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Case Report

Acute stent occlusion due to secondary thrombosis in a patient with romatoid arthritis

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Abstract. It has been known that rheumatoid arthritis is a risk factor for cardiovascular events. However, secondary thrombocytosis is not accepted as a risk factor for acute myocardial infarctus. We presented a case of acute myocardial infarction associated with secondary thrombocytosis. Primary percutaneous transluminal coronary angioplasty and stenting were performed due to acute myocardial infarction. Then, patient started to receive clopidogrel, aspirin, glycoprotein IIb/IIIa receptor blocker (trofiban) and standard heparin. But, repeated coronary angiography was required due to stent thrombosis, because persistent chest pain and ST elevation at anterior leads were observed approximately 4-6 hours after primary percutaneous transluminal coronary angioplasty. Repeated angiograph revealed acute stent restenosis and then the revascularization was achieved by balloon angioplasty.

Key words: Secondary thrombocytosis and acute myocardial infarctus

1. Introduction

Rheumatoid arthritis (RA) is a chronic systemic inflammatory disease affecting approximately 1% of the adult general population. RA can affect the pericardium, myocardium, and endocardium (1). Moreover, RA increases cardiovascular morbidity and mortality by accelarating atherosclerosis (2). Cardiovascular mortality accounts for 40–50% of all deaths in RA. Patients with RA are at a twofold increased risk for myocardial infarction and stroke, with risk increasing to nearly three-fold in patients who have had the disease for 10 years or more (3). Rheumatoid arthritis is often complicated by thrombocytosis. Thrombocytosis usually occurs during the active clinical stages of RA. It is previously demonstrated that platelet counts correlate with disease activity in RA (4).

2. Case report

A 53-year-old man was admitted to the hospital with complaints of severe chest pain. He had suffered from RA for ten years. He had been treated with corticosteroids. Patient had no risk factors, except his age and gender. He had no family history of coronary artery disease.

Physical examination at the time of presentation revealed a blood pressure of 154/82 mm Hg and a heart rate of 102 bpm. Cardiac examination revealed normal first and second heart sounds. There were no third or fourth heart sounds and no murmurs. His lung examination was was normal. Hepatosplenomegaly was not detected. His electrocardiogram showed ST elevations and T wave inversions in leads V1 to V6 (Figure 1). Blood samples were taken for determination of biochemical parameters. Then, patient was transferred to angiography laboratory for primary percutaneous transluminal coronary angioplasty (PTCA).

Coronary angiography showed that proximal left anterior descending artery was totally occluded by thrombus. Any atherosclerotic lesion (narrowing or obstruction) was not detected in coronary angiography. PTCA and stenting were performed for acute anterior MI (Figure 2 a,b).

Patient started to receive clopidogrel (450 mg bolus and 75 mg/day), aspirin (300 mg/day), Glycoprotein IIb/IIIa receptor blocker (trofiban)

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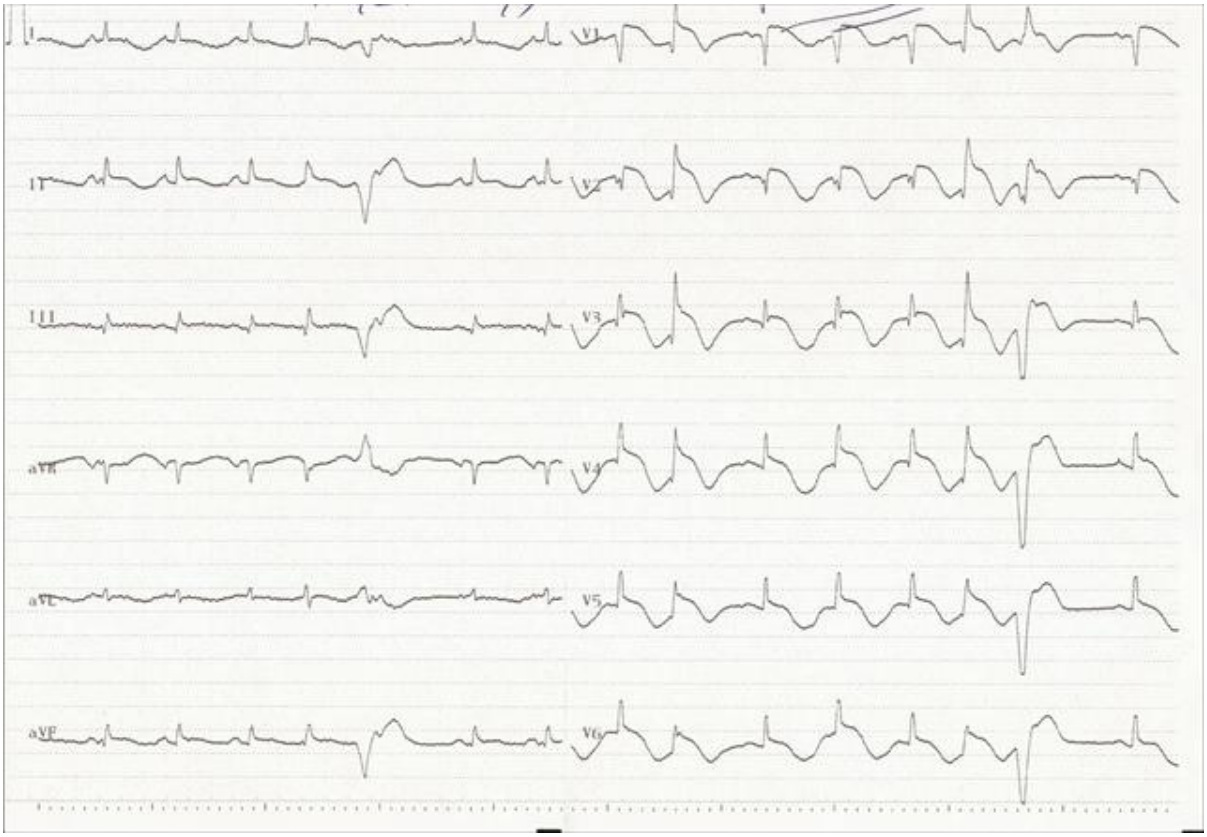

