

Hyperbaric oxygen therapy in a caustic wound

Oxigenoterapia hiperbárica em ferida cáustica

Vitorino Modesto Santos ¹, Mariely Fernanda Silva Helbingen ², Larissa Di Villeneuve
Caetano Pereira de Araújo ², Filipe Emanuel Fonseca Menezes ²

Abstract

A case of deep foot wound by caustic soda treated with adjunctive hyperbaric oxygen therapy and successful outcome is described in a 47-year-old man. The patient first searched for medical attention seven days after the accidental injury. With diagnosis of deep chemical wound caused by the strong alkali, he underwent debridement of cutaneous necrotic tissues, antibiotic therapy, and local wound care. The patient was also managed with 41 sessions of hyperbaric oxygen therapy, which has been considered a useful procedure aiming the improvement of wound healing. The evolution of tissue granulation was remarkable, with general good outcome. Despite of inherent weakness of a single case study, hyperbaric oxygen therapy might be an adjunctive tool for healing of chemical burns. Limitations for utilization include unavailability in small centers and its elevated cost.

Keywords: Caustic soda, chemical burn, hyperbaric oxygen therapy, pedal wound, sodium hydroxide

Resumo

Um caso de ferida profunda por soda cáustica no pé, tratada com oxigenoterapia hiperbárica adjuvante e resultado bem sucedido, é descrito em um homem de 47 anos de idade. O paciente procurou atendimento médico sete dias após a lesão acidental. Com diagnóstico de ferimento químico profundo causado pelo álcali forte, foi submetido ao desbridamento de tecidos cutâneos necróticos, terapia antibiótica, e tratamento local da ferida. O paciente também foi tratado com 41 sessões de oxigenoterapia hiperbárica, que tem sido considerado um procedimento útil visando a melhoria da cicatrização de feridas. A evolução do tecido de granulação foi normal, com boa evolução geral. Apesar da fraqueza inerente a um único estudo de caso, a oxigenoterapia hiperbárica pode ser uma ferramenta auxiliar para a cura de queimaduras químicas. Limitações para a utilização incluem indisponibilidade em pequenos centros e seu custo elevado.

Palavras chaves: Soda cáustica, queimadura química, oxigenoterapia hiperbárica, hidróxido de sódio

1. Professor Adjunto da UCB e Preceptor do Departamento de Medicina Interna do HFA

2. Médicos Residente de Clínica Médica do HFA

E-mail do primeiro autor: vitorinomodesto@gmail.com

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Introduction

Chemical burn wounds by sodium hydroxide (NaOH) are due to contact with this agent in industries (petroleum refining; soaps, rayon, paper, plastic/ cellophane manufacturing; chemical and metal processing; and rubber reclaiming); or domestic environment (v.g.: drain and oven cleaners).^{1,2} The lack of adequate first care of chemical burns by this strong alkali is the major risk factor of poor outcome.¹⁻³ Further occurrence of infection of necrotic tissues usually increases the wound depth. Rigid preventive measures must be followed when handling the caustic chemicals, as wearing protective gloves and boots, and utilizing less-irritating cleaning products.¹

Hyperbaric oxygen therapy (HOT) has been an adjunctive tool to control non-healing wounds, in association with antibiotic therapy and surgical debridement.⁴⁻⁸ HOT involves administration of 10% oxygen by intermittent sessions at 1.4 to 3.0 absolute atmospheres in hyperbaric camera.⁸ The therapeutic mechanisms include enhancement of tissue perfusion, fibroblast proliferation, collagen production and angiogenesis; and reduction of hypoxia and edema, and of inflammatory cytokines.^{7,8} HOT has been a safe modality of treatment for acute or chronic entities, including diabetic foot ulcers, Fournier's gangrene,

necrotizing infection, and thermal burns.^{9,10} The most common side-effects are barotrauma; neurological or pulmonary toxicity; visual disorder; hypoglycemia; dizziness; dyspnea; chest pain; and claustrophobia.⁹

The present case study aims to highlight the consequences of cutaneous corrosion by sodium hydroxide, and highlight the favorable role of adjunctive hyperbaric oxygen therapy.

