THE SILENT THREAT – ALKALI FOOT BURN

A CASE REPORT

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Introduction

Despite current workplace protection measures, chemical burn accidents are not uncommon. **(1)** Within chemical burns, alkali burns are the most challenging due to their silent behaviour and aggressive mechanism. Alkali chemicals cause damage by liquefactive necrosis, leading to saponification of fats and denaturation of proteins. **(2)** Characterized by its initial painless onset, allowing a prolonged exposure, they tend to result in deeper and more destructive burns, creating major reconstructive and therapeutic challenges. This case concerns a 53-year-old male who sustained a deep foot burn after prolonged contact with an alkali substance.

Case report

A previously healthy 53 year-old male, presented to the emergency department with a left foot burn. A detailed medical history revealed a prolonged contact with an alkali blend, containing sodium hydroxide, through his working-boots. The accident occurred by dropping off this product on his left boot while working, which the patient neglected. Within several hours he developed pain, took off his boots and noticed significant oedema and a wound on his left instep. He was then admitted in the emergency department with a full thickness burn of his left instep foot and on the dorsum of the 1st to 4th toes, corresponding to approximately 2% of body surface area **(Fig.1)**. Intravenous fluid resuscitation and copious wound irrigation were promptly applied and later admitted in the burn unit with silver sulfadiazine dressing. Then the wound was surgically debrided several times with tangential scarectomy with preservation of the tendinous and vascular-nervous structures, with exception of some superficial veins and cutaneous nervous branches **(Fig.2 and 3).** The skin defect was then repaired with expanded 1:1.5 partial thickness skin graft from the anterior surface of the left thigh **(Fig.4)**. Since this type of burn is associated with development of chronic neuropathic pain, it was crucial to treat it aggressively. Pain was successfully managed before and after the surgery with the use of multimodal analgesia with tramadol, morphine, ketamine and acetaminophen. Four weeks after surgery, it presents good healing evolution, complete graft integration and satisfactory aesthetic results **(Fig.5).** No relevant algic complaints or sensory changes were reported.

Discussion

In this case, the late onset of symptoms allowed a prolonged aggression, in combination with pathophysiology of alkali burn resulting in a deep burn. Early recognition and prompt management with copious and prolonged wound irrigation is paramount. **(3, 4)** As in this type of burn it is difficult to initially assess its true depth, even after initial surgical debridement, a more cautious approach is recommended. Even when only a limited body surface area is involved, some chemical burns can cause systemic toxicity, so identifying the products involved is crucial during initial approach. Chronic pain is associated with chemical burns **(5, 6)** and it should be treated early in the process with the use of multimodal analgesia, in order to prevent future complications. No matter the absence of major complaints in the 4 week-postoperative evaluation, the possible long-term consequences are still unknown. Despite the prolonged exposure time and the initial presentation with a deep burn, after several surgical debridement, preservation of major tendon and neurovascular structures was assured, which allowed a plain approach for reconstruction of the wound with a skin graft. The case illustrates different challenges associated with evaluation and treatment of patients with deep alkali burns. Also, usage auditing and awareness of regular users appear to be essential.

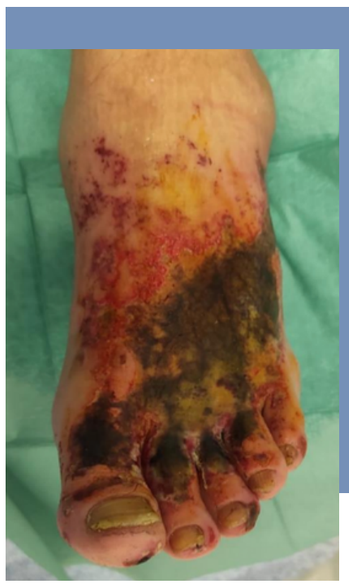
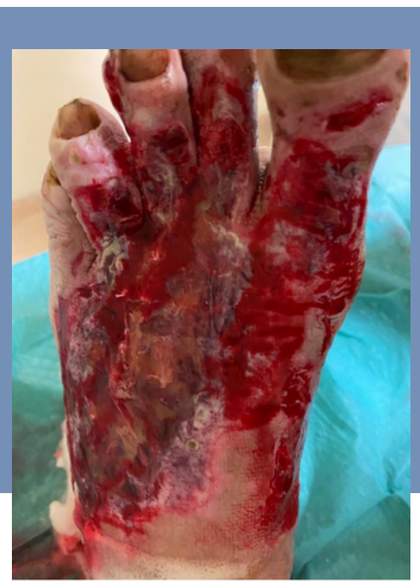


FIG1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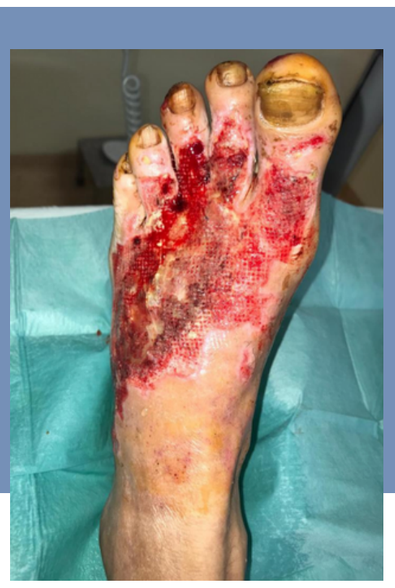
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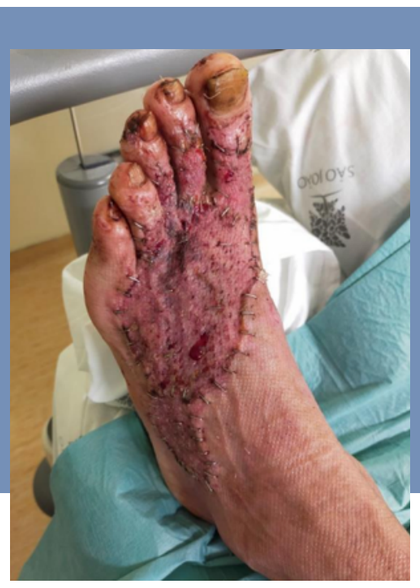
FIG4

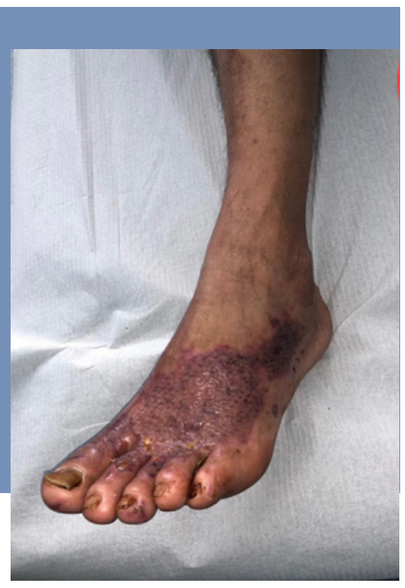
FIG5

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