Generalized seizure secondary to probable antivenom-induced anaphylaxis after treatment of sea-snake envenomation: a case report from Oman

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ABSTRACT

Background: Sea snake bites are uncommon but potentially life-threatening emergencies due to the neurotoxic and myotoxic effects of their venom. Antivenom remains the mainstay of treatment; however, it may rarely cause severe hypersensitivity reactions, including anaphylaxis.

Case Presentation: A 34-year-old previously healthy male presented to the emergency department in Oman 1.5 hours after a confirmed sea snake bite sustained while fishing. Initial examination revealed a fang mark on the right foot without significant local swelling. Shortly after initiation of intravenous antivenom, the patient developed vomiting, lethargy, hypotension, oxygen desaturation, and a generalized tonic-clonic seizure. Antivenom administration was immediately discontinued, and the patient was treated with intramuscular epinephrine, intravenous hydrocortisone, fluid resuscitation, and supportive care. The seizure resolved promptly, and he recovered without neurological sequelae. Laboratory investigations demonstrated mild leukocytosis and a transient rise in creatine kinase levels. Computed tomography of the brain was unremarkable. The temporal relationship to antivenom infusion, associated hemodynamic instability, and rapid response to epinephrine strongly suggested probable antivenom-induced anaphylaxis as the underlying mechanism.

Conclusion: This case highlights a rare presentation of probable antivenom-induced anaphylaxis manifesting as seizure following sea snake envenomation. Close monitoring during antivenom administration, prompt recognition of anaphylaxis, and immediate availability of resuscitative measures are essential to optimize patient safety.

Keywords: Anaphylaxis, antivenom, emergency medicine, hydrophiinae, pyrogenic reaction, sea snake, seizure.

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Background

Bites from sea snakes represent a serious medical emergency in coastal communities of the Indo-Pacific region. These marine reptiles possess highly potent venom that contains neurotoxins and myotoxins, capable of causing profound muscle damage, paralysis, and potentially fatal respiratory failure [1]. Unlike viper bites, sea snake envenomation often produces little local tissue injury, which can delay recognition and treatment [2].

Management relies on the timely administration of anti-snake venom (ASV), which is generally effective in reversing systemic toxicity. Nevertheless, ASV is not without risk; adverse events, including hypersensitivity reactions, anaphylaxis, and neurological complications, have been documented [3]. Seizures, in particular, are rarely reported following ASV infusion, and their occurrence poses both a diagnostic dilemma and a therapeutic challenge.

Here, we report a rare case of a healthy patient in Oman who sustained a sea snake bite and subsequently developed a seizure immediately after ASV administration. This case underscores the dual challenge of managing venom toxicity while remaining vigilant for potential iatrogenic complications of treatment.

Case Presentation

A 34-year-old male patient with no past medical history presented to the emergency department 1/2 hours after sustaining a sea snake bite on the right foot while fishing in coastal waters of Oman. The snake was not captured or photographed. However, the patient described a marine snake encountered during fishing, and a clear fang mark was observed on the medial aspect of the right sole on

presentation. The history of marine exposure, characteristic fang marks, and subsequent clinical manifestations were considered consistent with sea-snake envenomation. Species level could not be identified. He experienced mild pain at the bite site, followed by recurrent vomiting and pain with numbness in the left upper and lower limbs. On examination, his initial vital signs were normal. A fang mark was noted on the medial aspect of the right sole, with no associated swelling. The rest of the systemic examination was unremarkable. At this point, given the history of a suspected sea-snake bite and the development of systemic symptoms, including recurrent vomiting and limb pain with numbness, the patient was considered at risk of evolving systemic envenomation. In accordance with local clinical practice for suspected significant sea-snake envenomation, antivenom therapy was initiated without waiting for the development of advanced neurotoxicity, rhabdomyolysis, respiratory compromise, or renal dysfunction, as these manifestations may occur later and can progress rapidly. A total dose of 100 ml of Saudi polyvalent antivenom was diluted in 200 ml of normal saline and administered intravenously over a planned duration of 1 hour.

