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Case Report

19 Years Old Male with ST Elevation Myocardial Infarction as an Initial Manifestation of Primary Antiphospholipid Syndrome

Abstract

Primary Antiphospholipid antibody syndrome is a rare disease associated with thromboembolic events which may affect either the arterial or the venous vasculature. Acute myocardial infarction is rarely associated with APS, especially as first manifestation. We present here a case of primary antiphospholipid antibody syndrome in young man, which present as acute myocardial infarction.

Introduction

Antiphospholipid antibody syndrome (APS) is a systemic, autoimmune, acquired disorder characterized by venous and/or arterial thrombosis and/or pregnancy morbidities [1]. Although detectable at 1–5% of asymptomatic subjects, persistent antiphospholipid antibodies (aPL) are significantly associated with recurrent arterial/venous thrombosis and pregnancy morbidity [2]. The 2006 International consensus Statement on an Update of the classification criteria recognize those antiphospholipid antibodies (aPLs): lupus anticoagulant (LA), anticardiolipin antibodies (ACA) IgM or IgG and antibody to β 2 glycoprotein 1 (β –2–GP I) [3]. Those should be confirmed at least two occasions, 12 weeks apart [1]. Primary APS has generally been defined as the presence of aPL in patients with idiopathic thrombosis, but no evidence of autoimmune disease or other inciting factor, such as infection, malignancy, hemodialysis or drug-induced APL. Primary APS was largely seen in young women, with a male-to-female ratio of 3.5:1, and the age of first thrombosis in APS was predominantly between 15 and 50 years. The most common features of thrombotic disorders in APS are deep-vein thrombosis, pulmonary thrombo-embolism, and stroke. Cardiac manifestations in APS include valve abnormalities (valve thickening and vegetations), occlusive arterial disease, intracardiac emboli, ventricular dysfunction, and pulmonary hypertension. Antiphospholipid antibodies may be associated with accelerated atherosclerosis in APS patients [4]. There are probably three relationships between aPL antibodies and coronary artery disease: first, aPL antibodies can cause thrombosis in normal vessels; secondly,

they may be associated with accelerated atherosclerosis; and lastly, in some individuals, transient aPL antibodies may arise at the time of MI because of vascular injury and exposure of neoantigens. The possible mechanisms of thrombosis in APS include the effects of APL on platelet membranes, on endothelial cells and on clotting components such as prothrombin, protein C and protein S. The antiphospholipid antibodies persistence for years, possibly for a lifetime. Recently circulating procoagulant microparticles are found to contribute to thrombotic propensity in patients with APS [5].

Case Presentation

A nineteen years old Caucasian male with no significant previous medical history, non-smoker and without known drug allergies has presented in the emergency department (ED) with ongoing left sided chest pain that has started 30 minutes before presenting to the ED. Three days before coming to the ED was diagnosed with a chest infection and treated with oral antibiotics. Day before presented to ED had a several episodes of chest pain that has been lasting up to 30 minutes with spontaneous resolution. The symptoms were not triggered by physical activity. On physical examination, general condition was good and vital signs were stable. Initial ECG in ED showed, sinus rhythm with ST elevation in II, III, aVF leads and depression in V1, V2, V3 (Figure 1). Laboratory findings on admission: CK 844 U/L; CK-MB 61 U/L; troponin I 11,383 μ g/L. His resting echocardiogram showed normal findings, with no regional contractility failure of the left ventricle. His left ventricular ejection fraction (LVEF) was 63%. We performed coronary angiography. His left and right coronary system

were normal, the proximal portion of the circumflex artery is present eccentric plaque with the non-significant stenosis (Figures 2,3). He was treated with aspirin, low molecular weight heparin, bisoprolol, and angiotensin converting inhibitor, statines and antibiotics (amoxicillin/clavulanic acid). The

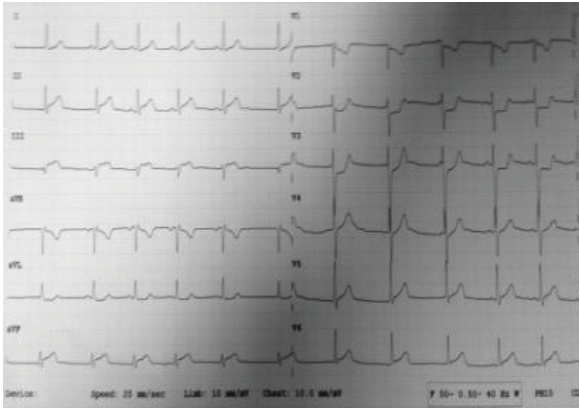


Figure 1: Electrocardiogram at hospital admission.



Figure 2: Left coronary arteriogram.



Figure 3: Right coronary arteriogram.

rest of his stay was uneventful, and he was discharged home after 8 days. The patient discharged with a recommendation following treatment: Aspirin 100 mg and simvastatin 40 mg. The patient's antinuclear antibody, anti-DNA were negative, but LA was positive 1.66. His aPL screening revealed ACA IgG 20,9 U/ml (n = 0.1–15) and β -2-GP I was negative. A repeat LA after 3 months was positive 1.46; his ACA IgG had come down to 12.8 U/ml. After the control examination recommended the introduction of anticoagulant therapy, warfarin while maintaining PV 2,5–3,5 INR. On follow up appointments he remains stable and free of chest pain and dyspnoea.

Discussion

The clinical presentation of APS at times can be very difficult to diagnose at the first presentation in the absence of classical symptoms. Primary APS is a rare disease in children as well as adolescents compared with APS associated with either autoimmune disease. Also previous studies associate cardiac manifestation of APS with older age, in this case major event occur in 18 years old male. Certainly a major cardiovascular event in this young man requires life-long treatment, anticoagulation therapy, such as in this case recommended warfarin. Our patient did not have any cardiovascular risk factors, In this case, mild chest infection was a breaking point in triggering myocardial infarction. Is it possible, that in the setting of aPL antibodies even mildest increase in inflammatory markers can disrupt the balance in-between anticoagulant factors and procoagulant factors. Although the presentation of a major cardiovascular event as a primary manifestation of APS is rare, myocardial infarction in young people, and not related to any cardiovascular risk factors absolutely indicates the diagnostic screening for APL.

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