



RESEARCH ARTICLE

KOUNIS SYNDROME FOLLOWING A SINGLE BEE STING: A RARE CASE REPORT

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ABSTRACT

The syndrome of allergic angina was first reported nearly six decades ago. Since then, several agents have been known to cause this condition out of which Hymenoptera sting is still uncommon and not much reported. We report a case of a 23-year-old male who developed chest pain, vomiting, dizziness, and transient loss of consciousness following a single bee sting. Subsequent Electrocardiography showed ST elevation in leads II, III and AvF with elevated Troponin T (0.168→0.315 ng/mL). No history of pre-existing coronary artery disease and the absence of any risk factors led to the unusual diagnosis of Type-I Kounis syndrome and the patient improved significantly with antihistamines and vasopressor support. Given the dearth of standard guidelines to treat Kounis syndrome and the dynamic nature of its presentations which may lead to delay in diagnosis and treatment, the authors hereby attempt to address the gap in this regard and hence report this case.

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INTRODUCTION

The concept of an allergic reaction presenting as an angina was first described in 1991 with the possible mechanism being the role of histamine in provoking a coronary arterial spasm⁽¹⁾. Kounis syndrome is defined as the existence of acute coronary syndromes including coronary spasm, in the setting of allergic or hypersensitivity and anaphylactic insults.⁽²⁾ KS can be triggered by various allergens such as drugs, animal stings or venom, food allergens, various diseases and environmental factors. Among these, Hymenoptera stings are uncommon and underreported. Timely and accurate diagnosis and management of coronary events are necessary to save patient's life and avoid complications. Varied and rare presentations of such common conditions should also be kept in mind while delivering patient care. Addressing the existing literature gap in this regard is what the authors attempt by reporting this case.

CASE PRESENTATION

We report a case of a male patient in his 20's who presented to the emergency department with the complaints of chest pain, dizziness and vomiting following a bee sting on his forehead along with pain and itching over the sting site. On examination, he was conscious but hypotensive (BP 70/50 mmHg) and tachycardic (136/min). Local

respiratory and systemic examinations were unremarkable. There was no history of any preexisting heart disease or of any similar complaints in the past. The ECG on admission showed ST elevation in leads II, III and AvF and ST depression in leads V4-V6. A repeat ECG four hours later demonstrated hyperacute T waves in V2-V5, suggestive of evolving inferior wall myocardial infarction. Troponin T was initially 0.168 ng/mL, which increased to 0.315 ng/mL. Routine hematological and biochemical parameters were normal. Workup for other hypercoagulable was done which was normal. The patient received injection pheniramine and dexamethasone intravenously and tablet levocetirizine (5 mg) for his symptoms. Vasopressor support by nor-adrenaline (1 ampule in 50cc of normal saline) was initiated for hypotension; adrenaline was avoided due to concern for coronary vasospasm. He improved clinically within 24 hours and was discharged with advice for allergen avoidance and cardiology follow-up.

DISCUSSION

Effect of Hymenoptera stings (bee, wasps, ants) have been known since age immemorial. These include intense itching, pain and possible systemic complications like hypotension and anaphylactic shock in case of sensitivity to sting venom⁽³⁾. Bee venom contains complex biological compounds namely histamine, phospholipase A2, melittin, leukotrienes and thromboxane among others which have been implicated in the development of acute coronary syndrome

⁽⁴⁾Kounis syndrome is caused by coronary vasospasm or non-vasospastic response (plaque rupture) during an allergic insult which may include other arterial circulations and ischemia/infarction of the vital organs supplied⁽⁵⁾. The complex interplay of mediators particularly histamine and leukotrienes can cause vasospastic angina in such situations as noted by Braunwald et al⁽⁶⁾. There are three known variants of Kounis syndrome. Type I is defined as angina following an acute allergic reaction in patients with no coronary disease in whom the culprit remains coronary vasospasm which may progress to ischemia; type II includes known cases of atheromatous disease, either previously quiescent or symptomatic, in whom allergic insult might cause plaque erosion or rupture. A new and more recent variant includes type-III Kounis syndrome seen in patients of pre-existing coronary artery disease treated with drug eluting stents and then presenting with stent thrombosis.

The possible pathophysiology of KS stems from mast cell degranulation during which in turn results in the release of inflammatory mediators that include histamine, cytokines, proteolytic enzymes and hematopoietic factors⁽⁷⁾. There is now an increasing degree of evidence suggesting the role of these chemicals particularly histamine in the progression and erosion of atheromatous plaque or in inducing coronary vasospasm.⁽⁸⁾ Studies have also shown the presence of mast cells in the coronary intima of patients who died due to acute myocardial infarction thus suggesting a common pathway between allergic and non-allergic coronary insult⁽⁷⁾. Our patient had focal allergic reaction which was followed by signs and symptoms of acute myocardial infarction as confirmed by elevated cardiac enzymes and ECG changes. Given the history and the age of the patient aligning with no evidence of pre-existing coronary artery disease the diagnosis of Type-I kounis syndrome has been postulated. Multiple triggers have been implicated in the development of kounis syndrome which include drugs like penicillin, diseases (angioedema, asthma etc.) and environmental exposures like food allergies, animal bites and venom and even contrast exposure. Owing to its varied presentations which may include dizziness, loss of consciousness, hypotension, retrosternal pain and even cardiopulmonary collapse, a set of standard guidelines enumerating the basic classification and treatment becomes necessary for ethical and quality medical practice. Furthermore, the exact pathophysiology behind what goes inside a patient during allergy induced angina needs to be studied in order to delineate possible triggers to facilitate timely suspicion and referral, if necessary, by primary care physicians. Previous nationwide studies done on western population have estimated the prevalence of kounis syndrome to be nearly 1.1% among all the hospitalised cases of allergic reactions⁽⁹⁾. Such studies need to be carried out in India given the huge and widely distributed population majority of which is rural who may also have a high risk of exposure to bee stings. There have been reports of similar instances by Puttegowda et al, Bharadwaj et al, Sunder et al. Santosh et al reported wolf Parkinson white syndrome induced by bee sting. Other reported cases of KS include triggers like contrast dye, cobra venom and drug exposure.

CONCLUSION

Increased evidence in literature is necessary in order to treat such unusual but preventable manifestations like that of kounis syndrome. High degree of suspicion and increased awareness towards rare differentials during treatment like KS, which are frequently misdiagnosed or confused is only possible through repeated reporting and discussions on such topics through. Given the varied presentations and triggers with a possibly high prevalence it is important to achieve an accurate diagnosis so as to both conduct further research and formulate possible guidelines and to arrive at the same. Addressing this gap the authors hereby attempt to do their part and report this case. Classic angina pectoris can have a multimodality approach involving interplay of various mediators like histamines, platelet activating factors and a variety of other biological chemokines released during allergic inflammation^(10,11).

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