is also a former smoker with a 20-packyear history.

In the middle of 2019, 4 years before our treatment plan began, the patient started experiencing joint pain in the elbows, hands, and knees. Around the same time, multiple erythematous papulonodular lesions appeared on the elbows. As a result, a peripheral blood sample was collected, and both rheumatological and dermatological examinations were conducted. X-rays of the hands and feet revealed no abnormalities, and the peripheral blood smear along with blood tests also showed no

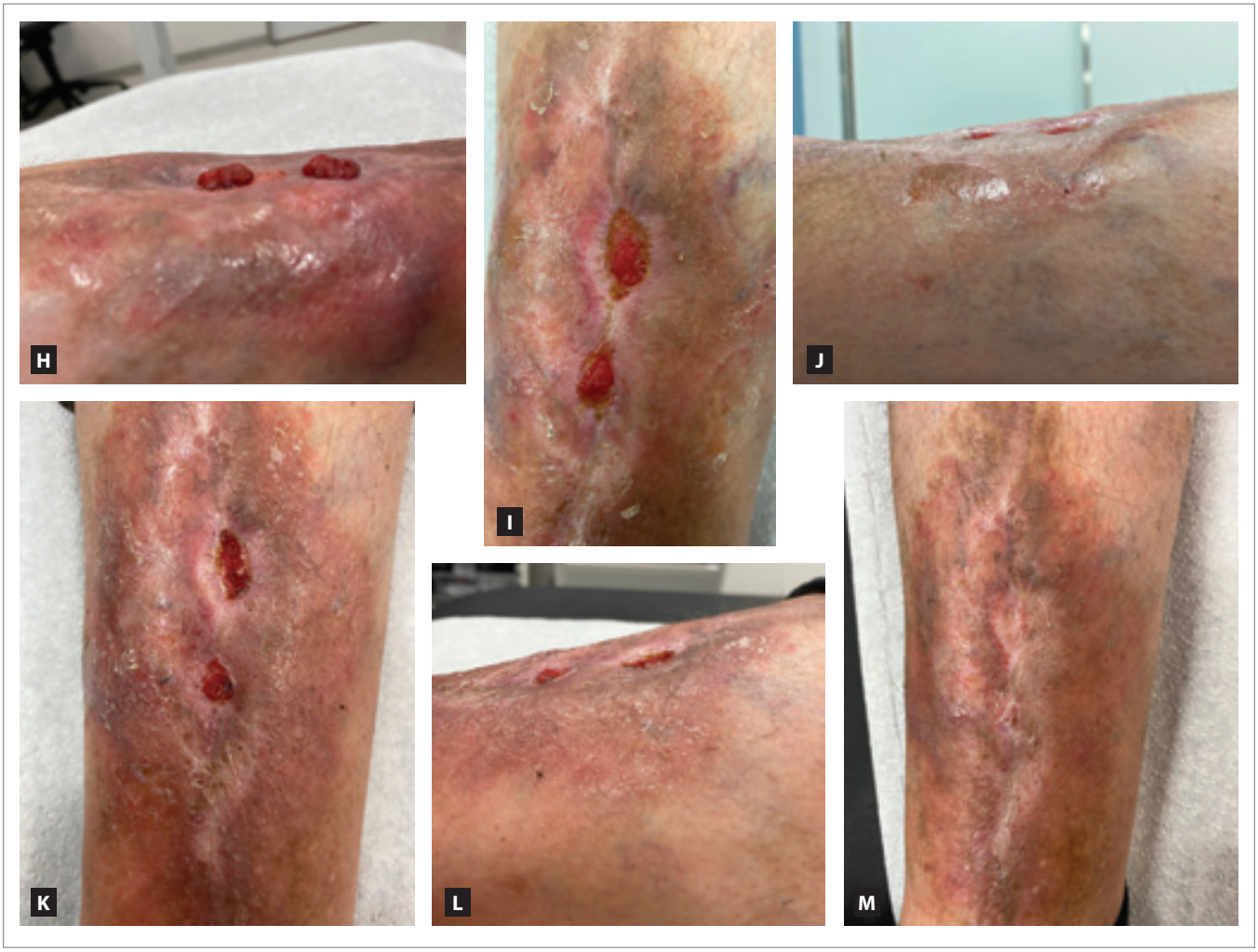


**Fig. 1 – continuing. G, H) 16 weeks post burr hole placement. Formation of hypergranulation tissue above skin level. At this point, treatment with silver nitrate was started. I, J) 18 weeks post burr hole placement. Hypergranulation has flattened and is nearly managed. K, L) 20 weeks post burr hole placement. The hypergranulation tissue had fully flattened. M) 24 weeks post burr hole placement. Fully healed wound after cortical bone fenestration and secondary intention healing.**

signs of rheumatological disease. A biopsy of one of the lesions was performed, and the histological examination revealed a pattern resembling erythema elevatum diutinum, suggestive of LCV. Immunosuppressive therapy with corticosteroids was initiated to alleviate and manage the patient's symptoms.

In early 2021, approximately 1.5 years after the diagnosis of EED lesions, the patient consulted the University hospital due to pretibial chronic ulcers on both lower legs. The patient reported that the ulcers had developed from papulonodular lesions, suggesting that LCV was the likely cause. Consequently, corticosteroids were replaced with methotrexate (supplemented with folic acid), and topical treatment with hydrogel was initiated. This treatment regimen led to an improvement in the pretibial ulcers.

