Kounis Syndrome: A Rare Case of Allergic Angina Secondary to Loxoscelism

Abstract

Kounis syndrome or allergic angina is characterized by a sudden transient or permanent myocardial dysfunction caused by inflammatory mediators such as histamine, leukotrienes, platelet-activating factor, neutral proteases, and a number of cytokines and chemokines. Herein, we discuss a case of Kounis syndrome, which was brought on by loxoscelism.

Keywords: *Allergic angina, Kounis syndrome, loxoscelism*

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Introduction

Kounis and Zavras first proposed the concept of "allergic angina" in 1991.[1] Inflammatory mediators such as histamine, leukotrienes, platelet-activating factor, neutral proteases, and a number of cytokines and chemokines of an allergic reaction, have been proposed to be the cause of allergic angina, also referred to as Kounis syndrome. These mediators have the capacity to either cause an immediate coronary thrombosis or coronary artery spasm, both of which may result in early myocardial damage.[1-3]

Because Kounis syndrome may be fatal, the disease must be ruled out in any patient with systemic allergic reactions having the signs of acute coronary syndrome (ACS). Often cases of Kounis syndrome go unidentified or undiagnosed, and in the absence of early intervention, many a times the patients succumb to their illness.[4,5] Herein, we discuss a case of Kounis syndrome, which was brought on by a loxoscelism.

Case Report

Three hours after being bitten by a spider on the foot, a relatively healthy 43-year-old farmer, from the rural area of the foothills of Aravalli, Jodhpur district, who had no substantial cardiac history, presented to the medical emergency department. She complained of throat and chest discomfort and respiratory distress. On cutaneous examination, the patient also had widespread urticaria. She had no prior history of anaphylaxis or spider bites. She denied having any food or medication sensitivities. Her initial vital signs indicated severe hypotension (60/40 mmHg). An electrocardiogram (ECG) measurement revealed V1–V6 ST-segment coving and elevation [Figure 1]. Troponin I level was under baseline (0.03 nanogram/mL). Two-dimensional Doppler sonography revealed normal wall motion and retained left ventricular function.

Although the patient did not bring the spider with her, the culprit spider was identified as the recluse spider by the patient based on the description of the spider and on showing her the representative images of the spider. The patient received intravenous hydrocortisone and intravenous pheniramine maleate along with intravenous fluids for anaphylaxis. Five days after the initiation of treatment, there was an eschar formation over the site of the dermonecrosis caused by the spider bite [Figure 2]. Her ST wave alterations on ECG immediately returned to normal as a result, and her blood pressure also increased. Due to concerns regarding worsening myocardial ischemia, higher myocardial oxygen demand, and coronary vasoconstriction, she did not get an epinephrine injection.

She was admitted to the hospital so that her hemodynamic condition could be monitored and any additional episodes

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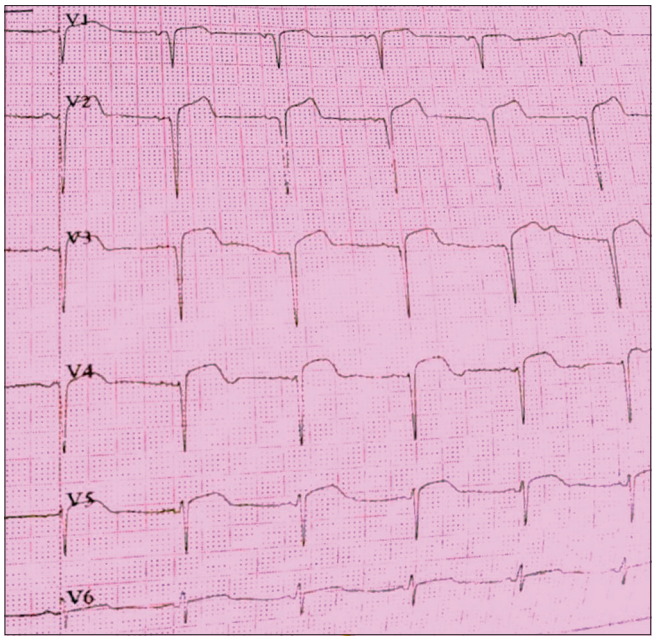

Figure 1: An ECG measurement revealing V1–V6 ST-segment coving and elevation

could be treated. Complete blood count (which revealed no eosinophilia), basic metabolic panel, and serial troponin I level (0.01 nanogram/mL) stayed unremarkable. Serial ECGs revealed a normal sinus rhythm without any noticeable ST-T wave alterations. Formal transthoracic echocardiography showed no anomalies in regional wall motion and a normal ejection fraction. Her brief ischemia ECG alterations led to the diagnosis of type 1 Kounis syndrome triggered by loxoscelism, which was treated successfully.

