Cerebellar Ataxia following Snake Bite

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Abstract

Neurotoxic snake envenomations are frequently encountered in medicine emergencies. Here we report a case of snakebite who presented with neuromuscular paralysis and respiratory failure, showed full recovery after effective treatment. Patient however developed cerebellar ataxia possibly due to delayed neurotoxicity of venom.

Introduction

Snake bite is an important health problem in India, particularly rural and farming areas. Of the 52 poisonous species in India, majority of bites and consequent mortality is attributable to 5 species viz. Ophiophagus hannah (king cobra), Naja Naja (common cobra), Daboia russelii (Russell’s viper), Bungarus caeruleus (krait) and Echis carinatae (saw-scaled viper). Common neurotoxic envenomations have the potency to cause a broad spectrum of presentations varying from ptosis and ophthalmoplegia to respiratory arrest which present as acute medical emergencies and show a good response if effective treatment is given in time. Other end of spectrum is formed by delayed neurotoxic manifestations which include peripheral neuropathies, occasionally optic neuritis and cortical blindness. Cerebellar involvement is very uncommon. On extensive medline search delayed neurotoxicity presenting as cerebellar ataxia has been reported very occasionally.¹ We report a case of cerebellar ataxia as delayed neurological complication following snake bite.

Case Report

A 26 year old female labourer, presented to the emergency department of our hospital with history of unidentified snake bite on the toe of left lower limb of 16 hours duration. She was bitten by snake while she was asleep which was not associated with any paraesthesia or bleeding at local site. She went off to sleep after a brief interruption during night. Next morning she became disoriented after she got up following an apparently short normal period. She was brought to hospital with history of rapidly developing ptosis, diplopia, breathlessness and weakness of all four limbs. There was no history of any addiction or drug intake. On general examination patient was unconscious unresponsive to deep painful stimuli, with unrecordable pulse and blood pressure and poor respiratory effort with a respiratory rate of 12 per min. along with pallor. Icterus, cyanosis, clubbing, oedema and lymphadenopathy were absent. There was no swelling or bleeding at local site of bite. On systemic examination cardiovascular, respiratory and abdominal examinations were essentially normal. Central nervous system examination revealed generalized hypotonia with power of grade 0 to 1 in all four limbs. All cranial nerves were normal. Light reflex, deep tendon reflexes and superficial reflexes were absent. Pupil was normal in size with sluggish reaction. A possible diagnosis of neurotoxic snake envenomation was made. Patient was mechanically ventilated because of poor respiratory efforts. She was given antisnake venom (150 ml) after sensitivity testing, in addition to antibiotics. Prothrombin time, partial thromboplastin time, bleeding time and clotting time were also normal. Other laboratory investigations included hemoglobin of 9.5 gm/dl, total leukocyte count 12,600/ mm³, platelet count 2 lacs/mm³. Plasma glucose, blood urea, serum creatinine, electrolytes including serum sodium, serum potassium, serum bilirubin, AST, ALT, total serum protein, albumin/globulin ratio, arterial blood gases and electrocardiogram were within normal limits. During her stay of two months her ataxia did not show any improvement, later she left against medical advice and was lost to follow up.

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Discussion

Snake bite is relatively uncommon in urban areas. Tertiary care centers in metropolitan cities like Delhi get these patients from adjoining rural and semi-urban areas as referred cases. Common neurotoxic snakes in India include Cobra (Naja naja) and Krait (Bungarus caeruleus). Krait bites are commonly reported in night. Those sleeping on the floor are at a greater risk and they show paucity of local tissue reaction. All these facts were conspicuous in this case.¹²

Common neurological symptoms in decreasing order of frequency include ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and neck muscle weakness (7.1%). These are experienced usually within 6 hours of the bite.³ Following administration of antivenom, the signs of recovery become evident within a few hours to several days.⁴ Our patient developed most of the neurological symptoms described above.

