Acute myocardial infarction in a young man with large left ventricular thrombus and the antiphospholipid syndrome

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ABSTRACT

Antiphospholipid syndrome is an autoimmune disorder that presents clinically with venous and/or arterial thrombosis and can affect any organ. Coronary artery involvement and left ventricular thrombus are rare presentations. We report the case of a 23-year-old male smoker with a strong family history of premature coronary artery disease who presented with acute anterolateral ST elevation myocardial infarction and large left ventricular thrombus. He was treated with primary percutaneous coronary intervention with thrombectomy without stenting. He was later diagnosed with antiphospholipid syndrome.

KEYWORDS Acute myocardial infarction; antiphospholipid syndrome; autoimmune disorder; thrombus

Antiphospholipid syndrome (APS) is a rare autoimmune disorder characterized by the presence of antiphospholipid antibodies, hypercoagulability, and vascular thrombosis. APS is more common in young women and can affect any organ. Coronary artery involvement and intracardiac thrombus are very rare manifestations of APS yet are serious complications. The treatment and diagnosis of these complications are clinically challenging.

CASE PRESENTATION

A 23-year-old male smoker with a strong family history of premature coronary artery disease presented to the emergency department with new-onset severe chest pain of 1-hour duration. His blood pressure was 120/85 mm Hg; pulse, 112 beats per minute; respirations, 12 breaths per minute; and oxygen saturation, 98% on room air. He was diaphoretic; precordial examination disclosed no abnormalities, and the rest of the physical examination was unremarkable. Electrocardiogram in the emergency department

showed ST segment elevation and T wave inversion in the anterolateral leads (*Figure 1*). He was rushed into the catheterization lab and found to have acute occlusion of the distal left anterior descending artery (*Figure 2*). The patient underwent primary percutaneous coronary intervention (PCI) and was treated with thrombectomy without stenting due to the small caliber of the distal vessel. Intracoronary imaging was not performed due to the distal location of the thrombus. An echocardiogram revealed a large left ventricular apical thrombus (*Figure 3*). The left ventricular ejection fraction was estimated to be 35% to 40% with apical hypokinesis. Complete blood count, comprehensive metabolic panel, and lipid profiles were normal.

Given his young age, family history, and presence of intracardiac thrombus, the patient underwent evaluation for a hypercoagulable state, which revealed positive antiphospholipid antibodies, including anticardiolipin antibody and antibodies against B2-glycoprotein I. His prothrombin time, activated partial thromboplastin time, protein C, protein S, antithrombin III, Factor V Leiden, and fibrinogen levels were

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Figure 1. Electrocardiogram showing ST segment elevation in the anterolateral leads and T wave inversion in the inferior leads.

Figure 2. Coronary angiogram, anteroposterior cranial view, showing distal left anterior descending (LAD) occlusion (yellow arrow).

all within normal limits. He was diagnosed with APS and started on warfarin, with an overlap with therapeutic heparin, in addition to other anti-ischemic medications. Though the etiology of his ST elevation myocardial infarction (STEMI) could be secondary to in situ coronary artery thrombosis in the setting of APS, embolus from his left ventricular thrombus was most likely the cause. The patient was pain free with no more thrombotic events after 1 year of follow-up.

Figure 3. Echocardiogram, apical four-chamber view, revealing a large left ventricular apical thrombus (star).

DISCUSSION

The diagnosis of APS can be established according to the following criteria: one clinical criterion related to a venous or arterial thrombotic event and/or thrombotic events related to pregnancy and the presence of one or more specified anti-phospholipid antibodies (anticardiolipin, anti-beta-2-glycoprotein-1 antibodies, and/or lupus anticoagulant) on two or more occasions at least 12 weeks apart.¹

Cardiac manifestations of APS vary from asymptomatic valve thickening to serious nonbacterial endocarditis (Libman-Sacks endocarditis), ventricular dysfunction, intra-cardiac thrombosis, and accelerated atherosclerosis.²⁻⁴ Myocardial infarction is diagnosed in about 5% of patients

with APS.⁵ Interestingly, in patients with APS, acute coronary syndrome can occur in normal coronary arteries with or without thrombus formation at the time of coronary angiogram. Acute myocardial infarction is usually caused by thromboembolism, microvascular thrombosis, or accelerated atherosclerosis with plaque rupture.^{3,6} Different treatment modalities have been used in patients with APS and STEMI, including isolated thrombolysis, balloon angioplasty, thrombectomy, and stent placement, with variable results.⁶ Although most patients with STEMI and APS undergo primary PCI with stenting, there are reports of an increased risk of stent thrombosis in such patients.^{7,8} In a recent retrospective study, the investigators reported that patients with APS who undergo PCI have worse long-term outcomes, driven by higher rates of stent thrombosis and repeat revascularization.⁹

Another serious complication of APS is intracardiac thrombosis. Our patient was found to have a large apical left ventricular thrombus, which could be related to a combination of a prothrombotic state related to APS and anterior STEMI with severe apical hypokinesis.

The optimal intensity of anticoagulation for the prevention of recurrent thrombosis in patients with APS is uncertain. Studies found that high-intensity anticoagulation (international normalized ratio >3) was associated with a higher bleeding risk and was not superior to moderate-intensity anticoagulation (international normalized ratio = 2–3).^{10,11} Lifelong use of anticoagulation is reasonable due to the high recurrence rate of thrombotic events.¹² At the present time, the data on the efficacy and safety of novel oral anticoagulants in patients with APS are insufficient, and further studies are needed before these agents can be widely recommended.^{13,14} Modification of concomitant risk factors for thrombosis, such as hypertension, dyslipidemia, and smoking cessation, must also be addressed. The patient was treated with warfarin therapy and remained asymptomatic at his 2-year follow-up visit. Repeat echocardiogram revealed resolution of left ventricular thrombus.

Acute myocardial infarction and intracardiac thrombus are very rare in young adults, and prothrombotic conditions such as APS should be considered as a possible etiology. Due to the significantly increased risk of stent thrombosis in patients with APS who present with STEMI, thrombectomy without stent placement may be considered a reasonable treatment option.

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