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Original Research Paper

Cardiology

ALLERGIC MYOCARDIAL INFARCTION – KOUNIS SYNDROME

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ABSTRACT We studied case series of 6 patients admitted with hypersensitivity and acute coronary syndrome. The local manifestation of a generalised hypersensitivity reaction is the Kounis syndrome. Although Kounis syndrome (allergic angina/allergic myocardial infarction) is not uncommon, it is underdiagnosed and infrequently identified. The diagnostic biomarker used to identify Kounis Syndrome is serum tryptase. Antihistaminic, adrenaline, and steroid treatments were given to the patients. Kounis syndrome should be suspected with a high index of suspicion in any patient experiencing a hypersensitive reaction brought on by a variety of triggers.

KEYWORDS : acute coronary syndrome, allergic angina, allergic myocardial infarction, kounis syndrome, serum tryptase, hypersensitivity

INTRODUCTION:

The Kounis Syndrome, also known as allergic myocardial infarction, was first identified in 1991 as "the allergic angina syndrome," which could lead to acute myocardial infarction. Kounis and Zavras first defined KS as the occurrence of acute coronary events and anaphylactic or l anaphylactoid allergic responses at the same time in 1991. In a 1998 editorial, Braunwald stated that mediators like histamine and leukotrienes from allergic reactions can cause vasospastic angina. 2 operating on the coronary arteries' smooth muscle Acute coronary syndromes have gained accepted as a new cause of coronary artery syndrome, and Kounis syndrome is a local manifestation of generalised hypersensitivity reaction linked to allergic reactions. KS has been divided into three types: Type I (without coronary disease): Chest discomfort during an acute allergic reaction in people without risk factors or coronary lesions, when the allergic event triggers coronary spasm that results in chest pain and electrocardiographic alterations secondary to ischemia. Chest pain during an acute allergic reaction in people with pre-existing atheroma disease is type II (with coronary disease) (whether known or otherwise).

Type III includes coronary artery stent thrombosis cases where the presence of mast cells and eosinophils was shown by aspirated thrombus specimens stained with hematoxylin and eosin and giemsa, respectively. Kounis syndrome pathophysiology Food, insect venom, iodine contrast media, and medications are examples of allergens that cause mast cell degranulation, which releases several vasoactive mediators (histamine, leukotrienes, serotonin) and proteases (tryptase, chymase). While tryptase and chymase activate the metalloproteinases, causing collagen degradation and erosion of the atheroma plaque which in turn causes the coronary event, histamine and leukotrienes are powerful coronary vasoconstrictors. Each of the four types of receptors that cardiac histamine works upon can affect how severely allergic myocardial damage manifests. While the H2 receptors play a minor role in coronary relaxation, the H1 receptors mediate coronary vasoconstriction. A reduction in diastolic blood pressure and an increase in pulse pressure are brought on by the interplay between the two receptor functions. The H4 receptors control mast cell, eosinophil, and lymphocyte chemotaxis—producing a change in eosinophil morphology and favouring 4 molecular adhesion. The H3 receptors, in turn, suppress noradrenaline release. Histamine can also stimulate platelets, improve the aggregation response to other agonists like adrenalin or thrombin, and decrease tissue factor expression and activity. This enzyme is a crucial part of the coagulation cascade and favours the nascent thrombin production.

MATERIAL AND METHODS:

Six patients who were admitted to our hospital over the past 2.5 years were the subjects of our study. These patients experienced acute onset chest discomfort, electro cardiographic alterations, and increased cardiac enzymes in response to allergy stimuli. Table 1 lists the traits of the patients who experienced KS. Drugs were the etiological agent in two patients, snake bites in two patients, and radiocontrast material in one patient. None of these conditions—allergy, bronchial asthma, dermatitis, eczema, or coronary artery disease—had ever affected them in the past. All patients had coronary angiography and an echo cardiographic evaluation. While coronary angiography found non-critical plaques in every patient, echocardiography demonstrated segmental wall motion abnormalities.

No.	1	2	3	4	5	6
age	52	26	43	61	22	39
sex	male	male	female	male	female	male
Allergic insult	Inj.Ceftriaxone	Snake bite	Radio contrast (iohexol)	Radio contrast (iohexol)	Amoxycillin-clavunlenic acid	Insect bite
ECG changes (ST elevation in)	V1-V4	V1-V4	II,III aVF	V3-V4	V5-V6	II,III,aVF
Symptoms	Erythematous rash Dyspnea Hypotension	Chest pain	Pruritic rash Chest pain	Erythematous rash Dyspnea Chest pain	Erythematous rash Dyspnea	Pruritic skin rash
Toponin T	Positive	Positive	Positive	Positive	Positive	Positive

TOTAL eosinophil LEVEL (30-350)	850	820	730	690	900	910
Sr tryptase level (5-5ng/ml)	40	42	36	31	55	47
Wall motion abnormality On echocardio- graphy	Anterior and septal	Anterior	Inferior	Anterior	Anterior	Inferior
Coronary angiography	Left anterior descending (30% stenosis)	Left anterior descending (30% stenosis)	Right Coronary artery (40% stenosis)	Left Anterior Descending (0% stenosis)	Left Anterior Descending (30% stenosis)	Right coronary (30% stenosis)

