

Acute Myocardial Infarction Induced by Thrombi within the Coronary Artery Aneurysms of a Young Male SLE Patient

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ABSTRACT

We describe the case of a 30-year-old man with systemic lupus erythematosus (SLE) and he was struck with non-ST segment elevation myocardial infarction: this was due to the presence of multiple coronary artery aneurysms those were full of thrombi. A diagnostic coronary angiogram revealed huge dilatations in the proximal three coronary arteries with multiple filling defects and a decreased flow rate, and these were suggestive of thrombi within the coronary artery aneurysms. An intravascular ultrasound (IVUS) examination revealed huge aneurysmal dilatations with movable thrombi in three coronary arteries. He had an uneventful recovery without us having to perform any percutaneous coronary intervention. (**Korean Circulation J 2006;36:72-75**)

KEY WORDS : Coronary disease ; Aneurysm ; Myocardial infarction ; Lupus erythematosus, systemic.

Introduction

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease that is characterized by the presence of immune complexes, and the various clinical features of the patients with SLE are due to their autoantibodies. The patients with SLE have increased risks for acute myocardial infarction (AMI) and cerebrovascular accident (CVA).^{1,2)} The major coronary abnormalities associated with myocardial injury in SLE patients include premature atherosclerosis, coronary arteritis and less frequently, coronary aneurysms. There have been some cases of SLE-associated premature atherosclerosis,³⁾ and SLE associated coronary aneurysms have been previously reported on.⁴⁻⁶⁾ However, there is no previous report on AMI associated with SLE-associated coronary artery aneurysm and thrombus that was diagnosed by coronary angiogram and intravascular ultrasound.

We report here on an unusual case of a young male patient who had SLE. He was struck with AMI, and this was due to thrombi within multiple aneurysms in three different coronary arteries. We diagnosed this case by performing coronary angiogram and intravascular ultrasound.

Case

A 30-year-old man with a 5-year history of systemic lupus erythematosus (SLE) visited to the emergency department with chest pain he had suffered with for 12 hours. He had experienced long-term treatment with prednisone and he had undergone right total hip replacement a year ago due to avascular necrosis of the femoral head. He had no complaints that were consistent with a past history of cardiac disease. There was no other significant medical or family history. He was current 5 pack-year smoker.

On the physical examination, he was afebrile with a blood pressure of 110/70 mmHg, a heart rate of 86/min, and a respiratory rate 24/min. The heart sounds were regular without gallops, murmurs or rubs. The lungs were clear upon auscultation. The rest of his examination was normal.

His initial electrocardiogram showed a normal sinus

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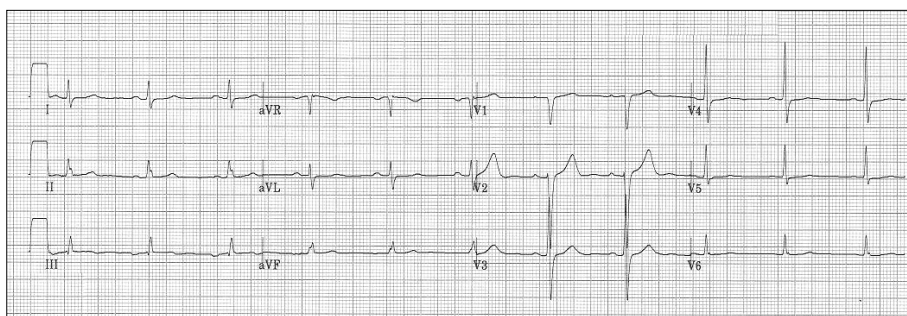


Fig. 1. Electrocardiogram showing normal sinus rhythm and the Q wave in V₁.

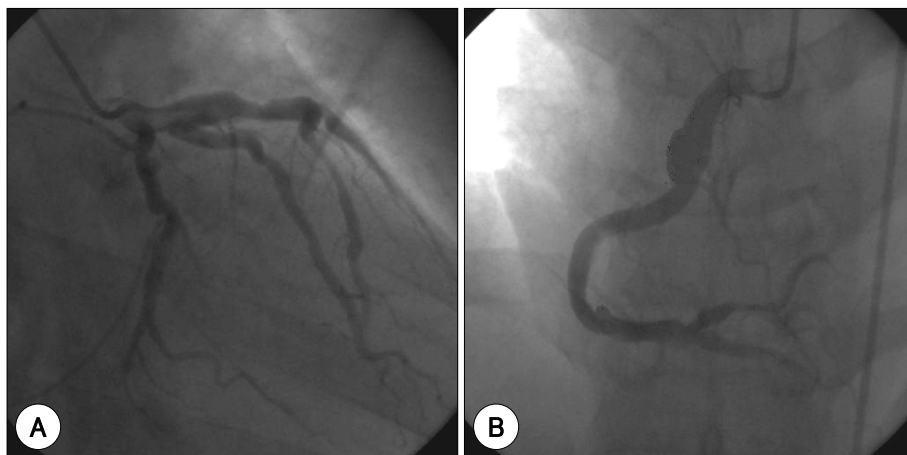


Fig. 2. A diagnostic coronary angiogram (left and right, A and B) revealed huge dilations in the proximal three coronary arteries with multiple filling defects, which was suggestive of thrombi within coronary artery aneurysms.

