Case Report

Cardiac MRI-Proven Myocarditis Mimicking ST-Elevation Myocardial Infarction after a Cobra Bite

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Keywords

Animal bite · Cardiovascular disease · Poisoning/overdose

Abstract

Cardiac complications following snake envenomation occur infrequently. Myocarditis, as a consequence of the toxic effect of the snake venom, is rare, and only a few cases have been reported. We present a case of an 84-year-old man who was envenomed by a cobra. In addition to respiratory failure due to neurotoxicity of the venom, cardiac abnormalities including elevated cardiac markers and abnormal electrocardiogram were found. The findings on cardiac magnetic resonance imaging confirmed the diagnosis of acute myocarditis. The patient spontaneously recovered without any momentous events. To our knowledge, this is the first reported case of myocarditis associated with cobra envenomation. Physicians should be familiar with potential life-threatening cardiac effects of these toxins.

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Introduction

Cobra is one of the most well-known snakes due to its fatal neurotoxic envenomation. Severe neuromuscular junction destruction leads to profound muscular weakness and results in serious life-threatening conditions such as respiratory failure. Cobra bites are related to a high mortality rate throughout the world [1, 2]. Cardiac complications following cobra venom are rare and the pathogenesis is unclear. There have been reported cases of Takotsubo cardiomyopathy [3] and ventricular bigeminy [4] following patients who were envenomed by a cobra. We present a case of an 84-year-old man, who, apart from neurotoxicity, was diagnosed with acute myocarditis after being attacked by a cobra. To our knowledge, this is the first reported case of acute myocarditis after a cobra bite.

Case Presentation

An 84-year-old Asian man presented to the emergency room after being bitten by a cobra in his right thumb an hour prior to the presentation. The snake was the patient's domestic animal and identified by him as a cobra. Upon arrival, the patient felt dizzy. No medical history could be obtained other than the underlying diseases, which include hypertension and a remote history of a cerebrovascular accident. Physical examination revealed an elevated blood pressure at 190/80 mm Hg, a respiratory rate of 22 breaths per minute, and pulse oximetry of 70%. There was no muscular tone of both upper and lower extremities. Palpebral fissures were less than 0.5 cm. Cardiovascular examination was unremarkable. There were no abnormal auscultations of the heart and lungs. Other examinations revealed no abnormalities except for fang marks with superficial skin necrosis localized at the lateral side of his right thumb.

The diagnosis of neurotoxic cobra envenomation was made. A reconstituted purified equine immunoglobulin cobra antivenin (QSMI, Thai Red Cross; Bangkok, Thailand) was given intravenously at a dose equivalence to neutralize the activity against 6 mg of cobra venom. He was intubated and supported by positive ventilation. Nitroglycerine was given intravenously to rapidly decrease blood pressure. An electrocardiogram (ECG) was performed and showed 1-mm, concave, ST-segment elevation and PR depression on leads II, III, and aVF (Fig. 1). Serial high-sensitive cardiac troponin T assays were 12.8 and 332 ng/L (normal reference value is less than <14 ng/L) at 0 and 5 h, respectively. The echocardiogram showed normal left ventricular size and function with left ventricular ejection fraction of 55–60% without significant regional wall motion abnormalities. There was no pericardial effusion. With abnormal cardiac markers and ECG changes, acute myocarditis was considered. The differential diagnosis includes acute coronary syndrome and perimyocarditis. A serial ECG did not show significant ST-segment changes, T wave inversion or Q wave. A high-sensitivity troponin I assay revealed abnormal results of 2,244 and 4,157 pg/mL at 12 and 36 h after admission, respectively.

Within the first day at the hospital, the motor power in all extremities was improved. He was extubated. Superficial skin necrosis localized at the right thumb, which was complicated by necrotizing fasciitis and *Enterococci faecalis* infection, was treated by surgical debridement and ceftriaxone.

Cardiovascular magnetic resonance imaging (CMR) was performed on day 6 and revealed a left ventricular ejection fraction of 58% with a hyperintense signal on the T2-weighted image with hypokinesia at the lateral wall from basal to the mid parts, compatible with myocardial edema. No late gadolinium enhancement was detected. No pericardial effusion was observed (Fig. 2). These findings confirm the diagnosis of myocarditis according to the Lake Louise criteria [5].

The patient was discharged uneventfully after 8 days. He was asymptomatic and able to perform daily activities without chest pain or dyspnea. High-sensitivity troponin I was tentatively reduced to 56 pg/mL on the day of discharge (Table 1). At the 6-month follow-up after the index hospitalization, there was no clinical suggestion of residual heart dysfunction or repeat hospitalization.

Discussion and Conclusion

It is well documented that spider and scorpion envenomations are associated with myocarditis [6, 7]. This is due to the direct cardiotoxic effect of the venom and hyperadrenergic state. Snake-venom-induced myocarditis has been reported in viper and krait envenomations with only a total of 2 definite and 1 probable reported cases worldwide [8–10] (Table 2). To our knowledge, this is the first reported case of myocarditis associated with cobra envenomation. The mechanism of snake-venom-induced myocarditis remains unknown. Several in vivo and in vitro studies have suggested that cobra venom could affect myocytes molecularly and morphologically. Hypersensitivity reaction, inflammation, and apoptosis have been proposed as parts of the pathogenesis [11, 12].

