



Letter to the Editor

Acute ST-segment elevation myocardial infarction complicating
amoxicillin-induced anaphylaxis: A case reportFrancesca Del Furia^{a,*}, Lorenzo Querceto^a, Sergio Testi^b, Giovanni Maria Santoro^a^a Cardiology Unit, Nuovo San Giovanni di Dio Hospital, Florence, Italy^b Allergology Unit, Nuovo San Giovanni di Dio Hospital, Florence, Italy

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Abstract

A 70-year-old man experienced an amoxicillin-induced anaphylactic reaction complicated by acute inferior myocardial infarction with transient ST-segment elevation. There was a spontaneous resolution of ST-segment elevation and the patient was treated for anaphylaxis. Coronary angiography showed severe obstructive coronary atherosclerosis, but not involving the infarct-related artery. Percutaneous coronary intervention of the affected artery was then performed and the patient was discharged three days later.

Acute ST-elevation myocardial infarction has been described as one of the severe, still rare cardiovascular complications of anaphylaxis. In the present case, according to the previous reports, the main pathogenetic mechanism involved appears to have been coronary vasospasm probably caused by the release of potent vasoactive mast cell derived mediators in the setting of anaphylaxis.

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A 70-year-old man collapsed while he was driving his car a few minutes after the assumption of a pill of amoxicillin (500 mg). Before collapsing he presented prodromic signs like pruritus, warmth, flushing, lips and hands swelling and dyspnea. The physician of the Mobile Emergency Service found him unconscious with spontaneous breath. The patient's skin was diffusely erythematous and warm. A 12-lead ECG showed junctional rhythm at a rate of 90 beats/min, with ST-segment elevation of 3 mm in II–III–aVF and ST-segment depression of 2 mm in I–aVL (Fig. 1). The ECG changes spontaneously resolved in 10 min. In the hypothesis of an anaphylactic reaction induced by amoxicillin, the physician immediately administered intravenous glucocorticoids and fluids and transferred the patient to the nearest hospital.

On admission to the Emergency Department, the patient was conscious. In the history he reported having had a

similar, still softer, reaction after the ingestion of the same antibiotic two years before. He also reported essential systemic hypertension in medical treatment and previous smoking habit. He denied any history of heart disease, chest pain or effort limitation. On physical examination diffuse erythema was present without other abnormalities. Vital parameters were normal: heart rate 90 bpm, blood pressure 100/60 mmHg, oxygen saturation 98%, no fever. He was given the following drugs: chlorfenamine maleate (40 mg i. v.), methylprednisolone (100 mg i.v.), ranitidine (900 mg i. v.), aspirin (100 mg orally), nitrates (50 mg orally) and intravenous fluids. An hour later the erythema resolved. Echocardiography performed at admission revealed hypokinesis of the basal inferior wall, with preserved systolic left ventricular function. Six hours after admission an increase of troponin-I was observed (0.31 ng/mL, normal value in our center <0.1 ng/mL). The patient was transferred to the Coronary Care Unit. Cardiac catheterization, performed 24 h later, showed a severe stenosis of the mid left anterior descending (LAD) artery; the right dominant coronary artery and the left circumflex were apparently not affected by

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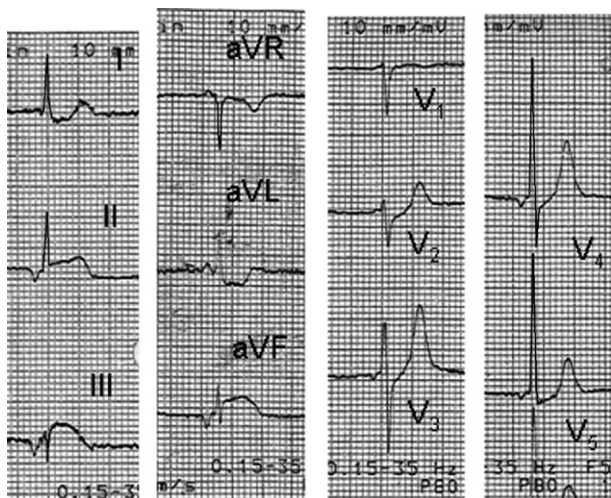


Fig. 1. The 12-lead ECG registered by the Mobile Emergency Service shows junctional rhythm at a rate of 90 beats/min with the typical ECG findings of acute inferior transmural ischemia: ST-segment elevation with positive T waves in the inferior leads and reciprocal ST-segment depression in the lateral leads (the V6 lead is missed because of the suboptimal quality of the ECG for the emergency situation in which it was obtained).

atherosclerotic disease (Fig. 2). Percutaneous coronary intervention with stent implantation of the LAD artery was performed, with final optimal angiographic result. The patient recovered completely and was discharged from the hospital three days after admission. Echocardiography performed before discharge revealed a complete recovery of the left ventricle's wall motion abnormality. The patient was then referred to the Allergy Department of our hospital. Prick Skin Test (PST) for amoxicillin hypersensitivity performed one month later was strongly positive.

