

# The role of pyridostigmine in recovery from motor paralysis in a snakebite patient with an allergic reaction to anti-snake venom

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## Abstract

A 30-year-old woman presented as an emergency with a history of snakebite 5 h previously with signs of bulbar palsy, ptosis, respiratory distress and weakness of all four limbs. Mechanical ventilation, anti-snake venom (ASV) and supportive management were immediately instituted. With the third dose of ASV, an early anaphylactic reaction ensued. Subsequent management with corticosteroids and antihistamines over the next few days allowed consciousness to return but muscle power did not improve beyond 2/5. A trial of intravenous neostigmine with glycopyrrolate, however, improved motor power in all four limbs to 3/5. Oral pyridostigmine at 60 mg every 8 h allowed subsequent full motor recovery in all four extremities. We suggest consideration of pyridostigmine to promote motor recovery after an allergic reaction to ASV.

## Keywords

Pyridostigmine, snakebite, anti-snake venom, recovery

## Introduction

An Elapid (cobra and krait) snakebite may cause neurotoxic muscle paralysis which may prove fatal. Anti-snake venom (ASV) should be ideally administered within 4 h of the bite but is still effective if given within 24 h. On average, 10 vials are administered for neurotoxic bites and 20 vials for viper bites. However, ASV may cause early anaphylactic reactions in 11% and pyrogenic reactions in 25% of victims.<sup>1</sup> A poor outcome of ASV treatment is usually associated with its delayed initiation and associated complications of high dosage, related to allergic reactions.<sup>2</sup>

## Case report

A 30-year-old woman presented with a history of snakebite 5 h previously in a semi-conscious state. She had ptosis, bulbar palsy, respiratory distress and absent motor power (upper limbs 0/5 and lower limbs 0/5). Mechanical ventilation, anti-snake venom (ASV) and supportive management were initiated. A polyvalent ASV infusion with a loading dose of 50 mL was followed by one vial (10 mL), every 8 h, for the first 48 h. However, on account of an allergic reaction to ASV after the third dose, exemplified by tachycardia,

hypotension, urticaria and fever, the ASV was immediately stopped and corticosteroid and antihistamines administered. In the next few days, she regained consciousness, but her muscle power did not improve beyond 2/5. A trial of intravenous neostigmine 1 g with glycopyrrolate every 2 h was found to improve motor power immediately in all four limbs to 3/5. Thus, pyridostigmine bromide 60 mg every 8 h was continued orally. Gradually, over the next 3–4 days, complete motor function returned in all four extremities. Her laboratory investigations, electrocardiogram and chest radiographs were all within normal limits. She remained asymptomatic after discharge.

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## Discussion

Rapid airway obstruction and diaphragmatic paralysis occurs due to the neuro-paralytic effect of an  $\alpha$ -toxin. On average, each millilitre of ASV neutralises 0.6 mg of Indian cobra venom and 0.45 mg of common krait venom. However, in an anaphylactic reaction, ASV needs to be stopped immediately and supportive anti-allergy management given.<sup>3</sup>

In this situation, the treatment of choice for neurological weakness is not evidence-based. In an earlier case report, oral pyridostigmine 30 mg every 6 h was administered in two patients with descending motor paralysis, where neurotoxic envenomation or myasthenia gravis were possible causes. Some authors have reported false-positive elevated titres of Anti-AChR Ab, possibly following ASV. Nonetheless, the important observation was that patients showed neurological motor recovery with ASV and pyridostigmine.<sup>4</sup>

Pyridostigmine is a synthetic quaternary acetyl cholinesterase inhibitor used in myasthenia gravis. Anticholinesterases have been beneficial in cases of neurotoxic envenoming by Asian Cobra, as they lead to an increase in acetylcholine at the neuromuscular junction synapse, enabling the neuromediator to stimulate unblocked receptors and attenuate the toxicity of the  $\alpha$ -neurotoxins. Better results obtained with pyridostigmine, compared to neostigmine, are due to better circadian cover and the longer half-life of pyridostigmine. Concomitant administration of atropine reduces adverse effects of anticholinesterase drugs.<sup>5</sup>

The benefit of pyridostigmine is also expected in cobra envenoming, but lacks benefit in presynaptic toxicity with kraits, taipans and coral snakes.<sup>5</sup> Thus proper identification of snake type is needed to identify the group of patients who may benefit from such treatment.

Different mechanisms of neurotoxicity with snake bite and allergy of ASV make management of these

patients challenging. More research into recovery at the neuromuscular junction and neurotoxic manifestations following envenoming is required to substantiate alternate modalities for snake bite treatment other than ASV.

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