Fig. 1. ECG of the patient in emergency room

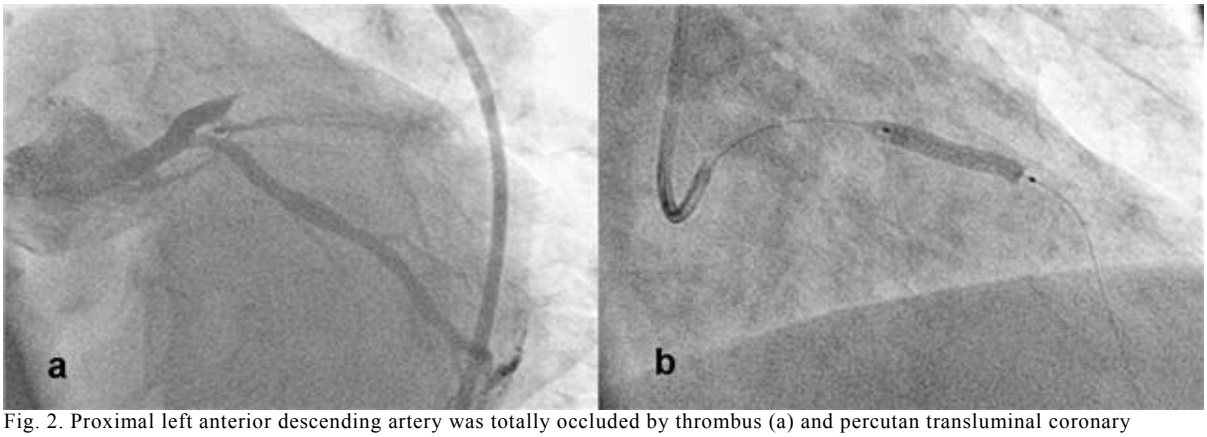

Fig. 2. Proximal left anterior descending artery was totally occluded by thrombus (a) and percutan transluminal coronary angioplasty (b)

and standard heparin (5.000 U bolus and 1.000U/h, as appropriate aPTT). However, chest pain was persisted and ST elevations at anterior leads were observed approximately 4-6 hours after primary PTCA (Figure 3). Therefore, a repeated coronary angiography was performed due to suspected stent thrombosis. Acute stent thrombosis was observed and then the revascularization was achieved again by balloon angioplasty (Figure 4 a,b). The cardiac markers were high whereas other biochemical parameters such as total cholesterol, LDL, triglyceride was normal in the blood samples of patient. However hematological parameters such as white blood cells (16500) and platelet (664000 per ml) were also high. Rheumatoid factor was positive (77, 6 per ml).

