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Letter to the Editor

# Acute ST segment elevation myocardial infarction after sulbactam–ampicilllin induced anaphylactic shock in an adult with significant coronary artery disease: A case report

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**Abstract**

Myocardial injury may complicate allergic reactions caused by several medications. We evaluated a case of a myocardial injury with transient ST segment elevation in a 72 year-old man presenting with collapse caused by sulbactam–ampicilllin assumption. The purpose of this report is to present this interesting case and revise the classification of Kounis syndrome.


*Keywords:* Myocardial infarction; Sulbactam; Ampicilllin; Anaphylactic shock; Coronary artery disease

## 1. Introduction

Anaphylactic reactions are acute and potentially life-threatening medical emergencies. Several drugs, especially penicillin derivatives may trigger anaphylactic reactions. Acute cardiac injury has been reported to complicate these allergic reactions, which is also named as Kounis syndrome. Although several have been proposed to explain this association, the underlying mechanisms have not been fully enlightened. In this report, we aimed to report a 72 year-old man presenting with collapse and subsequent myocardial injury occurred after sulbactam–ampicilllin (SAM) assumption.

## 2. Case report

A 72 year-old man was brought to the emergency room of our institute after a syncopal attack, which developed 15 min after ingesting a pill. He was confused and hypotensive with a systolic blood pressure of 80 mm Hg on the admission. His pulse rate was approximately 150. He had respiratory distress with bronchospasm and rales at the base of both lungs.

A 12 lead ECG obtained showing atrial fibrillation and ST segment elevation in leads D2, D3 aVF, and ST segment depression in the leads V1-4, D1, aVL indicating acute inferior injury (Fig. 1). There was no sign of right ventricular infarction on the ECG. A percutaneous coronary angioplasty was planned with the diagnosis of inferior MI. A bolus dose of heparin (5000 IU), ASA and nebulised mixture of salbutamol and ipratropium were administered. But during the preparation phase of primary PTCA; ST segment elevations resolved, sinus rhythm restored and blood pressure improved to a systolic value of 115 mm Hg. was transferred to the coronary care unit to be followed as a case of coronary spasm. Elevated cardiac markers were indicative of subsequent myocardial injury.

Because he was confused at the admission, the detailed history could only be taken 8 h after the symptoms to reveal SAM ingestion 15 min before the syncopal attack. After the suspicion of an allergic event; a tryptase level was obtained. The result was in normal limits with a value of <1.0 mcr/l

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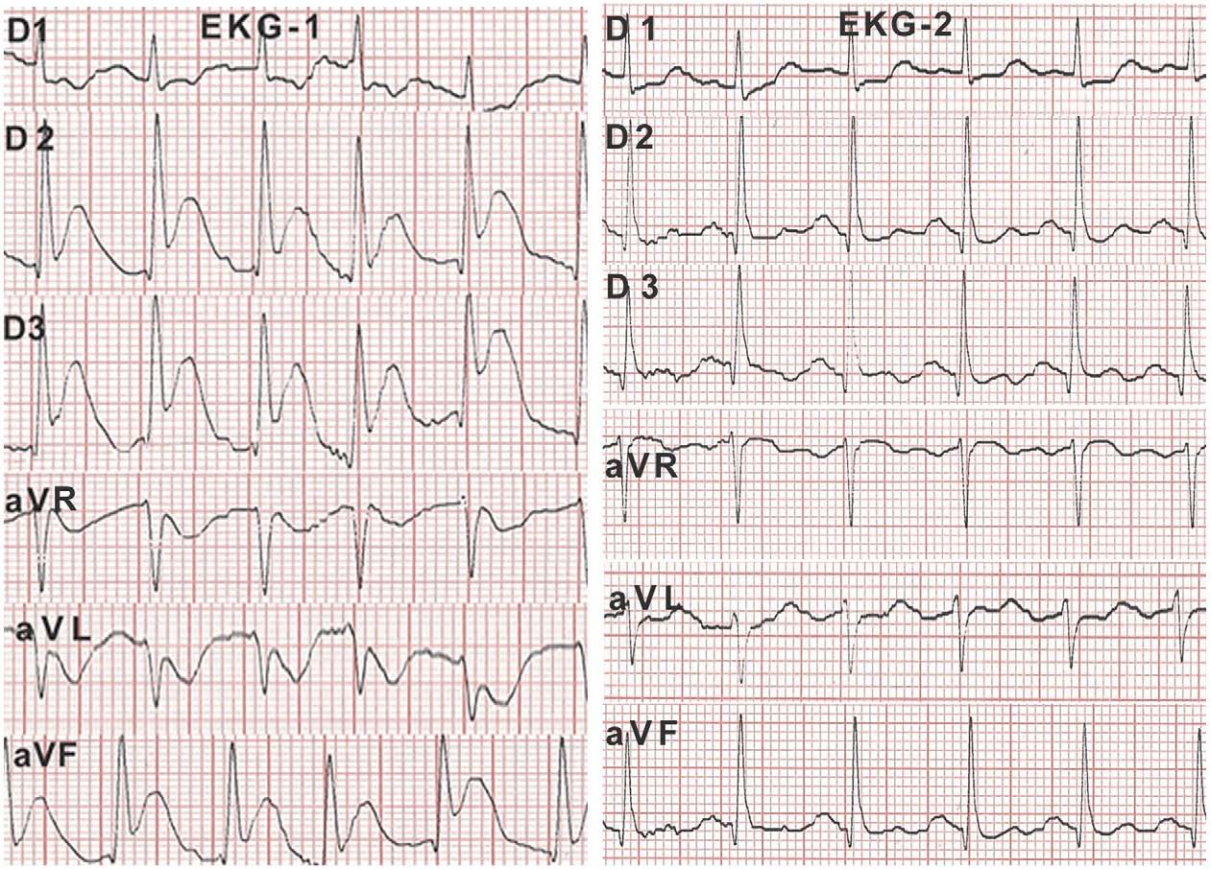