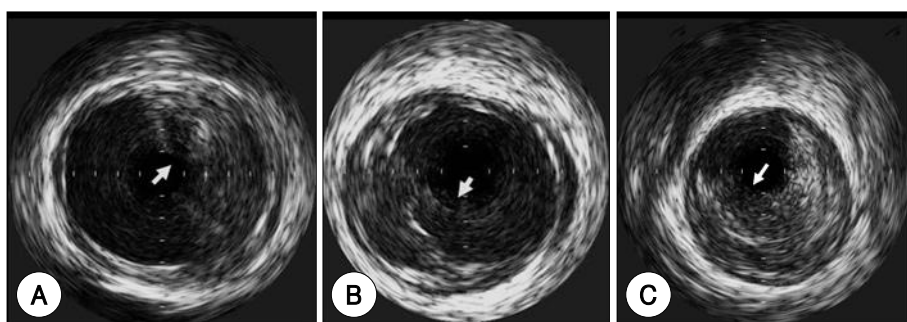


Fig. 3. Intravascular ultrasound (IVUS) revealed movable echogenic masses in the coronary aneurysms of the proximal right coronary artery (A), left anterior descending artery (B) and circumflex artery (C).

rhythm and a Q wave in V₁ (Fig. 1). An emergent echocardiogram was obtained, and it demonstrated hypokinesia of the inferior walls. On the laboratory studies, the hemoglobin was 14.2 g/dL, the white blood count was $4.6 \times 10^3/\mu\text{L}$ and the platelets were $169 \times 10^3/\mu\text{L}$. The initial coagulation studies showed a partial thromboplastin time of 39.4 seconds (normal: 26.5 to 41.1) and an international normalized ratio of 1.02 (normal: 0 to 1.2). The peak level of creatine kinase (CK) was 2347 U/L (normal: 35 to 172), CK-MB 95.4 U/L (normal: 2.3 to 9.5), troponin-I 38.18 ng/mL (normal: 0 to 0.05) and troponin-T 2.52 ng/mL (normal: 0 to 0.1). The antinuclear antibody test was positive at 1 : 160 with a homogeneous pattern. The double-stranded DNA was negative and the anti-phospholipid antibody panel was negative. The levels of complement were low, with the levels of C3 at 58

mg/dL (normal: 90 to 180) and C4 at 9.31 mg/dL (normal: 10 to 40).

We performed a coronary angiogram procedure, and it revealed severe aneurysmal dilations of the three proximal coronary arteries with low Thrombolysis in the myocardial infarction (TIMI) flow (Fig. 2).⁷⁾ He was treated with intravenous heparin for one week. The follow-up CAG and intravascular ultrasound were done at seven days after the heparinization. An intravascular ultrasound examination revealed huge aneurysmal dilations with thrombi in all three coronary arteries (Fig. 3).

The patient received medical therapy instead of percutaneous coronary intervention. He had an uneventful recovery and there were no cardiac events during the one-month clinical follow-up.

Discussion

Cardiovascular disease is the most common cause of death for patients with long-standing SLE, and among the different types of heart disease, coronary artery disease (CAD) is a major source of mortality.¹²⁾ A little less than 20% of SLE patients suffer with CAD such as angina or myocardial infarction.⁸⁾ For patients with SLE, atherosclerosis, embolism and vasculitis of coronary arteries are the causes of ischemic syndrome.⁹⁾¹⁰⁾

Despite the young age of many of the patients with lupus, atherosclerosis remains the most common cause of ischemic cardiac disease.¹¹⁾ The risk factors for coronary artery disease include the disease duration, the period of treatment with corticosteroids, the postmenopausal status and hypercholesterolemia. As patients are now living longer with their disease, the prevalence of major clinical events will surely increase.

Immune mediated endothelial injury, hypertension and hyperlipidemia have been suggested to contribute to the progression of atherosclerosis.¹²⁾ Corticosteroid therapy may also contribute to progression of atherosclerosis via the steroid increasing the serum triglyceride and low-density lipoprotein levels.¹³⁾ Peri et al¹⁴⁾ have reported that a longer mean duration of prednisone use was associated with hypertension, hypercholesterolemia and obesity, as well as known CAD risk factors for patients with SLE.

Our patient had no history of hypertension or hypercholesterolemia despite the long duration of his corticosteroid use, but the intravascular ultrasound examination of his coronary arteries showed a large number of thrombi.

The major cause of the acute coronary syndrome in this patient may have been thrombosis, which often related to the presence of anti-phospholipid antibodies (APLAs). The presence of APLAs predisposes some patients to thrombosis, and it has also been associated with valvular thickening and nonbacterial endocarditis.¹⁵⁾ Anti-endothelial antibodies may accelerate atherogenesis. The possibility that intimal proliferation and enhanced atherogenesis are due to primary stimulation of the vascular endothelium by immunologic factors has been supported by evidence from an experimental animal model.¹⁶⁾ In this regard, APLAs are regarded as an independent predictor of CAD.¹⁷⁾

Coronary aneurysms are a rare finding in patients with SLE. Aneurysmal dilatation of the coronary arteries has been commonly described in patients with polyarteritis nodosa, Takayasu's disease and Kawasaki's disease, and they have been reported to be associated with atherosclerosis.¹⁸⁾ Aneurysm formation associated with SLE is thought to be a complication of inflammation and fibrosis in the media.¹⁹⁾ The myocardial injury from coronary aneurysms may be associated with throm-

bosis.²⁰⁾

We performed a coronary angiogram and intravascular ultrasound for our patient, and this revealed huge aneurysmal dilatations with thrombus burdens in three major coronary arteries. This case report describes a young male SLE patient who was struck with AMI, and this was due to thrombi within multiple coronary aneurysms.

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