Case Report

J Trauma Inj 2022;35(3):209-214
ttps://doi.org/10.20408/jti.2021.0048

Journal of Trauma and Injury JTI

pISSN 2799-4317 • eISSN 2287-1683

Hyperbaric oxygen therapy for the treatment of a crush injury of the hand: a case report

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We describe a case of hyperbaric oxygen therapy (HBOt) as an adjunct to treatment of a crush injury to the hand. A 34-year-old male paramedic was involved in a motor vehicle accident and admitted for diagnosis and surgical treatment. He sustained a crush injury to his right hand and presented with significant muscle damage, including multiple fractures and dislocations, an avulsion injury of the flexor tendons, and amputation of the distal phalanx of the little finger. He underwent reconstructive surgery and received HBOt over the following days. In the following 2 months, he lost the distal and middle phalanges of the little finger and recovered hand function. Posttraumatic compartment syndrome responds well to HBOt, which reduces edema and contributes to angiogenesis, as well as promoting the cascade of healing events. High-energy trauma causes massive cell destruction, and the blood supply is usually not sufficient to meet the oxygen demands of viable tissues. Hyperbaric oxygenation by diffusion through interstitial and cellular fluids increases tissue oxygenation to levels sufficient for the host's responses to injury to work and helps control the delayed inflammatory reaction. HBOt used as an adjunct to surgical treatment resulted in early healing and rehabilitation, accelerating functional recovery. The results suggest that adjunctive HBOt can be beneficial for the treatment of crush injuries of the hand, resulting in better functional outcomes and helping to avoid unnecessary amputations.

Keywords: Hyperbaric oxygenation; Cruush injuries; Surgical flaps; Compartment syndrome; Case reports

Received: July 6, 2021
Accepted: September 1, 2021

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INTRODUCTION

Crush injuries of the hand have become a relatively common phenomenon due to modern lifestyle habits and are a common cause of amputation or historically poor outcomes in non-specialized services [1]. Usually caused by a high-energy mechanism, a compressive force crushes and leads to a rise in tissue pressure and damage of varying severity to multiple tissue types [1]. Crush injuries of the hand pose a challenge to the hand surgeon, whether it is a minor fingertip injury sustained by the hand being squashed or a high-pressure compression injury involving the palm or wrist [2,3].

A crush injury of the hand is defined as compression of the distal extremities of the upper limbs causing muscular, neurological, vascular, and bone disturbance and producing damage to tissues. The degree of damage is proportional to the amount of force applied and the duration of compression.

A wide zone of injury results from a delayed inflammatory reaction involving the bordering zone, which may initially belie the severity of the damage. Profuse edema and inflammation increase the volume within the fascial compartments, decreasing perfusion to soft tissues and nerves and progressively reducing tissue viability [1].

Debridement is the cornerstone of surgical treatment of open traumatic injuries. All devitalized tissue and foreign bodies should be removed, and all viable, well-vascularized tissue should be preserved. However, it is not always possible to assess tissue viability intraoperatively and sometimes, depending on the surgeon's experience, an inappropriate decision may be made.

Notwithstanding this observation, experience and knowledge of the beneficial effects of hyperbaric oxygen therapy (HBOt) in the rescue of hypoxic tissue can contribute substantially not only to saving the limb, but also to earlier healing. Thus, a well-trained and experienced team of surgeons, as well as physicians with knowledge of hyperbaric medicine, can provide a successful alternative for salvaging tissues and body segments when compared to services that do not offer hyperbaric facilities.

CASE REPORT

A 34-year-old male paramedic was involved in a motor vehicle accident and admitted for diagnosis and surgical treatment. He sustained a crush injury to his right hand with significant muscle damage, including multiple fractures and dislocations, an avulsion injury of the flexor tendons, and amputation of the distal phalanx of the little finger (Fig. 1).

