A 33-year-old man was admitted to the internal medicine department with fever and fatigue. He was treated with intravenous (IV) methimazole and cefazolin for an upper respiratory tract infection. During his treatment, he complained of a self-terminating chest discomfort. The electrocardiogram (ECG) showed 0.5 mm ST segment elevation in I-II-III-aVF, and V3 through V6. Troponin I assay revealed elevated levels (4.15 ng/mL). He was then transferred to the coronary care unit. A transthoracic echocardiography (TTE) examination showed interposterolateral wall hypokinesia and normal left ventricular systolic functions with ejection fraction of 55%. His past medical history was unremarkable with no cardiovascular risk factors. Standard treatment for ACS was initiated. Treatment of upper respiratory tract infection was also continued with orally administered amoxicillin-clavulanic acid. His chest discomfort was relapsed in spite of the anti-anginal therapy regimen. A repeated ECG showed prominent ST segment elevation in the same leads (Fig. 1). A single 2 mg IV dose of morphine was administered for relieving his pain. Shortly after administration of morphine, erythematous lesions evolved on most part of his skin, and chest discomfort increased. An allergic condition was suspected, and he was given IV saline, methylprednisolone, famotidine and phenirammine maleate. Then, his chest discomfort relieved within minutes. By the way, the patient’s past medical history was negative for atopic diseases. The following day, a left heart catheterization demonstrated normal left and right coronary arteries (Fig. 2 A-B and Video 1-2. See corresponding video/movie images at www.anakarder.com). Following catheterization, his medical treatment was continued with orally nitrate, histamine 1 and 2 receptor blockers. It was probable that cefazolin caused the first anginal attack, clinical manifestation relapsed because of another β-lactam antibiotic, and administration of IV morphine aggravated the clinical picture. The measurements of serum specific IgE directed to the β-lactams, serum chymase and tryptase levels were not performed (due to a lack of laboratory support). The patient refused to undergo an allergy testing. The limitations of the presented case may be lack of these tests. Both repeated TTE and ECG were in normal limits. No recurrent angina was observed after the catheterization procedure, and the patient was discharged on the fourth day without any symptoms. Our final diagnosis was Kounis syndrome secondary to the β-lactam antibiotics. The coincidence of evident hypersensitivity reaction following IV administration of morphine was considered as aggravation of Kounis syndrome.

A case of Kounis syndrome aggravated by administration of morphine

Morfin uygulanmasıyla ağırlaşan bir Kounis sendromu vakası

Especially in the young ages, non-atherosclerotic coronary artery diseases must be considered in acute coronary syndromes (ACS). In the case presented here, morphine unmasked Kounis syndrome (1).

References

Activation of mast cells and the systemic release of histamine are common side effects of morphine. In addition to other side effects, cutaneous changes may occur as manifested by peripheral vasodilation and flushing of the skin with urticaria, a response to the histamine releasing properties of the morphine. This case calls attention to the Kounis syndrome which was induced by two other β-lactam antibiotics and aggravated by morphine.

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References


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Echocardiographic assessment in children with Gaucher disease receiving enzyme replacement therapy

Gaucher hastalığı olan ve enzim replasman tedavisi alan çocukların ekokardiyografik değerlendirilmesi

Cardiac involvement is rare in Gaucher disease and may be in the form of pulmonary hypertension, constrictive pericarditis, pericardial calcifications, various valvular lesions and infiltration of the myocardium. Pulmonary hypertension in Gaucher disease is not common but it is shown to be secondary to interstitial or perivascular infiltration of Gaucher cells or primary in patients exposed to enzyme replacement therapy (ERT). Valvular lesions are seen as calcifications of aortic and mitral valves and these are mainly reported in patients with D409H homozygosity (1).

We investigated echocardiographic findings in our pediatric patients while receiving ERT. Patients with Gaucher disease who received ERT for at least six months were assessed. A Vingmed (GE, Horten, Norway) Vivid-5 echocardiography equipment with 2.5, 3.5 and 5 MHz transducers were used for echocardiographic evaluation. M-mode, 2-dimensional, color Doppler, pulsed wave (PW) Doppler and continuous wave (CW) Doppler examinations were performed in each patient. Echocardiographic assessment was done by the same pediatric cardiologist and tricuspid regurgitation gradient of 30 mmHg was considered as upper limit of normal as it was known to reflect pulmonary pressure in the absence of ventricular outflow obstruction. Other abnormal findings were also recorded.