Kounis 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 hyperperfusion 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 vasodilation.

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

Early dissemination seeds metastasis in breast cancer

Accumulating data suggest that metastatic dissemination often occurs early during tumor formation, but the mechanisms of early metastatic spread have not yet been addressed. By studying metastasis in a HER2-driven mouse breast cancer model, Hosseini and colleagues show that progesterone-induced signaling triggers migration of cancer cells from early lesions shortly after HER2 activation, but promotes proliferation in advanced primary tumor cells. The switch from migration to proliferation was regulated by increased HER2 expression and tumor-cell density involving microRNA-mediated progesterone receptor downregulation, and was reversible. Cells from early, low density lesions displayed more stemness features, migrated more and founded more metastases than cells from dense, advanced tumors. Notably, we found that at least 80% of metastases were derived from early disseminated cancer cells. Karyotypic and phenotypic analysis of human disseminated cancer cells and primary tumors corroborated the relevance of these findings for human metastatic dissemination.

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Birth defects among fetuses and infants of U.S. women with evidence of possible Zika virus infection during pregnancy

Understanding the risk of birth defects associated with Zika virus infection during pregnancy may help guide communication, prevention, and planning efforts. In the absence of Zika virus, microcephaly occurs in approximately 7 per 10,000 live births. Honin et al. tried to estimate the preliminary proportion of fetuses or infants with birth defects after maternal Zika virus infection by trimester of infection and maternal symptoms. Among 442 completed pregnancies in women (median age 28 years, range 15–50 years) with laboratory evidence of possible recent Zika virus infection, birth defects potentially related to Zika virus were identified in 26 (6%, 95%CI, 4%–8%) fetuses or infants. There were 21 infants with birth defects among 395 live births and 5 fetuses with birth defects among 47 pregnancy losses. Birth defects were reported for 16 of 271 (6%, 95%CI 4%–9%) pregnant asymptomatic women and 10 of 167 (6%, 95%CI 3%–11%) symptomatic pregnant women. Of the 26 affected fetuses or infants, 4 had microcephaly and no reported neuroimaging, 14 had microcephaly and brain abnormalities, and 4 had brain abnormalities without microcephaly. Reported brain abnormalities included intracranial calcifications, corpus callosum abnormalities, abnormal cortical formation, cerebral atrophy, ventriculomegaly, hydrocephaly, and cerebellar abnormalities. Infants with microcephaly (18/442) represent 4% of completed pregnancies. Birth defects were reported in 9 of 85 (11%, 95%CI 6%–19%) completed pregnancies with maternal symptoms or exposure exclusively in the first trimester (or first trimester and periconceptional period), with no reports of birth defects among fetuses or infants with prenatal exposure to Zika virus infection only in the second or third trimester.

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