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CASE REPORT

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Acute myocardial infarction in a patient with post-splenectomy thrombocytosis: A case report and review of literature

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Abstract

Unlike essential thrombocytosis, which is associated with thrombotic and hemorrhagic complications, reactive thrombocytosis (RT) is usually considered a benign process without thrombotic complications.

We describe a case of acute myocardial infarction in a young heavy smoker with RT following splenectomy. Coronary angiography showed a linear filling defect at mid-part of left anterior descending artery. We performed balloon angioplasty and stenting for this lesion. Aspirin and clopidogrel were administered. His in-hospital course was uneventful and platelet count returned to the normal range at four month follow-up. We concluded that RT may not be an entirely benign process, especially in patients with a history of smoking. Regular monitoring of platelet count, and possibly antithrombotic agents like aspirin prescription for high risk patients with moderate thrombocytosis, may be useful. (Cardiol J 2010; 17, 1: 79–82)

Key words: myocardial infarction, thrombocytosis, angioplasty, splenectomy

Introduction

Thrombocytosis, often found incidentally, is most commonly a reactive or secondary response to chronic inflammation, iron deficiency, active malignancy, infection, trauma, or splenectomy [1]. In a small number of cases however, thrombocytosis may be attributable to a clonal process, such as one of the four well-described classic myeloproliferative disorders: chronic myelogenous leukemia, polycythemia vera (PV), agnogenic myeloid metaplasia, and essential thrombocytosis (ET). Thrombocytosis is a rare cause of myocardial infarction in patients with intrinsically normal coronary arteries, and especially very rare in patients with reactive thrombocytosis [2].

Case report

A 34 year-old male presented to the emergency department with acute retrosternal chest pain radiating to his left arm associated with cold sweat and nausea which woke him at 4 am, three hours before his arrival at emergency department. He had undergone distal pancreatectomy with splenectomy, for chronic pancreatitis and pancreatic pseudocysts 12 days earlier. His past medical history also included migraine headaches. He had also been a heavy smoker. He didn't have a family history of coronary artery disease. He had a blood pressure of 130/80 mm Hg, and heart rate of 80/min. His physical examination showed no pathological findings except for a recent laparatomy scar. His first

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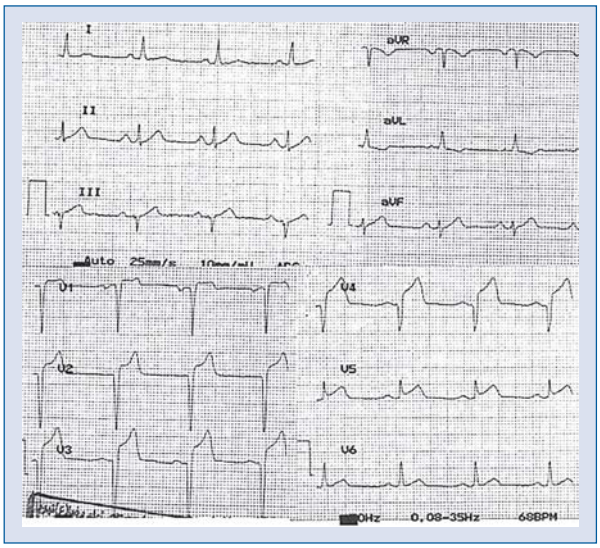

Figure 1. Electrocardiography shows ST segment elevations in leads V1–V5.

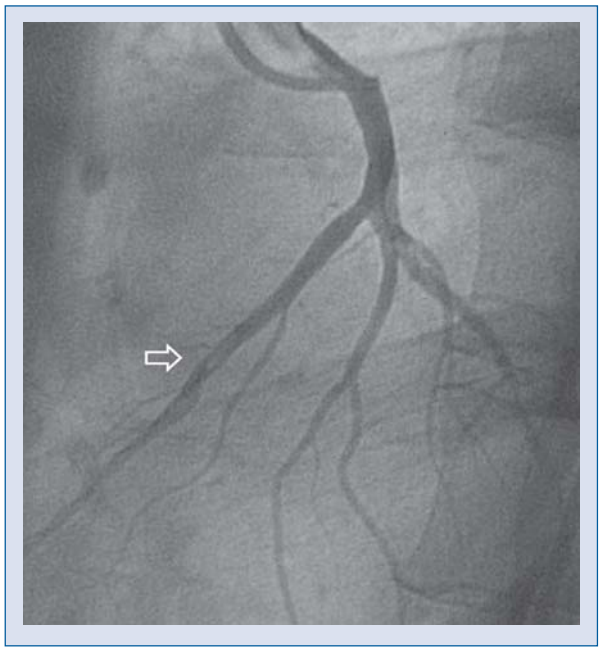

Figure 2. Left anterior oblique view of left coronary system. Linear filling defect indicating thrombosis is seen within the left anterior descending (arrow).

