

Acute myocardial infarction as the first manifestation of antiphospholipid syndrome

Esmeralda García Padilla^a, MD; Eder N. Flores López^b✉, MD; David L. Aguilar de la Torre^c, MD; Luis C. Álvarez Torrecilla^c, MD; and Rogelio López Alfárez^d, MD

^a Facultad de Estudios Superiores Iztacala UNAM. Tlalnepantla, State of Mexico.

^b Department of Internal Medicine. Hospital General de Cuautitlán ISEM "General Vicente Villada". Cuautitlán, State of Mexico.

^c Department of Interventional Cardiology. Affiliated to Hospital General de Cuautitlán ISEM "General Vicente Villada". Cuautitlán, State of Mexico.

^d Clínica Florida Satélite. Naucalpan, State of Mexico.

Este artículo también está disponible en español

ARTICLE INFORMATION

Received: February 2, 2014

Accepted: April 10, 2014

Competing interests

The authors declare no competing interests

Acronyms

AMI: Acute myocardial infarction

APS: antiphospholipid syndrome

On-Line Versions:

Spanish - English

✉ EN Flores López
Hospital "General Vicente Villada"
Alfonso Reyes S/N, Santa María,
Cuautitlán, CP 54820
Estado de México.
E-mail address:
dr.eder.natanael@msn.com

ABSTRACT

Antiphospholipid syndrome is an autoimmune disease characterized by the combination of venous and arterial thrombosis, whose presentation is very diverse. Acute myocardial infarction is one of the more unusual presentations. This article presents a case with this diagnosis and intracavitary thrombus as presentation form of antiphospholipid syndrome.

Palabras clave: Antiphospholipid syndrome, Acute myocardial infarction, Thrombus

Infarto agudo de miocardio como primera manifestación de síndrome antifosfolípido

RESUMEN

El síndrome antifosfolípido es una enfermedad autoinmune caracterizada por la combinación de trombosis venosa y arterial, cuya forma de presentación es muy diversa. El infarto agudo de miocardio es una de sus presentaciones más inusuales. En este artículo se presenta un caso con este diagnóstico y trombo intracavitario, como forma de presentación del síndrome antifosfolípido.

Palabras clave: Síndrome antifosfolípido, Infarto agudo de miocardio, Trombo

INTRODUCTION

The antiphospholipid syndrome (APS) is an autoimmune disease characterized by the combination of venous and arterial thrombosis, and recurrent fetal loss, which is usually accompanied by thrombocytopenia and elevated antiphospholipid antibodies¹.

Acute myocardial infarction (AMI), as presentation form of this syndrome is very rare. There are clinical and serological criteria to determine its existence. Within the latter the presence of lupus anticoagulant (LA) is required, as well as that of anticardiolipin (ACA) IgG or IgM (higher titers at 40 GPL or MPL) or anti-beta2-glycoprotein I (beta2-GPI) positive IgG or IgM (higher titers at 99th percentile) on at least 2 occasions, separated by 12 weeks according to the modified Sapporo criteria².

The diagnostic criteria for AMI according to the third universal definition of myocardial infarction are³:

1. Detection of an increase or decrease of cardiac biomarkers values (preferably troponin) with at least a value above the 99th percentile of upper reference limit and at least one of the following:
 - Symptoms of ischemia.
 - New or presumably new significant ST-T changes or new left bundle branch block.
 - Appearance of pathological Q waves on the electrocardiogram.
 - Imaging tests of new loss of viable myocardium or new regional abnormalities in wall motion.
 - Identification of an intracoronary thrombus on angiography or autopsy.
2. Cardiac death with symptoms of myocardial ischemia and alleged new ischemic changes on the electrocardiogram or new left bundle branch block, but it occurred before determining cardiac biomarkers or before their values increased.

The primary antiphospholipid syndrome is largely seen in young women with a ratio 3.5:1 compared to men, and the age of first thrombosis is between 15 and 50 years. AMI in the presence of APS occurs in young patients with clinical manifestations similar to the general population. In the Euro-Phospholipid project, AMI was the presentation form of APS in 2.8% of patients⁴.

