Scorpion envenomation-induced acute thrombotic inferior myocardial infarction

Akrep sokmasına bağlı gelişen akut trombotik inferiyor duvar miyokart enfarktüsü

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Summary– The occurrence of a serious cardiac emergency following scorpion envenomation has rarely been reported and, when so, mostly presented as non-ST segment elevation myocardial infarction, cardiogenic shock, or myocarditis. Possible mechanisms include imbalance in blood pressure and coronary vasospasm caused by the combination of sympathetic excitation, scorpion venom-induced release of catecholamines, and the direct effect of the toxin on the myocardium. We report a case of a 55-year-old man who presented with acute inferior wall myocardial infarction (MI) within 2 h of being stung by a scorpion. Coronary angiogram revealed total thrombotic occlusion of the left circumflex artery, which was treated successfully with glycoprotein IIb/IIIa inhibitor, thrombus aspiration, antivenom serum, and supportive therapy. Therefore, life-threatening MI can complicate the clinical course during some types of scorpion envenomation and should be managed as an acute coronary syndrome.

Özet– Akrep sokması sonrasında ortaya çıkan ciddi kardiyak komplikasyonlar nadir olarak bildirilmekle birlikte, kendisini genellikle ST segment yükselmesiz miyokart enfarktüsü, kardiyojenik şok ya da miyokardit şeklinde gösterir. Muhtemel mekanizmalar arasında, akrep zehiri tarafından aktive edilen sempatik deşarj ve katekolamin salınımına bağlı gelişen kan basıncı ile koroner vazospazm dengesinin kaybolması ya da toksinin miyokart üzerine direkt etkisi yer alır. Bu yazıda, akrep sokmasından iki saat sonra akut alt duvar miyokart enfarktüsü gelişen 55 yaşında erkek hasta sunuldu. Yapılan koroner anjiyografide saptanan sol sirkumfleks arterdeki total trombotik tıkanıklık; glikoprotein IIb/IIIa inhibitörü, trombüs aspirasyonu, antitoksin serum ve destek terapisi ile başarılı bir şekilde tedavi edildi. Sonuç olarak, akrep sokması neticesinde ortaya çıkan hayatı tehdit edebilecek miyokart enfarktüsü klinik seyri daha da kötüleştirebilir ve bu sürecin yönetimi akut koroner sendromlardakine benzer şekilde yapılmalıdır.

Although more than one million cases of scorpion stings are reported annually worldwide, incidence is highest in rural regions of countries with hot climates.[1] The most common manifestations are local symptoms consisting of severe pain, erythema, and burning sensation at the site of the sting.[2] However, severe cardiac complications such as myocardial infarction (MI), acute pulmonary edema, cardiogenic shock, myocarditis, and even death occur infrequently.[3,4]

Abbreviations:

Cx	*Circumflex*
IgE	*Immunoglobulin E*
MI	*Myocardial infarction*
STEMI	*ST segment elevation myocardial infarction*

We present a case report of a scorpion sting complicated by acute inferior ST segment elevation myocardial infarction (STEMI) in an adult patient treated successfully with antivenom, thrombus aspiration, and glycoprotein IIb/IIIa inhibitor.

CASE REPORT

A 55-year-old man with no previous disease or cardiac risk factor except smoking was stung in the right