Emergency reconstructive surgery was performed. The distal phalanx of the fifth finger did not satisfy the minimum conditions for replantation, and the presence of debris in the wound and an unviable aspect of the thenar musculature were also noted. The wound was then abundantly washed with saline, and debridement was performed to clean the wound from unviable tissue. The interphalangeal joint dislocations of the middle and little fingers were reduced and fixed using Kirschner wires under image intensifier guidance. Tenorrhaphy of the distal flexor digitorum profundus stumps and proximal flexor digitorum superficialis (FDS) stumps of the middle and ring fingers was performed, followed by transosseous suturing of the distal FDS stump to the middle phalanx of the avulsed little finger. Grafting of the distal FDS stump of the little finger to the proximal FDS stump of the ring finger was achieved with tendon transfer. The wound was closed using a plane suture as satisfactorily as technically possible (Fig. 2).

Volume replacement therapy, pain medication, broad-spectrum antibiotics, and nutritional support were initiated as per the service's routine. The wound showed progressive tissue damage, manifesting mainly as a change in the color of the skin associated with progressive edema (Fig. 3).

Despite the delayed start of HBOt, the color in some of the ischemic and hypoxic superficial tissues improved within the first days of adjunct treatment (day 6), after only three HBOt sessions.

Fig. 1. Right hand on day 1 in the emergency box.

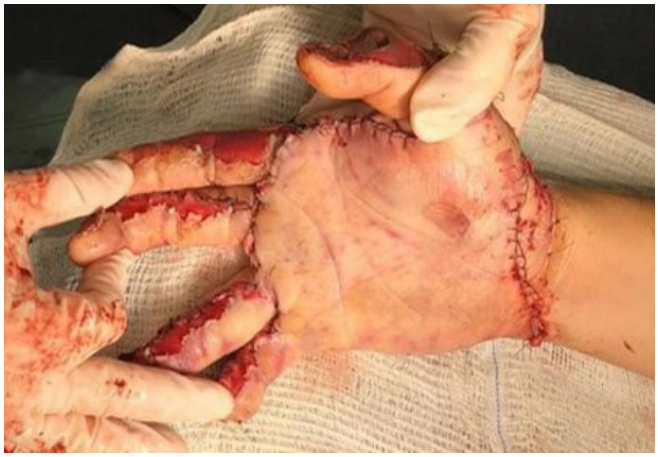

Fig. 2. Right hand on day 1, on the immediate postoperative period.

https://doi.org/10.20408/jti.2021.0048

Fig. 3. Right hand on day 3 after reconstruction.

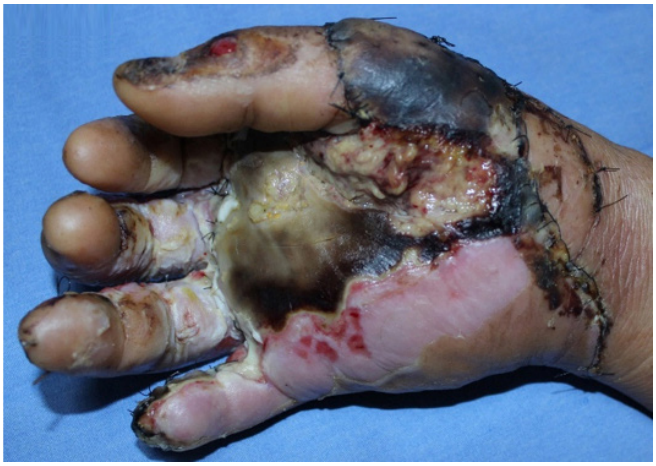

Fig. 5. Right hand on day 20 after 17 hyperbaric oxygen therapy sessions.

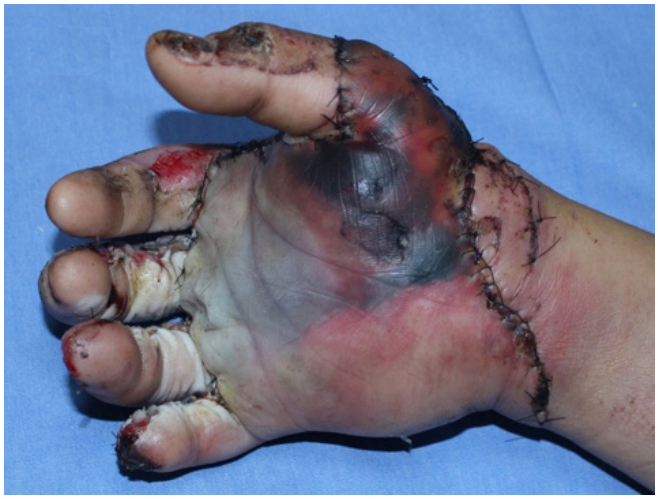

Fig. 4. Right hand on day 6 after three hyperbaric oxygen therapy sessions.

