Case Report

Non- ST-elevation Myocardial Infraction in a Patient with Essential Thrombocytosis: Successful Treatment with GP-IIbIIIa Inhibitor

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ABSTRACT

We treated a 37 years old female who has essential thrombocytosis and developed NSTEMI with GPIIb IIIa inhibitors in addition to standard treatment with dual anti platelet therapy, low molecular heparin, beta blocker and ACE inhibitor and had good recovery of left ventricular function without any bleeding complication. Since these patients have both bleeding and thrombotic complications, it is prudent to carefully follow these patients during treatment and on follow up. (Rawal Med J 2008;33:262-263).

Key Words: Essential thrombocytosis, NSTEMI, EF, Von Willibands Factor, coronary angiography, angiotensin converting enzyme inhibitors.

INTRODUCTION

Essential thrombocytosis (ET) (Essential thrombocythemia, Idiopathic thrombocytosis, hemorrhaghic thrombocythemia.) is a clonal disorder of unknown etiology involving multipotent haemopeotic progenitor cell and manifests clinically by the over production of platelets without a definable cause. ET can occur at any age in adults and often occurs without symptoms or disturbance of haemostasis with preponderance in females.[1]

Pulmonary embolism and deep vein thombosis are less common in ET than arterial thrombosis.[2] ET was described as hemorraghic thrombocytosis, but is now well established that thromboembolic complications, especially thrombus in cerebral, peripheral and coronary arteries are more common in patients with ET.[3] Acute coronary syndrome in ET was reported around 9.4% and acute myocardial infarction was more common in patients older than 40 years of age.[4] There is evidence that hydoxyurea is effective in preventing thrombus in ET patients at high risk for systemic artery disease.[5] Patients with dyslipidemia, hypertension, smoking and elevated platelet counts have been treated with low dose aspirin and reduced the coronary thrombosis without increasing the bleeding complications.[4] There are no well established guidelines to treat patients with acute coronary syndrome, with few case reports of treating acute myocardial infarction with GPIIbIIIa inhibitors.[6] We present a case of NSTEMI with ET who was treated with GPIIbIIIa inhibitor, tirofiban.

CASE REPORT

A 37 years old female gravida three visited department of cardiology Sher e Kashmir Institute of Medical Sciences with chief complaint of severe chest pain for last eight hours with crescendo pattern increasing with less than ordinary work. She was nonsmoker, normotensive without any significant family history of heart disease. Her pulse was 84bpm, regular and blood pressure 130/70mm Hg. ECG showed significant ST- T wave changes in V1 to V6. Her hemoglobin was 11.5g/dl, WBC 10.7mm/dl, platelets 946000/mm^3, CK was 350 IU, blood sugar 106mg/dl and serum Troponin levels were high. Von Willebrand factor was elevated. Echocardiography at admission revealed Ejection Fraction of 51% with reduced contractility. Coronary angiography on 5^{th} day of

admission revealed normal coronaries. Patient was treated with aspirn, clopidogrel, enoxaparin, ACEI, β-blockers and IV nitroglycerine. Despite the treatment, she continued with chest pain and was treated with IV tirofibane (GPII bIIIa inhibitor) for 24 hours. This resolved her chest pain with significant clinical improvement. She was placed on hydroxyurea in addition to low dose aspirin/clopidogrel and ACEIs and plate count came down to around 500,000/cmm.

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DISCUSSION

Fewer than 20 cases of angina pectoris and myocardial infarction have been reported in association with ET. The aggressive inhibition of platelet aggregation and production may play an important role in treating ET associated with ischemic syndrome and coronary thrombosis. As our patient had high levels of VW factor, she was placed on hydroxyurea therapy. It is well known that platelets are major source of VW Factor,[6] we tried to control platelet count in our patient. When platelet count exceeds 600,000/cmm, ET may cause coronary thrombosis involving platelet activation as a result of endothelial injury, prolonged arterial spasm with subsequent thrombosis and increased activity of platelets with changes in the glycoprotein of the platelet granules.[7]

Scheffer et al reported a series of seven patients, six with ET. Five of them had unstable angina and two had acute myocardial infraction. These patients were lass than 50 years of age and coronary angiography revealed either normal or single vessel disease. The thrombotic complications were successfully controlled with aspirin and busulfan.[8] This is similar to our patient. We used of hydroxyurea in our patient, in addition to

aspirin/clopidogrel therapy. In conclusion, we used GP IIbIIIA inhibitor successfully in the setting of persisting chest pain and NSTEMI in a patient with ET.

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