development right ventricular failure in patients with RA is not well known. The presence both of these diseases in this patient bring to our mind the role of the inflammation or the presence of shared genetic predisposition. The role of infection or inflammation in the etiology of ARVD was proposed and thought is the remnant of myocarditis. Fontaine et al. (1) in examining 27 patients, they found inflammatory infiltrations in eight of them. In other site of view, the major genetic risk factor for RA is class II histocompatible complex alel HLA-DR4. Meanwhile ARVD shows different type of family genetic transmission in which the plakophilin (PKP2) mutation has been found as a major cause of ARVC/D with prevalence of mutations among unrelated index cases as high as 43% (3, 4).

It is unexplained whether unknown genetic mutation or the effect of inflammation played a main role in the etiology of our case. However, it is logical to keep in our mind the diagnosis of ARVD in-patient with rheumatoid arthritis when presented to us with malignant ventricular arrhythmias. In these conditions clinical evaluation with meticulous echocardiography examination, cardiac MRI and right ventriculography should be done and if it is possible to be supported by genetic study.

Diyar Köprülü, Halit Zengin[1], Zeydin Acar[2], Sabri Demircan[1]
Clinic of Cardiology, Ordu State Hospital, Ordu
[1]Department of Cardiology, Faculty of Medicine, Ondokuz Mayıs University, Samsun
[2]Clinic of Cardiology, Ahi Evren State Hospital, Trabzon-*Turkey*

References

1. Fontaine G, Fontaliran F, Lascault G, Frank R, Tonet J, Chomette G, et al. Congenital and acquired right ventricular dysplasia. Arch Mal Coeur Vaiss 1990; 83: 915-20.
2. Harris: Kelley's Textbook of Rheumatology, 7th ed, 696-97.
3. Dalal D, Molin LH, Piccini J, Tichnell C, James C, Bomma C, et al. Clinical features of arrhythmogenic right ventricular dysplasia/cardiomyopathy associated with mutations in plakophilin-2. Circulation 2006; 113: 1641-9. [CrossRef]
4. van Tintelen JP, Entius MM, Bhuiyan ZA, Jongbloed R, Wiesfeld AC, Wilde AA, et al. Plakophilin-2 mutations are the major determinant of familial arrhythmogenic right ventricular dysplasia/cardiomyopathy. Circulation 2006; 113: 1650-8. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Diyar Köprülü, Ordu Devlet Hastanesi, Kardiyoloji Kliniği, Ordu-*Türkiye*
Phone: +90 452 234 95 44 E-mail: drkoprulu@gmail.com
Available Online Date/Çevrimiçi Yayın Tarihi: 07.02.2012

doi:10.5152/akd.2012.051

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).

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 inferoposterolateral 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, erythematosus lesions evolved on most part of his skin, and chest discomfort increased. An allergic condition was suspected, and so he was given IV saline, methylprednisolone, famotidine and pheniramine 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.

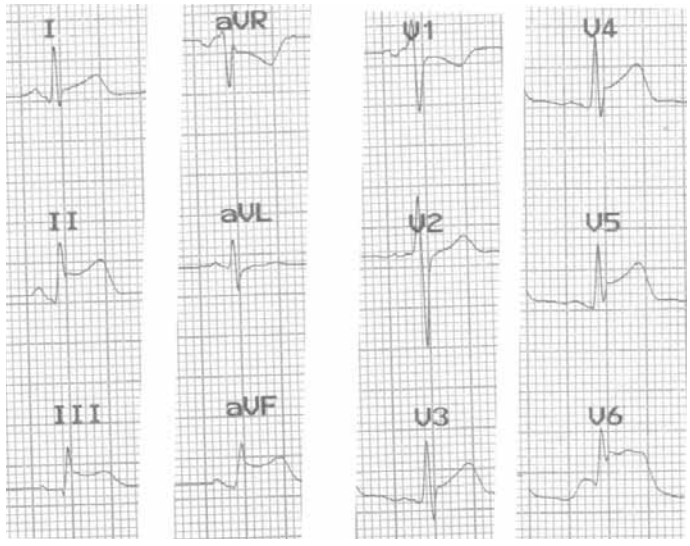

Figure 1. Prominent ST segment elevation in electrocardiogram leads I-II-III-aVF, and V3 through V6