Approximately 5 minutes after initiation of antivenom infusion, the patient developed recurrent vomiting and lethargy, followed by hypotension with a blood pressure of 80/57 mmHg and oxygen desaturation to 80% on room air. Shortly thereafter, he experienced a generalized tonic-clonic seizure associated with urinary and bowel incontinence. No skin rash, urticaria, or angioedema was observed. Following the onset of symptoms, antivenom administration was immediately discontinued. The patient received two doses of epinephrine (0.5 mg each, IM), intravenous hydrocortisone 200 mg, and intravenous fluid resuscitation. Clinical improvement was observed shortly thereafter, with resolution of the seizure and normalization of vital signs within minutes. No benzodiazepines or other antiseizure medications were required, and the seizure resolved following treatment of the presumed anaphylactic reaction.

He then had a generalized tonic-clonic seizure associated with urinary and bowel incontinence. The ASV was stopped immediately. Two doses of epinephrine (zero point 5 mg each) and hydrocortisone 200 mg were administered, along with a normal saline bolus. The seizure resolved, and the patient's vital signs normalized. He was admitted for observation and received intravenous hydration at 63 ml/hour. No additional ASV was given during admission. He was discharged on the second hospital day with an excellent recovery.

Laboratory investigations are summarized in Table 1. Notably, the creatine kinase level increased from 104 to 244 units/l within 9 hours of the bite. He also had neutrophilic leukocytosis. The coagulation profile was normal apart from a reduced activated partial thromboplastin time. Electrocardiogram showed sinus bradycardia (Figure 2). Head computed tomography revealed no acute intracranial insult (Figure 1).

The patient remained hospitalized for approximately 48 hours following the bite. During this period, serial renal function testing remained within normal limits, and creatine kinase increased only mildly from 104 to 238 U/l without evidence of progressive myotoxicity or renal impairment. The patient remained clinically stable, with no muscle weakness, respiratory compromise, or other manifestations of evolving systemic toxicity. Based on the

Table 1. Laboratory investigations

TEST PARAMETERS	DAY 1	DAY 2	REFERENCE RANGE
WBC	18.6	15.4	2.2-10 $\times 10^3/\mu l$
Hemoglobin	11.9	11.6	11.5-15.5 g/dl
Platelets	310	287	140-400 $\times 10^3/\mu l$
Sodium in serum	141.06	138	136-145 mmol/l
Potassium in serum	4.11	4.27	3.5-5.1 mmol/l
Chloride in serum	110.05	104.6	97-107 mmol/l
Creatinine	84.36	83.80	62-106 umol/l
eGFR.MDRD	> 90	> 90	Normal > 90
ALT	15.99	-	0-41 U/l
ALP	135	-	40-129 U/l
PT	11.70	10.70	9-14 seconds
APTT	21.8	20.3	24-28 seconds
Fibrinogen	N/A	-	1.5-4.2 g/l
C-reactive protein	N/A	-	0-5 mg/l
Troponin T hs	5.83	-	0-14 pg/ml
Creatine kinase	104.60	238.5	30-200 U/l