CASE REPORT

A 25 year old male with no relevant antecedents comes to the office with chest pain of 10/10 intensity at rest, which began during the morning, with adrenergic discharge. In the emergency department an electrocardiogram was performed where ST segment elevation in extensive anterior face (V1-V6) was found; also enzyme elevation with total CPK of 1600 U, and its MB fraction of 210 U as well as positive quick test of

troponin I were demonstrated.

Two hours had gone by from the onset of symptoms, so it is decided to transfer the patient to the Interventional Cardiology Service for possible primary angioplasty. In the coronary angiography a thrombus image occupying part of the proximal and middle segments of the left anterior descending artery is located (**Figure 1**), with a TIMI III blood flow.

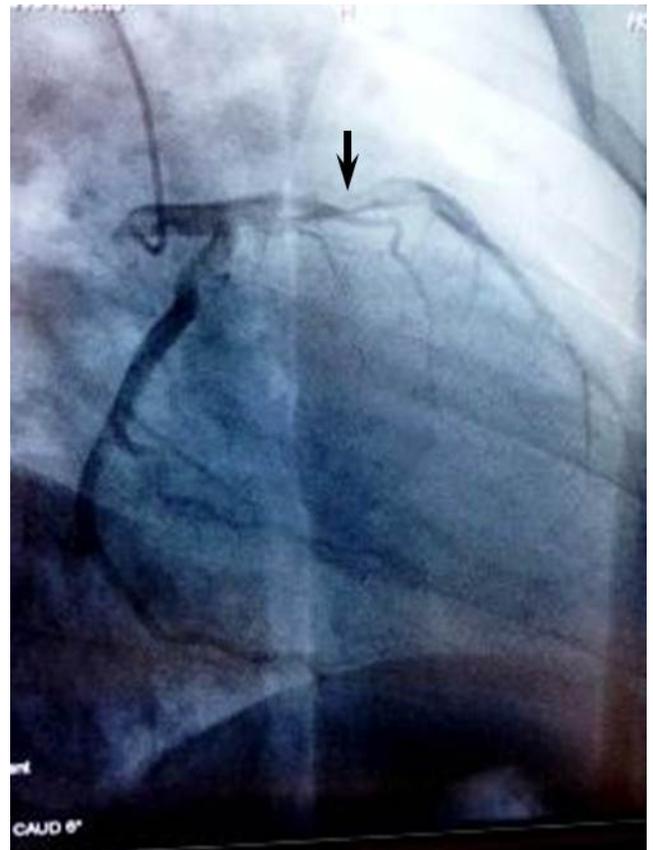


Figure 1. Cardiac catheterization showing the left coronary artery (right anterior oblique with caudal angulation) with thrombus image in proximal and middle segments of the left anterior descending artery (arrow), and TIMI III flow

Due to the risk of fracture and thrombus embolism, no interventional procedure is performed and the patient is taken to the intensive care unit, where the echocardiography performed reports: akinesia of apical region, of the middle and distal thirds of the septum, and anterior, inferior, posterior and lateral walls; also an organized thrombus of 22 x 22 mm in apical region (**Figure 2**).

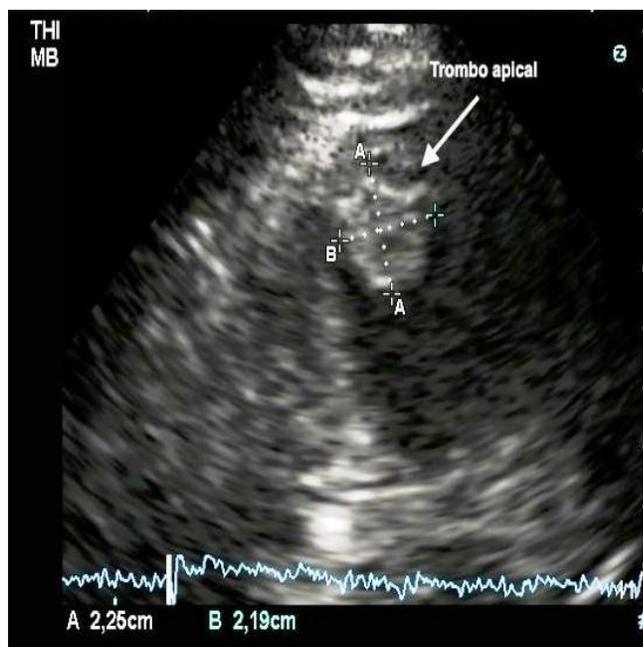


Figure 2. Echocardiographic image of apical thrombus. 2-chambers apical view.

