Hyperbaric oxygen in the treatment of venomous snake bites

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Venomous snakes can be found on every continent with the exception of Antarctica. Bites from these snakes present a significant public health concern. While data is somewhat limited, estimates are that 421,000 envenomations occur, with 20,000 deaths annually worldwide [1]. While antivenom is the mainstay of treatment for venomous snake bites, there is growing evidence to suggest the use of hyperbaric oxygen (HBO₂) therapy as an adjunctive treatment. Goldfrank’s Medical Toxicology currently states that HBO₂ should be considered an “experimental” treatment for snake envenomation [2]. With a growing body of evidence to suggest otherwise, should HBO₂ therapy still be considered “experimental” in treating snake envenomation?

Primary treatment of snake envenomation is directed at neutralization of the venom with antivenom. However, adjunctive therapy must often still be employed to manage the tissue toxicity inflicted by the envenomations. Typically, surgical intervention has been the treatment of choice for both compartment syndrome (fasciotomy) and extensive wound necrosis (debridement) [7]. The success of HBO₂ therapy in injury patterns similar to the complications of snake envenomation, albeit from a different cause, suggest that HBO₂ therapy may have a role in treating snake envenomation. It is well known that HBO₂ has an established role in decreasing both morbidity and mortality when added to conventional medical and surgical treatment in traumatic compartment syndrome and management of wounds [3,4]. In addition, there is evidence that HBO₂ therapy is beneficial in treating necrotic arachnidism caused by Loxosceles reclusa – both in animal models and in patients [5,6].

However, early attempts at using HBO₂ therapy in the setting of poisonous snake envenomation yielded mixed results in animal models. A rabbit model was used to evaluate the effects of HBO₂ therapy on myonecrosis secondary to Western diamondback rattlesnake (Crotalinae atrox) envenomations. After three 90-minute treatments of HBO₂ at 2.4 atm with antivenom, no difference in the amount of skeletal muscle myonecrosis was found when compared with antivenom alone or no treatment [8]. Investigations using a mouse model that compared the effect of varying HBO₂ treatments on both myonecrosis and tissue edema caused by Crotalinae atrox envenomations demonstrated no significant difference in tissue edema. However, there was a significant improvement in the resolution of myonecrosis and tissue healing found in mice treated with higher pressures of oxygen, with more treatments and a greater length of treatment [9].

Experience in patients is limited, but there is some anecdototal evidence to support the use of hyperbaric oxygen. There is a report of one case of early compartment syndrome caused by the Western diamondback rattlesnake (Crotalinae atrox) bite which was treated with antivenom and four treatments of 100% HBO₂ (one dive to 2.4 atm for two hours, and three dives at 2.0 atm). Symptoms resolved successfully without surgical intervention [10].

In this issue there are two articles – a case report and a retrospective case series – that present evidence in support of the use of HBO₂ therapy as an adjunctive treatment for snake bite envenomations. Rainer and colleagues (Pages xxx-xxx) report a case of puff adder (Bitis arietans) envenomation, in which 100% HBO₂ at 2.5 atm is used in conjunction with antivenom and other supportive measures. The patient received 10 treatments, each 90 minutes in duration, from the second to eleventh day post-bite. The treatment was started prior to the development of compartment syndrome,
so it is difficult to say with certainty that the HBO₂ therapy prevented it, as not all bites progress to compartment syndrome. However, upon starting hyperbaric oxygen therapy, the patient appears to have improved symptomatically, and the local toxic effects appear to have started to regress instead of progressing [11].

Hochedez and colleagues present a retrospective review of five patients treated with hyperbaric oxygen therapy in addition to traditional antivenom after sustaining Bothrops lanceolatus envenomation. The injury patterns ranged from compartment syndrome (two cases) to abscesses (two cases) to necrotizing fasciitis (one case). All patients had at least one surgical intervention. HBO₂ therapy was not used as a deterrent to surgical intervention but rather as an adjunct to surgical treatment in these cases. Patients received between 1 and 15 treatments of 100% HBO₂ therapy at 2.2 atm. This case series, too, demonstrated a favorable outcome when the use of HBO₂ therapy was added to standard treatments of snake envenomations [12].

While antivenom is the mainstay of treatment for venomous snake bites, these two articles add to a small collection of data that support the use of HBO₂ therapy in the treatment of complications from venomous snake bites. Compartment syndrome, delayed wound healing and necrosis, skin and soft tissue infections and necrotizing fasciitis are all indications for HBO₂ There is no evidence that hyperbaric oxygen should still be considered experimental when any of these are the result of snake venom. These articles add to the growing body of evidence supporting its use specifically in snake envenomations.

REFERENCES