

Case Report

Severe rhabdomyolysis following a possible sea snake bite: A case report

Pavithera Packiyarajah*, Maheshwaran Umakanth, MSM Nusair


Teaching Hospital, Batticaloa, Sri Lanka.

Abstract

Rhabdomyolysis is a potentially life-threatening condition characterized by muscle necrosis and the release of muscle constituents into the circulation. Rhabdomyolysis has several causes, however many of them are complex or ambiguous. Rhabdomyolysis can lead to complications such as acute kidney injury (AKI), electrolyte abnormalities, compartment syndrome, and disseminated intravascular coagulation (DIC). The mainstay of management principle is the prevention of acute kidney injury by hydration, alkalization and diuresis, with correction of electrolyte imbalances and acidosis.

In our case, a 38-year-old man presented with generalized body swelling, body pain and dark urine after a possible sea snake bite when he visited Mannar Gulf. Blood workup showed significantly elevated creatinine phosphokinase (CPK). With proper hydration and alkaline diuresis, the patient did not develop AKI or electrolyte imbalances and he was discharged after 2 weeks with a significant reduction in CPK and clinical improvement.

Keywords: Rhabdomyolysis, Sea snake bite

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Introduction

Rhabdomyolysis is caused by traumatic, ischemic, pharmaceutical, toxic metabolites and infectious causes which influence the loss of integrity of the plasma membrane and cause toxic intracellular content to appear in the circulation [1]. It can result in severe electrolyte imbalance, acute kidney injury, and disseminated intravascular coagulation (DIC) [2]. Identifying and managing rhabdomyolysis promptly is crucial to prevent further deterioration.

Sea snake bite is one of the known causes of rhabdomyolysis. Sea snakes are extremely venomous and live in Sri Lanka's estuaries, lagoons, and coastal waterways [3]. Sea snakes have been encountered more commonly in northern coast including Mannar, although the bites are rarely reported [4].

In this report, we present a rare case of severe rhabdomyolysis after a possible sea snake bite at Mannar Gulf.

Case presentation

A 38-year-old previously healthy male patient, a heavy vehicle driver, presented with generalized body swelling and body pain with passing dark urine for 5 days duration.

Five days before the admission, he had visited the Gulf of Mannar. When he was in the shallow seawater he had felt a bite on his right fourth toe. Soon he had noticed a blister. The next day the patient developed bilateral lower limb and then upper limb swelling with generalized body pain. He had not developed any detectable bleeding or had no evidence of having ptosis or ophthalmoplegia. The pain at the bite site worsened with time. He came back to Trincomalee where he resides and he was admitted to the surgical unit of District General Hospital Trincomalee on 3rd day of bite. He has undergone incision and drainage of a right toe abscess under local anaesthesia. The patient was discharged on the second day of surgery with oral antibiotic ciprofloxacin. Following that, generalized body swelling and pain worsened and he noticed passing dark urine. He did not develop a noticeable reduction in his urine output. The patient was admitted to District General Hospital Trincomalee on the 6th day of the bite and transferred to Teaching Hospital Batticaloa on the next day. He did not have a history of fever. He had no history of trauma, burns, or exposure to illicit drugs, toxins or medications, except for local anaesthetic agent lignocaine and oral antibiotic ciprofloxacin. He had no history of heavy work or seizures.

On admission at Teaching Hospital Batticaloa, he was alert and orientated. He had generalized body swelling, which was more prominent on both upper limbs extending up to the shoulders (Figure 1A). The bilateral upper limbs were very tense and tender during palpation. His bilateral upper and lower limb distal oxygen saturations were above 98% on room air. He had evidence of an incision at the right 4th toe (Figure 1B). He did not have any rashes. His pulse rate was 100 beats per minute and his blood pressure was 140/ 90 mmHg.

The power of the upper limbs was 3 proximally and 4+ distally while the power of the lower limbs 4+ proximally and distally according to MRC muscle power grades. Except for muscle power reduction, the rest of the nervous system examination was normal. Cardiovascular, respiratory and abdominal examinations were unremarkable.



