

letter

Kounis syndrome and antibiotics

To the Editor: We read with interest the article recently published by Saleh entitled "Kounis syndrome: acute inferior myocardial infarction with atroventricular node block due to ceftriaxone: a first reported case."¹ However, we have some concerns about the article.

Inflammatory mediators, adhesion molecules of neutrophils, and monocytes have been shown to be increased in the plasma of patients presenting with acute coronary syndromes. Anaphylaxis is a systemic, immediate hypersensitivity reaction caused by rapid, immunoglobulin (Ig) E-mediated release of mediators from mast cells and basophils. Kounis syndrome (KS) is the coincidental occurrence of these two distinct conditions accompanied by clinical and laboratory findings of angina pectoris caused by inflammatory mediators released during an allergic insult.² Allergic angina can progress to acute myocardial infarction, which is named "allergic myocardial infarction." Three variants of KS have been described.³ The type I variant (coronary spasm), which might represent a manifestation of endothelial dysfunction or microvascular angina, includes patients with normal coronary arteries without predisposing factors for the coronary artery disease. In these patients, 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. The type II variant (coronary thrombosis) includes patients with culprit but quiescent pre-existing atherosclerotic disease. In these patients, the acute release of inflammatory

mediators can induce either coronary artery spasm with normal cardiac enzymes and troponins, plaque erosion, or rupture manifesting as acute myocardial infarction.

The type III variant (drug-eluting stent thrombosis) includes patients with stent thrombosis. In these patients, thrombus harvesting and staining with hematoxylin-eosin and Giemsa shows the presence of eosinophils and mast cells, respectively.

Although Saleh claimed the current patient as the first KS patient associated with ceftriaxone use, this is not true. Çağlar et al described an 85-year-old woman with ST-segment elevations on the anterior precordial leads after the use of ceftriaxone.⁴ Yurtda and Aydin also reported a 42-year-old man with no history of known atopy or cardiovascular risk factors presented the emergency department with the chest pain, severe dyspnea, sweatings, nausea, vomiting and, urticarial and edematous lesions approximately 20 minutes after the use of ceftriaxone.⁵ Electrocardiogram was compatible with acute inferior myocardial infarction. There are several causes that have been reported as capable of inducing KS.⁶ These include a number of conditions, several drugs, foods, insect stings, etc.^{6,7} After the first report of allergic myocardial infarction, nearly 350 cases with KS have been reported in the published studies. In summary, in the investigation or interpretation of the published studies, authors should be careful and reviewers must be qualified to evaluate a component of the research.

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