King Cobra envenoming

S. Larréché, [1] G. Mion, [1] J.L. Daban, [1] M. Goyffon. [2]

(1) Sébastien Larréché (MD), Jean Louis Daban, Resident, Georges Mion (MD), Professor of anaesthesiology.
Hôpital d'instruction des armées du Val-de-Grâce,
Département d'Anesthésie-réanimation.
74 Boulevard de Port Royal.
75230 Paris Cédex 05 – France.

(2) Max Goyffon (MD), Professor,
Muséum d'Histoire Naturelle
57 rue Cuvier. 75005 Paris – France.

Correspondance to : Professor Georges Mion (MD)

e-mail address : georges.mion@club-internet.fr

Hôpital d'instruction des armées du Val-de-Grâce,
Département d'Anesthésie-réanimation.
74 Boulevard de Port Royal.
75230 Paris Cédex 05 – France.

Dear Dr Bogod,

We read with a great interest the paper of Veto et al. which reports the first case of King Cobra (*Ophiophagus hannah*) envenoming in the United Kingdom (1). The successful medical handling of a bite by one of the most dangerous *Elapidae* in the world has to be apologised. We definitely agree with the fact that exotic snakebites are an increasing risk in developed countries. This is alarming because physicians are not trained for treating such cases (2). It is thus desirable to propose formations and to diffuse medical recommendations about envenoming.

Several points of the treatment described in the paper have nevertheless to be discussed. Knowing that the venom of *Ophiophagus hannah*, like that of Naja, has a curare-like effect, related to the presence of post-synaptic neurotoxins, it would be of interest to know why this patient received vecuronium, a post-synaptic neuromuscular blocking drug ? Did he benefit from a monitoring of the neuro-muscular junction?

The authors describe an atypical symptom of the ophidian envenoming in this intubated patient, namely a hypertensive crisis, treated by labetalol. It is known that snake bites rather generate hypotension, even a shock state, through hypovolemic, vasoplegic or vagal mechanisms. One may wonder whether this hypertensive crisis may have been in connection with an insufficient sedation by morphine. On the other hand, the use of sufentanil, which is a more potent and no histamino-liberator drug, would have been perhaps more appropriated. The use of a β-blocking agent appears poorly indicated in this asthmatic patient. Moreover, we may suppose that labetalol was probably the true origin of the bronchospasm, rather than antivenom. To use a calcic inhibitor, like nicardipine, would certainly have been more appropriated.

Whereas the effectiveness of antivenom is not to demonstrate any more in 2007, its indications are poorly known by physicians. The paper of Veto et al. which confirms the effectiveness of this treatment in an elapid envenomation is of great interest.

Major loco-regional symptoms (extensive oedema or necrosis), bleeding or coagulopathy (a thrombopenia, a drop in coagulation factors or an hypofibrinogenemia), neurological or cardio-circulatory disorders, a rhabdomyolysis or an acute renal failure are indications to an immediate anti-venomous immunotherapy (2). On the other hand, hyperleucocytosis, common in this setting, does not represent per se an indication to treat. Lastly, it will be retained that a bradycardia, disorders of conduction or of repolarisation, are evocative of envenomation by Atractaspis, and may be indications, too. The patient should be assessed every 4 hours, as recommended by Chippaux, and should receive a supplemental dose of antivenom if he still exhibits one of the above signs (3).

The adverse effects induced by immunotherapy, most dramatic of which are anaphylactic shock and serum disease, became rare since the use of highly purified immunoglobulin Fab'2 fragments. Such antivenins are unfortunately not available for all snake species and the physician has sometimes to use products of poor quality only. In this last case, especially, he encompasses the risk to deal with anaphylactic reactions. The treatment of an anaphylactic shock is the intravenous administration of epinephrine, in boluses, then in perfusion with an electric syringe. On the other hand, the anaphylactic shock cannot be prevented by either epinephrine, hydrocortisone nor antihistaminic agents.

Lastly, the effectiveness of immunotherapy, managed six hours after the bite of this elapid, and the early weaning from artificial ventilation is of particular interest. It has been shown in vitro that immunotherapy remains effective against neurotoxic venoms containing curarizing toxins (Naja sp's venoms) (4); on the other hand, it has been demonstrated in the clinical setting that immunoglobulins loose their effectiveness when injected more than 4 hours after bites of the australian Elapidae, Oxyuranus scutellatus. In this last setting, treatment then rests on symptomatic measures such as artificial ventilation continued until the natural elimination of toxins (5).

(Best regards)

Larréché S., Mion G., Daban JL., Goyffon M.

1. Veto T, Price R, Silsby JF, Carter JA. Treatment of the first known case of king cobra envenomation in the United Kingdom, complicated by severe anaphylaxis. *Anaesthesia* 2007 ; **62** : 75-8.
4. Warrell DA. Treatment of bites by adders and exotic venomous snakes. *BMJ* 2005 ; **331** : 1244-7.
5. Chippaux JP. The developpement and use of immunotherapy in Africa. *Toxicon* 1998 ; **36** : 1503-6.
6. Boulain JC, Menez A. Neurotoxin-specific immunoglobulins accelerate dissociation of the neurotoxin-acetylcholine receptor complex. *Science* 1982 ; **217** : 732-3.
7. Trevett AJ, Lalloo D, Nwokolo NC et al. The efficacy of antivenom in the treatment of bites by the Papuan taipan (*Oxyuranus scutellatus canni*). *Trans R Soc Trop Med Hyg* 1995 ; **89** : 322-5.