Figure 1: A, Significant swelling of the upper limb of the patient, up to the shoulders; B, Evidence of incision at the right fourth toe.

Laboratory findings of the patient are presented in Table 1. In summary, the patient had very high Creatinine phosphokinase, AST and myoglobin levels. However, his serum creatinine and electrolytes were normal throughout.

From the history, we excluded the intake of toxins, elicit drugs, excess alcohol, extreme physical exertion, prolonged immobilization, seizures, burn injury, electrical injury and trauma. Although he has been exposed to local anaesthesia lignocaine and antibiotic oral ciprofloxacin, he developed symptoms of rhabdomyolysis before the exposure to local anesthesia. The patient didn't develop fever and his CRP was normal throughout. Investigations revealed metabolic disorders and connective tissue diseases were unlikely. With the history of a feeling of a bite while in sea water at the Gulf of Mannar, the diagnosis of rhabdomyolysis possibly due to a sea snake was made and management was started.

Since the only available antivenom, Indian polyvalent antivenom does not cover the sea snakebites, no antivenom was given. During the ward stay his vital parameters, distal oxygen saturation and urine output were carefully monitored. His serum creatinine and electrolytes were monitored daily.

A multidisciplinary team meeting was arranged and expert opinions were obtained from the consultant nephrologist, consultant rheumatologist, consultant chemical pathologist and consultant surgeon.

Forced alkaline diuresis with intravenous 8.4% sodium bicarbonate (NaHCO₃) and 0.9% saline with intravenous furosemide infusion was started and a target urine output of 200- 300 ml per hour was maintained. Compartment syndrome of bilateral upper limbs was excluded.

CPK gradually started to drop after 5 days of management. Clinically, limb swelling and pain were reduced and CPK came down to 5438 U/L on the 13th day of admission. He was discharged after 13 days of in-ward care. The patient was reviewed after 1 week of discharge, generalised swelling and pain resolved completely and

CPK came down to 657 U/L. After 1 month the patient was reviewed again, he was clinically well and CPK came down to 430 U/L.

Table 1: Laboratory findings of the patient at the Teaching Hospital, Batticaloa

Investigation (normal range)	Day since admission											
	1	2	3	4	5	6	7	8	9	10	11	13
WBC ($4-10 \times 10^3/\mu\text{L}$)	12	11		16		23	24	18		15	15	9
HB (12-16g/dL)	16	15		15		12.8	12.2	11		11	11	12
PLT ($150-450 \times 10^3/\mu\text{L}$)	128	145		153		147	171	182		148	193	189
CPK (U/L)	78943	69340	76598	89392	115917	101479	85548	64764	47991	42474	34706	5438
AST (15-37U/L)	1857		1774	1983	2251	2484		1745	1715	1917	1594	523
ALT (12-78U/L)	309		303	337	370	421		396	303	397	374	
Creatinine (53-88 $\mu\text{mol/L}$)	74	61	50	59	72	55	126	62	68		76	75
Na ⁺ (136-145mmol/L)	127	127	129	133	127			138	138			
K ⁺ (3.5-5.1mmol/L)	4.9	4.2	3.9	3.6	3.1			3.2	3.2			
Alb (34-50g/L)	28		27	27		30		35		37		
LDH (140-280U/L)	1869			1897								
Ca ²⁺ (2.1-2.5mmol/L)	2.2			2.3			2.2			2.4		
PO ₄ ³⁻ (0.8-1.6mmol/L)	1.4				1.5					1.3		
ANA	negative											
Urine myoglobin	3+											
Serum myoglobin (25-72ng/mL)	315											
CRP (0-5mg/L)	5		7					7				
FBS (3.9-5.6mmol/L)		5.1										
Venous pH	7.38		7.4		7.42		7.4		7.4			
Serum cortisol (123-626 nmol/L)					362							
Magnesium (0.7-1mmol/L)					0.8							
ESR	46				42				38			

Discussion

This case emphasizes the possibility of severe rhabdomyolysis after the sea snake bite and elaborates on the approach, challenges and management. Rhabdomyolysis is a syndrome, characterised by muscle necrosis and the release of muscle constituents into the circulation and presents with muscle pain, weakness, swelling and dark urine [5].

