CASE REPORT - OPEN ACCESS

International Journal Of Medical And Clinical Case Reports

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International Journal Of Medical And Clinical Case Reports

Kounis Syndrome-Anaphylactic Insult on Heart

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ARTICLE INFO

Article history:
Received 02 January 2021
Accepted 28 January 2021
Available online 05 February 2021

1. Introduction

Kounis syndrome is a rare case introduced in the year of 1991 by Kounis and Zavras. Allergic reaction triggering ST segment elevation myocardial infarction is referred as Kounis syndrome. The pathophysiology is by release of histamine due to anaphylactic reaction, which induces coronary artery spasm and atheromatous plague rupture. This disease is frequently misdiagnosed as myocardial infarction.

Intense coronary disorder going with pole cell actuation from unfavorably susceptible, extreme touchiness, or anaphylactoid responses was first portrayed by Kounis and Zavras in 1991 and has been alluded to as “hypersensitive angina” or “hypersensitive myocardial localized necrosis” [1,2]. The system of Kounis condition (KS) includes arrival of provocative cytokines through pole cell enactment, which prompts coronary course vasospasm or potentially atheromatous plaque disintegration or crack [2]. KS has been depicted with various conditions, including an assortment of ecological openings and medications [3]. All the more as of late, variation introductions of unfavorably susceptible angina have been depicted. For this situation arrangement, we depict three variable case introductions of anaphylactic ST rises, which incorporate ST-rise myocardial dead tissue (MI) within the sight of fundamental coronary corridor sickness and ST-height MI without hidden coronary conduit illness (unadulterated vasospasm). We additionally quickly audit the current writing on KSVV [4-8].

2. Case Representation

Patients Details & Presenting History

Patient is a 37 year old female presented to our casualty with history of bee sting around 7 pm at her residence following which she developed complaints of puffiness of face , swelling of tongue , breathlessness , chest pain and palpitations .Patient has no known other allergies , medical comorbidities and significant past history of any cardiovascular disease There was no significant history cardiac disease occurring at a early age in the family.

3. Examination

Patient was conscious: Tachypnea +Afebrile No pallor/icterus/cyanosis/clubbing/lymphadenopathy Facial puffiness + Swollen lips + Swollen tongue + Conjunctival suffusion+ Uvula was edematous. BP- 100/ 50 mm Hg PR– 100/ min Spo2- 94 @ room air RR– 31/ min Temperature -98.6 degrees CVS – S1 S2 heard tachycardia+, RS- normal vesicular breath sounds heard polyphonic biphasic wheeze heard all over the chest; P/A– bowel sounds + soft ; CNS - No focal neurological defect.

4. Course in Hospital

Injection adrenaline 0.5 mg was given via intramuscular route (1:1000 dilution). Injection hydrocortisone was given intravenously (100 mg). Injection CPM 2cc was given intramuscularly, Nebulization was initiated .Fluids were started on flow (0.9% NaCl).

Cardiac enzymes were within normal limits. Other routine investigations like CBC, RFT, LFT, Serum electrolytes, FLP and chest x ray were normal. There was no eosinophilia in peripheral blood ECHO at acute presentation was normal.

Initially patient was diagnosed to have a acute allergic reaction to bee sting and was promptly managed. As the treatment was administered all of the symptoms like breathlessness, facial puffiness, swollen tongue and palpitations settled with time except for the chest pain which remained the same and worsened on mild exertion - ECG was obtained and it showed ST segment elevation in all leads - Immediately a diagnosis of acute coronary syndrome was made. Patient was put in continuous cardiac monitoring , propped up position and complete bed rest Sublingual nitroglycerine 5 mg was given TDS. Intravenous fluids were given at 100 ml/hour. Injection hydrocortisone 50 mg intravenously TDS. Injection RANTAC 50 mg was given intravenously BD.

Patient improved remarkably with the given treatment and chest pain slowly reduced and completely abolished after 5 days of initiation of treatment .Follow up ECG showed settling of ST segment changes. Follow up ECHO was normal. In between a coronary angiography was performed which revealed no significant abnormality in the vasculature .Patient is asymptomatic now performing her day to day activities without any difficulty.

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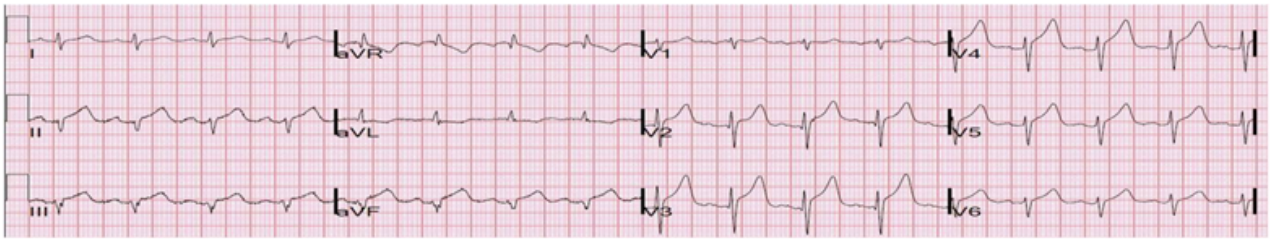
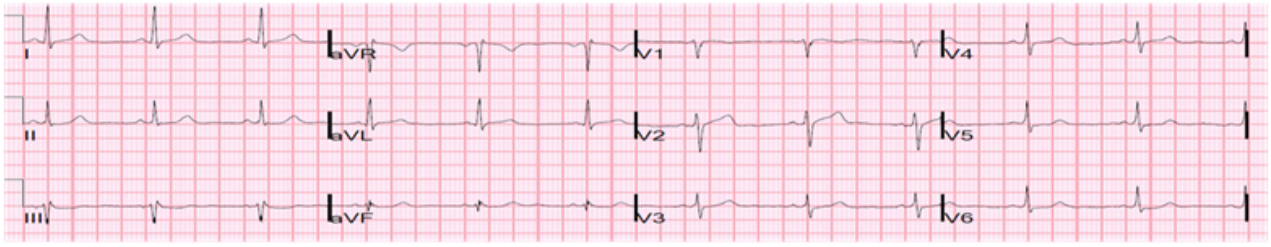


ECG on Admission



Follow up ECG after stabilization

5. Discussion

Kounis syndrome is a common yet infrequently diagnosed and reported condition in day to day practice. A multitude of etiological factors cause Kounis syndrome , few are certain foods like fish (scombridae species), shellfish ,kiwi fruit and Raw fish. Many drugs like anesthetics, analgesics and antibiotics can trigger Kounis syndrome. Mast cells play a key role in the pathogenesis of kounis syndrome their interaction with macrophages, T lymphocytes and a specific subset of platelets are the key in the activation of a cascade of events that lead to Kounis syndrome. It is of three variants type 1 (Vasospastic type) Type 2 (quiescent disease getting triggered) Type 3 (stent thrombosis). Type 1 – intravenous corticosteroids, H1 and H2 blockers, vasodilation with calcium channel blockers or nitrates. Type 2 – acute coronary event protocol with concurrent usage of corticosteroids and antihistaminic drugs Type 3 – current acute MI protocol with aspiration of thrombus, if patient develops symptoms after the stent implantation then administration of corticosteroids, anti histaminic drugs and mast cell stabilizers can be tried, if symptoms persist after adequate therapy then stent extraction is the only option.

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