CorSalud 2013 Oct-Dec;5(4):388-392

CorSalud

Cuban Society of Cardiology

Case Report

Cardiocentro ERNESTO CHE GUEVARA SANTA CLARA CUBA

Acute coronary syndrome of non-atherosclerotic origin

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ARTICLE INFORMATION

Received: December 12, 2012
Modified: March 21, 2013
Accepted: May 23, 2013

Competing interests
The authors declare no competing interests

Acronyms
ACS: acute coronary syndrome
LA: left atrium

On-Line Versions:
Spanish - English

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ABSTRACT
Coronary embolism is a rare cause of acute coronary syndrome. Among the various types of embolic material is that of tumoral origin. These emboli can cause myocardial ischemia of varying intensity, from angina to acute myocardial infarction or even sudden death. The case of a 58-year-old woman who presented unstable angina episodes with electrical changes with no risk factors and no history of ischemic heart disease is presented. By means of coronary angiography, the presence of normal coronary arteries was showed. Transesophageal echocardiography showed the echogenic polylobulated and pedicled image towards the septal surface of the left atrium (possible myxoma); thrombi in the cardiac chambers were not observed. The patient underwent surgery (surgical removal of the tumor), had a good progress and was transferred to her hospital of origin 72 hours later.
Key words: Acute coronary syndrome, Normal coronary arteries, Myxoma

Síndrome coronario agudo de causa no aterosclerótica

RESUMEN
La embolia coronaria es una causa poco frecuente de síndrome coronario agudo. Dentro de los varios tipos de material embólico se encuentra el de origen tumoral. Estos émbolos pueden ocasionar isquemia miocárdica de intensidad variable, desde angina de pecho hasta infarto agudo de miocardio o incluso, muerte súbita. Se presenta una mujer de 58 años de edad, que presentó episodios de angina inestable con cambios eléctricos sin factores de riesgo y sin antecedentes de cardiopatía isquémica, que en la coronariografía se demostró la presencia de arterias coronarias normales. El ecocardiograma transesofágico informó imagen ecogénica polilobulada y pediculada hacia la superficie septal de la aurícula izquierda (posible mixoma), sin observarse trombos en las cavidades cardíacas. La paciente fue operada (exéresis quirúrgica del tumor), evolucionó favorablemente y fue trasladada a su hospital de origen 72 horas después.
Palabras clave: Síndrome coronario agudo, Coronarias normales, Mixoma

INTRODUCTION

Acute coronary syndrome (ACS) is a group of diseases that represent different stages of a single pathophysiological process: acute myocardial ischemia[1]. Such ischemia usually occurs secondary to coronary atherosclerosis, complicated with thrombotic phenomena that cause different degrees of obstruction to coronary blood flow, and depending on the degree of obstruction and the previous state of the myocardium, it will clinically present as unstable angina, acute myocardial infarction with or without ST elevation, or sudden death[2,3].

The diagnosis of ACS is based on examination of the patient, as the classic clinical description of "pain" or "angina" due to coronary insufficiency (from the Greek ***ankhein***: choking) that was made by William Heberden in 1768[4,5] remains valid, together with the information of the electrocardiogram (ECG) and the identification of myocardial necrosis markers. The latter are very important for the possibility of atypical manifestations such as dyspnea, palpitations and epigastric pain without accompanying crushing chest discomfort[4,6].

This article presents the case of a 58-year-old woman with no history of ischemic heart disease who was urgently hospitalized due to peripheral embolic phenomena, and during admission, she presents episodes of typical chest pain with electrical changes, normal coronary angiographic examination and echocardiographic diagnosis of intracardiac mass.

CASE REPORT

58-year-old white female, with a history of mild mitral valve disease with several years of evolution and previous episodes of uncomplicated peripheral embolisms, who was urgently hospitalized due to new similar embolic episodes, this time to the brain and lower limbs, the latter required surgical treatment. In studies performed, an intracardiac mass in left atrium (LA) of about 3-4 inches was detected by echocardiography, so she was assessed by the Cardiovascular Surgery Service, and transferred to our center to complete diagnosis studies and define definitive treatment.

During admission, the patient began to show typical symptoms of angina with electrocardiographic changes manifested by the presence of negative T waves in V_4-V_6. It was decided to perform emergency coronary angiography which showed coronary arteries with no angiographic lesions, so these episodes of angina were interpreted as ACS without ST-segment elevation by microembolizations originated from intracardiac mass. Subsequently, the patient decompensated hemodynamically, and new ECG changes with diffuse disorder of ventricular repolarization were detected [negative T waves in DI, DII, aVL, aVF, V2 -V6 (**Figure 1**)]. After her hemodynamic stability was achieved the emergency surgery was performed with the preoperative diagnosis of LA tumor (myxoma vs. thrombus).