A larger prospective clinical study in Srilanka showed alteration in level of consciousness in 71%, autonomic disturbance in 66%, anterograde memory loss in 40% and delayed neuropathy in 22%.¹³

Anti snake venom (ASV) forms mainstay of therapy and doses up to 400 ml have been used.¹ In our patient a dose of 150 ml was used. Polyvalent antivenom has no significant benefit in reversing respiratory paralysis and preventing delayed neurological complications. Polyvalent ASV is relatively safe, and allergic reactions after ASV injection can be prevented by...
premedication with adrenaline, intravenous hydrocortisone and antihistaminics. Ventilatory support forms a corner-stone of krait envenomation therapy. Incidence of complication is directly proportional to the duration of venom in blood. Respiratory failure is the most common cause of mortality and morbidity in victims bitten by snakes. A mortality rate of 7.6% was observed in patients on intensive care management. A prompt recognition of respiratory failure and timely mechanical ventilation can decrease morbidity and mortality. But due to poor availability at periphery and at larger district centres ASV still remains mainstay of therapy. Therefore, suspected snake envenomation should be treated empirically with intravenous polyvalent ASV.

Other causes of death are complications of mechanical ventilation, shock, intracerebral haemorrhage, wound complications, tetanus, cortical venous thrombosis, renal failure, and hypoxic brain damage.6

Features of delayed neurotoxicity have been postulated due to structural damage to nerve endings, nerve fibres or demyelination. Neurotoxin probably causes ultrastructural damage to motor nerve endings.1 Similar pathogenesis could be true for development of cerebellar ataxia in our patient although brain imaging was normal. Direct toxic effects of snake venom on brain parenchyma have not been reported in literature.6 The reason why cerebellar ataxia developed as a late neurological manifestation is unknown. The other possibilities for delayed neurological symptoms which can be considered include secondary to hypoxic brain damage and ASV hypersensitivity following snake envenomation.

The hypoxic effect on the brain is more likely related to respiratory paralysis and cardiac arrest that occurs after neurotoxic envenomation. Prolonged respiratory paralysis following snake bite results in widespread cerebral hypoxia which on recovery usually manifests as focal neurological deficit which can be further documented in neuroimaging.7 This possibility seems unlikely in our case as the patient presented very early after onset of symptoms and an isolated episode of cardio respiratory arrest was managed immediately with life support, central nervous system examination soon after regaining consciousness did not show any focal neurological deficit which are frequently associated with hypoxic brain damage and neuroimaging was also normal.

Though treatment by ASV still forms the mainstay of treatment of a poisonous snake bite we should be aware of its possible reactions. The precise mechanisms responsible for these reactions have not been clearly established, although there is strong evidence pointing to direct complement activation, effects of contaminating pyrogens, and reactions to immune complexes. Delayed (serum sickness type) reactions usually occurs 1 to12 days after treatment. Characterized by fever, arthralgia, myalgia, nausea, vomiting, diarrhea, recurrent urticaria, lymphadenopathy, periarticular swellings, proteinuria, with immune complex nephritis and rarely uveitis, neurological manifestations include mononeuritis multiplex, encephalopathy and optic neuritis.8 No documentation of cerebellar ataxia resulting from ASV could be found.

Some newer, less allergenic substitutes are being developed, namely more purified ASV using polyacrylamide gel affinity chromatography, purified antigen-binding fragments (Fab) of TgG, monoclonal antibodies and highly refined purified antivenom from sheep or chicken.

Delayed central nervous system manifestations following snake envenomations include nerve conduction defects in ulnar, median and common peroneal nerve. There can be sensory loss at site of bite. Glove and stocking type of sensory motor neuropathy has also been seen. Ophthalmic neurotoxicity include optic neuritis, retinal and optic nerve oedema, pupillary changes, optic atrophy and cortical blindness.1,7

In view of these known complications and possible multiple etiologies a possibility of cerebellar ataxia induced by ASV was considered. The unusual finding and rarity prompted us to document this case. On an extensive review of literature we could only come across one patient with cerebellar ataxia in whom cerebellar features persisted for two years on follow up.1 Therefore, evolution of cerebellar ataxia in patient with definitive history of snakebite adds to profile of complications of snake bite.

References