Case report: Hyperbaric oxygen in the treatment of puff adder *(Bitis arietans)* bite

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ABSTRACT

Introduction: The puff adder *(Bitis arietans)* is a venomous viper mainly found in sub-Saharan Africa. Due to its common occurrence and potent venom, it is considered to be the most dangerous snake in Africa, responsible for most snakebite fatalities there. Puff adder bites outside Africa are rare and involve captive vipers. We present the unusual case of puff adder envenomation in an Austrian man.

Case Report: A 26-year-old Austrian man was bitten by a puff adder that he kept illegally in his home. On admission he showed signs of local and systemic toxicity. He was stabilized with antivenom, intravenous fluids, catecholamines and packed platelets. Hyperbaric oxygenation was begun due to incipient compartment syndrome on the second day and continued until the eleventh day, when the patient had recovered completely and could be discharged.

Discussion: The venom of *Bitis arietans* can cause serious systemic and local complications. Our patient suffered from both. Systemic signs included hemodynamic as well as hemostaseologic impairment. Local effects included swelling and incipient compartment syndrome. Systemic and local treatment, including hyperbaric oxygenation, effected a full recovery. We suggest that, whenever feasible, hyperbaric oxygenation should be considered as adjunct treatment in snake bites to avert adverse outcomes. ❖

INTRODUCTION

The puff adder *(Bitis arietans)* is a venomous snake belonging to the family of *Viperidae* and found in the grasslands of sub-Saharan Africa and the Arabian peninsula. Due to its widespread occurrence it is responsible for a large number of bites in both humans and livestock in Africa and poses a public health concern there [1,2].

Puff adder bites in America and Europe occur with snakes held in captivity, whether legally or illegally, and are a rare event with which clinicians usually have no experience. The viper's complex venom contains hemorrhagins, which inhibit platelet function, induce thrombocytopenia and may also interfere with plasmatic coagulation [3-8]. When puff adder venom causes shock, vascular permeability is increased, leading to extravasation of plasma and, later, red blood cells, followed by hypovolemic shock and death [9]. Cardiac arrhythmias, ECG changes and chronotropic incompetence (no pulse acceleration in relation to hypovolemia) suggest that as-yet unidentified cardiotoxins could be involved [1,9].

Local complications of envenomation such as inflammation, bleeding, edema, pain and, finally, necrosis are caused by hemorrhagins, proteases and fibrinolytic venom constituents.

Recommended first aid measures are immobilization of the patient, including fixation of the bitten limb and urgent transfer to a hospital [10,11]. Antivenom should be administered when there are systemic signs of poisoning or serious local involvement (extension over half the affected limb, rapid swelling) [11]. Obtaining antivenom within a reasonable time often poses a logistic problem,

although there are online databases providing information where antivenom is on stock and when it expires [12]. To date there have been no reports about hyperbaric oxygenation as an adjunctive treatment in puff adder bites.

CASE REPORT

A 25-year-old man incurred a bite to his left hand while trying to catch a puff adder that had escaped from its case. The man lay down while friends caught the snake, called the ambulance and applied a tourniquet to his forearm.

After initial treatment in the nearest hospital, where he received intravenous analgesia, colloidal fluids, 250 mg of prednisolon and a tetanus shot, the patient was transferred to our university medical center for intensive care. On admission four hours after the bite, the patient was awake and alert. He complained of severe pain in his left hand.

Four fang marks were visible between his left thumb and index finger. The hand was swollen beyond the elbow, red and hot to the touch. The patient was able to move his fingers, and sensation was preserved. Capillary refill time was within normal limits. His blood pressure was 159/69 mmHg and pulse rate 86 beats per minute. No bleeding stigmata could be found.

Initial laboratory testing revealed severe thrombocytopenia (23,000/µl – normal range is 140,000-440,000/µl); plasmatic coagulation parameters were within normal limits. We were able to obtain one ampoule of SAIMR polyvalent snake antivenom and 10 ampoules of FAV-Afrique, Aventis Pasteur snake antivenom, but all ampoules were beyond their expiry dates.