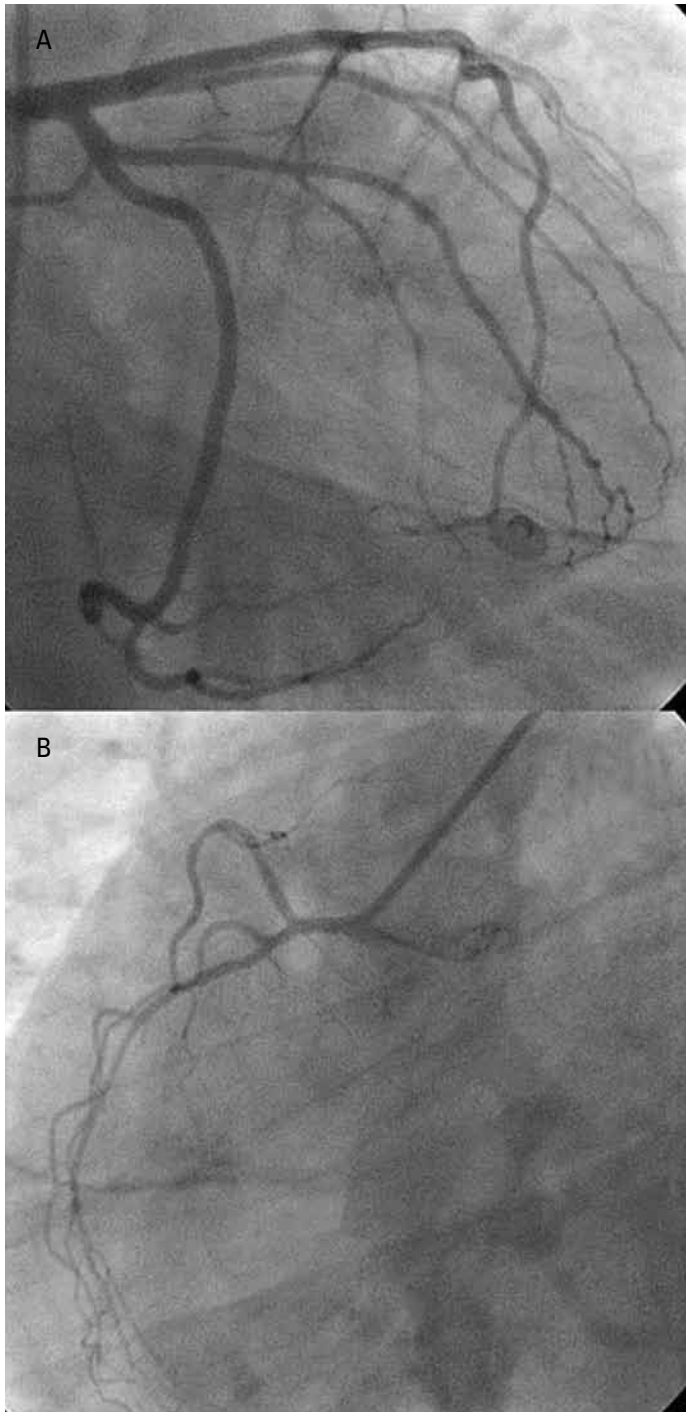

Figure 2. Coronary angiography images of normal left (A) and right coronary (B) arteries

The concurrence of ACS with conditions associated with allergic or hypersensitivity and anaphylactic or anaphylactoid reactions constitutes the Kounis syndrome (1). Two variants of Kounis syndrome have been described (2). The type I variant includes patients with normal coronary arteries without predisposing factors for coronary artery disease. The type II variant includes patients with active or quiescent preexisting atheromatous disease. The type III variant has been proposed recently (3). A number of conditions, several drugs, foods and venom and toxins have been reported as capable of inducing Kounis syndrome (1, 2).

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 vasodilatation 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.

Abdullah Uluçay, Mehmet Faruk Aksoy
Clinic of Cardiology, Defne Hospital, Hatay-*Turkey*

Video 1, 2: Angiographic views of the left and right coronary arteries

References

1. Kounis NG. Kounis syndrome (allergic angina and allergic myocardial infarction): a natural paradigm? Int J Cardiol 2006; 110: 7-14. [CrossRef]
2. Nikolaidis LA, Kounis NG, Gradman AH. Allergic angina and allergic myocardial infarction: a new twist on an old syndrome. Can J Cardiol 2002; 18: 508-11.
3. Biteker M. A new classification of Kounis syndrome. Int J Cardiol 2010; 145: 553. [CrossRef]

Address for Correspondence/Yazışma Adresi: Dr. Abdullah Uluçay,
Özel Defne Hastanesi, Kardiyoloji Bölümü, 31030, Hatay-*Türkiye*
Phone: +90 326 221 11 00 Fax: +90 326 221 44 45
E-mail: ulucaytr@hotmail.com
Available Online Date/Çevrimiçi Yayın Tarihi: 07.02.2012

doi:10.5152/akd.2012.052

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.