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CASE REPORT

In-hospital acute myocardial infarction: A case of type II Kounis syndrome☆

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KEYWORDS
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Abstract Kounis syndrome (KS) is the coincidental occurrence of acute coronary syndrome (ACS) and anaphylactic or allergic insult. It results from mast cell degranulation with subsequent release of numerous inflammatory mediators, leading to coronary vasospasm or atheromatous plaque rupture. Diagnosis is clinical and based on the temporal relationship between the two events. Despite the growing number of reported cases, especially in southern Europe, the lack of awareness of this association may lead to under-reporting in Portugal. Recognition of KS, even if retrospective, has clinical implications since individual atopy must be investigated and desensitization measures should be employed, if possible, to prevent future events. We report the case of a 70-year-old man who was admitted to hospital because of generalized exanthema and itching and onset of chest pain while under observation. Coronary angiography confirmed coronary artery disease and ACS and he was diagnosed as having type II KS.

PALAVRAS-CHAVE
Síndrome de Kounis;
Enfarte agudo do miocárdio;
Reação alérgica;
Atopia

Enfarte agudo do miocárdio intra-hospitalar: um caso de síndrome de Kounis tipo II

Resumo A síndrome de Kounis (SK) define-se como a ocorrência simultânea de uma síndrome coronária aguda (SCA) e de um insulto anafilático ou alérgico. Resulta da desgranulação de mastócitos e subsequente libertação de mediadores inflamatórios, responsáveis pelo vasospasmo coronário ou rutura da placa de ateroma. O diagnóstico é clínico e baseia-se na relação temporal entre os dois eventos. Apesar do número crescente de casos descritos, principalmente

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nos países do sul da Europa, o desconhecimento médico pode levar ao subdiagnóstico em Portugal. O reconhecimento da SK, mesmo realizado *a posteriori*, tem implicações clínicas, nomeadamente na investigação da atopia e medidas de dessensibilização, se possíveis, com vista à prevenção de novos eventos. Os autores relatam o caso clínico de um doente de 70 anos, que recorreu ao serviço de urgência por exantema generalizado e pruriginoso e iniciou dor torácica enquanto estava em observação. A angiografia confirmou SCA com evidência de doença coronária e subsequente diagnóstico de SK tipo II.

Introduction

Kounis syndrome (KS), first described in 1991 by Kounis and Zavras,[1] is defined as the coincidental occurrence of acute coronary syndrome (ACS) and anaphylactic or allergic insult. It results from mast cell degranulation with subsequent release of numerous inflammatory mediators, leading to coronary vasospasm or atheromatous plaque rupture. Diagnosis is clinical and is based on symptoms and signs of an acute allergic reaction – cutaneous (rash, itching, hives, angioedema), respiratory (dyspnea, wheezing, stridor) or gastrointestinal (abdominal pain, nausea, vomiting) – simultaneously with a setting suggestive of ACS. According to Cepeda et al.,[2] the clinical history is essential for establishing a cause-effect relationship in time with the possible triggering factor, and so the patient's allergic history should be investigated, including allergy to latex, nuts or drugs, insect stings or bites, or recent use of new medications.

As KS represents a cluster of cardiovascular symptoms that result from an allergic insult, in most cases the clinical diagnosis is made retrospectively. There is no diagnostic test pathognomonic of KS, but there are some studies that suggest measuring levels of tryptase (at onset of symptoms and after two and 24 hours), histamine, complement, eosinophils and total immunoglobulin E (IgE). However, normal values of these parameters do not rule out the possibility of a previous allergic reaction.[2] At the same time, given the suspicion of an ACS, an electrocardiogram (ECG) and serial measurement of cardiac enzymes should be performed.

There are currently no specific clinical guidelines for the treatment of KS, which should consist of treating the ACS and the allergic reaction in accordance with the type and severity of each. Although the two conditions should be treated appropriately, accurate diagnosis of the syndrome, even after the event, has clinical implications. The patient's atopy should be assessed and desensitization measures should be taken if necessary, given the risk of future allergic reactions that could trigger an ACS.[3]

The authors present a case of KS following a probable insect sting.

Case report

J. H. B. C., male, 70 years, went to the emergency department due to exanthema and generalized itching. His symptoms had begun after a bicycle ride. He reported two previous episodes of generalized maculopapular erythema and itching after insect stings in the outer ear, five and two years before, which improved with medication. In the present episode, he had not detected an insect sting, and reported consuming no unusual foods or taking new drugs. He reported a personal history of hypertension and dyslipidemia, medicated with ramipril 5 mg daily and simvastatin 20 mg daily, respectively.

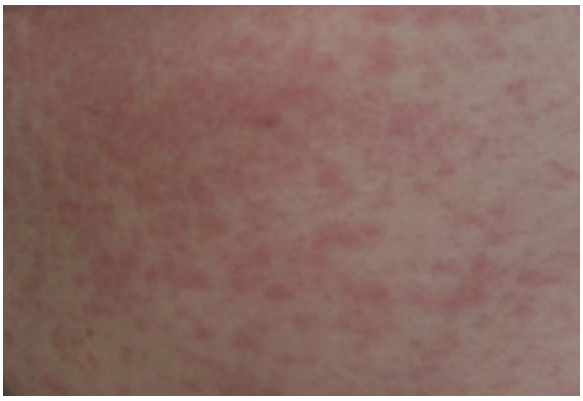

Figure 1 Erythematous maculopapular exanthema.

On arrival at the emergency department, around 30 min after symptom onset, he began to experience constricting chest pain radiating to the left arm, with no relieving or aggravating factors and not improved by sublingual nitroglycerin. He also reported general malaise but no fever, dyspnea, cough, expectoration, nausea, vomiting or other symptoms.

On physical examination the patient was apyretic and hemodynamically stable, with blood pressure 140/70 mmHg and heart rate 65 bpm. He presented generalized maculopapular erythema (Figure 1) and itching that spared the palms, soles and face. Cardiac auscultation revealed rhythmic heart sounds and no murmurs, while pulmonary auscultation detected normal breath sounds with no adventitious sounds. His abdomen was painless on palpation and there was no edema or other alterations in the lower limbs.

The ECG showed sinus rhythm with heart rate 72 bpm and ST-segment elevation of around 2 mm in V2-V6, DI and aVL (Figure 2). A provisional diagnosis was made of anterolateral ST-elevation myocardial infarction.

The hemodynamic team was activated and medication was begun with a loading dose of aspirin 300 mg, ticagrelor