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

### *Alerjik reaksiyonların gözden kaçan komplikasyonları: 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, antineoplastics, contrast 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 pre-existing atheromatous disease (5). In type I variant acute allergic reaction may progress either to vasospastic angina or acute myocardial infarction (eg. ST elevated or non-ST elevated myocardial infarction). This may reflect an endothelial dysfunction or microvascular angina. In type II variant spasm of coronary artery may result in atheromatous plaque disruption and thus manifests as an acute myocardial infarction.

In this letter, we aimed to remind allergic angina and allergic myocardial infarction by presenting a young patient suffered acute anterior-inferior ST elevated myocardial infarction following intravenously penicillin administration. The patient was a 21 years old male. He presented to the emergency service with loss of consciousness, clammy and cold skin, respiratory distress with bronchospasm and with all signs of cardiogenic shock. The electrocardiogram obtained following the initial interventions to provide airway, breathing and circulation was revealing hyperacute T wave on V2-V6 and ST segment 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. 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.

Aforementioned complications and allergic myocardial angina are not uncommon clinical conditions in daily practice because of high likelihood of developing allergic reaction to a wide range of drugs administered intravenously. It will certainly be lifesaving to the sufferer if those complications be minded, evaluated and intervened earlier.

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## Incomplete Kawasaki disease: a pediatric diagnostic conflict

### *İnkomplet Kawasaki hastalığı: Pediatrik tanısal zorluk*

Dear Editor,

Kawasaki disease (KD) can be difficult to diagnose; there is no diagnostic laboratory test and there is a 25% chance of serious cardiovascular damage if the treatment is not administered at an early stage. No difficulty exists in diagnosis in the patients with full criteria, but some patients who don't fulfill the criteria have been diagnosed as having "incomplete" Kawasaki disease (IKD). It should be considered in all children with unexplained fever for >5 days associated with 2 or 3 of the principal clinical features of KD. (1, 2).

Two cases of IKD are presented in this letter.

**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

diagnosed with tonsillitis and prescribed antibiotics. Because of persistent fever, the physician referred him to our hospital. The results of the laboratory tests conducted were as follows: hemoglobin: 12 g/dl, white blood cell (WBC): 16.700 mm<sup>3</sup>, thrombocyte: 820.000 mm<sup>3</sup>, erythrocyte sedimentation rate (ESR) 125 mm/h, C-reactive protein (CRP): 84 mg/dl. All viral markers were negative. Two-dimensional echocardiogram was normal. He was diagnosed with IKD and therefore administered 2 gr/kg single dose intravenous immunoglobulins (IVIG) and 3 mg/kg aspirin. Rapid improvement of the general condition and disappearance of fever were observed two days later.

**Case 2.** A four-year-old boy had fever and abdominal pain for 5 days. He was diagnosed with urinary infection at another hospital. Because of fever and presence of jaundice, he was diagnosed with viral hepatitis and transferred to our hospital. Hematological findings were as follows; Hb: 9.6 g/dl, WBC: 8.000 mm<sup>3</sup>, thrombocyte: 245.000 mm<sup>3</sup>, ESR: 115 mm/h, CRP: 200mg/dl, AST/ALT: 124-240 IU/L, total/conjugated bilirubin: 5.2 -3.2 mg/dl, excess leukocyte in the urine exam. All viral markers and cultures were negative. Echocardiogram was normal. The diagnosis was IKD, so the patient was administered 2gr/kg IVIG and 3 mg/kg aspirin. After the administration of IVIG, fever disappeared. Three days later, maculopapular rash was observed in the perineal area with desquamation. After 6 days, biochemistry values were normalized.

The IKD may display different symptoms such as jaundice like in the second case. Rash, which was not present at the beginning, may be easily confused with the drug eruptions, just like in the first case. Sterile pyuria may be mistaken for partially treated urinary tract infection with sterile urine cultures like in the second case.

Kawasaki Disease Research Committee published new diagnostic criteria in 2002 (3). Major alterations are interpretation of cases with 4 or fewer febrile days shortened by early IVIG treatment and the importance of IKD. Cases with 4 or less febrile days shortened by early IVIG treatment were proposed to be equivalent to cases with 5 or more febrile days in the previous criteria (4). Then, the American Heart Association published the latest diagnostic criteria and the importance of IKD was emphasized in this guide. The conventional diagnostic criteria should be viewed as guidelines that are particularly useful in preventing overdiagnosis but may result in failure to recognize incomplete forms of illness.

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## Transposition of the great arteries in a newborn whose mother was treated with carbamazepine during pregnancy

### *Gebelikte karbamazepin kullanan annenin bebeğinde büyük arter transpozisyonu*

A male infant born by cesarean section from the first pregnancy of a 26-year-old mother as the first living child on the 38th gestational week with a birth weight of 2800 gram, and followed-up at the hospital of birth for cyanosis following birth for three days was referred to our hospital when he was 14 days old due to continuing cyanosis. There was no relevant family history for consanguinity or congenital heart disease. The mother was epileptic and did not have any maternal history of interest except for one seizure and carbamazepine usage during pregnancy. There was no maternal gestational diabetes. The baby's antenatal follow-up and fetal echocardiography had been normal.

Physical examination revealed a body temperature of 36.5°C, apical heart rate of 130/min, respiratory rate of 38/min and a body weight of 2550 grams. The only positive finding was cyanosis with normal cardiac and respiratory findings. Blood gas analysis revealed a PaO<sub>2</sub> value of 40 mmHg. There was no significant change in the oxygen saturation following 100% oxygen administration. Anteroposterior chest X-ray showed egg-shaped heart with narrowing of the mediastinum and mild cardiomegaly (Fig. 1). Echocardiography showed transposition of the great arteries (TGA) and the patient was referred to another center for surgery.

As maternal seizures during pregnancy are associated with an increased risk of miscarriage, preterm labor, intracranial hemorrhage in the fetus, stillbirth, and possible developmental or learning difficulties; control of seizures with maintaining appropriate anticonvulsant therapy during pregnancy is essential. (1). Many of the antiepileptic drugs are established teratogens. The risk of birth defects in offspring of mothers who have seizure disorders treated with antiepileptic drugs is two to three times that of the general population (1,2). The factors causing this increase are not well defined (1). The risk of malformations has been shown to be higher following exposure to anticonvulsant drugs during polytherapy than for monotherapy. Carbamazepine is the first choice for monotherapy in an epileptic mother during pregnancy. Although many authors have suggested that carbamazepine is not a teratogen and it is the first choice for women who require an anticonvulsant during pregnancy, some studies have shown that carbamazepine is associated with major malformations (3, 4). Carbamazepine is a folic acid antagonist and increases the risk of



**Figure 1.** Anteroposterior chest X-ray shows mild cardiomegaly, narrow mediastinum and normal pulmonary blood flow