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Acute myocardial infarction complicated by sudden cardiac arrest in a patient with ulcerative colitis

Ülseratif kolitli hastada ani kardiak arrest ile komplike olmuş akut myokard infarktüsü

To the Editor,

Arterial and venous thromboembolism is a common complication of inflammatory bowel disease (IBD) (1). This complication most commonly occurs in the lower extremities (1, 2); however, coronary embolization is very rare (1, 3, 4). We report a 50-year-old man with ulcerative colitis who had inferoposterior myocardial infarction.

A 50-year-old man was admitted to our clinic with bloody diarrhea and abdominal pain. He had a history of bloody defecation with mucus for about one month, 15-20 times per day on a limited scale. The patient had no risk factor for coronary artery disease except male gender and age. On physical examination, his blood pressure was 110/80 mmHg and pulse rate was 76 beats/min, and the bowel sounds were slightly increased. ECG and chest X-ray were normal. Laboratory parameters were as follows: Fasting blood glucose 105 mg/dl, total cholesterol 88 mg/dl, high density lipoprotein (HDL)-cholesterol 24 mg/dl, low density lipoprotein (LDL)-cholesterol 49 mg/dl, hematocrit (Hct): 37%, platelet (Plt): 639,000 /uL, and C-reactive protein (CRP) 16 mg/dl. Other biochemical para-

meters were normal. Microscopic analysis of the feces showed abundant leukocytes and erythrocytes, but there was no bacterial growth. Colonoscopy revealed that the mucosal appearance of the left colon was hyperemic, edematous, granular,

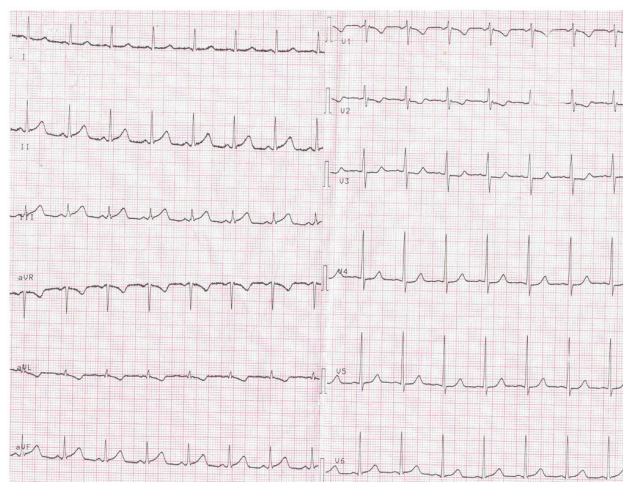


Figure 1. ECG showing ST elevations in the inferior leads and increased R wave amplitude in V2.

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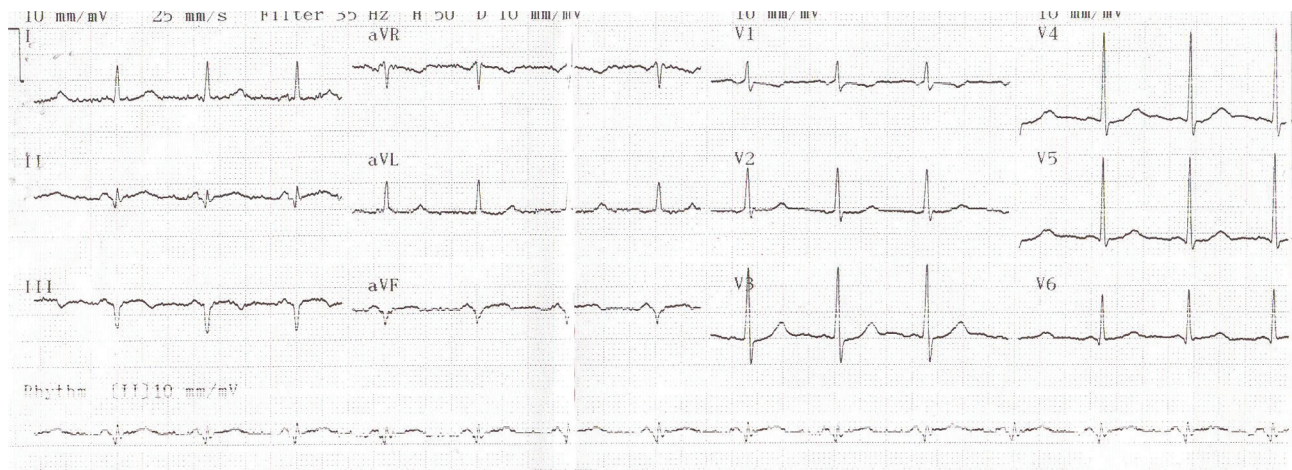