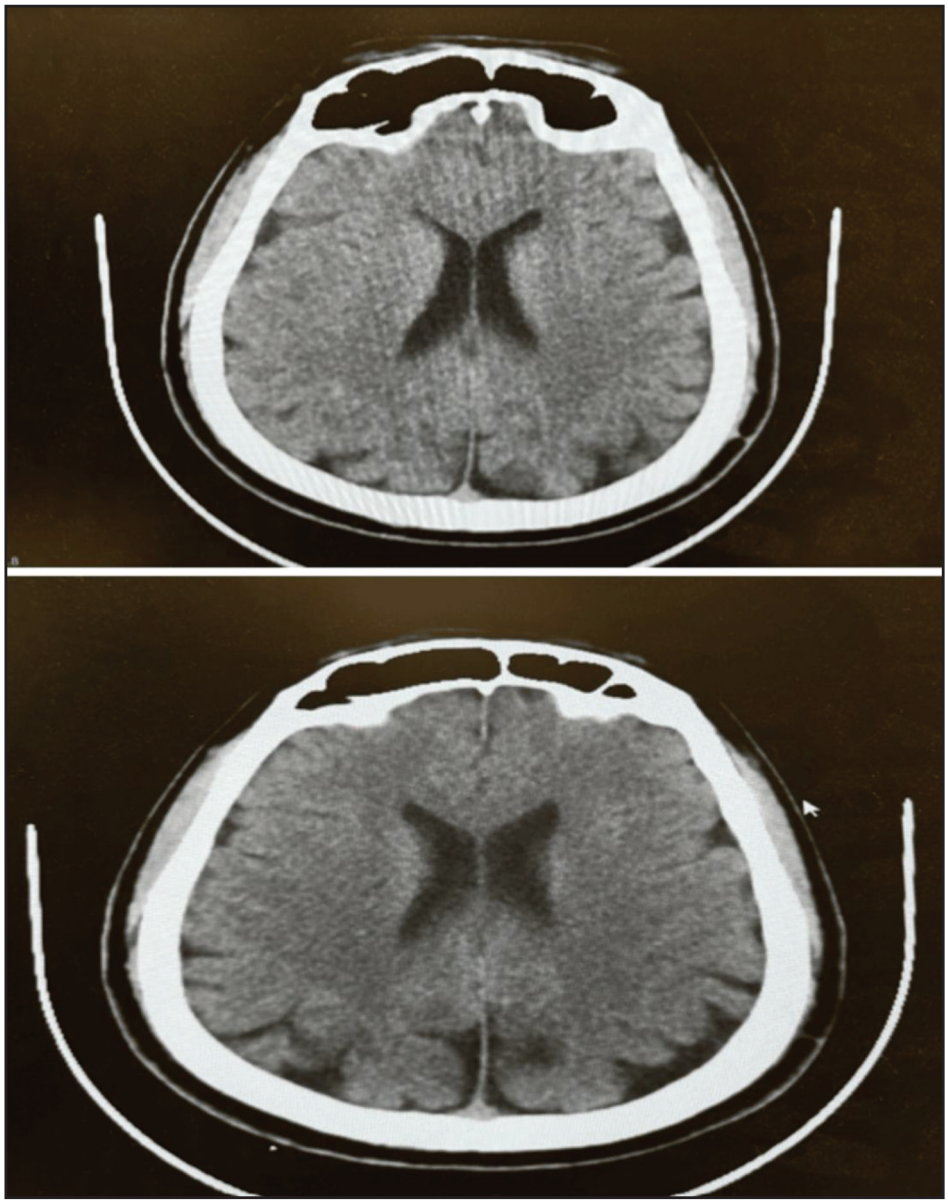

Figure 1. *Head computed tomography.*

absence of clinical deterioration, preserved renal function, and only a mild increase in creatine kinase, discharge on hospital day 2 was considered appropriate. We acknowledge that longer follow-up and additional investigations such as serial CK measurements beyond discharge and urine myoglobin testing would have strengthened the assessment of delayed myotoxicity, and we have added this as a limitation of the report.

Discussion

Snake envenoming is a major health problem in tropical regions. Viper bites often cause coagulopathy and tissue necrosis, while elapid bites, including those from sea snakes, primarily lead to systemic neurotoxic or myotoxic syndromes [1,2].

Sea snakes are widely distributed across the Indo-Pacific region, including the Arabian Gulf and Oman. Their bites often leave small fang marks without swelling but can cause severe systemic effects due to potent myotoxins and neurotoxins [4,5].

Convulsions have been described in severe envenoming, including a fatal *Laticauda colubrina* bite with convulsions and respiratory failure [6], as well as pediatric cases presenting with tonic spasms [7]. Proposed mechanisms include hypoxia from respiratory paralysis, metabolic derangements such as hyperkalemia secondary to rhabdomyolysis, and cerebrovascular complications [5].