Case report

An African descent male with 47 years of age presented with tropical spastic paraparesis associated with HTLV infection that was diagnosed in April 2016. The disease began 30 months ago with paresis in the left leg, and one year later, the paresis developed in the right leg with clonus. The symptoms worsened four months ago and required utilization of a wheelchair, and urinary incontinence occurred. As a consequence of the positive serologic test for HTLV 1 and 2 on April 25, he underwent a course of pulse therapy with methylprednisolone (1g daily) from May 3rd to May 7th. On May 12th he was admitted to hospital with pain and swelling in the left lower limb and fever (38.5°C) lasting four days. Accidentally, he had a burning in the left foot with caustic soda a week before admission. Physical examination showed accentuated edema up to mid-calf, without signs of venous

thrombosis, and an area of fluid collection at the instep, Osteomyelitis was discarded by radiographic studies, including magnetic resonance. Laboratory tests on May 11th showed – red cells: 4.65 million /mm³; hemoglobin: 12.9 g/dL; leukocytes: 28.100 /mm³; platelets: 193,000 /mm³; erythrocyte sedimentation rate: 140 mm/1st hour; glucose: 67 mg/dL; urea: 29.8 mg/dL; creatinine: 0.8 mg/dL; calcium: 1.15 mmol/L; sodium: 138 mmol/L; potassium: 4.7 mmol/L; triglycerides: 214 mg/dL; total cholesterol: 152 mg/dL; HDL: 30 mg/dL; ALT: 63 IU/L; AST: 40 IU/L; C-reactive protein: 25.8 mg/dL, and procalcitonin: 1.66 ng/mL. Surgical debridement of the lesion was performed on May 17th (Figure 1A and B), followed by treatment with hyperbaric chamber, and bandages with petroleum jelly. Additionally, vancomycin was initially used and then replaced by ciprofloxacin after culture of skin fragment revealed a sensitive *Proteus sp.* (MIC < 1). Moreover, 41 sessions of HOT were done from May 20th to July 20th, until hypergranulation was observed on the ulcer (Figure 1 C to F). Blood tests on August 6th showed – red cells: 5.51 million /mm³; hemoglobin: 13.7 g/dL; leukocytes: 6,253 /mm³; platelets: 246.000 /mm³; erythrocyte sedimentation rate: 6 mm/1st hour; urea: 43 mg/dL; creatinine: 0.75 mg/dL calcium: 1.31 mmol/L; sodium: 140 mmol/L; potassium: 4.8

mmol/L, and C-reactive protein: 0.3 mg/dL. Currently, he is undergoing dressing daily topics in the residual lesion of the instep.

Discussion

Isolated feet burns affect adults and children with variable frequency and in the Welsh Centre for Burns (2007) the distribution in adults was scald (35%) and chemical (32%) burns; with prolonged hospital stay and complication rate of 18%.¹¹ The male to female ratio described between adults with burn wounds was 3.5:1; and complications include hypertrophic scars, graft loss, delayed healing and infections.¹¹ Shoen et al. (1996) retrospectively reviewed 33 isolated burns of the foot at the University of Chicago Burn Center and reported the following distribution of causes of the burns: scald (70%), grease (9%), hot solid (9%), flame (6%), and other (6%).¹²

Accidentally, the Brazilian male patient herein described had an instep chemical burn wound by sodium hydroxide (also called lye or caustic soda).^{1,2} Local bleeding, predominance of bloody scabs and deep ulcers were the consequences of cutaneous corrosion by this chemical agent.^{2,3} The dissolution of caustic soda in water gives origin to a highly exothermic reaction; and the exposition of cutaneous tissues to elevated concentrations of hydroxide ions gives origin to saponification of

fat and formation of soluble alkaline proteinates.² These strong alkali actions often promote deep liquefactive tissue necrosis that frequently requires extensive surgical debridement of the affected skin structures; moreover, either grafting or long standing conservative management are further necessary until obtaining the complete epithelialization of the wound.¹ Appropriate first local care of this chemical burn is the mainstay of successful outcome,¹ and the occurrence of infection usually favors the increase in wound depth. Graft procedure has been a major primary

treatment option; nevertheless, repeated lysis of skin graft may occur.² In caustic chemical lesions, the tissue destruction persists after the initial exposure; consequently, a late healing of defect site can evolve without esthetic appearance.² Therefore, some additional resources could yield fast wound epithelialization; as the example of HOT,⁴⁻⁶ which might contribute to improve the tissue granulation. In the present case, 41 sessions were done in concomitance with local bandages containing petroleum jelly.



Figure 1. **A** and **B**: Initial features of the instep wound before and just after the deep necrotic tissue debridement; there is evident exposure of muscles and tendons; and **C** to **F**: comparative images of the development of tissue hypergranulation, in addition to contraction of the wound area following the hyperbaric oxygen therapy.

Conclusions

In spite of inherent weakness of single case study, the authors believe that HOT could constitute efficacious adjunctive tool for obtaining increased rate of tissue granulation and healing of the chemical burns. Limitations for utilization include unavailability in small centers and elevated costs.^{8,10}

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