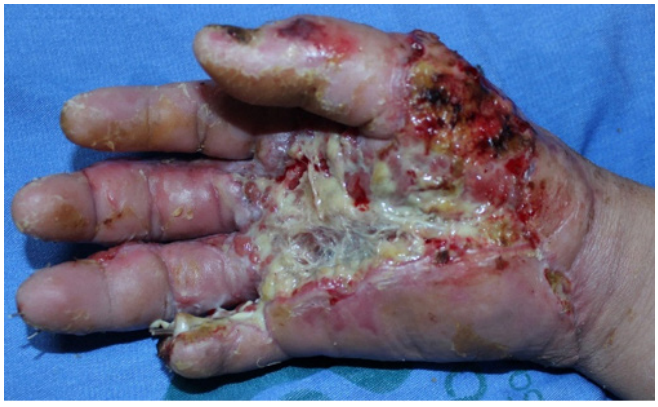

Fig. 6. Right hand on day 32 after 29 hyperbaric oxygen therapy sessions.

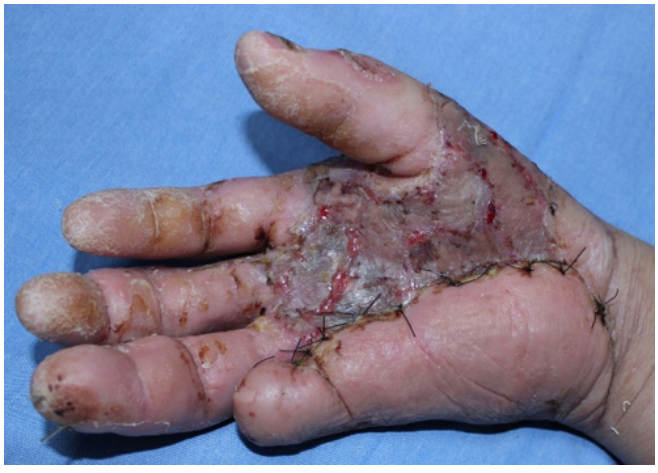

Fig. 7. Right hand on day 40 after 37 hyperbaric oxygen therapy sessions.

In addition, the posttraumatic edema also improved markedly with HBOt (Fig. 4). The lesion progressed to soft tissue necrosis in the thenar region with superficial spreading towards the base of the fingers (Fig. 5).

Over the ensuing days, occlusive dressings were changed daily and unviable tissue was removed, eventually revealing wet necrosis and infection in the middle phalanx of the little finger (Fig. 6). The patient was taken to the operating room for amputation of the distal phalanx of the little finger, and a thenar skin flap was taken from the right forearm (Fig. 7).

Concurrently, an early rehabilitation program was initiated with an excellent functional outcome and full restoration of hand function at the end of treatment, despite the loss of the distal and

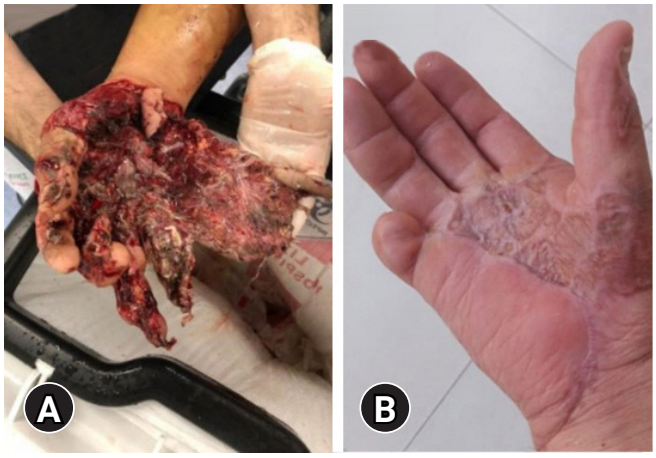

Fig. 8. Right hand (A) on the day of crush injury and (B) after recovery.

middle phalanges of the little finger (Fig. 8).

