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Antiphospholipid Syndrome: A Rare Cause of Myocardial Infarction in Young Adults (Case Report and Review of the Literature)

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ABSTRACT

Acute coronary syndrome rarely occurs in young adults and is infrequently associated with antiphospholipid syndrome. We report the case of a 28-year-old man who presented for an acute myocardial infarction with ST-segment elevation myocardial infarction. He benefited from an urgent percutaneous transluminal coronary angioplasty. Hypercoagulability status studies were performed after the event, establishing the diagnosis of antiphospholipid syndrome. Our case highlights this syndrome as a rare cause of myocardial infarction in young adults.

KEYWORDS: Antiphospholipid syndrome, Acute myocardial infarction, Coronarography

I. INTRODUCTION

Antiphospholipid syndrome is an autoimmune disease characterized by vascular thrombosis (arterial, venous, or small vessel) and elevated serum levels of antiphospholipid antibodies (anticardiolipin, lupus anticoagulant, or anti-2 glycoprotein I) [1].

The most common manifestation of antiphospholipid syndrome is deep vein thrombosis (31.7%). Myocardial infarction is a rare manifestation of this syndrome with an overall prevalence of 5.5%, but with a relatively good hospital prognosis (92.5% survival rate) [2]. The median age of diagnosis is 34 years.

However, this rare manifestation can lead to a fatal outcome. We report the case of a 28-year-old man who presented with inaugural chest pain. He was diagnosed with an inferior myocardial infarction due to antiphospholipid syndrome.

II. CASE REPORT

An A 26-year-old man presented to the hospital with 3 hours of chest pain associated with episodes of vomiting and sweating. He had no history of diabetes, smoking, hypertension, hyperlipidemia, family history of early coronary artery disease or sudden cardiac death. He denied using illicit drugs.

Physical examination revealed tachycardia at 110 bpm and a 120/70 mmHg blood pressure. Cardiac and pulmonary auscultation were both normal.

A 12-lead electrocardiogram showed ST-segment elevation in the inferior leads and ST segment depression in extensive anterior (Figure 1).

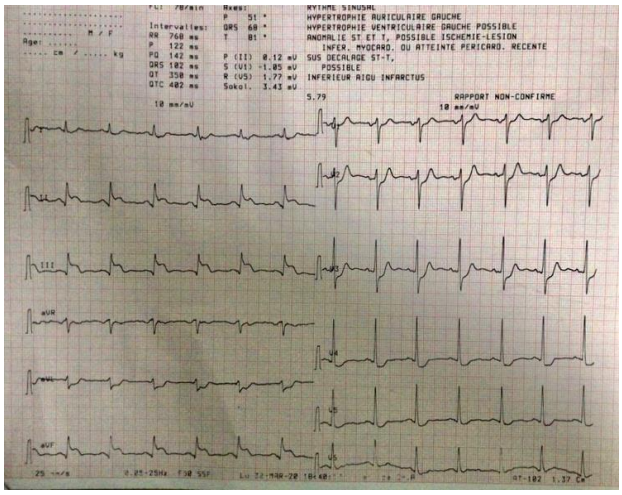

Figure 1. Electrocardiogram showing ST segment elevation inferior and extensive anterior ST segment depression

The patient was admitted to the cardiac catheterization suite, where an emergency coronary angiogram was performed. We found a middle-segment occlusion of the right coronary artery that required percutaneous coronary angioplasty with direct stenting (Figure 2 and 3). The patient received intavenous anticoagulation for the next 10 days, combined with clopidogrel, aspirin, and pravastatin.

An echocardiogram performed afterward showed a decreased left ventricular ejection fraction of 43% and inferior wall hypokinesis.

Because of the young age of our patient, we performed a thrombophilia test: protein C and S, antithrombin III, factor V and factor II mutation, which came back normal, as well as the platelet count and serum protein electrophoresis.

Anticardiolipin antibodies, on the other hand, came back positive with an IgG level of 42 GPL and an IgM level of 19 MPL.

However, antinuclear antibodies were negative, as were circulating lupus anticoagulants and anti-2 glycoprotein I.

After 12 weeks, anticardiolipin antibody assay was performed and was always positive.

These data confirmed the diagnosis of antiphospholipid syndrome. The later was considered primary because of the absence of typical signs of other autoimmune diseases, particularly systemic lupus erythematosus.

We decided to put the patient on oral anticoagulant therapy for life.

The patient remained stable and did not present a recurrence. The stress test, performed 3 and 6 months after angioplasty, was negative.