Fig. 1. Electrocardiographic images. EKG-1:ST segment elevations in the inferior leads, EKG-2: Records obtained after the resolution of the ST elevations.

(normal < 13.1). Circulating specific IgE levels (UNICAP100 Pharmacia Diagnostics) for the ampicillin was moderately positive (class 2). Specific IgE levels for the penicilloyl G, penicilloyl V, cefaclor and amoxycillin were also determined showing low positive (class 1) values for the former two and negative values for the latter two (class 0). For the safety reasons, we did not perform intradermal tests. A coronary angiogram was performed on the 5th hospital day revealing multivessel disease (Fig. 2). RCA was non-dominant. Although RCA, LAD and LCx had multiple critical lesions, none of them was interpreted as the infarct related artery with the images obtained by the angiography. Ventriculography was normal. Because of the multivessel disease, a coronary artery bypass graft surgery was recommended to the patient.

## 3. Discussion

The concurrence of allergic reactions and acute coronary syndromes is called Kounis syndrome (KS) [1,2]. Several mechanisms have been proposed to explain this relationship between the allergic insult and the myocardial injury. Coronary spasm as a result of vasoactive peptides released from circulatory and/or cardiac mast cells involved in the allergic process may be the underlying mechanism. Plaque rupture induced by the allergic insult may be another cause. Platelet aggregation and thrombus formation facilitated by the vasoactive peptides may accompany these events. Prolonged hypotension and subsequent myocardial hypoperfusion or epinephrine administration are the other proposed mechanisms [1,3–10]. The latter is clearly not the case in this patient.

Although there are many causes of KS including drugs, environmental exposure, food allergy, KS induced by SAM ingestion is very rarely reported in the literature [1]. To our knowledge, only one patient has been reported and our patient is the first case with significant coronary disease [11].

In this case, diagnosis of a KS is supported by the short time interval between the ingestion of the related drug and the symptoms; and also the ampicillin specific IgE levels detected in the serum. However, we found a normal tryptase level. Tryptase, a neutral protease in the mast cells, serves as marker of mast cell activation. Despite it may be present in the circulation for longer periods depending on the magnitude of the initial responses, because of its short half-live, best time to obtain samples for tryptase determination was suggested 1–2 h after the initiations of the symptoms [12]. Nevertheless, in our case, the clinical picture did not allow us to take a detailed history on admission and the sample for tryptase determination was taken 8 h after the collapse to find normal levels. Furthermore, we should also mention that a negative tryptase test does not exclude anaphylaxis.