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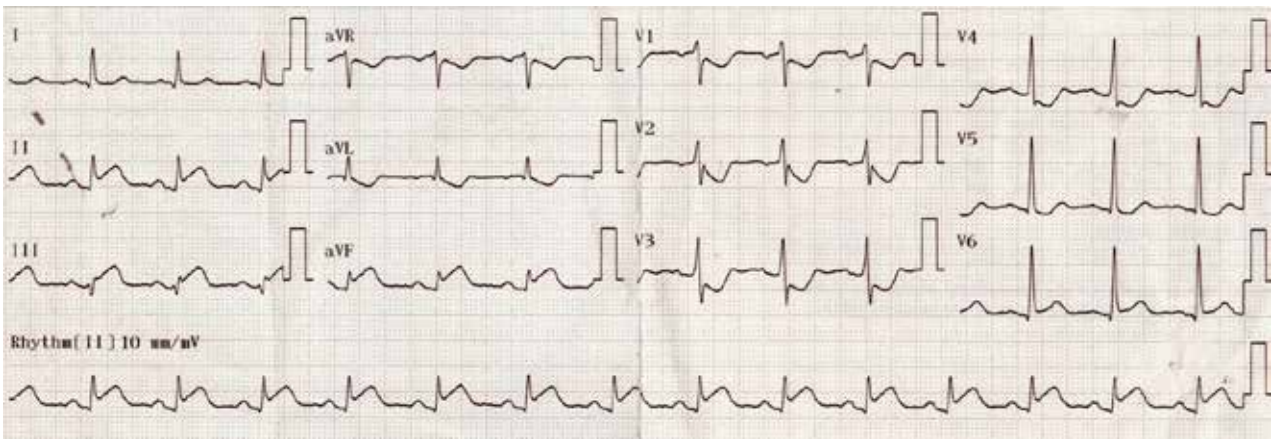

Figure 1. Electrocardiogram of a 55-year-old man showing ST segment elevation in leads DII, DIII, aVF, and V6, as well as reciprocal ST segment depressions in leads aVL, V1, V2, V3, and V4.

leg and right flank by a scorpion while sleeping in bed at night. A few minutes after the sting, he experienced severe local pain, erythema of his right leg and flank, as well as several episodes of emesis, vomiting, dizziness. He was referred to an emergency department where he received analgesic and primary medical care and was later brought to our cardiology emergency department 2 h later due to severe anginal chest pain.

Upon admission, the patient's heart rate was 92 bpm, blood pressure was 142/75 mmHg, and respiratory rate was 24 breaths/min, with mild respiratory distress. Electrocardiogram revealed sinus rhythm with ST segment elevation in leads DII, DIII, aVF, and V6; reciprocal ST segment depressions were found in leads aVL, V1, V2, V3, and V4 (Figure 1). Transthoracic echocardiography demonstrated hypokinesia

Table 1. Laboratory findings on admission and during follow-up

Variables	Admission	24 hours later	Discharge	Normal range
Glucose (mg/dl)	133	102	98	74–106
Potassium (mmol/L)	4.7	4.0	4.2	3.5–5.1
Sodium (mmol/L)	133	138	130	136–145
AST (U/L)	369	110	78	0–40
ALT (U/L)	286	58	41	0–41
Albumin (g/dl)	40.4	4.2	4.1	3.5–5.5
CRP (mg/dl)	10.6	1.8	1.2	3.5–5.2
Hemoglobin (g/dL)	15.1	14.8	14.9	11.9–15.4
WBC (10^3/uL)	22.2	18.2	13.8	3.9–8.8
Eosinophil (10^3/uL)	00.009	0.1	0.12	0.03–0.4
Lymphocyte (10^3/uL)	20.46	3.29	3.57	0.8–3
Neutrophil (10^3/uL)	18.9	14.3	8.95	1.8–7.4
NLR	70.7	4.3	2.5	–
Troponin I (ng/mL)	00.07	50	29	0.02–0.06
CK-MB (ng/mL)	60.81	300	2.75	0–5
Pro-BNP (pg/mL)	–	140	–	0–100
Total IgE (IU/mL)	–	86	–	0–100

AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; CK-MB: Creatine kinase-MB; CRP: C-reactive protein; IgE: Immunoglobulin E; NLR: Neutrophil-to-lymphocyte ratio; pro-BNP: Pro-brain natriuretic peptide; WBC: White blood cell count.

of the inferior and posterior walls, with mild mitral regurgitation and left ventricular ejection fraction of 42%. Results of initial laboratory tests including serum cardiac biomarker levels (creatine kinase-MB and troponin I), creatinine, glucose, and electrolytes were in the normal range. However, marked increase was observed in serum liver tests, C-reactive protein, leukocyte count, and neutrophil-to-lymphocyte ratio (Table 1).