In the present case, the temporal sequence of events suggests that drug-related anaphylaxis was the triggering factor of acute ST-segment elevation myocardial infarction. Only few cases have been reported of anaphylactic reactions triggering cardiovascular events, including myocardial infarction [1]. Acute myocardial infarction has been described as a consequence of an anaphylactic reaction complicating insects' sting [2–4], shellfish ingestion [5] and administration of drugs [6–13]. The main underlying pathogenetic mechanism of anaphylaxis-induced acute coronary syndromes has not been fully elucidated, even if some hypotheses have been suggested. The presence of mast cells within the human heart, strategically located perivascularly, has been demonstrated in close proximity to myocytes, in the arterial intima and in the shoulder region of coronary atheromas [14]. According to the previous observations, these findings support the hypothesis that heart may be the target as well as the site of anaphylaxis [15–17]. Coronary vasospasm, induced by potent vasoactive mast cell derived mediators, has been proposed as the main pathogenetic mechanism of anaphylaxis-related acute coronary syndromes. Moreover, mast cell may induce plaque activation, especially in patients with underlying coronary atherosclerosis. There are evidence that

mast cells are an integral component of the inflammatory infiltrate of the eroded or ruptured coronary plaques [16]. The ability of activated mast cells to initiate matrix degradation suggests that mast cells may actively participate in the local weakening of inflamed atherosclerotic lesions that ultimately leads to erosion or rupture of the plaque. Another pathogenetic mechanism proposed for acute coronary syndromes complicating anaphylaxis is prolonged hypotension, especially in patients with critical coronary stenoses, in whom hypotension may precipitate an abrupt reduction of coronary flow and provoke myocardial ischemia in the

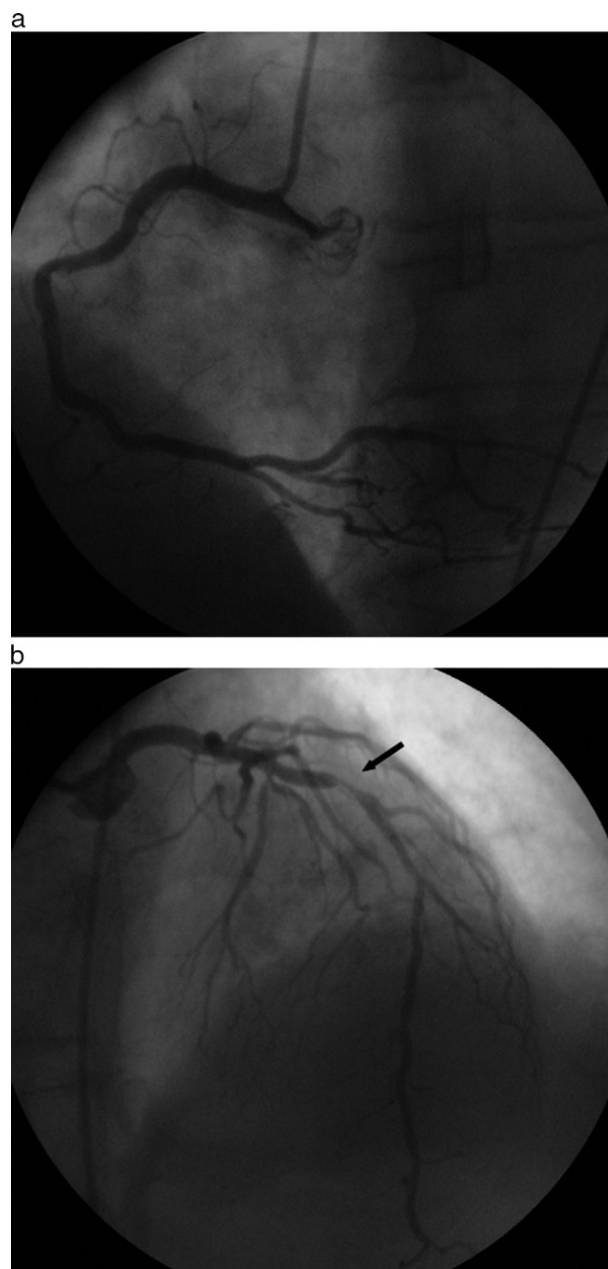


Fig. 2. Coronary angiography. a. Left anterior oblique view shows the right dominant coronary artery not affected by significant stenosis. b. Right anterior oblique cranial view shows subocclusive stenosis of mid left anterior descending artery (look at the black arrow).

territory of the stenotic artery. Finally, there are cases of acute coronary syndromes caused by the administration of drugs like epinephrine [18], which is a life-saving drug in case of anaphylaxis but can aggravate a condition of coronary vasospasm induced by mast cell derived mediators [5].

In our patient coronary spasm was almost surely the main pathogenetic mechanism involved. Indeed, the patient presented an acute coronary syndrome with transient ST-segment elevation in the inferior ECG leads, which indicates transmural ischemia in the inferior wall. The subsequent occurrence of parcellar myocardial necrosis was confirmed by the increase of troponin-I. Coronary angiography showed right dominant coronary artery and left circumflex without stenosis and a severe stenosis of the left anterior descending (LAD) artery. The discrepancy between the vessel affected by the fixed stenosis and the territory involved by the acute ischemia led to exclude a causal role for the LAD stenosis, which was however treated with stent due to the severity of the vessel narrowing.

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