Case report

Severe lower limb crush injury and the role of hyperbaric oxygen treatment: a case report

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Abstract

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Open fractures with severe soft-tissue injury and critical local ischaemia of the lower limbs are usually difficult to treat and require a multidisciplinary approach. A 33-year-old Caucasian female with crush injury of the right foot (Gustilo IIIC) was admitted to hospital after a car accident. Despite surgical interventions, a persistent state of hypoxia was present because of the severe vessel injury, and amputation was suggested. Seventy-two hours after admission she was referred to the hyperbaric medicine unit for hyperbaric oxygen treatment (HBOT) to define the limits of viable tissues prior to amputation. After six sessions, clinical improvement was so obvious that the decision to amputate was rejected and she underwent a total of 32 HBOT in addition to frequent debridement and administration of antibiotics. After the HBOT course, she underwent successful surgical reconstruction with a vascularised cutaneous flap. Full healing was achieved. Given the fact that hyperbaric oxygen mechanisms of action target the pathophysiology of crush injuries it should be considered not only for the definition of viable tissue limits but also to enhance viability, even in the most serious situations. HBOT may prove a valuable supplement in the therapeutic armamentarium of these patients.

Key words

Hyperbaric oxygen therapy, trauma and stress, wounds, case reports

Introduction

Open fractures with severe soft-tissue injury and critical local ischaemia of the lower limbs are often difficult to treat and require a multidisciplinary approach. When crush injuries are severe, the rate of complications, including infection and non-healing of fractures and tissues, range up to 50 to 60% with a high amputation rate.[1,2] In these cases, adjuvant treatment with hyperbaric oxygen (HBOT) may be of crucial importance, based on sound pathophysiological rationale and growing experimental and clinical evidence. We present a case of lower limb crush injury that highlights many of these issues.

Case report

A 33-year-old Caucasian female was admitted to the emergency department with a crush injury of the right foot received in a car accident. She had open fractures with dislocation of the tarsal and all the metatarsal bones (rupture of the ligaments – Lisfranc) and a severe crush injury of the dorsal surface of the right foot. (Gustilo IIIC; Figure 1) The dorsalis pedis artery had been dissected and angiography revealed blockage of the posterior tibial artery with little collateral circulation. Emergency surgery with fixation and stabilization using Kirschner wires was performed, but there was no rheological improvement, as shown in repeat angiography (Figure 2). Transcutaneous oximetry (TCOM) of the right foot showed an intense hypoxic state (almost zero).

Due to the progressive deterioration of the injured foot and the persistent hypoxia, amputation was proposed. Therefore, she was referred to the hyperbaric medicine unit 72 hours after admission in order to improve the local metabolic processes and define the limits of necrotic versus viable tissues. HBOT was administered twice daily for the next three days at a pressure of 243 kPa. Each HBO session consisted of two 40-minute periods of 100% oxygen via an oronasal mask, with a 5-minute air break.

Despite the initial poor prognosis, after these six HBOT, clinical improvement was obvious. There was also a

Figure 1

Photo of the injured foot on admission to the emergency department; despite the poorly-focused image the severe extent of the injury can be clearly seen

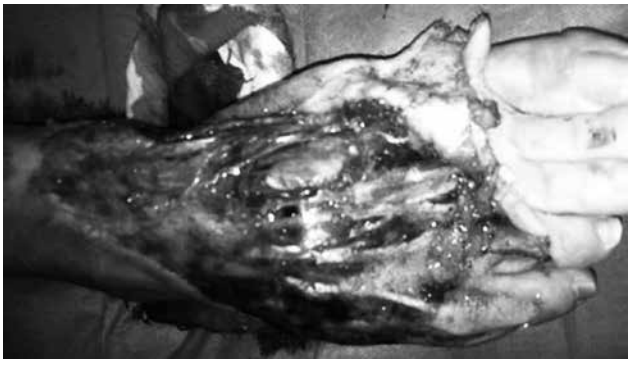


Figure 2
Angiography performed after emergency surgical interventions; the dorsalis pedis artery is dissected (left arrow) and there is blockage of the posterior tibial artery (right arrow) and little collateral circulation can be seen

