CASE REPORT

A leukocytoclastic vasculitis successfully treated with hyperbaric oxygen

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ABSTRACT

We are presenting a case of a 27-year old female suffering from non-healing, recurrent, painful ulcers on her left lower leg, affected by minor trauma, and resulting from underlying cutaneous leukocytoclastic allergic vasculitis. After two years of conventional systemic therapy and aggressive topical wound care she was presented to the Division of Hyperbaric Oxygen Therapy. A hyperbaric oxygenation was applied as mono-therapy. After 40 sessions a complete regression of all skin defects was achieved. Several previous good experiences and this successful treatment encourage us to advocate for the hyperbaric oxygen as a mono or adjuvant therapy for such a hard-to-treat problem.

Key words: hyperbaric oxygenation; leukocytoclastic vasculitis; cryoglobulinemia, leg ulcers; limb ischemia

Leukocytoclastic vasculitis (LCV) is the most frequent form of cutaneous vasculitis. There is always an exogenous (infections, drugs, chemicals, foodstuff allergens) or an endogenous (malignant neoplasm, connective tissue disease) trigger (1).

A pathogenesis of LCV is immune complex form related. Laboratory examination should reveal a mixed cryoglobulinemia type III referring to the presence of polyclonal immunoglobulins in the serum. (2). Deposits within the small- and medium-sized vessel walls may lead to the complement activation, intravascular and perivascu-

lar fibrin deposits, subsequent tissue hypoxia and necrosis (1). Erythematous maculae and palpable purpura occurring on lower legs are usually observed. The lesions, ranging from pinpoint to few centimetres, may progress from papulonodular, vesicular, bullous, pustular, to ulcerative (1). The progression of LCV leads to ischemic lower limb, sometimes demanding a surgical approach, including debridement, removal of necrosis, sympatectomy and amputation at the end stage.

We are reporting on a patient who had suffered from persistent non-healing wounds for 2
years, occurring after a minimal traumatic damage to her ankle (Figure 1). She recovered completely after HBO mono therapy was applied. This article emphasizes the role of HBO therapy for LCV.

CASE REPORT

A 27-year-old woman was presented to the Division for hyperbaric oxygen therapy (HBOT) with a history of recurrent painful ulcers on her left lower leg. Two years before in terms of a medical aspect she had suffered a minor trauma of her left ankle in the form of small laceration, which later turned into an inflamed area with irregular 1.5-2 cm ulcer, surrounded with palpable purpura. Histopathological findings referred to LCV. She reported no history of allergic reactions. A direct immunofluorescence demonstrated cryoglobulinemia type III, not related to viral hepatitis infection type C or type B. At a late stage of disease, there were signs of secondary sideropenic anemia (serum Fe 4 μmol L⁻¹).

Based on isolated microorganisms (Staphylococcus aureus, Pseudomonas aeruginosa, Enterobacter sp.) she was given antibiotics: amoxicillin with clavulonic acid, cloxacillin, trimethoprim-sulphomethoxazole, ciprofloxacin, clindamycin, gentamycin and finally sulfone, used as leprostatic. Corticosteroids, non-steroid analgesics, acetylsalicylic acid, and adjuvant therapy consisting of ranitidine, diosmin, hesperidin and iron derivatives were used too. At the end stage of treatment azathioprin, and warfarin were given, without any results. Local wound care included corticosteroid and antibiotic ointments, hydrocolloid dressing, hydrogen and normal saline compresses.

The ulcerations and pain deteriorated over time despite the conventional therapy. The next step planed was surgical sympathectomy, and amputation of affected limb was considered in case of persistent pain. Social and psychological disorders superposed to her of non-healing wound problem. She walked with crutches, could not sleep, could not work, became depressive, frustrated, anxious and had suicidal ideas.

Upon admission to the HBOT unit, she had several serpiginous ulcers with necrotic purplish edge and sloughy base. The surrounding skin was covered with polymorphic lesions like erythematous and purpuric papules, hyperpigmentation, brown crusts, and white atrophic scars (Figure 1). The ankle and foot were edematous, whereas pain was grade 8 according to visual analogue scale.

These skin defects were further complicated by an increased drainage from the wound and bacterial infection, which were treated by gentamycin 160 mg IM day⁻¹ and methylprednisolone 8 mg day⁻¹.

Based on the clinical and histological diagnosis of LCV, a decision was made to stop the entire ongoing therapy. The HBO as a monotherapy at

Figure 1. Before hyperbaric oxygen therapy: heterogeneous lesions from “palpable purpura”, as clinical hallmark of early LCV, to the painful, swelling, ischemic limb, with deep, suppurating, stinking ulcers (I. Takač, 2005., with patient’s permission).

Figure 2. After 40 hyperbaric oxygen therapy sessions: white scars and hyperpigmentation persisted over the intact surface of affected lower leg (I. Takač, 2005., with patient’s permission).
the regime of 2.2 bar (223 kPa), 60 minutes day⁻¹ was applied. The wound was dressed with moist compresses of normal saline. After only three therapy sessions, a surprisingly early regression of ischemic pain was observed. Subsequent HBOTs resulted in the rapid healing of migrating ulcers and regression of edema.

After 40 HBOTs a complete regression of lesions and good leg function were realized. Cryoglobulins were no longer detectable in the patient’s plasma (Figure 2). One year after the treatment she had no recidive changes.

Local hypoxia and infection are the primary underlying substrate in non-healing wounds (compromised diabetic foot, ischemic ulcers with underlying cutaneous vasculitis, venous ulceration, and radiation necrosis). (3). In the LCV a differentiation between disease-related and treatment-induced leg ulcers is always difficult and sometimes may not be possible. Cessation of the drug typically leads to wound healing in this hypersensitivity disorder.

HBOT is defined as a mode of medical treatment in which the patient breathes 100% oxygen intermittently at a pressure greater than one atmosphere (at least 1.4 or 3 atmospheres) (3). It increased partial pressure of oxygen in all the tissues of the body, mostly by increasing a dissolved fraction of oxygen in blood (4). Under physiologic conditions the amount of oxygen physically dissolved in plasma is 0.3 ml dl⁻¹, and at pressure of 2.2 bar reaches 4.8 ml dl⁻¹ of blood. An increase in the oxygen diffusion gradient between blood and tissues enables satisfactory oxygenation in the low perfused tissue (5). The beneficial effects are: wound healing; increased neutrophil bactericidal capacity; direct toxic effect against anaerobic bacteria; arteriolar vasoconstriction with subsequent edema reduction, collagen synthesis, and neovascularization in ischemic tissues (4). Complications of HBOT are related to pressure changes and middle ear barotraumas, sinus pain, pulmonary barotraumas, oxygen seizures, decompression sickness and claustrophobia.

HBOT is currently accepted as the primary therapy in patients with carbon monoxide poisoning, decompression sickness, and arterial gas embolism. HBOT is also indicated in radiation-induced tissue injury, thermal burns, and acute traumatic ischemia, compromised grafts and possibly ischemic-reperfusion injury (3).

Many recent case reports indicate that the HBOT may be a useful technique in the management of wound problems precipitated by hypoxia and/or infection. Opportunity to apply HBOT as mono therapy resulted in the excellent overall response in our patient: the cryoglobulins and clinical symptoms disappeared. The presented case should encourage clinicians to use HBO as mono therapy in the treatment of non-healing painful ulcers resulting from underlying cutaneous LCV. High-quality randomized controlled prospective trials are needed to evaluate the short- and long-term risks and benefits of HBOT in this form of vasculitis.

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Written consent was obtained from the patient for publication of the Figure 1 and Figure 2.

REFERENCES