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Case Report Open Access

Recurrent ST-elevation Myocardial Infarction in a Patient with Systemic Lupus Erythematosus

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Abstract

Increased atherosclerosis and thrombosis in patients with systemic lupus erythematosus (SLE) commonly cause cardiovascular complications such as myocardial infarction. Mechanism of myocardial infarction is different: Some immunological and genetic causes such as antiphospholipid syndrome, increased inflammation, immune complexes, elevated homocysteine level and Factor V Leiden mutation, play pivotal role in this group of patients. So its treatment shows difference too. Anticoagulant drugs are not recommended for treatment of myocardial infarction in current practice however, main treatment protocol should contain oral anticoagulants such as warfarin after myocardial infarction in this group of patients. Herein, we present a 30 year old SLE patient with recurrent ST-elevation myocardial infarction due to lack of oral anticoagulant treatment. With the initiation of anticoagulation, the patient has not experienced myocardial infarction again. In this case, we underline the importance of oral anti-coagulant therapy after myocardial infarction in patients with SLE.

Keywords: Systemic lupus erythematosus; Acute myocardial infarction; Oral anticoagulant

Introduction

SLE is an autoimmune disorder with multiple organ involvement [1]. Nearly half of these patients die from cardiovascular diseases [2], which are particularly related to increased atherosclerosis and thrombosis. Thrombosis has been reported 27 to 43–fold higher in patients with SLE than in the general population [3]. SLE itself is an independent risk factor for developing thrombotic events. It may further increase when accompany with other risk factors such as nephrotic syndrome, Infections and antiphospholipid-antibody syndrome [4]. Thrombosis is primarily venous and usually occurs as deep vein thrombosis however, arterial thrombosis is also encountered in this group of patients.

Venous and arterial thrombosis has different risk factors. While older age, longer disease duration, disease activity, smoking and presence of hypertension are risk factors for arterial thrombosis, factor V Leiden, nephrotic syndrome, current dose of prednisone and shorter disease duration are risk factors for venous thrombosis [5]. Arterial thrombosis develops from plaque rupture and leads to serious conditions such as myocardial infarction, stroke and peripheral artery disease which enhance mortality [2].

Case Presentation

A thirty-year-old male patient visited emergency room with complaint of chest pain for last 30 minutes. Physical examination of the patient was unremarkable and the electrocardiogram was consistent with acute inferior ST elevation myocardial infarction (STEMI). Anamnesis revealed that patient had Systemic Lupus Erythematosus (SLE) and hypertension for six years. He experienced coronary angioplasty for two times, 48 and 18 months ago for an acute STEMI, and three drug-eluting stents had being placed in the right coronary artery during the second angioplasty (Figure 1). He has been smoking a pack of cigarettes daily for the past five years. He stopped taking clopidogrel one year after stent placement and was receiving ASA 100 mg daily.

The family history revealed recurrent deep vein thrombosis in his brother and stroke for two times in his mother. After being pre-diagnosed as very late stent thrombosis, the patient was admitted to the catheter laboratory. In stent 100% thrombosed lesion was detected in the right coronary artery; stent was placed over the lesion and TIMI-3 flow was established (Figure 2). Dual anti-platelet therapy (acetylsalicylic acid, clopidogrel) with an oral anticoagulant was planned at the time of discharge. Homozygous factor V Leiden mutation was determined in patient by investigation of predisposing genetic factors for thrombosis.

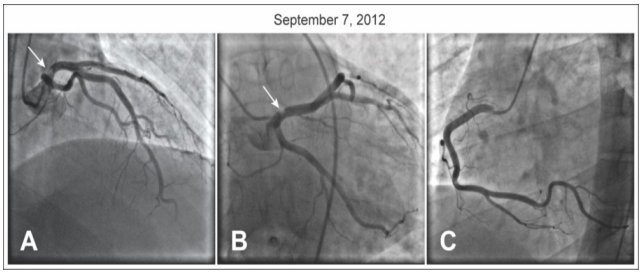

Figure 1: The first presentation with acute anterior ST elevation myocardial infarction; (A) Thrombus in the proximal and distal left anterior descending coronary artery; (B) Thrombus in the proximal and distal left anterior descending coronary artery; (C) Normal right coronary artery.

