Case Report
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Secondary hyperkalemic paralysis and acute kidney injury due to rhabdomyolysis caused by sea-snake envenomation

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Abstract

Sea-snakes are a common occupational hazard to fishermen in Asian coastal waters.Sea snake venom contains both myotoxins and neurotoxins. Hyperkalemia is a rare cause of acute flaccid paralysis. Myotoxicity result in rhabdomyolysis due to muscle necrosis, acute kidney injury(AKI) and hyperkalemia sometimes resulting in flaccid paralysis. We report here a 50 year old fisherman who presented with muscle pain, AKI,hyperkalemia and quadriparesis with respiratory paralysis. History revealed sea snake bite on his right hand. His serum creatine kinase and lactate dehydrogenase enzymes were elevated several fold.Urine myoglobin was positive. He was treated with hemodialysis and other supportive measures. He required total of 7 hemodialysis sessions and was discharged with serum creatinine of 2mg/dl on day 25.His muscle paralysis was attributed to hyperkalemia as he recovered dramatically with hemodialysis support.

Keywords: acute kidney injury, hyperkalemia, myotoxicity, sea snake

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1. Introduction

Sea-snakes do not bite humans unless provoked. Potent myotoxins in the sea snake venom cause significant muscle necrosis, with consequent myoglobinuria . Hyperkalemia is a rare but important cause of acute flaccid paralysis which is rapidly reversible and can simulate Guillain Barré syndrome [1]. Here,we describe sea snake bite in a fisherman who presented with rhabdomyolyis, AKI and respiratory failure due to secondary hyperkalemic paralysis. He improved with hemodialysis.

2. Case report

A 50-year-old man who was a fisherman by occupation along the coasts of Indian ocean, admitted with weakness of all four limbs associated with muscle pain, dysphagia, dark colored urine and oligo-anuria for two days. He was not a diabetic or hypertensive and no prior such episodes of weakness. His blood pressure was 130/90 mm of Hg, respiratory rate-18/min and pulse rate- 92/min. Examination revealed flaccid quadriparesis with power of 2/5, absent tendon reflexes and muscle tenderness. There was no ptosis or ophthalmoplegia.All sensations were normal. He developed respiratory paralysis and was put on mechanical ventilation. Investigations revealed trace proteinuria; haemoglobin: 12gm/dl; white blood cell count: 9200/cu; blood urea: 86mg/dl; serum creatinine :6.5mg/dl; sodium: 130mEq/L; potassium: 8.6mEq/L; total bilirubin:0.9mg/dl; aspartate amino transferase:1625IU/L; creatine phosphokinase : 29970IU/L; lactate dehydrogenase :10170 IU/L;corrected calcium : 8.1mg/dl; phosphorous: 5.8mg/dl; uric acid 13 mg/dl. Urine myoglobin was positive. Blood coagulation parameters were normal. Electrocardiogram showed tall T waves and wide QRS complex. Diagnosis of acute kidney injury due to rhabdomyolysis was made. On re-eliciting history he revealed sea snake bite in the right

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hand two days back while handling fishing net . The sea-snake was around one metre long and the species was not identified. Bite site did not reveal any fang marks but for mild fading erythema. Sea snake antivenom was not given due to non-availability.He was started on hemodialysis. He was weaned off from ventilator the next day and became ambulant in two days. He required total of 7 hemodialysis sessions over 15 days and was discharged on 25th hospital day with serum creatinine of 2mg/dl. His creatinine normalised at the end of two months and maintaining normal creatinine till last followup.

3. Discussion

Sea snakes are large, paddle-tailed marine snakes that are immobile at land, due to absence of ventral scales [2]. They are found in the warm waters of Indo- Pacific Ocean. Sea-snakes are more venomous than land snakes. Bite is usually painless with no local signs. Bite is rarely fatal and cause of death is acute hyperkalemia , bulbar and respiratory muscle weakness.

Sea snake venoms contain both myotoxins, which are basic phospholipase A_2 and polypeptide neurotoxins (homologous to elapid neurotoxins). The venom causes respiratory failure, myonecrosis, myoglobinemia and acute renal failure. The neurotoxin also can cause paralysis by competitive binding to postsynaptic nicotinic acetylcholine receptors at the neuromuscular junction. Antivenom is indicated in patients with signs of envenomation [3 . Benefits may be seen up to 36 hours after the bite. The agent of choice is polyvalent sea snake antivenom (Commonwealth Serum Laboratories, Melbourne, Australia), a hyperimmune horse globulin prepared against the venoms of Enhydrina schistosa and N scutatus.

Alternatively, tiger snake antivenom can be substituted because of similarity of tiger snake and sea snake venoms.Unfortunately, land-snake antivenoms do not neutralize sea-snake venom.If sea snake antivenom is not available,dialysis should be considered as toxin is dialyzable (low molecular weight) and may be life saving in cases of severe hyperkalemia [4] .

Sitprija et al [5] reported two cases of sea-snake bite presented with myoglobinuria, renal failure and muscular weakness. Pathological study by biopsy showed renal tubular necrosis and myonecrosis. Both patients recovered completely after hemodialysis. It was postulated that muscular paralysis was the effect of hyperkalaemia and uraemia on the muscles sensitized by the toxic effects of sea-snake venom.Removal of potassium and uraemic factors by dialysis thus led to the rapid improvement of the muscular activity.

A study of 860 snakebite victims in Sri Lanka revealed one case of sea snake (Hydrophis spiralis) bite without signs of local envenomation [6].Patient had muscle tenderness, and neurotoxic signs but did not develop respiratory failure or AKI. Muscle paralysis without ptosis in our case is attributed to hyperkalemia secondary to AKI and rhabdomyolysis which improved dramatically with hemodialysis. Sea-snake bite should be thought of, when fisherman present with renal failure or paralysis. Early dialysis may be life saving in sea-snake bite with renal failure and hyperkalemia.

4. Conflict of Interest

None Declared

5. References

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