Immediately, scorpion antivenom was intravenously administered, and the patient was admitted to the catheterization laboratory. Coronary angiogram revealed total occlusion of the left circumflex (Cx) artery, whereas other coronary arteries were normal (Figure 2). Intracoronary nitroglycerine was administered to exclude coronary vasospasm. Subsequently, the completely occluded segment was easily crossed by a floppy coronary guidewire, and a lesion with high thrombus burden was observed. After thrombus aspiration, tirofiban, an intracoronary glycoprotein IIb/IIIa inhibitor, was administered and maintained as an intravenous infusion for 24 hours. Additionally, he received dual antiplatelet therapy (acetyl salicylic acid and clopidogrel), low molecular weight heparin, angiotensin-converting enzyme inhibitor (ramipril), and a beta-blocker (metoprolol).

On the second day, he underwent a second coronary angiography, which revealed total recanalization of the lesion in the Cx artery with no additional coronary stenosis (Figure 3). Moreover, electrocardiogram revealed normal sinus rhythm with the resolution of ST segment elevation and Q waves in leads DII, DIII, and aVF (Figure 4). An increase was observed in serum cardiac biomarkers 24 hours after the index event (creatine kinase-MB: 300 ng/ml; troponin I: 50 ng/ml). At that time, his symptoms were resolved, and his clinical condition improved considerably.

On the fifth day, the patient was totally symptom free and discharged from the hospital. Echocardiography revealed mild hypokinesia of the inferior and posterior walls with preserved ejection fraction.