RESULT:

In this study, we discussed 6 patients with a kounis syndrome diagnosis. Out of 6 patients, 2 are female and 4 are male, with a median age of 44 years. The patients' ages range from 22 to 62 years. The radiocontrast agent (iohexol) in two patients, a snake bite in one patient, an insect bite in one patient, amoxycillin-clavulanic acid in one patient, and ceftriaxone in one patient were identified as the etiological agents for generating allergic reactions. All patients' initial complaints were allergic rashes, along with one patient's chest pain and hypotension. Three patients had ST elevation in the anterior lead, two in the inferior lead, and one in the lateral lead. ECG alterations are consistent with echocardiographic findings. All patients had positive qualitative troponin T results. All patients had elevated eosinophil levels, indicating an allergic cause of myocardial infarction. Our diagnosis is supported by the elevated serum tryptase levels in all patients (normal 5–15 ng/ml). All patients underwent coronary angiography, and all of them had non-critical stenosis in their left anterior descending arteries and right coronary arteries. All patients receive steroid and antihistaminic therapy.

DISCUSSION:

The first description of Kounis syndrome was given by Kounis and Zafras in 1991. They defined it as "the concomitant occurrence of chest pain and allergic reactions accompanied by clinical and laboratory findings of classical angina pectoris caused by inflammatory mediators released during an allergic insult [1]. Three different kinds of KS have been identified. Patients with normal coronary arteries experience type 1 disease. Patients who already have atheromatous coronary artery disease experience type 2. Stent thrombosis will result from type 3. Angina and myocardial infarction can result from any of the three situations. Snake and bee sting bites, radiocontrast agent (iohexol), amoxicillin, and ceftriaxone are all indicated as etiological agents in our study. In our investigation, 2 cases of amoxycillin-clavulanic acid and ceftriaxone-induced Kounis syndrome were found. Cases of drug allergies to specific medications, such as antibiotics, were found by Kulratne et al. Fagley et al. described the allergic vasospasm caused by rocuronium. (6) Rocuronium and succinylcholine are the NMBs that are most frequently implicated. Shibuya et al. describe 11 cases connected with contrast induce, of which three cases are related to iopomide, three are related to iohexol, while two are related to iopomidol, and the remaining contrast agent is unknown (7). These include cobra bites that cause anaphylaxis and viper venom, according to a case study by Priyankara et al. (9) Manjur et al. describe a vasospastic angina case associated with scrombide use. (8) Frengid et al. describe vasospastic angina caused by shellfish eating. (8) Only a few examples of type III KS have been documented in the literature. A few occurrences of KS have also been linked to particular kinds of contrast materials. Although low-osmolar non-anionic contrast materials are less hazardous than anionic and monomeric contrast materials, low-osmolar non-ionic contrast materials were responsible for the majority of the KS cases reported in the literature (.7) In our investigation, there were two cases of kounis, and the contrast agent in both cases was iohexol. In our investigation, two patient coronary arteries are normal and the others have non-critical plaque; therefore, type I Kounis syndrome is suspected in the first two cases, while type II Kounis syndrome is suspected in the remaining cases. 11 examples of radio contrast-induced kounis syndrome were studied in the literature by K. Shibuya et al., with 6 cases being type II KS and 4 cases being type II KS (7). Both of them are type II in our investigation. Additionally, a case series of 6 patients with insect bite-related kounis syndrome by Rao et al. was analysed, of which 4 patients had type II KS. One case involves a snake bite that caused anaphylactic shock. Ruth and colleagues discovered a hypersensitivity among cobra handlers to spitting airborne cobra venom. (10) Due to different components such poisonous peptides, phospholipase A2, and other proteins found in its venom, snake venom can cause allergic or anaphylactic reactions [11]. There have been reports of potential causes including vasospasm, hypercoagulability, direct cardiotoxicity, and hypovolemia. Viper bites are more frequently reported than cobra bites as causing myocardial infarction [11]. individuals with the Kounis syndromeTypically, all myocardial infarctions affect the inferior heart. 14 Out of the six patients in the current investigation, three had anterior wall infractions, two had inferior wall myocardial infractions, and one had anteroseptal wall infractions. whereas most studies include the right coronary most frequently (12). We discovered that the RCA was affected in two patients and the left anterior descending artery in four patients. Histamine levels were found to be increased in one incidence of scromboid consumption that was examined in the literature and had vasospastic angina (1347). (8). This study did not involve any of our patients, yet it is one of the diagnostic indicators for Kounis syndrome. . In the current investigation, patients were given aspirin, calcium channel blockers, steroids, and nitrate as antihistaminic, anti-ischemic, and anti-steroid medications.

Conclusion The local manifestation of a generalised hypersensitivity reaction is the Kounis syndrome. Although not uncommon, Kounis syndrome (allergic myocardial infarction) is underdiagnosed and infrequently identified. The diagnostic biomarker used to identify KS is serum tryptase. Antihistaminic, adrenaline, and steroid treatments were given to the patients. Kounis syndrome should be suspected in all patients who have a hypersensitive reaction caused by a variety of stimuli with a high index of suspicion.

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