Informed consent for publication of the research details and clinical images was obtained from the patient.

DISCUSSION

Skeletal muscle-compartment syndrome (SMCS) is a consequence of trauma, but in this situation the affected tissues are muscles and nerves. Edema and/or bleeding within the confines of the fascial envelope can increase the pressure within the skeletal muscle compartment. When the tissue fluid pressure within the compartment exceeds the capillary perfusion pressure to these tissues, they are rendered ischemic and manifest the signs and symptoms of SMCS. No means to arrest the progression of SMCS, especially in its stages before fasciotomy is required, exist other than HBOt [4].

Unfortunately, HBOt is neglected as an adjunct for managing crush injuries and SMCS. Arguments exist for its use based on evidence-based information and how HBOt mitigates the pathology of these conditions [4].

The literature regarding the use of HBOt in patients with SMCS is limited, mainly because this condition does not occur as a standalone event and is most frequently associated with hypovolemic shock, crush injuries, and polytrauma. Despite the paucity of research, the beneficial effects of HBOt as an adjunctive therapy have been demonstrated in both experimental and human studies, even though one study with a low level of evidence has reported that tissue necrosis progressed with HBOt. Bouachour et al. [5] conducted a randomized clinical trial with a high level of evidence and reported complete healing in 94% of patients receiving HBOt versus 34% in the control group ($P<0.05$). These findings are consistent with several reported clinical cases.

The underlying pathophysiology of crush injuries and SMCS is trauma with tissue hypoxia, which may lead to the continued evolution of the injury to an irreversible state or a self-perpetuating progression of edema, forming a vicious circle. The consequences of trauma include visible tissue damage, injury at the cellular level, and biochemical alterations. Immediate necrosis occurs in high-energy trauma cases and the only options in these circumstances are debridement or amputation.

Trauma to the vasculature at the microcirculation level leads to transudation of fluid with increasing edema, interstitial bleeding, slowed blood flow, venous stasis, clot formation, and vascular obstruction. The consequences are ischemia and hypoxia to the tissues perfused by the damaged vasculature. When this occurs, cells are no longer able to maintain their metabolic functions such as retaining intracellular water, which contributes to edema and third-spacing of fluid. If the edema occurs in a closed space, the increased pressure will collapse the microcirculation, eliminate oxygen transfer across the capillary endothelium, and contribute to the hypoxic tissue insult [4]. Considering that oxygen is required for all metabolic functions, the low oxygen tension from trauma and its consequences thwart the cascade of events involving wound healing, angiogenesis, bacterial killing by neutrophils, and the action of fibroblasts.

Events at the biochemical level, the ultimate determinants of outcomes, are manifested in two ways. First, oxygen is required for all cellular metabolic functions. If oxygen tension is insufficient, cell signaling factors, wound healing and angiogenesis responses as elaborated through fibroblasts, and bacterial killing by neutrophils are thwarted. A partial pressure of oxygen in the tissue fluids greater than 30 mmHg is required for these responses to occur.

The second biochemical event is that of ischemia-reperfusion injury (IRI) [6]. Once perfusion is temporarily interrupted, which occurs in varying degrees with crush injuries and compartment syndrome, the endothelium becomes sensitized to the hypoxic insult, resulting in the activation of adhesion molecules, which in turn leads to the attachment of neutrophils to the endothelium and the release of reactive oxygen species (ROS). The consequence is a cascade of biochemical events, whereby these ROS damage tissue further and cause severe vasoconstriction, defining the IRI and the no-reflow phenomenon associated with it.