Sea-snake envenomation is primarily associated with neurotoxicity and myotoxicity, and severe cases may result in respiratory failure, rhabdomyolysis, and, rarely, seizures. However, seizures in snakebite patients may also occur secondary to treatment-related complications, particularly severe antivenom reactions.

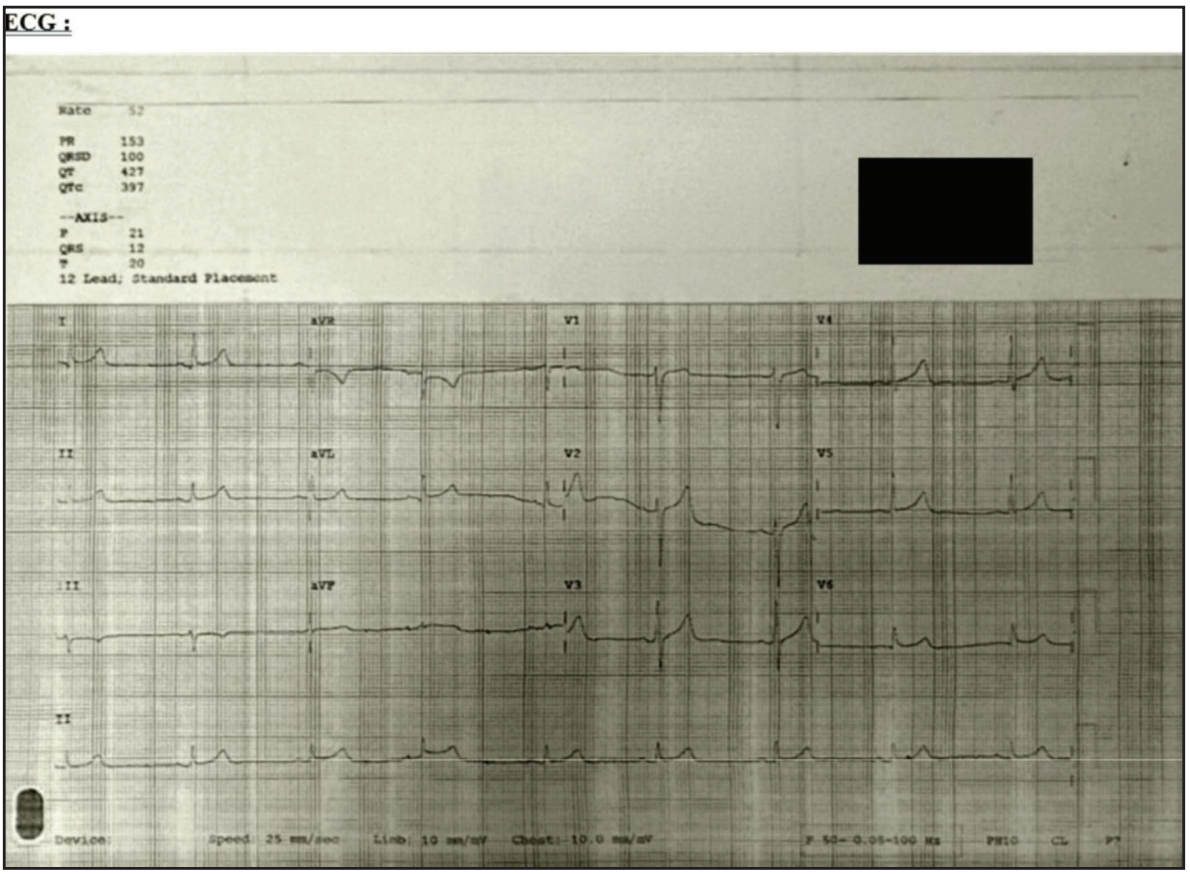

Figure 2. *ECG on the time of presentation.*

The distinguishing feature in this case was the timing of symptom onset. The patient developed vomiting, hypotension (80/57 mmHg), oxygen desaturation (80% on room air), and a generalized tonic-clonic seizure ~5 minutes after initiation of antivenom infusion. The abrupt onset immediately following antivenom administration, together with rapid improvement after epinephrine treatment and discontinuation of the infusion, strongly supports probable antivenom-induced anaphylaxis.

