Cardiorespiratory arrest after administration of an antibiotic

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CASE REPORT

An 82-year-old mentally retarded male presented to the emergency department with abdominal pain and dyspnoea. His medical history included volvulus for which he had undergone surgery. He did not smoke. Physical examination showed a blood pressure of 130/70 mmHg, heart rate of 95 beats/min, temperature of 39.0°C, and diffuse abdominal tenderness. The ECG was normal. Since the patient fulfilled the criteria for systemic inflammatory response syndrome and no drug allergy was known, we started intravenous administration of amoxicillin/clavulanic acid according to local guidelines before availability of laboratory results. Immediately after administration of the antibiotic, the patient experienced a cardiorespiratory arrest. The ECG recording showed ST elevation in the inferior leads and reciprocal depression in anteroseptal leads, aVR and aVL (figure 1). Under the suspicion of acute myocardial infarction, we started cardiac resuscitation, including administration of aspirin and heparin. After the patient was stabilised, we discovered a mild erythematous rash. Ten minutes after the administration of the antibiotic, the ECG normalised (figure 2).

WHAT IS YOUR DIAGNOSIS?

See page 532 for the answer to this photo quiz.
DIAGNOSIS

We suspected an allergic reaction to amoxicillin/clavulanic acid after the discovery of the rash, and administered clemastine and prednisolone. When the patient was stabilised, we excluded pulmonary and abdominal pathology by computed tomography. Coronary angiography did not show any coronary artery occlusion. We established the diagnosis of acute coronary spasm caused by an allergic reaction after the administration of β-lactam antibiotic. It is also known as the Kounis syndrome.

DISCUSSION

Kounis syndrome, or allergic angina, is a syndrome in which patients with a hypersensitivity reaction present with acute coronary syndrome. It was first described by Kounis and Zavras in 1991 and is associated with massive mast-cell degranulation during a type I allergic reaction. Mast cells release endogenous mediators, including histamine. Histamine increases cardiac contractility and heart rate, and causes vasospasms through H1 and H2 receptors. Histamine also induces tissue factor expression, which contributes to thrombus formation resulting in acute coronary occlusion.1,2 Two variants of the Kounis syndrome have been described.3 The first variant includes patients with normal coronary arteries without predisposing factors for coronary artery disease in which an allergic reaction induces coronary artery spasms. The second variant includes patients with culprit but quiescent pre-existing atheromatous disease. An allergic reaction can induce plaque rupture, which may result in an acute myocardial infarction.

The treatment of Kounis syndrome has two goals: 1) dilatation of the coronary vessels by vasospasmolytic agents, and 2) suppression of the allergic reaction by corticosteroids. The use of epinephrine is doubtful. It is life-saving in anaphylaxis, but it can aggravate ischaemia, prolong the QTc interval, and induce coronary vasospasms and arrhythmias.4

This case illustrates the potential harm of rapid antibiotic administration. Although rapid administration of antibiotics can be life-saving, physicians should carefully consider this use of antibiotics and need to be aware of the risks and adverse outcomes after administration.

REFERENCES