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Case Report

Coronary Arteritis in a 20-Year-Old Female

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Abstract

Coronary artery disease in systemic lupus erythematosus is commonly due to an atheromatous process. Coronary arteritis with aneurysms is a rare cause of myocardial ischemia in SLE. Confirmation of coronary arteritis has been made usually at post-mortem examination. Here, we report the case of a 20-year-old female who presented with acute myocardial infarction. She was subsequently diagnosed to have SLE complicated with coronary arteritis. This case illustrates that myocardial infarction can be the initial manifestation of SLE secondary to coronary arteritis with aneurysms.

Keywords: SLE; myocardial infarction; coronary arteritis

Introduction

Premature coronary artery disease (CAD) in Systemic Lupus Erythematosus (SLE) patients is a cause of substantial morbidity and mortality. Premature atherosclerosis is the main cause of CAD in SLE patients. Another important aetiology for myocardial infarction in SLE is coronary arteritis. Coronary arteritis and premature atherosclerosis have been always reported in patients with an established diagnosis of SLE.

Here, we report the case of a 20-year-old female who presented with acute myocardial infarction. She was subsequently diagnosed to have SLE complicated with coronary arteritis.

Case Report

A 20-year-old female presented with chest pain of 6 hours duration. Physical examination revealed malar rash and oral ulcers. Chest X-ray was normal and her electrocardiogram (ECG) showed ST segment elevations in leads V1-V6, I and a VL. An echocardiogram demonstrated hypokinesia of the anterior and lateral walls. Investigations demonstrated haemoglobin 9.2 g/dL with normal platelet and leucocyte counts. The erythrocyte sedimentation rate (ESR) was 98 mm/hr (normal 0 to 20) and Coomb's test was positive. Troponin-I was elevated 15.0 ng/mL (normal 0 to 0.05). The antinuclear antibody and double stranded DNA antibody were positive and the antiphospholipid antibody was negative. Her lipid profile and coagulation studies (protein C, protein S, homocysteine, antithrombin III, and lipoprotein-a) were normal. Coronary angiogram revealed aneurysmal dilatations of proximal coronary arteries and only moderate stenoses of the secondary branches (Figure-1). In view of these angiographic findings, coronary revascularization was not indicated and anticoagulant therapy was initiated in view of the presence of large aneurysmal coronary dilatations, which are predisposed to in situ thrombosis and distal embolization. The patient received standard regimen of dual antiplatelet (aspirin and clopidogrel) with heparin. The coronary vasculitis was treated with immunosuppressive therapy {steroids and mycophenolate mofetil (MMF)}. The patient was discharged on MMF, telmisartan, rosuvastatin and aspirin. She had good recovery and there were no cardiac events during clinical follow-up. Repeat coronary angiogram performed at 6-months after discharge revealed good distal flow with multiple aneurysms in the coronary arteries.

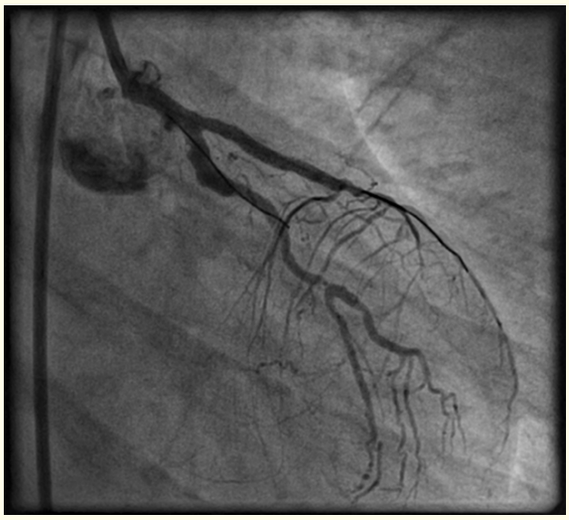

Figure 1: Coronary Angiogram Revealed Aneurysmal Dilatations of Proximal Coronary Arteries.

Discussion

The etiology of CAD in SLE may be varied; the most prominent being premature atherosclerosis, coagulopathy and coronary arteritis. Coronary arteritis has been been described in few case reports of SLE [1-4]. Coronary arteritis is far less common than premature atherosclerotic disease among SLE patients.

Pathologically, vessels in coronary arteritis are narrowed by cellular intimal fibrosis with areas of aneurysmal dilatation [5]. Coronary arteritis in SLE does not have any relationship between clinical or serologic disease activity and the development of symptomatic vasculitis [6]. As with other forms of vasculitis, it may be difficult to differentiate coronary arteritis from atherosclerosis exclusively on the basis of the angiographic appearance such as of beading of the vessels. The young age, new onset of SLE, absence of exposure to systemic corticosteroids, absence of visible intracoronary calcification on coronary angiography favoured the diagnosis of vasculitis in our patient. The different pathogenesis associated with coronary disease in SLE makes the treatment complex. Immunosuppressive therapy with corticosteroids and cyclophosphamide is the mainstay of SLE vasculitis therapy. Coronary artery bypass surgery can be performed with acceptable morbidity and mortality in patients with SLE vasculitis [7].

However, coronary artery bypass surgery should be accompanied by continuous immunosuppressive therapy. The clinical course and coronary angiographic appearance did not warrant revascularization in our patient. Measures aimed at prevention of atherosclerotic coronary disease such as optimization of serum lipid profile, blood pressure, body weight, and lifestyle modifications are an important component of a broader therapeutic approach in SLE patients

Conclusion

In conclusion, awareness of the association between SLE and CAD is essential while managing asymptomatic SLE patients. Possible pathophysiological mechanisms of CAD in SLE are premature atherosclerosis, arteritis, thrombosis and embolization. Immunosuppressive therapy is the mainstay of CAD complicated with coronary arteritis.

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Conflict of Interest

We affirm that we do not have any conflict of interest.

We verify that all the authors had access to the data and a role in writing the manuscript.

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