Discussion

Loxoscelism is caused by the bite of a recluse spider (genus *Loxosceles*). As the skin around the bite ages and dies, the region turns drab and develops necrosis. Phospholipase D, inhibitor cystine knot peptides, sphingomyelinase, and astacin-like metalloproteases are among the enzymes found abundantly in recluse spider venom.[2] Phospholipase D is specific to *Loxosceles* and has clinical significance. This enzyme causes damage in the human body by triggering complement activation, neutrophil chemotaxis, and keratinocyte death. Apart from causing cutaneous necrosis, the enzyme can also trigger catastrophic events like hemolysis, thrombocytopenia, and renal failure. Anaphylaxis is a very rare complication of loxoscelism.[6,7]

Kounis syndrome can be brought on by a variety of factors, including drugs, intravenous contrast, food allergies, and environmental exposures. It can afflict people of any age. Presenting symptoms might range widely, from moderate ones like flushing, vomiting, and chest constriction to serious ones like hemodynamic instability and sudden cardiac arrest.[3,8]

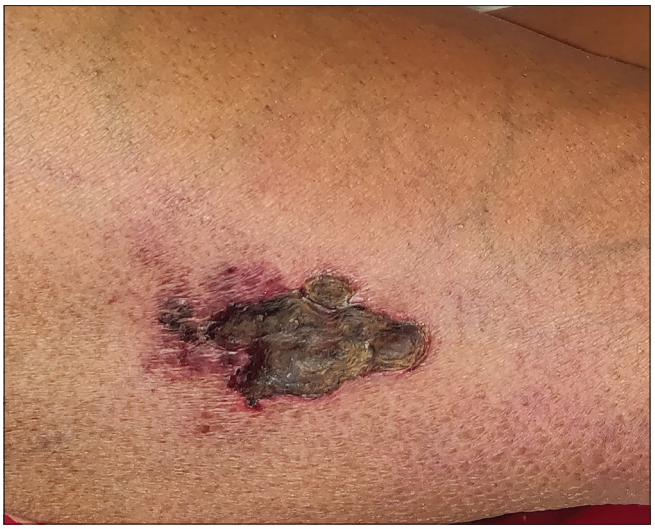

Figure 2: Dermonecrosis over the site of the cutaneous loxoscelism on day 5

Kounis syndrome has so far been described in three different forms. Patients with the type 1 variant have normal coronary vessels, and the sudden release of inflammatory mediators results in coronary vasospasm with or without an increase in cardiac enzyme levels. Patients with the type 2 variant have mediator-induced vasospasm together with abnormalities in vasculature. Coronary artery abnormalities include plaque erosion or rupture, which leads to an abrupt myocardial infarction. Patients with coronary artery stent thrombosis brought on by mast cell and eosinophil activation commonly exhibit the type 3 variation.[2]

Treatment of allergy while preserving appropriate cardiac perfusion are essential components of effective Kounis syndrome therapy.[2,5] While keeping in mind that some drugs may have an impact on coronary blood flow, the primary goal of treatment should be to prevent life-threatening anaphylaxis. Epinephrine is the preferred medication for anaphylaxis; however, it can increase coronary vasospasm and ischemia. Beta-blockers, on the other hand, can exacerbate coronary spasms because of the unchecked alpha-adrenergic receptors activation.

Kounis asserts that antihistamines and corticosteroids alone are sufficient for treating type 1 variation.[3] After the acute episode has passed, a complete cardiological examination that includes a 12-lead ECG, echocardiography, and the modification of cardiac risk factors is necessary. The ACS protocol should be used as the first line of treatment for type 2 variation, followed by corticosteroids and antihistamines. The type 3 variant demands immediate aspiration and histologic analysis of the stent thrombus in addition to the ACS regimen. Antihistamines and corticosteroids may be helpful for patients who experience allergy symptoms after stent insertion. Desensitization and stent removal should be taken into consideration if allergy symptoms continued despite treatment.

Conclusions

Although Kounis syndrome is not a rare disorder, there are few references to it in the literature. In clinical practice, it is crucial for doctors to recognize Kounis syndrome since it necessitates immediate treatment decisions. As dermatologists, we must always be on the lookout to identify loxoscelism as a potential trigger for Kounis syndrome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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