

# Kounis Syndrome: Acute ST segment Elevation Myocardial Infarction following Allergic Reaction to Amoxicillin

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**K**ounis syndrome is an acute coronary syndrome following an allergic reaction. We report a case of acute myocardial infarction (MI) complicated by ventricular fibrillation following an allergic reaction to amoxicillin.

## PATIENT DESCRIPTION

A 53 year old man swallowed a sip of pediatric solution of amoxicillin to show his son how to take the medicine. Within a few minutes he experienced diffuse urticaria and itch; 30 minutes later he felt chest pain and called for an ambulance. On arrival of the emergency medical services the patient's blood pressure was 110/84 and heart rate 90 beats/min; an electrocardiogram (ECG) showed sinus rhythm, ST segment elevation in leads II-III-AVF, depression in leads I-AVL and ventricular premature beats

[Figure 1]. Treatment with IV hydrocortisone 300 mg and intramuscular promethazine hydrochloride 25 mg was started. The patient had no history of cardiovascular disease, but he was allergic to penicillin.

During transportation to hospital he developed ventricular fibrillation and was successfully defibrillated. On hospital admission the chest pain decreased in intensity, and a new ECG confirmed the diagnosis of acute diaphragmatic myocardial infarction. Clinical examination revealed diffuse urticaria but no angioedema or bronchospasm; blood pressure was 110/70 mmHg, and the patient was in Killip class I. An additional intravenous bolus of 300 mg hydrocortisone was added to the treatment and primary percutaneous coronary intervention was performed 75 minutes after the beginning of chest pain. A 90% stenosis of the proximal right coronary artery was demonstrated and successfully treated by deploying a bare metal stent 4.0/18 mm. After the procedure the chest pain resolved completely and the ECG showed isoelectric ST segment. The maximal troponin I value was 1272 µg/L

(normal value 0–14 µg/L) and creatinine phosphokinase value was 574 U/L (normal 20–180 U/L). Follow-up at 6 months was uneventful.

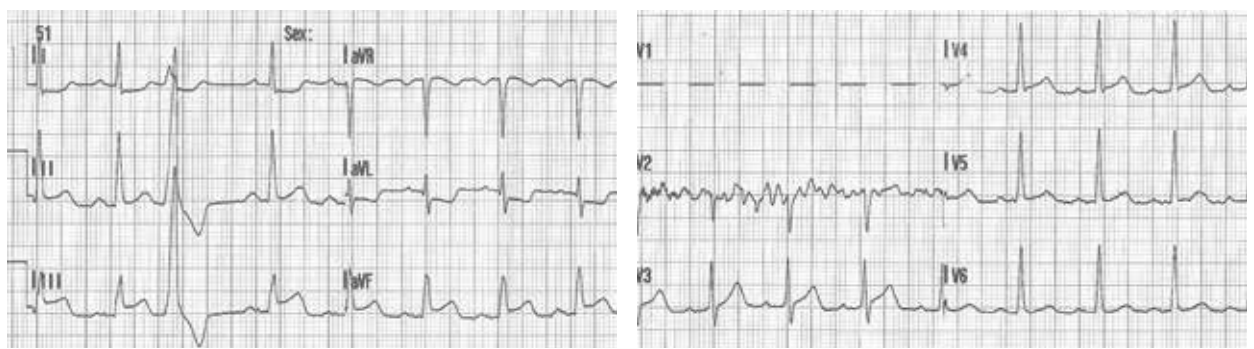
## COMMENT

Acute MI following an allergic reaction was first reported in 1950 [1]; allergic angina syndrome was later described by Kounis and Zavras in 1991 [2].

Following an allergic process activation of mast cells occurs with release of inflammatory mediators such as histamine, thromboxane, prostaglandins, leukotrienes and platelet activation factor; these mediators can induce either coronary artery vasoconstriction or atheromatous plaque rupture, culminating in coronary artery thrombosis [3].

Three variants of Kounis syndrome have been described: type I variant is observed in patients with normal or near normal coronary artery disease; in type II variant the patients have a preexisting atheromatous disease; type III variant includes coronary artery stent thrombosis [3].

**Figure 1.** Electrocardiogram on patient's admission (lead V1 is missing). ECG shows sinus rhythm, ST segment elevation in leads II-III-AVF, and ventricular premature beat



We previously described two patients with anaphylactic shock and transient ST segment elevation, and suggested that the ECG abnormalities were caused by histamine-induced coronary artery spasm and that coronary artery hypoperfusion following severe systemic hypotension may have had a role in producing myocardial ischemia [4]. However, our patient, described above, presented with an allergic reaction and ST segment elevation but no systemic hypotension. Previously reported findings [5] also refuted that myocardial damage during

anaphylactic shock is related to peripheral vasodilatation.

Our case shows that a non-life-threatening allergic reaction may be complicated by a cascade of events resulting in fatal arrhythmia and leads us to suspect that some cases of sudden death may be due to Kounis syndrome.

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### References

1. Pfister CW, Plice SG. Acute myocardial infarction during a prolonged allergic reaction to penicillin. *Am Heart J* 1950; 40: 945-7.
2. Kounis NG, Zavras GM. Histamine-induced coronary artery spasm: the concept of allergic angina. *Br J Clin Pract* 1991; 45: 121-8.
3. Kounis NG. Coronary hypersensitivity disorder: the Kounis syndrome. *Clin Ther* 2013; 35: 563-71.
4. Antonelli D, Kolton B, Barzilay J. Transient ST segment elevation during anaphylactic shock. *Am Heart J* 1984; 108: 1052-4.
5. Kounis NG, Soufras GD, Hahalis G. Anaphylactic shock: Kounis hypersensitivity-associated syndrome seems to be the primary cause. *North Am J Med Sci* 2013; 5: 631-6.