3. Discussion

The increased cardiovascular disease risk in RA patients seems to be independent of traditional cardiovascular risk factors. Pathogenic

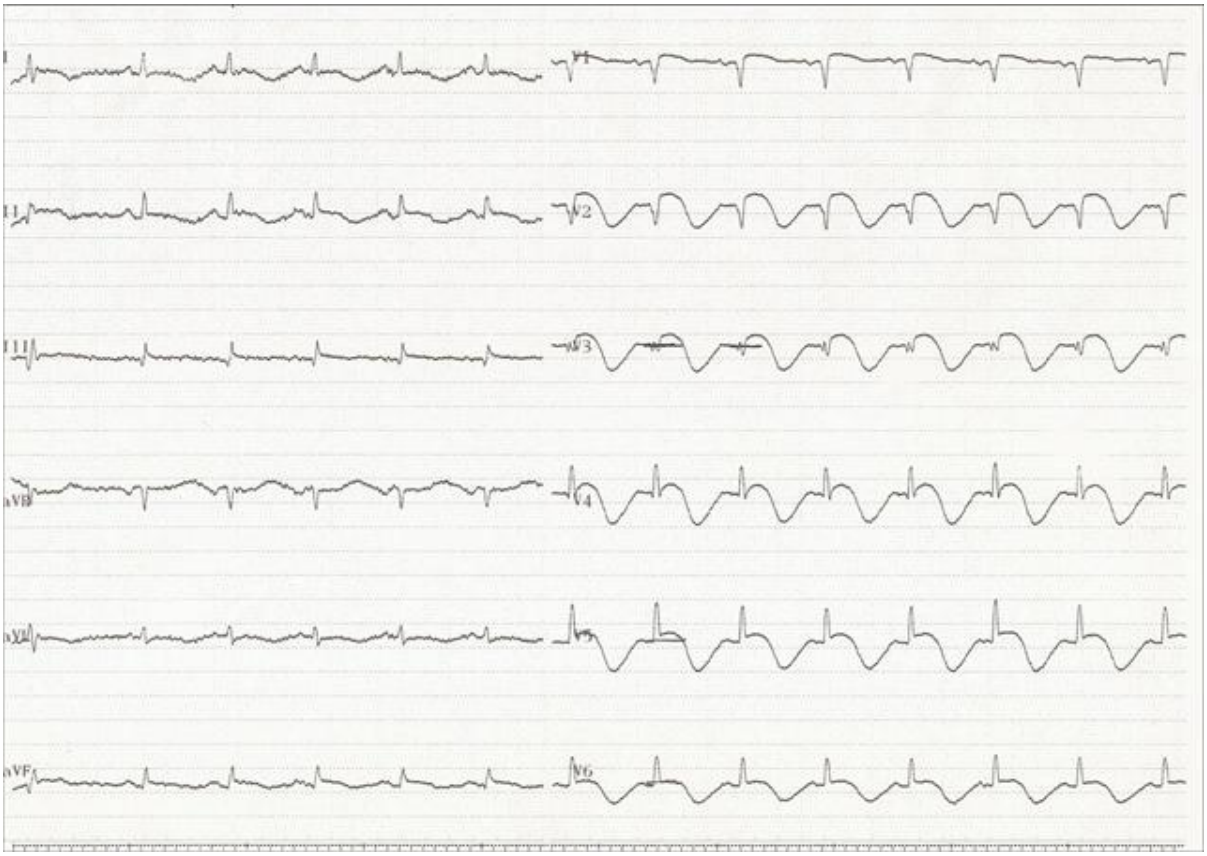

Fig. 3. ECG approximately 4-6 hours after primer PTCA.