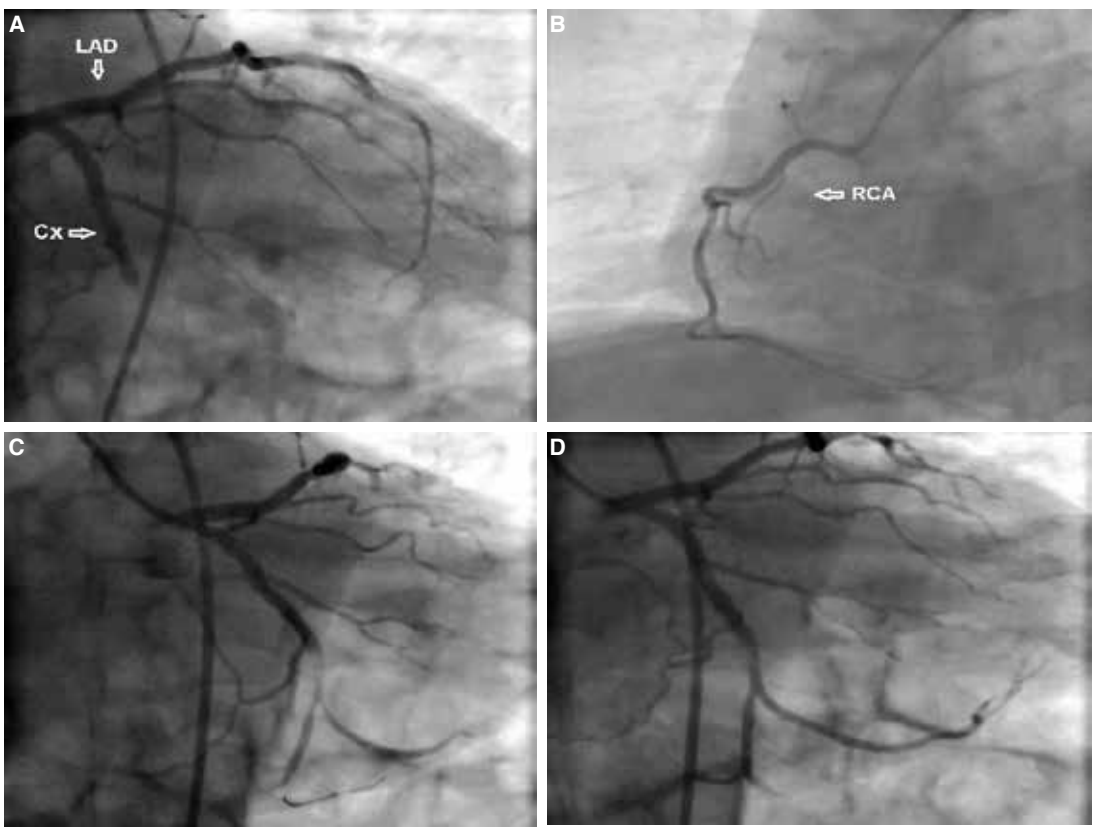

Figure 2. **(A)** Coronary angiogram revealed total occlusion of the left circumflex (Cx) artery, whereas left anterior descending (LAD) artery and **(B)** right coronary artery (RCA) were normal. **(C)** The completely occluded segment was easily crossed by a floppy coronary guidewire. **(D)** Cx artery after thrombus aspiration.

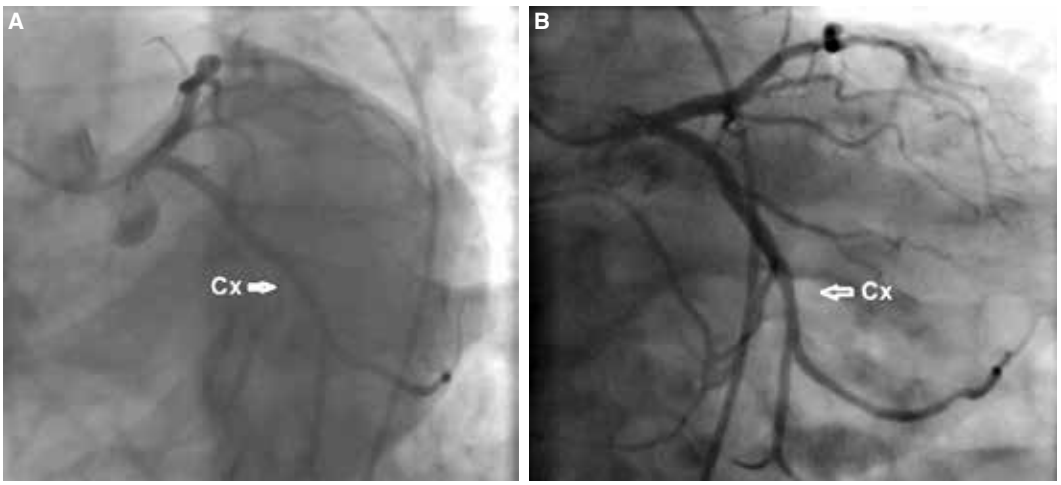

Figure 3. **(A, B)** Control coronary angiography revealed total recanalization of the lesion in the left circumflex (Cx) artery, with no additional coronary stenosis.