The presented case is the first case of snake-venom-induced myocarditis documented by CMR. Traditionally, the definite diagnosis of myocarditis is done pathologically by direct visualization of inflammatory cells invading the cardiac tissue obtained by endomyocardial biopsy or autopsy. The pathologic diagnosis was found to have low sensitivity and specificity [13]. CMR has recently become the diagnostic modality commonly used to investigate cardiac abnormalities because it has a superb multifaceted ability to evaluate cardiac morphology, function, and tissue characterization. CMR can be performed prior to endomyocardial biopsy owing to the ability to support the diagnosis of myocarditis and much less noninvasiveness. When acute myocarditis is suspected, rather than using single clinical or imaging findings to confirm the diagnosis, physician should incorporate available information to make a diagnosis.

The International Consensus Group on CMR Diagnosis of Myocarditis has developed recommendations for clinical use of CMR including the Lake Louise criteria for patients who are suspected for myocarditis. The acute myocarditis is diagnosed if at least 2 of the following criteria are present: (1) regional or global myocardial signal intensity increase in T2-weighted images; (2) increased global myocardial early gadolinium enhancement ratio between myocardium and skeletal muscle in gadolinium-enhanced T1-weighted images; and (3) there is at least 1 focal lesion with nonischemic regional distribution in inversion recovery-prepared gadolinium-enhanced T1-weighted images [5]. The presented patient met the first 2 criteria. Interestingly, a few studies have shown that myocarditis is occasionally found as a focal process in CMR. An endomyocardial biopsy was not performed in this patient. However, with the

CMR findings as presented, biopsy of the right ventricle could have led to a false-negative result.

Even though ST-segment changes in this patient are compatible with myocarditis (diffuse, concave, with PR depression), one should consider acute coronary syndrome from thrombosis or coronary spasm, which has been reported in hematologic toxin snakebite [14–16]. Retrospectively considering the coronary angiogram at the time of admission to rule out coronary artery diseases, the temporal relation of the ST-segment and cardiac marker normalizations in this case suggest the diagnosis of myocarditis rather than acute coronary syndrome.

In conclusion, other than the known complications of snake venom, the cardiac complications should be carefully sorted out since the management and prognosis are distinctive. Myocarditis is a rare consequence that can occur after a snakebite. The diagnosis can be suggested by clinical, ECG, and cardiac markers. CMR should be commonly used as a primary tool for noninvasive evaluation of myocardial inflammation, which was conventionally done invasively by endomyocardial biopsy.

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Statement of Ethics

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Disclosure Statement

The authors declare no conflict of interest.

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Author Contributions

Palapun Waitayangkoon drafted the manuscript and designed the figures. Suchai Suteparuk, Pairoj Chattranukulchai, and Aekarach Ariyachaipanich aided in interpreting the results and worked on the manuscript revision and final approval. All authors discussed the results and commented on the manuscript.

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Fig. 1. An ECG at the time of admission showing normal sinus rhythm with 1-mm, concave, ST-segment elevation and PR-segment depression in leads II, III, and aVF.

Fig. 2. There is a hyperintense signal on the T2-weighted image at the lateral wall in the short axis view (a, b) and four-chamber view (c), compatible with myocardial edema. LV, left ventricle; RV, right ventricle.

Table 1. Levels of cardiac markers during hospitalization

	Time after onset of the bite									
	1 h	6 h	12 h	36 h	day 3	day 4	day 5	day 6	day 8	
<i>Cardiac markers</i>										
CK-MB, U/L		196		50						
hs-cTnT, ng/L	12.38	332								
hs-cTnI, pg/mL			2,244	4,157	1,685	692	382	195	97	

CK-MB, creatine kinase-muscle/brain; hs-cTnT, high-sensitivity troponin T; hs-cTnI, high-sensitivity troponin I. Reference ranges are as follows: CK-MB, 0–24 U/L; hs-cTnT, <14 ng/L; hs-cTnI, <34.2 pg/mL.

Table 2. Reported cases of myocarditis following cobra envenomation

Ref	Snake	Venom type	Presentations	Investigation	Follow up
Bhatt et al. [8]	Viper (unknown spp.)	Hemotoxin	Severe hemotoxic (unknown onset from the time of snakebite) No cardiac symptoms Unknown onset of elevated markers after snakebite	12-lead ECG, troponin, echocardiogram	n/a
Agarwal et al. [9]	Common krait (<i>Bungarus caeruleus</i>)	Neurotoxin	Severe neurotoxic 6 h after snakebite Cardiogenic pulmonary edema 16 h after snakebite Unknown onset of elevated markers after snakebite	12-lead ECG, CK-MB, troponin, echocardiogram	6 weeks
Pillai et al. [10]	Sind krait (<i>Bungarus sindanus</i>)	Neurotoxin	Severe neurotoxic 3 h after snakebite Unstable tachycardia 9 h after snakebite Cardiac markers were elevated 6 h after snakebite	12-lead ECG, CK-MB, troponin, echocardiogram	n/a
Presented case	Cobra (<i>Naja siamensis</i>)	Neurotoxin	Severe neurotoxic 1 h after snakebite No cardiac symptoms Cardiac markers were elevated 6 h after snakebite	12-lead ECG, CK-MB, troponin, echocardiogram, CMR	6 months

ECG, electrocardiogram; CK-MB, creatine kinase isoenzyme MB; CMR, cardiovascular magnetic resonance imaging.