Despite initial improvement, by the end of 2021, large ulcers had developed on both lower legs. The wounds were covered with a thick white layer of fibrinous tissue. Systemic corticosteroid therapy was reinstated and combined with antibiotics and NSAIDs. Wet-to-dry debridement was performed, and within a few days, healthy granulation tissue had formed on the wound bed. Subsequently, the wounds were treated with split-thickness skin grafts. Although wound healing was delayed, the grafts adhered well in the center of the wounds. The patient was discharged with instructions for daily wound care and a tapering schedule for the corticosteroids. After a brief period of healing, the patient's ulcers deteriorated further, prompting a referral to the University hospital for comprehensive systemic analysis and treatment. It was decided to escalate the immunotherapy to a combination of corticosteroids and methotrexate.

Roughly 17 months after the initial presentation of the pretibial

ulcers, the pretibial ulcer on the right leg healed without any further intervention. Unfortunately, the left pretibial ulcer worsened and the tibial bone was exposed (Fig. 1A). After informed consent we removed the fibrotic tissue and removed the underlying cortex which created a cavity that measured 2 × 1 cm (Fig. 1B). In the following weeks, the wound was treated with Flaminal® Hydro, then covered with a paraffin gauze dressing and a dry bandage. Figures 1C–E show the wound at weeks 2, 3 and 8, respectively. Finally, tissue granulation appeared in the subsequent weeks. Within 3 weeks, the wound bed was entirely covered by granulation tissue, eliminating any exposure of bone. By the 12$^{th}$ week, the entire cavity had been filled with granulation tissue up to the skin level (Fig. 1F).

Over the subsequent weeks, an excess of granulation tissue developed above the skin surface (Fig. 1G, 1H), but this issue was effectively addressed through cautery with silver nitrate and almost resolved by week 18 (Fig. 1I, 1J). Figures 1K and 1L depict the wound at 20 weeks, with the granulation tissue fully flattened. By the 24$^{th}$ postoperative week, the wound had achieved complete closure (Fig. 1M).

## Discussion

The case presented demonstrates the complexity and challenges involved in the management of chronic pretibial ulcers, particularly in the context of underlying systemic conditions like LCV and EED. Despite initial improvement following immunosuppressive therapy including corticosteroids and methotrexate, the patient experienced recurrent ulcerations, culminating in infection and the need for surgical debridement which resulted in exposure of the bone tissue.

Chronic ulcers, such as those described in this case, often follow a prolonged and relapsing course. The fact that the ulcer on the right leg healed without further intervention after 17 months illustrates the unpredictability of wound healing in complex cases.

Identifying the underlying cause of chronic ulcers is crucial for developing an effective treatment strategy. While most leg ulcers are commonly linked to venous insufficiency, arterial insufficiency, diabetes, or a combination of these factors, rare conditions can sometimes be the root cause [12]. In this case, the diagnosis of EED was already confirmed through histological analysis, and the presence of papulonodular lesions on the tibia, preceding the ulcers, strongly suggested it as the cause.

Another rare cause of chronic ulcers is pyoderma gangrenosum (PG), a specific form of ulceration with non-leucocytoclastic vasculitis caused by an autoinflammatory process [13]. Although PG and EED may initially appear similar, there are several key differences between these two conditions. EED is a form of chronic LCV characterized by firm nodules or plaques that may gradually progress to ulcers, typically at a slow rate. In contrast, PG is a neutrophilic dermatosis that begins with nodules that quickly evolve into painful, rapidly progressing ulcers.

EED is associated with small vessel inflammation and can be triggered by infections, rheumatologic, and hematologic diseases. PG, however, is frequently associated with inflammatory bowel diseases, arthritis, hematological malignancies, and several other diseases, including auto-inflammatory disorders. Surgery or any skin trauma makes the disease worse. PG may even be triggered by skin trauma, a process known as pathergy [14].

Both conditions are managed with immunosuppressants, but in the case of PG, timely diagnosis and addressing the underlying disease is especially crucial due to the grave consequences of misdiagnosing the condition for necrosis or infection [13].

Worsening of chronic tibial ulcerations after surgical therapy or pathergy is not unique to PG.

Skin necrosis after trauma can also occur in Behçet's disease, calciphylaxis cutis, and critical limb ischemia [14].

In the future, every patient with a leg or foot should be examined extensively to determine whether the cause is arterial, venous or diabetic. If an ulcer appears in an unusual location, presents with uncommon clinical features or symptoms, and does not respond to standard treatments, it is important to consider the possibility of an atypical etiology [15]. For patients with an unclear diagnosis, tissue biopsy is often essential in narrowing down the differential diagnosis. Histological examination can help identify ulcers with vasculitic or vasculopathic origins, as well as those caused by infections or malignancies [16].

This case underscores the importance of a multidisciplinary approach in managing chronic, non-healing ulcers, especially when complicated by systemic diseases. Long-term immunosuppressive therapy, wound care, and timely surgical interventions were critical in trying to stabilize the patient's condition and preventing further complications. Future management should focus on preventing further ulcerations by closely monitoring the patient's condition and response to ongoing immunosuppressive treatment.

## Conclusion

Treating chronic ulcers with bone exposure on the lower leg poses a significant challenge, often necessitating local or free flap surgery. However, in certain cases where underlying systemic diseases are present, invasive flap procedures may need to be avoided. In this case, we highlight an older technique involving the decortication of exposed bone to encourage secondary wound healing. While this method has primarily been documented for scalp injuries, we have demonstrated its effectiveness in managing pretibial wounds as well.

**Roles of the authors**

Coskun Arkaz – concept, design, data acquisition, data analysis, manuscript preparation, manuscript editing;

Whitney Van Damme – literature search, data acquisition, data analysis, manuscript preparation; Geert Peeters – design, manuscript preparation, manuscript editing, manuscript review.

**Declaration of patient consent to participate and publish:** The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.
All procedures performed in this study involving human participants were in accordance with ethical standards of the institutional and/or national research committee and with the Helsinki declaration and its later amendments or comparable ethical standards.

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