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CASE REPORT

Acute myocardial infarction in a patient with sickle cell trait

Orak hücre taşıyıcılı bir hastada akut miyokart enfarktüsü

Oğuz Akkuş, M.D., Mustafa Topuz, M.D., Ali Deniz, M.D.[#]

Department of Cardiology, Adana Numune Training and Research Hospital, Adana, Turkey
[#]Department of Cardiology, Çukurova Universty Faculty of Medicine, Adana, Turkey

Summary– Presently described is the rare complication of sickle cell trait (HbAS) with acute inferior myocardial infarction in a young adult patient. Angiogram revealed microemboli in the distal thin segment of the circumflex (Cx) artery. Anticoagulant and anti-aggregant therapies were initiated to restore Cx artery flow. HbAS was detected as an underlying pathology. ST segments returned to the isoelectric line and the patient became asymptomatic.

Özet– Bu yazıda, genç erişkin bir orak hücre taşıyıcısında nadir bir komplikasyon olarak ortaya çıkan akut inferiyor miyokart enfarktüslü olgu sunuldu. Anjiyografide sirkumfleks arter distalinde mikroemboliler tespit edildi. Kan akımını iyileştirmek için anjiyoplasti ve stent uygulanmaksızın tıbbi tedavi başlandı. Altta yatan neden olarak orak hücre taşıyıcılığı saptandı. Elektrokardiyogramda ST segment yükseklikleri izoelektrik hatta döndü ve hastanın semptomları geriledi.

Sickle cell disease (SCD) (HbSS: homozygous; HbAS: heterozygous) is a rare, inherited, chronic, hemolytic red cell disorder that affects many systems.[1] Ischemic complications are the most common causes of morbidity and mortality in these patients.[2–4] In contrast to sickle cell trait (HbAS), which is commonly regarded as a benign disorder with the exception of rare cases, HbSS may be associated with shortened life expectancy due to vascular occlusions and myocardial infarction. Vulnerable atherosclerotic plaque and subsequent thrombosis generally do not occur in SCD patients with myocardial infarction. Myocardial damage is most commonly caused by microvascular disease related to vaso-occlusion.[5–7]

Abbreviations:

Cx	*Circumflex*
HbAS	*Sickle cell trait*
HbSS	*Homozygous sickle cell disease*
SCD	*Sickle cell disease*

CASE REPORT

A 27-year-old female patient was admitted to the emergency department with chest pain that had begun 2 hours prior. Blood pressure was 90/60 mmHg, and pulse rate was 73 beats per minute. Physical examination was normal, and initial electrocardiogram in the emergency ward demonstrated acute (inferior wall) ST elevation myocardial infarction with ST depression on anterior leads (Figure 1). Because clinical scenario was compatible with acute inferior ST elevation myocardial infarction, aspirin 300 mg and ticagrelor 180 mg were orally administered, and the patient was immediately transferred to the catheterization laboratory. Angiogram revealed microemboli in the distal segments of the circumflex (Cx) artery (Figure 2). The right and left coronary arteries were normal, and thrombus formation and spontaneous recanalization were suspected. Decision to initiate therapy of nitric oxide and anti-platelet agents was made. Intracoronary tirofiban was administered, as was infusion and antithrombotic therapy (acetylsalicylic acid and low-molecular-weight heparin), and the patient was transferred to the intensive coronary care unit. ST segments returned to the isoelectric line, and symptoms were relieved.

According to her history, the patient had no chronic disease, and had not been hospitalized due to cardiovascular disease. She did not smoke, and there was no

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Correspondence: Dr. Mustafa Topuz. Süleyman Demirel Bulvarı, Kurttepe Mevkii, Seyhan Uygulama Hastanesi, 01150 Adana, Turkey.
Tel: +90 322 - 355 01 01 e-mail: mtpuz@hotmail.com

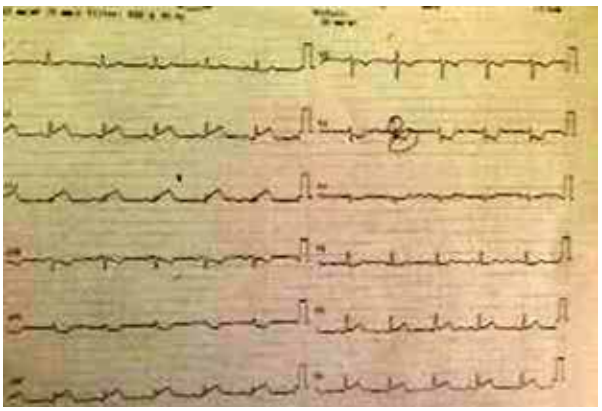

Figure 1. Acute inferior-posterior myocardial infarction.

