Acute myasthenic syndrome following coral snake envenomation in the Caribbean: a harbinger for admission to the intensive care unit

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DESCRIPTION

A 25-year-old Afro-Caribbean woman was bitten by a snake in her right inner thigh while in a forest reserve in Trinidad and Tobago. As an ardent nature lover and animal rights activist, she had entered the rainforest with companions to release a wounded owl that had been rehabilitated. The snake was photographed by digital phones and later identified by a toxicologist as *Micrurus lemniscatus*, a coral snake species (figure 1A). The patient experienced local minor bleeding, followed 5 min later by regional swelling, pain and paraesthesia radiating to her upper thigh. She arrived at our tertiary care hospital 4 hours later. She had a history of allergic reactions to insect bites manifesting as local swelling, but no history of consumption of medications, recreational drugs, alcohol or tobacco. On admission, vital signs were normal. Neurological examination demonstrated bilateral palpebral ptosis (figure 1B), slurred speech, dysarthria, dysphagia, sialorrhoea and flaccid quadriparesis (grade 4/5). The deep tendon reflexes were preserved with no sensory abnormalities. The right inner thigh puncture is shown in figure 1C. The rest of the physical examination was normal. Her course in the hospital is shown in table 1.

Laboratory studies revealed normal full blood count, erythrocyte sedimentation rate, C reactive protein, coagulation, and renal and liver function tests. Serum calcium, phosphorous, magnesium, creatine kinase and amylase were also normal. Arterial blood gas remained normal during hospitalisation. Chest X-ray, ECG and echocardiogram were normal. An ultrasound examination of the right leg was normal. Electromyography and edrophonium test were not performed.

Diagnosed with progressive descending myasthenic syndrome of coral snake envenomation, we administered 80 mL of monovalent equine lyophilised coral antivenom intravenously with no adverse reactions or significant improvement. Atropine 0.6 mg followed by neostigmine 1.5 mg were given intravenously stat. Half an hour later, atropine 0.6 mg followed by neostigmine 0.5 mg were given intravenously stat again. Improvement in single breath count from 12 to 35, muscle strength and sialorrhoea by more than 75%, and ptosis in more than 25% in 1 hour were documented. No further administration of atropine-neostigmine was necessary in our patient due to appropriate and significant response thereafter. When the symptoms

are not completely resolved, five additional doses of atropine-neostigmine (0.6 mg/0.5 mg) should be given every half-hourly intravenously and then 2–12 hourly according to recovery. For severe cases, intravenous infusion of neostigmine at 12 µg/kg/hour can be administered.^{1–3} Respiratory distress was resolved, and oxygen therapy and nebulisations were discontinued. The patient recovered and was discharged from hospital 5 days from admission.

Neostigmine is a reversible acetylcholinesterase inhibitor that restores neuromuscular transmission by increasing concentrations of acetylcholine at the neuromuscular junction, counteracting postsynaptic blockade at the nicotinic receptors. Neostigmine increases bronchial, gastric, sweat gland and salivary secretions at the muscarinic receptors. Atropine is an anticholinergic agent that inhibits smooth muscle and glands which are innervated by postganglionic cholinergic nerves, and is administered together with neostigmine most commonly to reduce the unwanted muscarinic effects of neostigmine by preventing excessive secretions.³

Coral snake venom contains α-neurotoxins of the three-finger (3FTx) protein superfamily that cause muscle weakness by competitive inhibition of the nicotinic acetylcholine receptors at the

Figure 1 Images of (A) *Micrurus lemniscatus* and the patient showing (B) bilateral palpebral ptosis and (C) a small puncture mark in the right inner thigh (black arrow) caused by the snake bite. The patient experienced full recovery of the ptosis 10 days after treatment with neostigmine (D).

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Table 1 Clinical manifestations of envenomation by *Micrurus lemniscatus* in a 25-year-old Caribbean woman