Several alternative causes of seizure were considered. Hypoxia and hypotension likely contributed to the event, as oxygen saturation decreased to 80% on room air and blood pressure fell to 80/57 mmHg immediately before the seizure. However, both abnormalities occurred in close temporal association with antivenom administration and are consistent with severe anaphylaxis. Serum sodium, potassium, renal function, and other routine biochemical parameters were within normal limits, making a major electrolyte disturbance less likely. There was no evidence of significant rhabdomyolysis, renal injury, or progressive neurotoxicity at the time of seizure onset. In addition, the seizure occurred only 5 minutes after antivenom infusion, which is earlier than would typically be expected for venom-induced neurological complications. Head computed tomography demonstrated no acute intracranial pathology. Although hypoxemia and hypotension likely contributed to seizure development, both occurred as manifestations of the anaphylactic reaction. Significant electrolyte abnormalities, renal dysfunction, and intracranial pathology were not identified. Furthermore, the absence of progressive neurotoxicity or severe myotoxicity at the time of seizure onset makes direct venom-induced neurological complications less likely.

Implications

This case highlights an important diagnostic challenge in snakebite management. Clinicians should recognize that neurological manifestations occurring shortly after antivenom administration may represent severe anaphylaxis rather than progression of envenomation. Continuous monitoring during antivenom infusion and immediate availability of epinephrine are essential to ensure prompt recognition and treatment of life-threatening adverse reactions.

Conclusion

This case describes a generalized tonic-clonic seizure occurring shortly after antivenom administration in a patient with presumed sea-snake envenomation. The close temporal relationship between antivenom infusion and the onset of hypotension, hypoxemia, and seizure, together with the rapid response to epinephrine and supportive treatment, suggests probable antivenom-induced

anaphylaxis with a secondary seizure rather than direct venom-related neurotoxicity. However, causality cannot be definitively established from a single case report, and alternative contributing factors cannot be completely excluded.

This case highlights the importance of considering severe antivenom reactions in the differential diagnosis of neurological deterioration occurring during or shortly after antivenom administration. Careful monitoring, prompt recognition of anaphylaxis, an2d immediate availability of epinephrine remain essential during antivenom infusion.

This case reinforces the importance of comprehensive observation protocols for snakebite management, integrating both rapid venom neutralization and proactive prevention of treatment-related complications.

What's new?

Reports a rare case of probable antivenom-induced anaphylaxis presenting with generalized tonic-clonic seizure after sea snake envenomation.

- Highlights that neurological deterioration occurring immediately after antivenom administration may represent anaphylaxis rather than progression of envenomation.
- Emphasizes the importance of close monitoring during antivenom infusion and prompt treatment with intramuscular epinephrine.

List of Abbreviations

ASV	**Anti-snake venom**
CK	**Creatine kinase**
CT	**Computed tomography**
ECG	**Electrocardiogram**
ED	**Emergency Department**
eGFR	**Estimated glomerular filtration rate**
IM	**Intramuscular**
IV	**Intravenous**
WBC	**White blood cell count**

Conflicts of interest

The authors declare that they have no conflict of interest regarding the publication of this case report.

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Summary of the case

1	Patient (gender, age)	34-year-old male
2	Final diagnosis	Probable antivenom-induced anaphylaxis presenting with generalized tonic-clonic seizure following treatment for presumed sea-snake envenomation
3	Symptoms	Sea snake bite, recurrent vomiting, limb pain and numbness, hypotension, hypoxemia, lethargy, generalized tonic-clonic seizure
4	Medications	Saudi polyvalent antivenom, intramuscular epinephrine (0.5 mg × 2), intravenous hydrocortisone, intravenous normal saline
5	Clinical procedure	Antivenom infusion discontinued immediately after onset of anaphylaxis; resuscitation with epinephrine, corticosteroids, intravenous fluids, observation, and supportive care
6	Specialty	Emergency Medicine