Figure 2. Follow-up ECG taken in the coronary care unit indicates Q waves in inferior leads and increased R wave amplitudes in V₁₋₂.

and fragile. The biopsy taken from this region was compatible with ulcerative colitis. Mesalamine, 4 g/day, was started. Methylprednisolone, 60 mg/day, was added in the first week since there was no improvement clinically. On the first day of steroid treatment, after receiving the first morning dose of 40 mg, he had sudden cardiac arrest. Cardiopulmonary resuscitation was done. ECG taken after the resuscitation showed ST elevation in the inferior leads and increased R wave amplitude in V₂ (Figure 1). Cardiac markers were as follows: troponin T >22.4 ng/ml (normal <0.03), mass CK-MB 111.5 ng/ml (normal 0-4.94), fibrinogen 4.531 g/L (normal 1.8-3.5), and D-dimer: 313 mg/dl (normal 63-298). The patient was transferred to the cardiology clinic with the diagnosis of acute inferoposterior myocardial infarction. Metoprolol and low molecular weight heparin were started, and methylprednisolone and mesalamine treatments were continued. Since he had no chest pain and ECG showed apparent Q waves, no thrombolytic therapy was given (Figure 2). Coronary angiography revealed a 20% stenosis on the left anterior descending coronary artery. The other coronary arteries were normal. Left ventriculography showed inferoposterior hypokinesia. Since circumflex and right coronary arteries were normal, it was speculated that the cause of myocardial infarction in this patient was thromboembolism. He was discharged with metoprolol, angiotensin converting enzyme inhibitor, methylprednisolone, and mesalamine treatment.

Thromboembolism is an extraintestinal manifestation and an important cause of mortality in IBD.

The risk of thromboembolism appears to be multifactorial and related to mucosal inflammatory activity in most patients (5). Cardiac complications of IBD are rare; the most common complication is pericarditis. Drugs have been implicated in the etiology of cardiac complication in IBD. Particularly, steroid therapy exacerbates diabetes, hypertension, congestive heart failure, and osteoporosis, or causes paradoxical depression. Baty et al. (3) suggested that high-dose steroid therapy given in a very short time may cause cardiovascular complications. Our patient had received only 40 mg methylprednisolone prior to his sudden cardiac arrest. Since we continued the steroid therapy, and he experienced no problem during the follow-up, we do not think that there was a relationship between the steroid therapy and myocardial infarction in our patient. Generally, thrombosis is seen with an increase of the hypercoagulability markers as thrombin, antithrombin III, prothrombin, and fibrinogen and the numbers of the platelets (6). In fact, the level of fibrinogen was above normal after the cardiac arrest and the platelet number was high, at $639 \times 10^3/\mu\text{L}$. Circumflex and right coronary arteries were normal in our patient; therefore, it was speculated that the cause of inferoposterior myocardial infarction in this patient was thromboembolism.

In conclusion, ulcerative colitis may rarely cause coronary thromboembolism. We report a patient with ulcerative colitis who presented with acute inferoposterior myocardial infarction, which was speculated to have been caused by coronary thromboembolism.

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