Diagnostic tests with determination by standard ELISA, of IgG type anticardiolipin antibodies (63 GPL, normal value: 0-18 GPL) and IgM (126 MPL, normal value: 0-18 MPL) presented elevated levels.

Lupus anticoagulant was positive in 3 cases by Dilute Russell's viper venom time. And finally, APS diagnosis was established by finding IgG B2 glycoprotein with 36.1 U/ml and IgM at 114 U/ml, also performed by standard ELISA.

Once the primary antiphospholipid syndrome was diagnosed, anticoagulation was added to antiplatelet therapy and treatment with the rest of the anti-ischemic drugs continued. Finally the patient was transferred to the Cardiothoracic Surgery Service where he progressed favorably, without surgery, since there were no embolic complications.

COMMENT

In the series of Davies and Hunt⁵, with more than 800 patients with APS, less than 1% had AMI; and the analysis of 59 patients with APS (27 primary and 32 secondary to systemic lupus erythematosus), performed by Miranda et al.⁶ proved that arterial thrombosis is more prevalent in primary APS; but in this series there were no cases with coronary involvement.

Possible mechanisms of thrombosis in APS include

effects of antibodies on the membrane of platelets, endothelial cells and coagulation components, such as prothrombin and C and S proteins which promote platelet aggregation and clot formation¹.

Three ways in which antiphospholipid antibodies may be associated with coronary heart disease can be stated: first, by thrombosis formation in normal coronary arteries, as in the case of our patient; second, by the possible association of antibodies with accelerated atherosclerosis; and third, by the transient induction of antibodies secondary to cell necrosis that occurs in the AMI⁷.

In this case, no interventional procedure was performed due to the great fracture risk and thrombus embolism, besides the presence of a TIMI III antero-grade flow. However, regardless of the success of treatment in this particular patient, it is important to note that in these cases one can indicate: thrombus aspiration, glycoprotein IIb/IIIa inhibitors⁸ and drug-eluting stents to prevent protrusion of the thrombotic material through its struts⁹.

REFERENCES

1. Prashanth P, Mukhaini M, Riyami A. A rare presentation of primary antiphospholipid syndrome. *Oman Med J.* 2009;24(4):300-2.
2. Miyakis S, Lockshin MD, Atsumi T, Branch DW, Brey RL, Cervera R, et al. International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS). *J Thromb Haemost.* 2006;4(2):295-306.
3. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, et al. Third universal definition of myocardial infarction. *Nat Rev Cardiol.* 2012; 9(11):620-33.
4. Cervera R, Boffa MC, Khamashta MA, Hughes GR. The Euro-Phospholipid project: epidemiology of the antiphospholipid syndrome in Europe. *Lupus.* 2009; 18(10):889-93.
5. Davies JO, Hunt BJ. Myocardial infarction in young patients without coronary atherosclerosis: assume primary antiphospholipid syndrome until proved otherwise. *Int J Clin Pract.* 2007;61(3):379-84.
6. Miranda CH, Gali LG, Marin-Neto JA, Louzada-Júnior P, Pazin-Filho A. Coronary thrombosis as the first complication of antiphospholipid syndrome. *Arq Bras Cardiol.* 2012;98(4):66-9.
7. Jurado OM, Durán J, Martínez A, Castellón JM, Gutiérrez MA. Acute myocardial infarction in a man

- without coronary atheromatosis and antiphospholipid syndrome: report of one case. *Rev Med Chil.* 2009;137(11):1478-81.
8. Kushner FG, Hand M, Smith SC, King SB, Anderson JL, Antman EM, et al. 2009 focused updates: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction (updating the 2004 guideline and 2007 focused update) and ACC/AHA/SCAI guidelines on percutaneous coronary intervention (updating the 2005 guideline and 2007 focused update) a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2009;54(23):2205-41.
 9. Romaguera R, Gómez-Hospital JA, Sánchez-Elvira G, Gómez-Lara J, Ferreiro JL, Roura G, *et al.* MGuard mesh-covered stent for treatment of ST-segment elevation myocardial infarction with high thrombus burden despite manual aspiration. *J Interv Cardiol.* 2013 Feb;26(1):1-7.