As the patient's blood pressure fell to 88/54 mmHg without adequate chronotropic response (heart rate 74 beats per minute) and the local swelling progressed, it was decided to use the antivenom. After administration of an antihistaminic (H1) agent, all 11 ampoules were added to 1000 ml of crystalloid fluid and infused over two hours, starting four and a half hours after the bite. Concurrently, the patient required norepinephrine at a dose of 0.12µg/kg/min. After two hours he was hemodynamically stable and catecholamines could be stopped. Two units of packed platelets were administered.

Follow-up laboratory tests seven hours after the first test showed a normal total white and red blood cell count. The wound was cleaned with alcoholic disinfectant and prophylactic antibiotic treatment was begun. Despite all measures, the left hand and forearm had not improved, and swelling still progressed toward a compartment syndrome. Thus, it was decided to apply hyperbaric oxygenation (HBO_2; 90 minutes, 2.5 bar absolute).

At the end of the first treatment session the patient felt a slight relief from pain and swelling. For the next nine days hyperbaric oxygen treatment was continued according to the same protocol, with one session per day. Once hyperbaric oxygenation had been initiated, there was no further progression of local toxicity, and daily reduction of edema became evident. The patient was discharged on the eleventh day. To date, he is well, with no sequelae.

DISCUSSION

The course of acute illness following puff adder bite often is impressive, with both systemic and local signs of envenomation developing within a few hours. While systemic signs, including bleeding, hemodynamic depression, cardiotoxicity and, in some cases, acute renal failure suggest a poor prognosis and demand immediate medical attention. Local signs like tissue swelling, compartment syndrome and, finally, necrosis worsen long-term outcome and are often difficult to treat or are even irreversible and may necessitate amputation[1,5].

Important initial measures are immobilization of the patient, including fixation of the bitten limb and urgent transfer to a hospital. Application of a tourniquet should be avoided [11]. In our case, acute illness arising from systemic poisoning could be adequately treated with intensive care measures within 24 hours.

Though administration of antivenom can reduce progression of initial tissue damage, it cannot reverse substantial tissue damage like inflammation, edema, necrosis and compartment syndrome [1]. The latter often develops because the body regions most prone to bites are the distal extremities. Their strong intermuscular fascial septa limit space, and swelling quickly impairs circulation. The necessity of fasciotomy or amputation following puff adder bites is not uncommon [5].

Our patient was treated conservatively. Whether initial systemic antibiosis was necessary remains an open question, since there was no signs of infection. Neither is the administration of steroids (which had been given in the first hospital) a general recom-

mendation in snake bites. Hyperbaric oxygenation is the treatment of choice for chronic wounds, compartment syndrome and gangrene, but has also been successfully applied in an animal model of brown recluse spider bites and western diamondback rattlesnake envenomations [13,14]. Though these problems have different etiologies, the underlying pathophysiological mechanisms of inflammation and tissue damage are similar. It thus seemed logical to include HBO_2 in our treatment regimen. Breathing of 100% oxygen under increased ambient pressure causes oxygen to dissolve in the plasma, resulting in paO_2 (arterial oxygen pressure) of 1500 mmHg at 2.5 bar absolute (normal values approximately 90–age/3 mmHg). At this level, oxygen shows pharmacological action such as prevention of reperfusion injury, reduction of edema and reversal of sublethal tissue damage.

Whether the local reaction in our patient would have progressed to full compartment syndrome and necrosis without HBO_2 is difficult to judge. The favorable, almost immediate, impact of HBO_2 on the local reaction was impressive, though, and exceeded the one usually seen when treating a post-traumatic compartment syndrome. If this effect is due to a specific interaction with the snake's toxin or its degradation products remains hypothetical, because there are neither clinical nor experimental reports on HBO_2 in this indication as yet. We do, however, believe that the unfavorable impact of local complications on clinical outcomes observed in puff adder bites [1] and the low rate of adverse effects of HBO_2 [15] justifies the empiric use of hyperbaric oxygenation in such cases. Considering the high incidence of this type of snake bite in some countries, further investigations about the usefulness of HBO_2 for this indication might be warranted.

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