Neurovascular paralysis in *viper aaspis* envenomation: pathogenetic mechanisms

Vipera aspis is the most common agent of snake envenomation in Italy and Western Europe. Its bite affects coagulation and causes a shock syndrome with severe cardiovascular failure.

Neurotoxicity, clinically characterised by external ophthalmoplegia, is uncommon (two cases out of 205 patients bitten by viper’s aspis) and difficult to explain because overt neurotoxic substances have not been detected in vipera aspis venom. Our case suggests that the venom is not neurotoxic.

A 20 year old herpetologist was bitten by a vipera aspis at the distal extremity of the index finger of the left hand. When he was admitted to the intensive care unit (50 minutes later) he was unconscious (Glasgow Coma Scale 7), pale, tachycardic (170 beats/min), tachypnoeic (50 breaths/min), without detectable peripheral pulses and blood pressure.

There was a metabolic acidosis (pH 7.24) and disseminated intravascular coagulation. The left hand was oedematous. Centrifugal venous compression was applied on the left arm. Shock, metabolic failure and disseminated intravascular coagulation syndrome were treated with fresh frozen plasma, albumin, dextran, dopamine and adrenaline, NaHCO₃ and heparin iv infusions. Cardiovascular and respiratory function, metabolic balance and consciousness returned to normal within the following three hours.

Neurological examination revealed facial diplegia, pharyngo-laryngeal paresis, bilateral ptosis and external ophthalmoplegia, with complete ocular closure.

The strength of the trunk, limb and respiratory muscles, deep tendon reflexes, plantar and abdominal reflexes, and sensory functions were normal. Symptoms were not modified by iv administration of 10 mg of edrophonium.

Neurophysiological studies of the facial nerves showed a low amplitude muscle action potential (0-9 mv-ns > 3 mv), with normal latency. Repetitive stimulation at low and high frequencies, tetanisation and stimulation with paired stimuli at stimulus intervals of less than 10 ms gave normal responses without signs of muscular transmission defects. Blink reflex showed responses with normal latencies. Similar neurophysiological studies performed on other nerves (median, common peroneal and sural) were normal.

Five days from the onset of the disease the patient improved considerably and after 10 days, neurological examination and neurophysiological tests were normal. He was discharged after 10 days.

The lack of clinical involvement of motor, sensory and cerebellar pathways within the brainstem, together with the normal latency of blink reflex responses in this case, do not suggest an involvement of the brainstem possibly caused by oedema and/or dissemi-
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