Case Report

A case of Kounis Syndrome

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Abstract:

Background: Acute coronary syndrome with allergic or hypersensitivity reactions due to various anaphylactic or anaphylactoid insults such as insect sting or environmental toxins is referred to as Kounis syndrome. We report a case of Kounis syndrome caused by wasp sting

Case presentation: A 34 year old male, smoker, non-diabetic, non-hypertensive was stung by multiple wasps all over the body. Patient developed ST segment elevation in anterior leads for which coronary angiography done and found that LAD was totally occluded. Patient was put on anticoagulants and antiplatelets. Repeat CAG done after 1 month and found that LAD was patent.

Conclusion: Wasp sting can induce acute coronary syndrome either by direct effect of venom constituents on coronary endothelium or through inflammatory mediators induced allergic reaction on coronary vasculature. Early recognition of Kounis syndrome is needed to implement necessary treatments.

Keywords: Kounis syndrome, Acute coronary syndrome (ACS), Wasp sting

INTRODUCTION:

Allergic reactions to chemicals, food products and insect stings are encountered all over the world with a variety of manifestations. Symptoms range from the minor rash to life threatening anaphylactic reactions. Sometimes, these allergic reactions can precipitate acute organ involvement - in our case, acute coronary syndrome. Acute coronary syndrome or ST-Segment elevation myocardial infarction resulting from an allergic reaction, also referred to as Kounis syndrome. We report a case of Kounis syndrome secondary to wasp sting.

CASE REPORT:

A 34 year old male, smoker, non diabetic, non hypertensive had multiple wasp sting all over the body. He had no h/o allergy, no relevant family history but he had past h/o wasp sting 8 years back with no evidence of chest discomfort or anaphylaxis. He developed pain all over the body with no itching or angioedema and taken to nearby private hospital where he received i/v steroids and s/c Adrenaline. Following which, he developed chest discomfort. Patient was referred to another hospital at with ongoing chest pain. In that hospital other ECG done showed frequent ventricular ectopics. Patient received i/v Metoprolol and started on antiplatelets, following which VPC’s reduced and chest pain subsided. Repeat ECG done showed ST elevation in anterior leads with cardiac injury markers positive. CAG was done showing complete occlusion of (LAD figure 1). Patient advised immediate revascularization, but relatives was not ready for the same.
DISCUSSION:

Coincidental occurrence of acute coronary syndromes with hypersensitivity reactions involving activation of interrelated and interacting inflammatory cells and including allergic or hypersensitivity and anaphylactic or anaphylactoid insults was first described and named after Dr. Nicholas Kounis in 1991 termed as “allergic angina”. Since then this condition has evolved to encompass a number of mast cell activation disorders which are associated with acute coronary syndrome.

There are two variants: Type I variant: patients with normal coronary arteries without predisposing factors for coronary artery disease in whom the acute release of inflammatory mediators can induce either coronary artery spasm without increase of cardiac enzymes and troponins or coronary artery spasm progressing to acute myocardial infarction with raised cardiac enzymes and troponins. Type II variant: patients with culprit but quiescent pre-existing atheromatous disease in whom the acute release of inflammatory mediators can induce either coronary artery spasm with normal cardiac enzymes and troponins or plaque erosion or rupture manifesting as acute myocardial infarction.

Pathophysiologically, Kounis syndrome results from mast cell degranulation in setting of allergic insult leading to release of various mediators such as tryptase and chymase that activate the zymogen forms of metalloproteinases such as interstitial collagenase, gelatinase, and stromelysin and can promote plaque disruption or rupture. Leukotrienes are powerful arterial vasoconstrictors and their biosynthesis is enhanced in the acute phase of unstable angina. Thromboxane is a potent mediator of platelet aggregation with vasoconstricting properties. Platelet activating factor in myocardial ischemia acts as proadhesive signalling molecule for activation of leukocytes and platelets to release other mediators. These factors ultimately lead onto coronary hypoperfusion with subsequent myocardial damage due to systemic vasodilation, reduced venous return, plasma and volume loss due to increased vascular permeability, depressed cardiac output.

Kounis syndrome may present with variety of symptoms such as chest discomfort, acute chest pain, dyspnoea, faintness, nausea, vomiting, syncope, pruritus, urticaria, hypotension, diaphoresis, pallor, palpitations, bradycardia, tachycardia, and anaphylaxis. This may present with variety of ECG changes such as T wave flattening or inversion, ST segment depression or elevation, QRS complex prolongation, QT segment prolongation, atrial fibrillation, ventricular ectopics, sinus bradycardia or tachycardia, nodal rhythm, bigeminal rhythm. Recent data have shown that a small but increasing number of patients develop stent thrombosis after insertion of drug eluting stents (DES) which is due to allergy to components of DES.

Treatment of Kounis syndrome is directed firstly towards the alleviation of the acute allergic insult and secondly the therapy of the acute coronary event. The first goal achieved by H1 and H2 blockers and corticosteroids which alone can be successful in cases of type I. When type I progresses to AMI with increased cardiac enzymes & in type II with plaque erosion or rupture manifesting as an acute MI then, antiiallergic treatment is combined with classical treatment of acute MI. Type 1 has a better prognosis than Type II.
CONCLUSION

Wasp sting can induce acute coronary syndrome either by direct effect of venom constituents on coronary endothelium or through inflammatory mediators induced allergic reaction on coronary vasculature. Early recognition of Kounis syndrome is needed to implement necessary treatments. Attention should be paid that recurrent use of or exposure to the agent that causes Kounis syndrome may lead to recurrent clinical pictures.

REFERENCES


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