Overlooked complications of allergic reactions: allergic angina and allergic myocardial infarction

Alerjik reaksiyondan gözden kaçan komplikasyonlar: Alerjik angina ve alerjik miyokard infarktüsü

Allergic reactions to certain drugs like penicillins (1), cephalosporins (2), ranging from urticaria to anaphylactic or anaphylactoid reaction, are increasingly encountered in the daily clinical practice. Recent increasing number of reported cases about the latter minds us that concurrence of acute coronary syndromes with those allergic reactions could be more in number than it was supposed. The drugs, reported to be accounted for allergic reactions and used widely in daily clinical application, are antibiotics, analgesics, antiinflammatory media, corticosteroids, intravenous anesthetics, non-steroidal antiinflammatory drugs, skin disinfectants, thrombolytics, and others (3, 4).

Allergic angina and allergic myocardial infarction, referred as “Kounis Syndrome”, have gained acceptance as a new cause of coronary artery spasm. Two variants of this syndrome were primarily described according to the findings of coronary angiography. Type I variant defines the patient having normal coronary angiography whereas type II requires a quiescent spasm. Two variants of this syndrome were primarily described according to the findings of coronary angiography. Type I variant defines the patient having normal coronary angiography whereas type II requires a quiescent coronary artery spasm manifesting as Kounis syndrome. Acta Cardiologica 2005; 60: 341-5.

Two cases of IKD are presented in this letter. The patients were young and required intensive care therapy. The first patient presented with ST elevation on derivations II, III and aVF. While interrogating the risk factors of premature coronary artery disease, we learned a penicillin drug was administered intravenously just before the clinical setting has initiated. We immediately transport the patient to the angiography laboratory to perform a primary percutaneous coronary angioplasty to the infarct related artery. Coronary arteries were completely normal on the angiography. The patient was transported to the intensive care unit on the support of inotropic medications. We planned the medical management in the intensive care unit with low molecular weight heparin (enoxaparin 80 mg/0.8 ml 2x1 SC), corticosteroid (methylprednisolone 80 mg 2x1 IV), mast cell stabilizer (ketotifen 2 mg tb, 1x1 PO), histamine (H2) antagonist (famotidine 20 mg tb 2x1 PO). ST segment elevation was regressed in a few hours despite the rise of cardiac markers minimally (eg cardiac troponin, CK-MB) as diagnostic criteria of myocardial infarction.

Case 1. A one-year-old boy with a history of 6-day fever and rash and reddening of lips for 2 days was admitted to the hospital. He was...