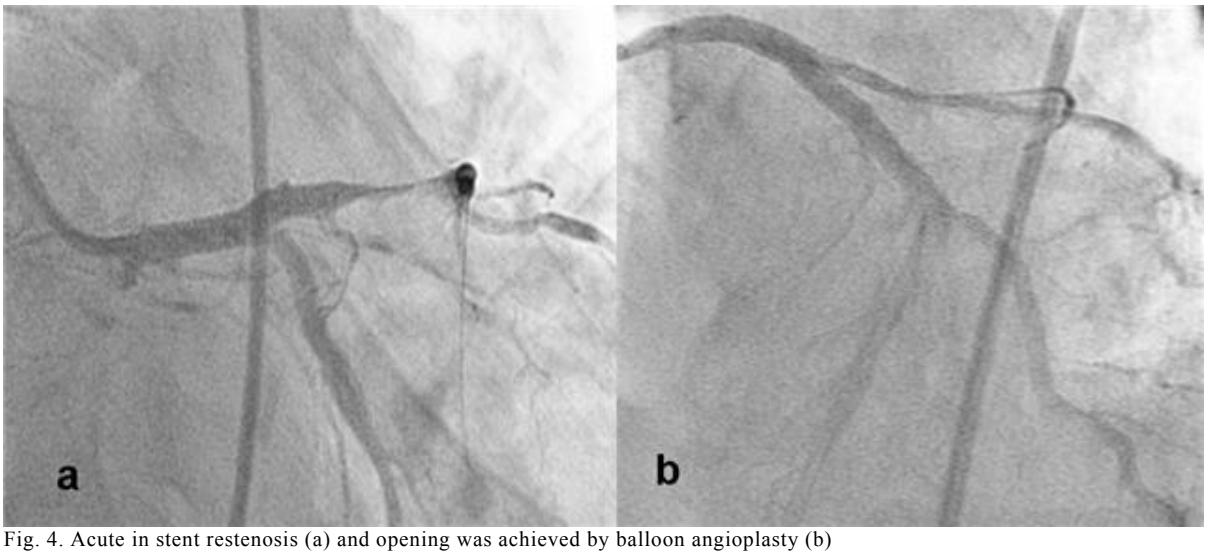

Fig. 4. Acute in stent restenosis (a) and opening was achieved by balloon angioplasty (b)

mechanisms include pro-oxidative dyslipidemia, insulin resistance, prothrombotic state, hyperhomocysteinemia, and immune mechanisms such as T-cell activation that subsequently lead to endothelial dysfunction, a decrease in endothelial progenitor cells, and arterial stiffness, which are the congeners of accelerated atherosclerosis observed in RA patients. (3,5).

In our case, the patient had no risk factors (except his age and gender) for atherosclerosis. Coronary thrombosis seemed to occur in normal coronary arteries in left anterior descending artery (LAD) because except in LAD thrombosis other coronary arteries were fully normal.

Rheumatoid arthritis is often complicated by thrombocytosis. The exact pathogenetic

mechanisms underlying the increased platelet counts in such patients remain undetermined. Persistent overproduction of certain thrombocytopoietic factors can induce megakaryocytopoiesis and thrombocytopoiesis (6). Thrombocytosis usually occurs during the active clinical stages of RA. Previously it was demonstrated that platelet counts correlate with disease activity in RA (4).

In our case, the patient had suffered from RA for ten years and had taken corticosteroid for treatment. But rheumatoid factor was + (77, 6 per ml), that is illness was in active phase.

There is no case of myocardial infarction associated with secondary thrombocytosis in literature, but nearly 20 cases of essential thrombocytosis (ET) with involvement of coronary arteries leading to acute coronary syndromes or myocardial infarction have been reported (7-9). The majority of the case reports about ET and acute coronary syndromes/acute myocardial infarction are due to the occlusion of the LAD (9) but other coroner involvement is rare (10). Similarly, our case had LAD occlusion leading to acute MI unrelated to atherosclerosis.

It is commonly believed that a high platelet count must cause intravascular stasis and thrombosis. The etiology of the thrombosis may be due to the dysfunction of the platelets rather than due to their increased counts in ET. However, very high platelet counts are associated primarily with hemorrhage while platelet counts of less than a million are often associated with thrombosis (11).

Deep venous thrombosis and pulmonary embolism are the most common thrombotic manifestations in ET (12). Arterial occlusions are primarily microvasculature; however, thrombosis may also occur in larger arteries. Thrombosis of the arterial system involving the coronary, renal, peripheral and digital arteries and thrombosis of the venous system involving the portal vein and sagital sinus have been reported. It is one of the rare causes of coronary arterial occlusion and acute coronary syndromes (11,13), as seen in our case.

The treatment modalities for MI in the ET have not been clearly defined yet. Primary angioplasty, intracoronary thrombolytic therapy and even coronary artery bypass surgery are of choice (14).

Michaels et al. (15) suggested that use of glycoprotein IIb/IIIa receptor-inhibiting monoclonal antibody drug followed by primary angioplasty might play an important role in the treatment of coronary thrombosis in ET. In a prospective study among the patients with ET and polycythemia vera, Rossi et al. (16) reported that a low dose of aspirin might be able to reduce the number of coronary thrombosis without increasing bleeding complications. The use of clopidogrel is strongly suggested in stent-implanted patients (17).

Although our patient had undergone angiography and he received clopidogrel (450 mg bolus and 75 mg/day), aspirin (300 mg/day), glycoprotein IIb/IIIa receptor blocker (trofiban 0.4 µgr/kg loading dose and 0.2 µgr/kg infusion) and standard heparin (1.000U/h, as appropriate aPTT), repeated coronary angiography was performed because chest pain and ST elevation at anterior leads were observed approximately 4-6 hours after primary PTCA. Then the revascularization was achieved by balloon angioplasty. According to our knowledge, this case may be first report of acute stent thrombosis owing to secondary thrombocytosis in spite of maximal anticoagulation therapy.

4. Conclusion

We thought that rheumatoid arthritis could be a risk factor for acute myocardial infarction in this case. However, although maximal anticoagulation therapy was performed after primary PTCA, acute occlusion developed in a short period of time. Therefore, stent occlusion could be related to secondary thrombosis.

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