Phase 1: initial manifestations, duration and treatment	Phase 2: neurological symptoms and signs before treatment, duration and treatment	Phase 3: neurological symptoms and signs after treatment, duration and treatment
<p>Onset: between 5 and 60 min after the accident: local small bleeding, mild local pain and paraesthesia, and radiation of pain and local myalgia. Transferred to the nearest hospital.</p>	<p>Onset: 1 hour after the bite, the patient showed bilateral palpebral ptosis, diplopia and dysarthria. Snake identification, toxicological centre contacted, antivenom requested and toxicologist informed.</p> <p>2 hours after the bite, she experienced dysphonia, difficulty walking and decreased strength in the upper limbs, generalised myalgia and muscle tremors, and headache and dizziness.</p> <p>Rest with immobilisation of the patient, local cleansing and antibiotic therapy (ceftriaxone 1 g intravenously daily) were instituted. A local incision with suction was out of therapeutic range (90 min post bite). Tetanus toxoid 0.5 mL was also given intramuscularly. Acetaminophen 1 g intravenously three times daily was given.</p> <p>3–6 hours after the bite, the sensorium was clear and the cranial and peripheral nerve functions were intact. However, there was severe difficulty speaking, dysphagia and profuse sialorrhoea. She developed mild shortness of breath. Oxygen therapy was given at 8 L/min via a face mask.</p> <p>The patient was put on NPO and a nasogastric tube was inserted. Nebulisations every 6 hours with epinephrine 5 mg were given. Pantoprazole 40 mg intravenously daily was given. 2 L of dextro-saline intravenous fluid was given daily for 2 days.</p> <p>Transferred to the ICU for close neurological and respiratory monitoring, possible intubation and ventilatory support. Constant mouth suctioning of secretions was instituted. ABG done was normal. A skin test for sensitivity to coral snake antivenin (Laboratorios Probiol, Colombia) was negative. This antivenom was manufactured using <i>Micrurus mipartitus</i> venom. One vial of this antivenom should neutralise at least 1 µg of the coral snake venom.</p>	<p>6–18 hours after the bite, antivenom was given (1 vial every 4 hours) intravenously. No improvement in the myasthenic syndrome was noticed. Constant mouth suctioning of secretions continued. The sensorium was clear and the cranial and peripheral nerve functions were intact. The shortness of breath continued but no deterioration was seen. Nebulisations every 6 hours with epinephrine 5 mg continued.</p> <p>18–34 hours after the bite, antivenom was given (1 vial every 4 hours) intravenously. No improvement in the myasthenic syndrome was noticed. Constant mouth suctioning of secretions continued. The sensorium was clear and the cranial and peripheral nerve functions were intact. The shortness of breath continued but no deterioration was seen. Nebulisations every 6 hours with epinephrine 5 mg continued. ABG was normal.</p> <p>Rapid and progressive recovery of the myasthenia: atropine-neostigmine therapy was administered. A progressive recovery of the patient was observed.</p> <p>Full recovery: atropine-neostigmine therapy was repeated half an hour later. Improvement continued. Full recovery was achieved.</p> <p>Discharged from the ICU: discharged from ICU 2 days from admission. Intravenous fluid was discontinued. The patient was placed on normal diet after removing NSG tube. Parenteral antibiotic, pantoprazole and paracetamol were discontinued.</p> <p>Discharged from the hospital: discharged on the fifth day postadmission, with improvement of the myasthenia, but still showing mild palpebral ptosis. Cefuroxime was given at 500 mg orally two times per day for 5 days.</p> <p>Clinical follow-up: moderate motor weakness lasted 7 days, and ptosis (figure 1D), ageusia and fatigability lasted 10 days. Transitory loss of taste lasted approximately 18 days.</p>

ABG, arterial blood gas; ICU, intensive care unit; NPO, nothing by mouth; NSG, nasogastric.

neuromuscular junction. Phospholipase A2 may cause myotoxicity and rhabdomyolysis.^{1–3}

Hypersalivation from envenomation by coral snakes is uncommon. In 2006 Bucarechi *et al*³ reported 1 case of moderate hypersalivation out of 11 (9.09%) cases of envenomation with *Micrurus* species in Brazil. The concomitant manifestation of excessive salivation and neuromuscular features in that patient was not described. The first line of therapy for the neuromuscular blockade manifestations of neurotoxic snake

envenomation is the specific snake antivenom. The administration of the combination of atropine-neostigmine is indicated when the antivenom is unavailable, if there is a delay in obtaining a sufficient amount, or in patients given the highest recommended doses of antivenom without improvement in paralysis or with delayed recovery. The glycopyrrolate-neostigmine combination can also be used with success.^{1–3}

M. lemniscatus is a highly venomous coral snake indigenous to the forests of Brazil, Bolivia, Colombia, Venezuela, Ecuador, Guyana, French Guiana, Suriname, Argentina, Paraguay, Peru and Trinidad and Tobago.^{1–3} *M. lemniscatus* characteristically has bright, bold-coloured rings, triad of black bands, small head, and a blunt and rounded snout (figure 1A).

This is the first published case from the Caribbean of acute myasthenic syndrome following coral snake envenomation causing respiratory paralysis. Timely intervention prevented death and disability.³ Snake bite is a neglected public health issue in the world, and education, improvement in healthcare and ongoing research are all essential.^{1–3}

Correction notice This article has been corrected since it was published Online First. The value of "Tetanus toxoid" has been corrected from "5 mL" to "0.5 mL" in table 1.

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Learning points

- ▶ Acute myasthenic syndrome has been reported as a result of envenomation caused by the Elapidae (coral and cobra snakes) and viper snakes (*Viper berus*) families, and even from the poisonous sea snakes of the warm waters of the Indian and Pacific Oceans.
- ▶ Progressive myasthenic syndrome associated with snake envenomation is an early warning sign of impending respiratory paralysis and can respond to the specific antivenom and/or atropine-neostigmine therapy.
- ▶ Neuromuscular manifestations of snake envenomation can include myokymia, myoclonus, myospasm, myositis and myasthenic syndrome.

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