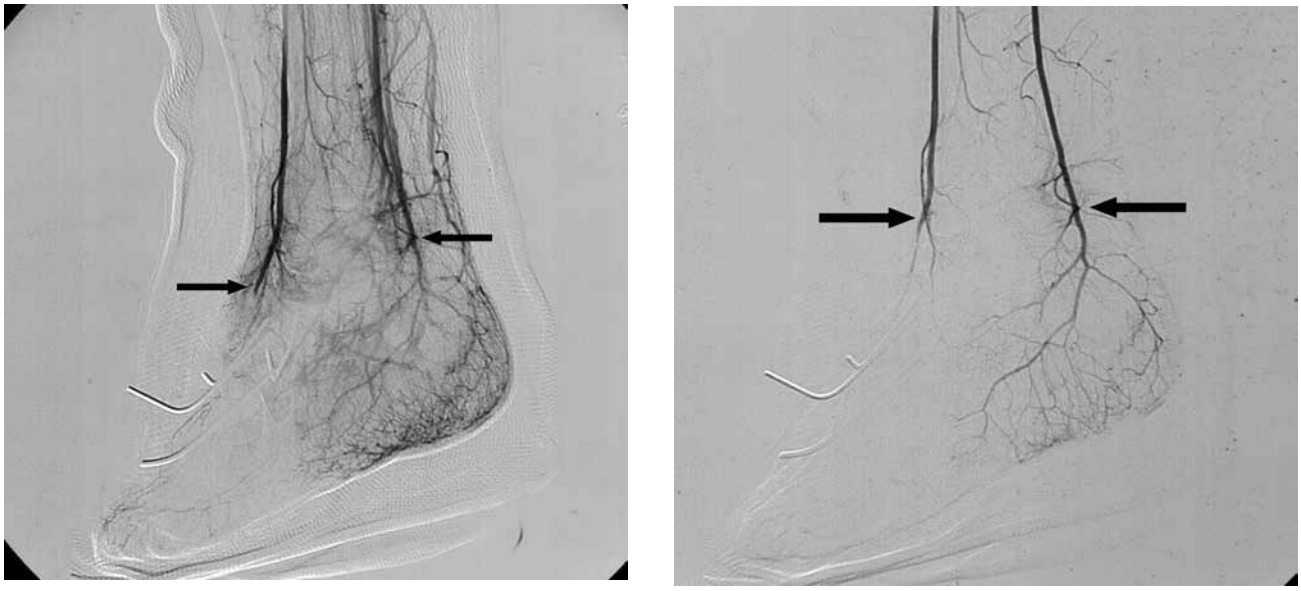
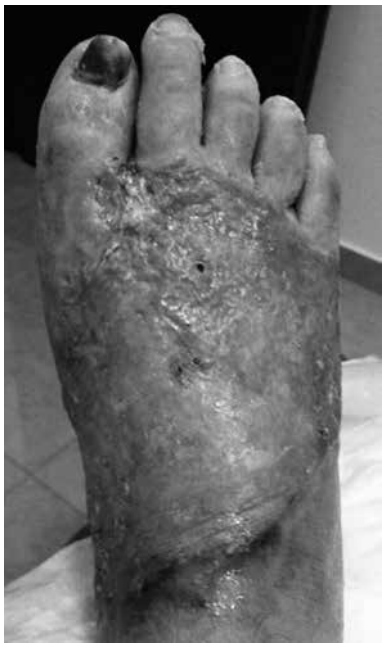


Figure 3
The foot at 2-months follow up after surgical reconstruction with a vascularised cutaneous flap



pronounced rise in the TCOM values measured adjacent to the injury to 300 mmHg on oxygen at 243 kPa. Oedema reduction, the decrease in the quantity and improved quality of exudates and the reduction in the inflammatory response were so apparent that the decision to amputate was postponed and an extended HBOT course proposed instead. The patient underwent a total of 32 HBOT, 26 on a daily basis, without complications, plus frequent debridement to remove necrotic tissue and exudates and to promote tissue granulation. Antibiotic treatment was modified to colistin as *Pseudomonas aeruginosa* was isolated from the wound.

A month later, the patient was submitted to removal of Kirschner wires. At six weeks post injury, a week after the end of the HBOT course, she underwent successful surgical restoration of the foot with a vascular skin flap. (Figure 3). At three months follow up, healing remained complete and with intensive physiotherapy, she had started walking again without the aid of crutches. At seven months follow up, healing remains intact, the patient walks without crutches and has returned to work (as a teacher) and everyday activities.

Discussion

Crush injury is characterized by a vicious cycle of ischaemia, hypoxia, oedema, disturbed microcirculation and secondary ischaemia in the area bordering the primary trauma.[2] HBOT ameliorates the effects of acute traumatic ischaemia by interrupting this cycle.[1] The therapeutic effects of HBOT are well recognized through experimental and controlled clinical trials in different kinds of ulcers.[1–4] However, clinical experience for its potential efficacy in crush injuries is sparse and there are few cases in the literature where scheduled amputations have been prevented. Current evidence suggests it should be started as soon as possible, preferably in the immediate postoperative period.[4]

In this case, the intensive hypoxia of the injured area despite the surgical interventions was reversed as soon

as HBOT commenced. The progressive increase in the diffusion of oxygen in the injured tissues overcame the "*circulus vitiosus*" of oedema and hypoxia and led to such an apparent clinical improvement that amputation was rejected. The multidisciplinary approach with HBOT, antibiotics, debridements and surgical reconstruction with a vascularised cutaneous flap resulted in the salvage of the foot with good functional recovery.

The oxygen gradient and thus diffusion in plasma and tissues is markedly raised during HBOT and can overcome a decreased but not obliterated perfusion. Injured but viable cells in the penumbra have increased oxygen needs. At a time when oxygen delivery is decreased by impairment of the microcirculation, survival of the cells is directly dependent on oxygen tension.[2] Secondary mechanisms via which HBO may help salvage severely ischaemic tissue include perturbation of ischaemia-reperfusion injury, rheological improvement, elimination of anaerobic bacteria, enhancement of antibiotic action and of the intracellular killing mechanisms of polymorphonuclear leukocytes and an anti-inflammatory effect by inhibition of specific cytokines.[1,5,6]

A distinct feature of hyperoxygenation, particularly with regards to crush injuries, is the pronounced reduction in oedema provoked by vasoconstriction and reduction of blood flow; the latter more than compensated by hyperoxia.[2] Furthermore, HBOT has been demonstrated to promote advancing angiogenesis in the margins of a lesion and this may be relevant in cases such as the one presented here.[5–8] Although each case is unique, we believe that even when further vascular surgery has been excluded, HBOT should be considered not only to define viable tissue limits but also to enhance viability even in the most serious situations. HBOT may prove a valuable supplement in the treatment algorithm of crush injuries.

TCOM is considered to be the gold standard in the evaluation of the response of the tissues to HBOT, and is an important tool both in the selection of patients suitable for treatment and in the prediction of the response to treatment.[3]

Conclusion

Given the fact that the mechanisms of action of HBOT target the pathophysiology of crush injuries, further evaluation by means of high-quality, randomized controlled trials is needed to determine the role of HBOT as an adjunctive treatment in the therapeutic armamentarium of these patients.

References

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