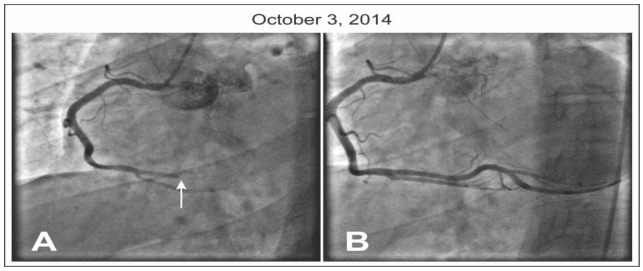

Figure 2: The second presentation with acute inferior ST elevation myocardial infarction; (A) Total thrombotic occlusion of distal right coronary artery; (B) Right coronary artery after stent implantation.

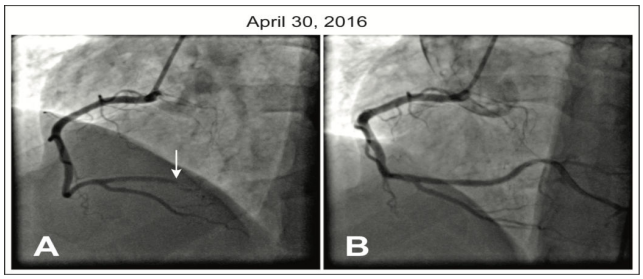

Figure 3: The third and the recent presentation with acute inferior ST elevation myocardial infarction; (A) Right coronary artery stent thrombosis; (B) Right coronary artery after stent implantation.

Discussion

Among the SLE patients, myocardial infarction risk is slightly higher. On the other hand, mechanism of myocardial infarction in this group of patient is different, so its treatment shows difference too.

While anti-platelet therapy is the mainstay of myocardial infarction and oral anticoagulant drugs are not recommend treatment of myocardial infarction in current practice, this quietly differs in SLE patients; main treatment protocol should contain oral anticoagulants such as warfarin for patients with SLE after myocardial infarction.

If therapy doesn't contain anticoagulants, there will be a new coronary event with high prospect due to lack of this therapy. In this paper, we report a young male patient with SLE who experienced recurrent myocardial infarction due to lack of oral anticoagulant drug after myocardial infarction. Clinicians should be alert for thrombotic events in SLE patients as these are life-threatening conditions.

Predisposing factors for thrombosis include antiphospholipid syndrome, increased inflammation, immune complexes, and elevated homocysteine level [6]. In these patients, oral anticoagulant therapy with warfarin should be started with a target INR of 2.0-3.0 in cases with venous thrombosis and INR of 3.0-4.0 in cases with arterial thrombosis and/or recurrent thrombosis [7].

In the present case, the 30-year-old male SLE patient has had arterial atherothrombosis (myocardial infarction) for three times. The factors those precipitated recurrent thrombosis in the present subject were considered due to SLE concomitant with hypertension, smoking, discontinuation of clopidogrel, lack of oral anticoagulant drug, and the presence of Factor V Leiden mutation [8].

Although dual anti-platelet therapy had been initiated, no anticoagulant therapy was given to the patient after previous coronary events. Derksen et al. followed SLE patients, who have had a thrombotic event and were receiving an anticoagulant drug, for eight years.

They could report no recurrent thrombotic events in any of the patients receiving oral anticoagulant therapy, whereas they reported 50% recurrence within two years in the patients that discontinued oral anticoagulant therapy [9]. Lack of oral anticoagulant drugs appears most likely cause of recurrent myocardial infarction in the present case.

Conclusion

Oral anticoagulant therapy should be initiated in patients with SLE who have arterial thrombosis such as myocardial infarction. Otherwise, recurrent myocardial infarction may be encountered frequently in such patients.

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