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Recurrent ST- Segment Elevation Myocardial Infarction a Young Male Patient With Essential Thrombocytosis

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İç Hastalıkları

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Özet

Esansiyel trombositoz (ET) (diğer isimleri, esansiyel trombositemi, idiopatik trombositoz, primer trombozis), sebebi bilinmeyen anormal megakaryosit proliferasyonu ve trombosit sayısında persistan artışla karakterize kronik myeloproliferatif bir hastalıktır. ET de koroner arter oklüzyonu ve miyokard infaktüsü (Mİ) nadir vakalar olarak bildirilmiştir. Bizim vakamız, ET nin ilk klinik bulgusu olarak akut anterior duvar Mİ ile gelip perkütan koroner girişim (PKG) ile başarılı bir şekilde tedavi edilen 30 yaşında erkek hastadır. Taburculuğundan 10 gün sonra, akut inferior duvar Mİ ve kardiyojenik şokla acil servisimize tekrar başvurmuştur.

Anahtar kelimeler: *Esansiyel Trombositoz, Miyokard İnfaktüsü Perkütan Koroner Girişim*

Abstract

Essential thrombocytosis (ET) (other designation include, essential thrombocythemia, idiopathic thrombosis, primary thrombosis) is a chronic myeloproliferative disorder of unknown origin characterized by a persistent increase in the platelet count and abnormal megakaryocyte proliferation. Coronary artery occlusion and myocardial infaction (MI) during ET are reported rarely and as single cases. We report a 30 year old-male patient an acute anterior wall MI has been successfully treated with percutaneous coronary intervention(PCI) as the first clinical signs of ET. After discharged 10 days later he was admitted to our emergency service because of acute inferior wall MI and cardiogenic shock

Keywords: *Essential Thrombocytosis, Myocardial Infarction Percutaneous Coronary Intervention*

Introduction

Esansiyel trombositoz (ET) is a clonal disorder of the myeloid stem cell that causes pathologic expansion of the megakaryocytic elements in the bone marrow, with a persistent increase in the platelet count. Platelet dysfunction in ET results in both hemorrhage and thrombosis[1]. It is commonly believed that a high platelet count must cause intravascular stasis and thrombosis. However very high platelet counts are associated primarily with hemorrhage while platelet counts of less than a million are often associated with thrombosis. Thrombosis is also the major cause of morbidity and mortality[2]. Coronary artery involvement leading the acute coronary syndrome is a rare complication.

Case Report

A 30 years old male patient without know cardiac disease admitted to the hospital with severe chest pain and dyspnea of 6 hour duration. He was nonsmoker and did not have a family history of heart disease or hemorrhagic disorder. Electrocardiography showed ST segment elevation in leads I, aVL, and V3-6 consisted with acute anterior myocardial infraction. The vital signs on admission were as follows: blood pressure 140/80 m/ Hg, heart rate of 82 bpm regular, were no abnormal heart sounds and murmur, temperature 36.5 C there. Routine laboratory evaluation revealed a platelet count of 1358 x 10 mm, white blood cell counts 24,000 / mm and hemoglobin level

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11.7 g/dl. Other laboratory studies were unremarkable. Aspirin 300 mg, clopidogrel 600 mg, i.v. metoprolol and enoxaparin 0.8 ml, was started immediately. Emergent coronary angiography revealed total thrombotic occlusion of the proximal left anterior descending artery (LAD) before the first diagonal branch (Figure 1A).

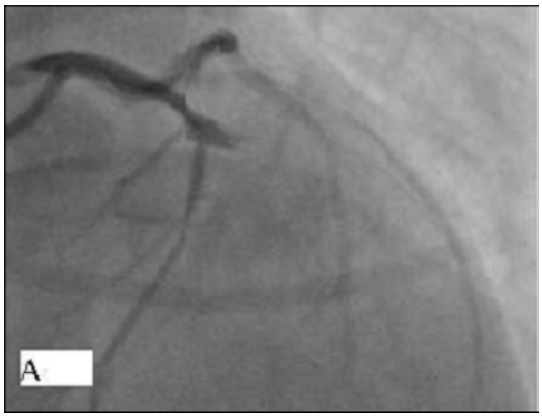

Figure 1A

Coronary angiography shows thrombotic narrowing of the proximal LAD

The circumflex (Cx) and right coronary arteries (RCA) were normal angiographically . The lesion was predilatated with 2,5x14 mm balloon and covered by a 3,5x 14 mm bare metal stent no residual lesion continued. A TIMI grade 3 flow was obtained (Figure 1B).

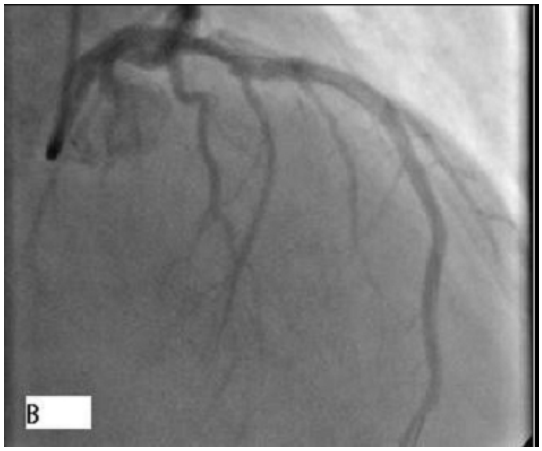

Figure 1B

After angioplasty and stent implantation shows TIMI grade 3 flow

The patient was discharged by taking the treatment of aspirin, clopidogrel, metoprolol and ramipril. He admitted to the emergency service with severe chest pain, dyspnea, nausea and sweating 10 days after discharged. Physical examination revealed blood pressure 70/40 mm/ Hg heart rate 110 bpm, bibasilar inspiratory crackles and cardiogenic shock . Electrocardiography showed ST segment elevation in leads II, III and aVF consisted with inferior wall myocardial infraction. I.v. tirofiban was started and emergent coronary angiography revealed total thrombotic occlusion without atherosclerotic plaques of the distal RCA (Figure 2).

