Learning Point for Clinicians

This report highlights that clinically significant and striking dysautonomia can rarely occur in elapid snakebites and when present in the absence of clear history of snakebite, can cause diagnostic confusion with other mimickers of neuroparalytic snakebite such as myasthenic crisis, Grave’s disease, botulism, Guillain–Barre syndrome and acute porphyria.

Case report

One early morning in August 2011, a previously healthy girl of 17 years was brought to the emergency medicine department of our hospital with history of sudden onset drooping of upper eyelids, diplopia, difficulty in speaking, swallowing and breathing and weakness of limbs for 2 h. These symptoms were preceded by abdominal pain and vomiting. She was apparently alright the previous night and had slept outside her kutcha house on the ground. There was no history of similar illness in family or deliberate self-poisoning.

On admission, she was found to be unconscious, cyanosed with poor respiratory efforts. Pupils were normal and reactive to light. She was intubated immediately and mechanically ventilated. After an hour, she regained consciousness and was noticed to have ptosis, complete external ophthalmoplegia and quadriplegia (muscle power: grade 2/5), with preserved tendon jerks and neck muscle weakness. With suspicion of neurotoxic snake bite, 10 vials of freeze dried polyvalent antisnake venom (ASV) antitoxin were administered, although no fang marks could be made out. Later, mechanical ventilation was continued in medical intensive care unit (MICU) under sedation and analgesia. Ten more vials of ASV were repeated 2 h after first dose. Serum potassium, calcium, phosphates, magnesium, arterial blood gas, renal, liver function, coagulation tests and serum cholinesterase levels were normal.

During the first 3 days in MICU, she had multiple unexplained episodes of dysautonomia, each lasting ~5–15 min, characterized by unexplained tachycardia (heart rate: 110–150 per minute), hypertension (BP: 150/100–190/110 mm Hg), profuse sweating and pupillary mydriasis. Hypoglycemia and hypoxia were ruled out during these episodes. These episodes were managed conservatively, without drugs. Electrocardiogram, chest radiograph, abdominal sonography and cardiac echocardiography were normal. She had complete recovery of muscle power and was extubated on day 6. Urinary porphobilinogen measurement (done on day 2), thyroid function tests (day 6), nerve conduction and repetitive nerve stimulation studies (day 7) were normal. Upon recovery, patient could recollect some bite on her right leg but was not aware of what had bitten her. Autonomic function tests done on day 12 and repeated at 6 weeks, 3 and 6 months follow-up were normal.

Discussion

Venomous snakebite is an important, yet neglected public health problem in tropical countries,
accounting for significant morbidity and mortality. In India, around 45 900 deaths are estimated to result annually from snakebites. The commonly encountered venomous snakes in India include common cobra (Naja naja), common krait (Bungarus caeruleus), Russell’s viper (Daboia russelli) and saw scaled viper (Echis carinatus), against which a locally prepared equine polyvalent ASV antitoxin is available. Neurological manifestations that follow envenoming by elapids (cobras and kraits) and less commonly Russell’s viper include ptosis, external ophthalmoplegia, paralysis of pharyngeal muscles, followed by respiratory and generalized muscle paralysis.

Our patient most probably had common krait bite with severe envenoming, as she had typical descending pattern of paralysis, culminating in respiratory paralysis in the early morning hours during sleep, preceded by abdominal pain and vomiting. This pattern of early morning onset neuroparalysis has been reported in krait bites. Krait bites usually occur at night, sometimes producing minimal or no pain and local reaction at bite site. Fang marks may not be appreciable. Hence, a sleeping victim might be unaware of bite. Sleeping outdoor on the ground adds to the strong possibility of snakebite.

Moreover, she resided in a part of southern India with high prevalence of venomous snakebites. As there was clinically significant autonomic dysfunction (AD) and no clear history of snakebite in the index case, other differential diagnoses such as myasthenic crisis, Grave’s disease with ophthalmopathy, botulism, Guillain–Barre syndrome (Miller Fisher variant) and acute porphyria were considered. These were excluded based on clinical features, course of illness and relevant investigations.

AD in victims of elapid snakebites has been under reported. AD can present as unexplained abnormalities in heart rate (tachycardia or bradycardia) or rhythm, hypertension or hypotension, pupillary abnormalities, episodes of unexplained sweating, lacrimation, salivation, vomiting, abdominal pain, paralytic ileus and constipation. AD, most often, is not a dominant clinical manifestation and is overshadowed by other serious neuroparalytic manifestations. In a study of common krait bites, 139 of 210 victims (66%) exhibited AD, which was more marked in those with severe envenomation.

The pathogenesis of AD may involve decreased parasympathetic activity, blockade of presynaptic alpha-2 receptors by neurotoxins causing sympathetic nervous system overactivity. AD is mostly self-limiting and needs no specific management, apart from ASV and supportive care.

Conflict of interest: None declared.

References