Eye muscle paralysis after *Vipera aspis* envenomation

G Re, G Trisolino, F Miglio

A 22 year old man was bitten on his left foot by a snake identified as *Vipera aspis*. He was admitted to our emergency department 40 minutes later. On arrival, the patient was confused (Glasgow coma scale 14), pale, and tachycardic (120 beats/min). His respiration rate was 40/min and blood pressure 95/50 mm Hg; temperature was 36.1°C. Peripheral perfusion was good. The left foot was oedematous. Neurological examination revealed facial diplegia, bilateral ptosis, external ophthalmoplegia (fig 1). A slight abnormality of pupillary accommodation was observed. The first cranial nerve was not involved and dysphonia was not observed. Limb and trunk strength was normal. Tendon, plantar, and abdominal reflexes were regular. Muscle enzymes were within the normal range (serum creatine phosphokinase: 192 U/l). Four litres of normal saline and two vials of antivenom were administered intravenously during the first two hours. The patient’s neurological picture improved steadily during the next two to three days, and two weeks after the snakebite neurological examination was normal.

*Vipera aspis* has been reported to be one of the most common snakes in the western part of southern Europe. Reactions to its venom include vomiting, diarrhoea, hypotension, shock, coagulopathy, thrombocytopenia, weakness, paraesthesias, and bleeding. Neurotoxicity has been known to occur but is unusual and difficult to explain, especially as the venom’s main components of geographically focal populations of *V aspis* may produce different neurotoxic activities. Recently, phospholipase A2 activities similar to vipoxin, which is known to be present in another European viper species (*V ammodytes meridianalis*), have been found in the venom of *V aspis*. In the original clinical case reports of neurotoxic symptoms documented in human beings after *V aspis* envenomation, the neurotoxicity was strictly localised in the cephalic muscles.

This case provides a clear description of neurotoxic envenomation involving muscles innervated by the third, fourth, and sixth cranial nerves in the absence of skeletal muscle damage. Extraocular muscle susceptibility in snake envenoming may be related to some basic properties of the neuromuscular junction anatomy and physiology, since other neuromuscular junctional disorders (such as myasthenia gravis and intermediate syndrome of organophosphorus insecticide) preferentially affect eye muscles.

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