There are many causes leading to rhabdomyolysis and often it is multifactorial or unclear. Causes can be divided into traumatic causes, non-traumatic exertional causes and non-traumatic non-exertional causes. Traumatic causes are direct trauma or direct muscle injuries such as crush syndrome, surgery, compartment syndrome, electrical injury and severe burn. Non-traumatic exertional causes are prolonged strenuous exercises, severe heat or humidity, seizures, metabolic myopathies, mitochondrial disorders, malignant hyperthermia and muscular dystrophy. Non-traumatic

non-exertional causes include drugs, alcohol, drugs of abuse, toxins, snake or insect venom, Infections, endocrinopathies and inflammatory myopathies.

Although rarely reported, fishermen who are fishing and people wading in the coastal sea of Sri Lanka are at risk of sea snake bites. Sea snakes are highly venomous but 80% fail to produce envenomation due to shallow fangs. Usually, bites do not produce pain or significant inflammation at the bite site.

Even though aetiology could differ, the ultimate pathogenesis is common and incorporates an increment in free ionized calcium within the cytoplasm [6]. It initiates a complex network of intracellular processes including the activation of phospholipase A2, prolonged contraction of muscles, mitochondrial dysfunction and generation of reactive oxygen species which initiate muscle damage and release of substances such as CPK, myoglobin, potassium, organic acids, other enzymes and

electrolytes into the circulation leading to clinical manifestation of rhabdomyolysis [7].

Complications of rhabdomyolysis are acute kidney injury and electrolyte disorders including hyperkalemia, hypocalcemia, hyperphosphatemia, compartment syndrome and DIC[2,8]. Acute kidney injury and hyperkalemia are the major factors that worsen the prognosis. The mechanisms of AKI in myoglobinuria include renal ischemia, the formation of myoglobin casts at the distal convoluted tubules, and the cytotoxic effects of iron on proximal convoluted tubules. Acidosis and hypovolemia make the situation worse [9].

The mainstay of management of rhabdomyolysis is fluid replacement to prevent AKI. According to the literature, intravascular volume expansion by 0.9% normal saline is essential to prevent myoglobinuric AKI by increasing renal blood flow, and consequently glomerular filtration rate and urination [9]. The objective is to achieve a minimum of 300ml /Hour urine excretion. Early recognition of AKI is essential to avoid excess fluid administration and pulmonary oedema [10].

The next management modality is urine alkalization by administration of sodium bicarbonate to prevent AKI. Cast formation is prevented by alkalization, which makes uric acid and myoglobin more soluble [9]. Additionally, it corrects metabolic acidosis and hyperkalemia. The objective is to maintain urine PH above 6.5 and serum PH in the range of 7.4-7.45. After hypovolemia is corrected and satisfactory hourly urine

output is achieved, diuresis is indicated and is carried out using mannitol or loop diuretics. Nephrotoxic drugs should be avoided.

Correction of electrolyte imbalances and acidosis is vital. Haemodialysis is indicated in severe resistant hyperkalaemia, a sudden increase in potassium level, persistent metabolic acidosis and ongoing AKI despite medical management [11].

Outcome

Although our patient had very high CPK from admission, his serum creatinine was persistently normal and he didn't develop AKI, electrolyte imbalance or acidosis throughout. He was discharged after 13 days of in-ward care with good clinical improvement and a significant reduction in CPK.

Conclusion

Sea snake bite-induced rhabdomyolysis is a rare but possible scenario mainly who work in the coastal sea of Sri Lanka. Early diagnosis and management are important in preventing complications of rhabdomyolysis. Proper hydration and alkaline diuresis can prevent or control the complications.

Consent: Informed consent has obtained from the patient. Patient has given his consent for his images and other clinical information to be reported.

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