During surgery, a greenish gray mass of 3 cm in diameter, macroscopically compatible with cardiac myxoma was found. It seemed fragmented with hemorrhagic areas on its surface (**Figure 2**). It was completely resected. Diagnosis was confirmed by histo-

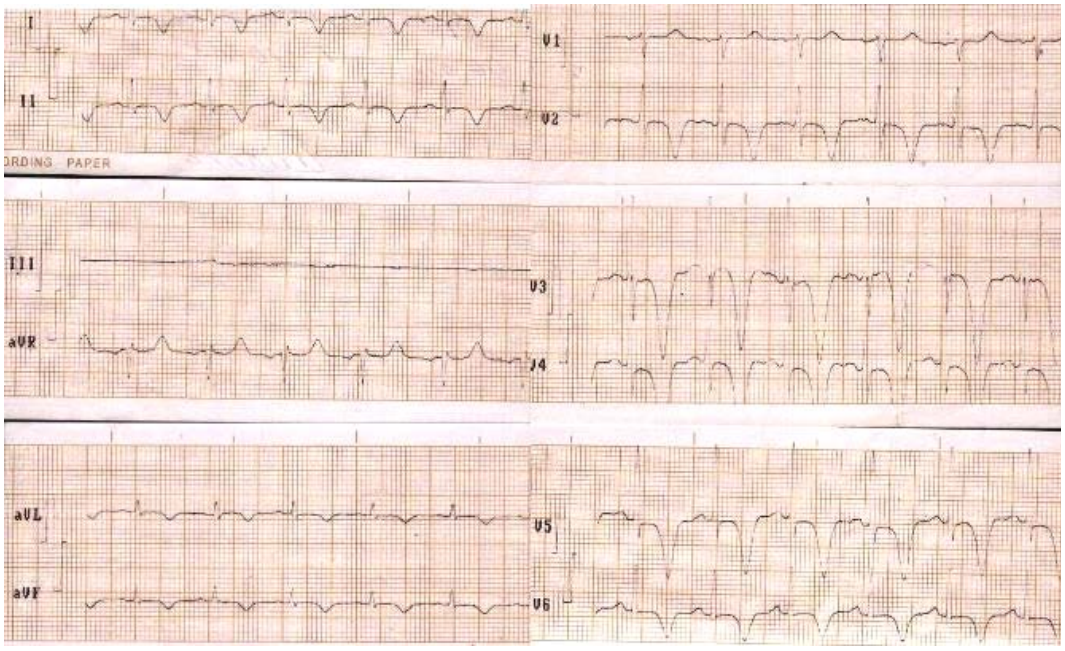

Figure 1. Electrocardiogram showing diffuse disorder of ventricular repolarization.

logical examination. The patient improved and was transferred to her home hospital three days after surgery.

COMMENT

The main cause of ACS are atherothrombotic phenomena. However, there are other congenital and acquired disorders that can produce it, such as:

1. Coronary occlusion secondary to embolization: nonbacterial infective or thrombotic endocarditis, and mural thrombi inside the cavity and tumors [10,11].
2. Inflammatory processes of the coronary arteries such as: viral diseases (particularly Coxsackie B), syphilis, Takayasu's arteritis, polyarteritis nodosa, Kawasaki disease and systemic lupus erythematosus[12-14].
3. Disproportion between the demand and the supply of oxygen by the myocardium: thyrotoxicosis, acute exposure to carbon monoxide, aortic stenosis, prolonged hypotension, acute blood loss [15].
4. Anatomical variations: Anomalous origin of coronary artery, coronary arteriovenous fistula or muscular bridge [16-18].
5. Drugs: Oral contraceptives, hormone replacement therapy[19].
6. Others: Cocaine, radiation, amyloidosis, Hurler syndrome, Fabry disease, homocystinuria [10,20-22].

Cardiac myxomas are benign tumors of endocardial origin. 75 % are found in the LA, pedicled to the interatrial septum. They mainly occur in women between 30 and 60 years of age. They are almost always unique and in 5 % of cases a dominant autosomal inheritance pattern has been observed[23,24].

These tumors can clinically present in various forms: asymptomatic, general symptoms, fever, peripheral embolisms (neurological, extremities, mainly), skin manifestations and cardiovascular symptoms[25-27]. The latter are mainly determined by the location of the tumor with subsequent involvement of adjacent structures, its size and degree of mobility. The most common clinical symptoms are those resulting from obstruction of the mitral valve (dyspnea, orthopnea, and chest pain), that appear in a mitral valve disease[28].