The immediate justification for using HBOt in crush injuries and compartment syndromes is twofold. First, HBOt supplements oxygen availability to hypoxic tissues during the early

https://doi.org/10.20408/jti.2021.0048

postinjury period, when perfusion is most likely to be inadequate and the oxygen requirement is greater. Second, HBOt increases tissue oxygen tension to sufficient levels for the host responses to function. Hyperbaric oxygen exposure at a pressure of two atmospheres absolute (202.65 kPa) increases the oxygen content (the combination of hemoglobin and oxygen) by 125%. The oxygen tension in plasma and tissue fluids is thus increased 10-fold—that is, by 1,000% [7,8]. Increased tissue oxygen tension results in a threefold driving force (mass effect), which compensates for the hypoxia resulting from the increased oxygen diffusion distance from the capillary to the cell through the edema fluids.

Edema reduction is a secondary effect of tissue hyperoxygenation. Hyperbaric oxygen induces precapillary vasoconstriction, which reduces blood flow by 20% [9,10]. Because inflow is decreased while outflow is maintained, the net effect is edema reduction of roughly 20% [10–12]. Edema reduction occurs because of decreased filtration of fluid from the capillary to the extracellular space as a consequence of vasoconstriction, resulting in decreased perfusion pressure in the capillary bed, while resorption of fluid at the capillary level is maintained by the oncotic pressure inside the capillary. Hyperoxygenation of the plasma maintains oxygen delivery to tissues in the presence of HBOt-induced vasoconstriction [9,13]. Another consequence of decreasing the interstitial fluid pressure through edema reduction is improved blood flow through the microcirculation. The reason for this is that, once the interstitial fluid pressure is reduced below the capillary perfusion pressure, the collapsed microcirculation can again open up and allow perfusion to resume. By reducing edema while supplementing tissue oxygenation, HBOt interrupts the self-perpetuating edema/ischemia vicious circle to prevent progression of the injury; therefore, SMCS and crush injuries are particularly amenable to HBOt.

Mitigation of IRI is another effect of HBOt for crush injuries and SMCS [13]. HBO interrupts the interactions between oxygen free radicals and cell membrane lipids, perturbing lipid peroxidation of the cell membrane and inhibiting the sequestration of neutrophils on postcapillary venules [14,15]. The latter effect occurs because HBO interferes with the adherence of neutrophils elaborated through the beta-2 integrin (cluster designation-11) on the sensitized capillary endothelium. The result is interruption of the interaction of superoxide anion with nitric oxide, which produces the highly reactive peroxynitrite radical [15]. Another benefit of HBOt for IRI is the generation, in an oxygenated environment, of enzymes such as superoxide dismutase, catalase, peroxidase, and glutathione reductase that detoxify ROS, improving patients' prognosis.

Fig. 9. Right hand 7 months after treatment.

The correlation of the pathophysiology of crush injuries with the physiological effects of adjunctive HBOt and the course of events in this case indicates that HBOt may be beneficial for arresting progressive tissue damage, reducing edema, and improving oxygenation and tissue perfusion. However, more well-designed studies with larger sample sizes are needed to establish high-level evidence to support its use.

HBOt used as an adjunct to surgical treatment resulted in early healing and rehabilitation, accelerating functional recovery. The results suggest that adjunctive HBOt can be beneficial in the treatment of crush injuries of the hand, resulting in better functional outcomes and helping to avoid unnecessary amputations (Fig. 9).

NOTES

Ethical statements

Informed consent for publication of the research details and clinical images was obtained from the patient.

Conflicts of interest

The authors have no conflicts of interest to declare.

Acknowledgments

The authors thank the healthcare teams from the emergency mobile care service of Cuiabá, Cuiabá Municipal Hospital, and Santa Rosa Hyperbaric Clinic, including physicians, nurses, physical therapists, and technicians who provided all the assistance required for the rescue and treatment of the patient.

Author contributions

Conceptualization: all authors; Data curation: all authors; Formal

analysis: PHN, ZB, AP; Methodology: all authors; Project administration: PHN; Visualization: PHN; Writing–original draft: all authors; Writing–review & editing: all authors.
All authors read and approved the final manuscript.

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https://doi.org/10.20408/jti.2021.0048