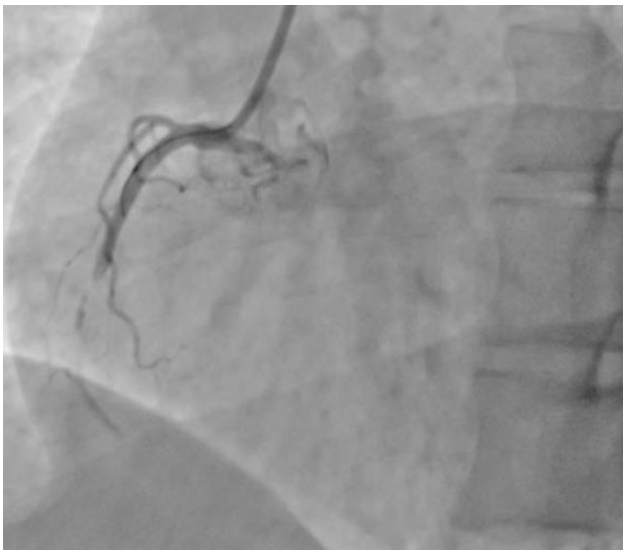

Figure 2. Occlusion of the second segment of the right coronary artery

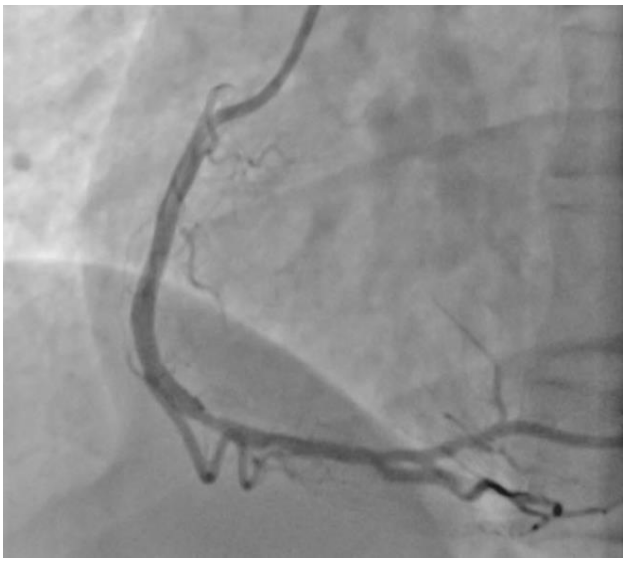

Figure 3. Implantation of a stent in the middle right coronary artery

III. DISCUSSION

Antiphospholipid antibody syndrome (APAS) is an autoimmune disease defined, according to Harris (1987) and Alarcon-Segovia (1992), by the combination of arterial or venous thrombosis, repeated miscarriage or fetal loss, and the presence of antiphospholipid antibodies [3], [4].

The heart is a prime target of antiphospholipid syndrome, both in the endocardium and the coronary circulation. Valvular damage is the most frequent injury, such as mitral valve prolapse or vegetations. Most valvular lesions observed concern left valves. The mitral valve tends to have greater impairment than the aortic one [5].

More rarely, the lesions may be coronary, as illustrated by our observation, with the formation of coronary thrombosis. In a ten-year follow-up of 39 patients with antiphospholipid syndrome, Erkan noted only one episode of myocardial infarction, i.e. 2.5% [6].

The other rare reported observations of myocardial infarction related to the antiphospholipid syndrome find, as in our observation, a major and extensive endoluminal thrombus developed on a coronary network that is either normal or the site of a minimal atheromatous plaque [7], [8].

Three molecular mechanisms explain thrombosis formation:

- The increase in thromboxane synthesis under the direct effect of antiphospholipid antibodies (hence the interest in using aspirin);
- Activation of protein C production (hence the interest in treatment with antivitamin K);
- Abnormal production of tissue factor by the injured endothelium in contact with antiphospholipid antibodies [9].

Treatment of myocardial infarction due to coronary artery disease focuses on coronary reperfusion, dual antiplatelet therapy, and antithrombotic therapy [10]. After coronary angioplasty, lifelong anticoagulation is generally not recommended. However, the mechanism of coronary stenosis or occlusion in antiphospholipid syndrome is primarily thrombotic. Because the treatment of antiphospholipid syndrome with thrombotic events is lifelong anticoagulation at initial presentation, myocardial infarction associated with the antiphospholipid syndrome should be subjected to lifelong anticoagulation.

Hydroxychloroquine, in addition to its protective action during lupus, may be effective in preventing antiphospholipid antibody-induced thrombi formation.

Statins have not been studied in controlled trials during antiphospholipid syndrome [11], but they are most certainly the treatment of choice in these at-risk patients. For example, fluvastatin may inhibit antiphospholipid antibody-induced endothelial cell activation [12].

IV. CONCLUSION

The Myocardial infarction is unusual in the young subject, antiphospholipid syndrome may be one of the etiologies. An

immunological workup should be performed early because an early diagnosis can reduce the burden of the disease. As in our case, percutaneous transluminal coronary angioplasty associated with antithrombotic treatment is effective. Long-term anticoagulant and antiplatelet therapy are recommended.

Given the increased mortality due to antiphospholipid syndrome in certain situations, this case highlights the importance of early diagnosis and effective treatment.

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