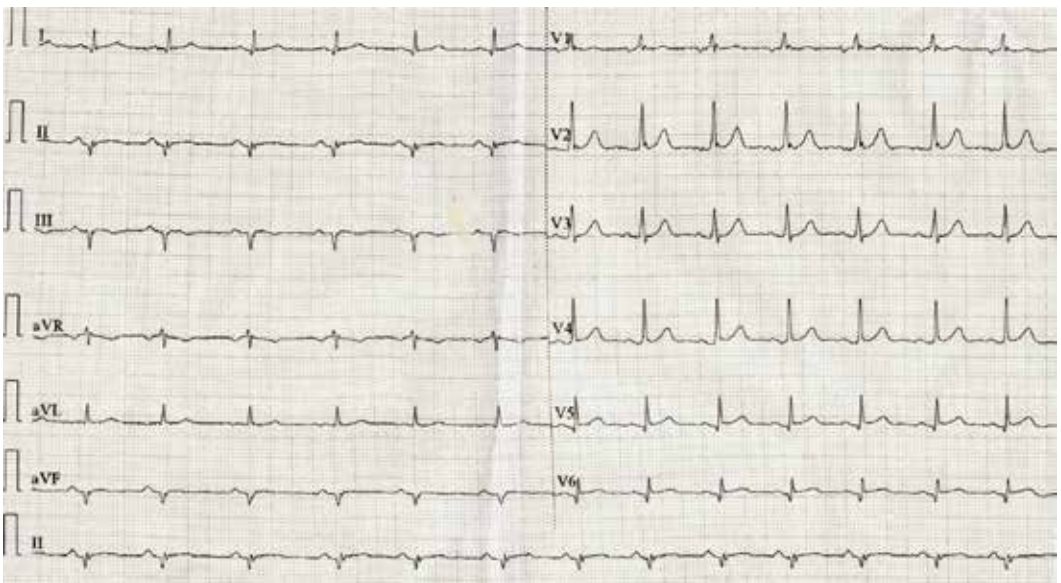

Figure 4. On the second day, electrocardiogram showing normal sinus rhythm with the resolution of ST segment elevation and Q waves in leads DII, DIII, and aVF.

DISCUSSION

We report a case of scorpion envenomation complicated by acute inferior STEMI as a result of thrombotic coronary total occlusion, treated successfully with thrombus aspiration, glycoprotein IIb/IIIa inhibitor, antivenom serum, and supportive therapy. To the best of our knowledge, a patient with thrombotic coronary occlusion after scorpion sting has not previously been reported. Scorpion stings are one of the most important health care problems in the rural areas of tropical and subtropical regions of the world.[5] The venom, a water-soluble antigenic complex, acts as a mixture of neurotoxin, cardiotoxin, nephrotoxin, hemolysins, phosphodiesterases, and histamine.[6] Cardiac consequences such as myocardial injury, pulmonary edema, and cardiogenic shock occur as a result of sympathetic excitation and the release of catecholamine in plasma. In the initial period, there is an increase in blood pressure and cardiac output, followed by diminished left ventricular function and hypotension. Possible mechanisms of myocardial

ischemia include imbalance in blood pressure and coronary vasospasm caused by the combination of sympathetic excitation and release of catecholamines induced by scorpion venom, and the direct effect of the toxin on the myocardium.

Furthermore, in cases of MI after scorpion sting, vasoactive, inflammatory, and thrombogenic substances–such as histamine, serotonin, bradykinin, leukotrienes, and thromboxane–are released. These substances act on coronary vasculature and may induce coronary artery vasospasm and platelet aggregation, as well as facilitate thrombus formation.[7] While there are few cases of acute MI due to scorpion bite reported in the literature, all of the patients had normal coronary angiogram.[8] There is only one report in the literature that shows coronary stenosis in a patient with acute anterior MI due to scorpion envenomation.[9] However, in that report, there was an established significant coronary artery disease, which became evident after severe scorpion envenomation. In contrast, in the report described here, we observed the total recanalization of the Cx artery, with no additional residual stenosis after treatment with thrombus aspiration, glycoprotein IIb/IIIa inhibitor, antivenom serum, and supportive therapy.

Another possible mechanism of this clinical scenario may be the immunoglobulin E (IgE)-mediated immediate hypersensitivity to the scorpion venom in susceptible persons, as described by Kounis.[10] However, previous exposure and IgE-mediated hypersensitivity is very rare with scorpion envenomation when compared with other insect stings.[11,12] In the present report, eosinophil count, eosinophil ratio, and total value of IgE were in the normal range. Therefore, the diagnosis of Kounis syndrome as the result of acute MI due to scorpion sting is unlikely in our case.

Conclusion

This is the first report in the literature of acute thrombotic inferior STEMI followed by a scorpion sting which dramatically responded to the administration of antivenom serum, glycoprotein IIb/IIIa inhibitor, and the removal of thrombus via thrombus aspiration. Therefore, life-threatening MI can complicate the clinical course during some types of scorpion envenomation and should be managed as an acute coronary syndrome.

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Keywords: Acute myocardial infarction; glycoprotein IIb/IIIa inhibitor; scorpion sting; thrombus aspiration.

Anahtar sözcükler: Akut miyokart enfarktüsü; glikoprotein IIb/IIIa inhibitörü; akrep sokması; trombüs aspirasyonu.