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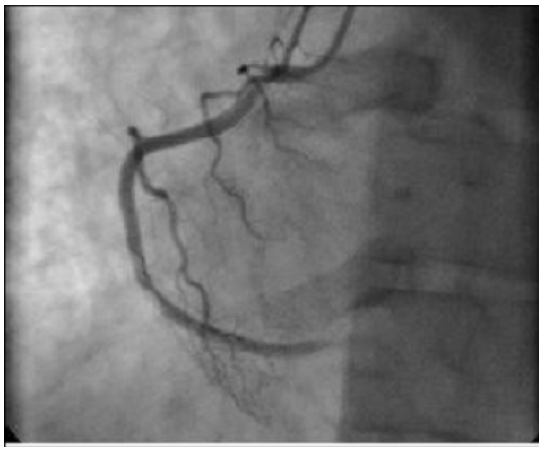

Figure 2

Coronary angiography shows thrombotic narrowing of the distal RCA

The Cx and LAD were normal. Coronary angioplasty were performed and TIMI grade 3 flow was obtained. Laboratory evaluation revealed a platelet count of 1316 x 10 mm, hemoglobin level 10,4 g/ dl, white blood cell counts 23,3 / mm, troponin I 3,64 ng/ ml. Hematology consultation and bone marrow biopsy was performed. Than hydroxyurea treatment was added. An echocardiography performed on the revealed inferior wall, posterior septum and anterior wall severe hypokinesis with a left ventricular ejection fraction of % 35. His platelet count is kept stable at approximately 600 x 10 mm after two mounts later and he was no cardiac complaint.

Discussion

ET may result in thrombus formation and development of myocardial infraction due to coronary artery involvement. Arterial thrombosis are reported 3 times more often than venous thrombosis. The most common sites for large arterial vessel thrombosis, in decreasing order of frequency are the heart, brain and extremities[3]. ET is primarily diagnosed in older (50-60 year old) patients, with equal percentages of males and females[4]. A second peak of incidence in younger patients with a female predominance has also been reported at around age 30[5]. Incidence of acute coronary syndrome in patients of the ET was reported % 9.4 and that of MI as more frequent in patients older than 40 years[6]. In MI associated with ET, coronary artery is often occluded with a large amount of thrombosis, thus a careful therapeutic strategy is required for successful revascularization. Coronary artery by-pass surgery, primary angioplasty, intracoronary thrombolytic therapy have been described in the literature[7]. Most of these patients are older than our patients and no one is recurrent myocardial infaction (MI) and ET. Smoking and hypertension are risk factors of arterial ischemic complications in patients with ET[8]. In patients with ET hypertension or smoke can cause the blood platelets facilitate the endothelial injury, which increases the possibility of thrombosis. Our patient was a nonsmoker and no additional risk factor for MI such as hypertension, hyperlipidemia and diabetes mellitus. Second MI wasn't prevented despite of taking a suitable medical treatment after first MI.

Especially, young patients who have acute coronary syndrome should be carefully evaluated for other rarely cardiovascular risk factors. Patients with high platelet counts and ET should be closely pursued. İt shouldn't be forgotten that ET may result in catastrophic complication as MI. Atherosclerotic plaques are not necessary for the development of MI in those patients. Percutaneous coronary intervention with GP II b /III a or thrombotic therapy may be considered for these patient groups.

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References

1. Schafer AI. Bleeding and thrombosis in the myeloproliferative disorders. Blood 1984;64:1-12.
2. Ozben B, Ekmekci A, Bugra Z, Umman S, Meric M: Multiple coronary thrombosis and stent implantation to the subtotally occluded right renal artery in a patient with essential thrombocytosis: A case report with review. J Thromb Thrombolysis 2006;22:79–84
3. Preston FE. Essential thrombocythaemia. Lancet 1982;1:1021.
4. Silverstein MN. Primary or hemorrhagic thrombocythemia. Arch Intern Med 1968;122:18-22.
5. Sanchez S, Ewton A: Essential thrombocythemia. Arch Pathol Lab Med 2006;130:1144–1150.
6. Rossi C, Randi ML, Zerbinati P, Rinaldi V, Girolami A. Acute coronary disease in essential thrombocythemia and polycythemia vera. J Intern Med 1998;244:49-53.
7. Watanabe T, Fujinaga H, Ikeda Y, Higashi T, Murakami M, et al.: Acute myocardial infarction in a patient with essential thrombocythemia who underwent successful stenting: A case report. Angiology 2005;56(6):771–774.
8. Mizuta E, Takeda S, Sasaki N, Miake J, Hamada T, et al.: Acute myocardial infarction in a patient with essential thrombocythemia: Successful treatment with percutaneous transluminal coronary recanalization. Circ J 2005;69(8):1000–1002.