electrocardiogram at the time of admission showed ST segment elevation of 3–4 mm in precordial leads (Fig. 1). The patient received a loading dose of aspirin 325 mg and clopidogrel 300 mg orally, heparin 5000 IU bolus IV injection, metoprolol 50 mg, atorvastatin 40 mg and was also placed on nitroglycerin and heparin infusion. Thrombolytic therapy was not administered as it was contraindicated for his recent major surgery history. His laboratory tests showed Hgb = 12.5 mg/dL, WBC = 11 000/mL, PLT = 792 000//mL, CK-MB = 110, cTNI = 8, ESR = 34, CRP: 1+ and the rest of routine laboratory studies were within normal limits. Further work-up for iron deficiency anemia was negative. Bedside echocardiography showed left ventricular ejection fraction of 40% with hypokinesia of anterior wall and apical segments. He underwent coronary angiography which showed a linear filling defect at mid-portion of left anterior descending artery (Fig. 2) which was treated with low pressure balloon inflation and then a 3 × 15 Prokinetik™ (Biotronik, Buelach, Switzerland) bare metal stent was deployed. His hospital course was uneventful and he was discharged in a stable condition on aspirin 325 mg, clopidogrel 75 mg, metoprolol 100 mg and atorvastatin 40 mg daily on the fifth day following admission. At four month follow-up the patient's general condition was good, left ventricular ejection fraction was about 50% and his platelet count was 410 × 10^3/mL.

Discussion

Thrombosis represents the second highest cause of mortality in patients with PV or ET [3]. In published series, the rate of cardiovascular complications related to the presence of ET has ranged from 4% to 21%. Thrombosis, when it occurs, is more common in the arterial vascular bed (75%) than in the venous circulation (25%) [4]. The overall risk of thrombotic events in ET, which may include myocardial infarction, stroke, pulmonary embolism, deep vein thrombosis, transient ischemic attack, retinal artery or venous occlusions, hepatic or portal vein thrombosis, and digital ischemia, has been found to be 6.6% per patient per year, in comparison to 1.2% per patient per year in the control population. Paradoxically, patients with ET are prone to hemorrhagic complications. Bleeding tends to occur spontaneously in superficial sites such as the skin and mucus membranes as well as the gastrointestinal or respiratory tracts [5].

Conversely, reactive thrombocytosis is a benign process and is not a risk factor for thromboembolic complications [6]. In the immediate post-operative period in uncomplicated splenectomy patients, the platelet count rises steeply with a peak

value at seven to 12 days. It usually subsides over the next two to three months. However in some patients the thrombocytosis persists indefinitely following splenectomy [1].

In a large retrospective study of 732 patients with thrombocytosis, 87.7% had reactive thrombocytosis. Thromboembolic complications were reported in 12.4% of patients with primary thrombocytosis compared with 1.6% in patients with reactive thrombocytosis. All patients with secondary thrombocytosis developing thrombotic complications had additional risk factors such as preceding surgery or a co-existing malignancy [7]. In one large retrospective study comprising 318 patients without myeloproliferative disorders who underwent splenectomy, thrombocytosis developed in 75% of the patients without a substantial increase in the incidence of thromboembolic events [8]. On the other hand, in a retrospective study of 129 patients with platelet counts of $1{,}000 \times 10^9$/L or more, Buss et al. [9] reported an equal frequency of thrombotic events among patients with myeloproliferative disorders and those with reactive thrombocytosis. In a retrospective study evaluating 80 splenectomized patients suffering from various types of hereditary hemolytic anemia, sideroblastic anemia, hemoglobinopathies, or thalassemia, Hirsh and Dacie found that 13% developed thromboembolic complications in association with persistent postsplenectomy thrombocytosis, two of whom died from these complications a long time after splenectomy. However, the authors suggested that post-splenectomy thrombocytosis should not be the only factor predisposing patients to thrombosis because many patients from their own series have had comparable increases in the post-splenectomy platelet count that have been maintained for many years and have not developed signs of thromboembolism [10].

To our knowledge, this is the third reported case of acute myocardial infarction in patients with reactive thrombocytosis [11, 12]. Our patient refused bone marrow biopsy. However, normal count of platelets at the time of surgery (256×10^3) with a dramatic rise to 792×10^3 on the twelfth postoperative day, together with the gradual decline to normal range during the next four months favor reactive thrombocytosis. Also, studies to evaluate a hypercoagulable state and iron deficiency anemia including factor V Leiden mutation, protein C and S, antithrombin activity and homocystein and fibrinogen levels, together with lupus anticoagulant, anticardiolipin and anti beta II glycoprotein I antibodies were all negative. Coronary angiography showed a linear filling defect at mid-part of left anterior descending artery consistent with a thrombosis. While coronary occlusion due to ET may occur as a result of thrombotic aggregation on the damaged endothelium in totally normal coronary arteries, it also could be due to the thrombotic material aggregation over the atherosclerotic lesions resulting in stenosis of unimportance [13]. During angioplasty, the balloon was fully expanded at very low inflation pressure of about 1–2 atmospheres and in coronary angiography images acquired just after this inflation there was no residual stenosis. This may indicate lack of any atherosclerotic stenosis in our patient's coronary tree. Our patient had a history of smoking which has been shown to be common in young patients with infarction and ET [14]. His past medical history also included migraine headaches which are reported to be associated with vascular reactivity and this may be an appropriate substrate for *in situ* thrombus formation [15].

Our case demonstrates the potential for a major vascular event in the setting of reactive thrombocytosis, especially in those with a history of smoking. While the preventive effect of anti-coagulant and anti-platelets drugs has not been well-established, regular monitoring of platelet count and possibly antithrombotic agents like aspirin prescription for high risk patients with moderate thrombocytosis may be useful. Due to a very low event rate in this setting, large studies are needed to evaluate this hypothesis.

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