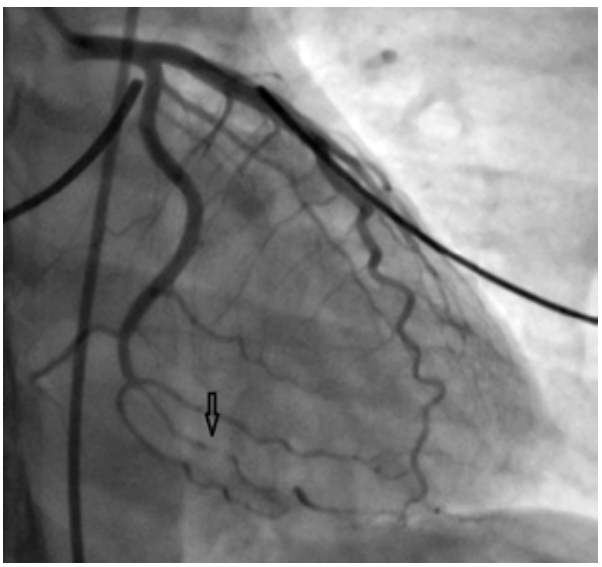

Figure 2. Microemboli at coronary angiography.

first-degree family history of cardiovascular disease. It was suspected that the complex findings may have been related to an underlying silent disease. Only mild anemia was detected on coagulation parameters (platelet count, international normalized ratio, factor V Leiden mutation, protein CS, antithrombin III, etc.), and optical coherence tomography test was normal. HbAS was identified via peripheral blood smear (Figure 3) and hemoglobin electrophoresis (Table 1). The patient was discharged with 100 mg acetylsalicylic acid.

DISCUSSION

Intravascular thrombosis is a well-known complication of HbSS and is associated with myocardial infarction. HbAS is thought to be a benign carrier condition, and is rarely associated with heightened coagulation tendency and shortened life expectancy in ordinary cases. Although HbAS patients have a higher quality of life with less cause for complaint than HbSS patients, they too may experience coronary thrombosis and myocardial infarction, as in the present case.

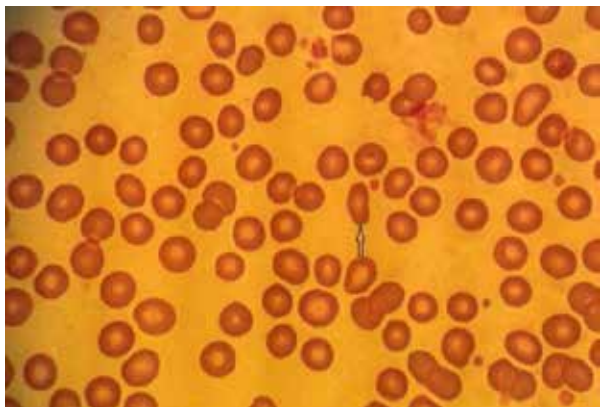

Figure 3. Sickle cell on peripheral blood smear.

Myocardial infarction in SCD is more likely caused by microvascular disease than epicardial coronary disease. The exact mechanism of myocardial infarction in SCD is unknown, though platelet abnormalities may play a role, given the combined stress of anemia-rheological, morphological, and biochemical effects of sickle cells. It is proposed that, in the present patient, tendency to hypercoagulation, increased arterial stiffness, and early vascular aging may have played a surrogate role in the coronary thrombus.[3]

Our patient had no history of exposure to foreign materials, drugs, or solvent or detergent chemicals, and no history of insect bite that may have played a crucial role in triggering heart attack.[8] Myocardial infarction due to coronary thrombosis should also be considered in SCD patients admitted with chest pain. It seems more appropriate that SCD patients with chest pain be closely followed in the intensive coronary care unit. Hypoxic patients should be promptly hydrated and oxygenated.[9] Absolute treatment of myocardial infarction in SCD is debated. Because of the initial Thrombolysis in Myocardial Infarction grade III flow to the distal vessel, the vessel diameter, and the partial resolution of the flow after intracoronary tirofiban, we agreed that the best clinical approach would be follow-up with anti-aggregant and tirofiban therapy. Control angiogram was not performed due to distal

thin vessel and asymptomatic course of patient. In addition to anti-aggregant agents, nitric oxide can be used during this period. Nitric oxide has been shown, in an in vitro study, to inhibit sickle erythrocyte adherence to the endothelium, and has been successfully used in the treatment of SCD with acute chest syndromes.[10]

Conclusion

Although HbAS is thought to be a benign carrier condition, its course may be complicated by myocardial infarction due to coronary thrombus. Hence, secondary etiologies must be kept in mind when encountering HbAS in a young patient with acute coronary syndrome.

Conflict-of-interest issues regarding the authorship or article: None declared.

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Keywords: Acute coronary syndrome; microemboli; sickle cell trait.

Anahtar sözcükler: Akut koroner sendrom; mikroemboli; orak hücre taşıyıcılığı.