Because the majority of these tumors are friable, a third of patients present peripheral or central embolic phenomena, either by tumor fragments or thrombi formed in its surface[29]. Coronary embolisms are rare, due to the location of coronary arteries which, during systole, are protected by the aortic valve leaflets[30]. However, they can occur and be the cause of an acute myocardial infarction; a possibility that should be considered especially in young patients without risk factors and normal coronary angiographic studies. Coronary embolization may involve embolisms exclusive to those arteries or associated with multiple embolisms, and may be spontaneous, as in the case presented, or secondary to surgical manipulation during surgery[29-31]. In this particular patient ACS must have been produced by microembolizations of the tumor, fragments of thrombi formed in its surface or vasoreact-

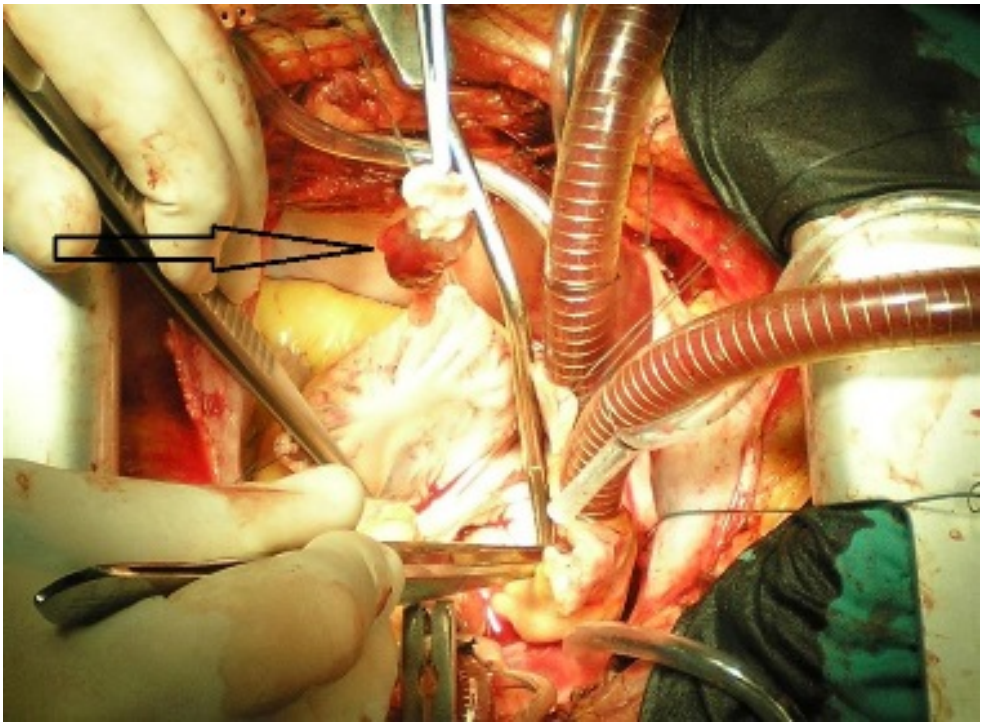

Figure 2. Anatomical piece (cardiac myxoma) resected from the left atrium (arrow).

ivity produced by a systemic inflammatory response syndrome, since the possibility of an occlusive thrombus in one of the epicardial arteries is discarded by the absence of ST-segment elevation and angiographic evidence of normal coronaries.

REFERENCES

1. Rizo Rivera GO, Ramírez Gómez JI, Gómez García Y. Enfoque actual sobre la fisiopatología del síndrome coronario agudo. Rev Cub Med. 2009;48(3):71-87.
2. Vilariño JO, Esper R, Badimón JJ. Fisiopatología de los síndromes coronarios agudos. Tres paradigmas para un nuevo dogma. Rev Esp Cardiol. 2004;4(Supl G):13-24.
3. García-Castrillo Riesgo L. Epidemiología del síndrome coronario agudo en los servicios de urgencias. Emergencias. 2002;14:S69-74.
4. Montero Hechavarría E, Rodríguez Leyva BA, Blanco Gómez L, Vidal Sigas VM, Mata Mendoza M. Enfoque diagnóstico y terapéutico del síndrome coronario agudo. MEDISAN [Internet]. 2010 [citado 2012 Nov 26];14(1):79-89. Disponible en: http://www.bvs.sld.cu/revistas/san/vol_14_1_10/san13110.pdf
5. Aguilar Pacín N. Enfermedades del Sistema Cardiovascular: Cardiopatía isquémica. En: Manual de terapéutica de Medicina Interna. La Habana: Editorial Ciencias Médicas; 2007. p. 18-40.
6. Ramos HR. Las Unidades de Dolor Torácico: una nueva solución para un antiguo problema. [Internet] 4to. Congreso Virtual de Cardiología, 2005 [citado 2012 Nov 26] Disponible en: http://www.fac.org.ar/ccvc/llave/c121/ramos.php
7. Fernández Portales J, García Robles JA, Jiménez Candil J, Pérez David E, Rey Blas JR, Pérez de Isla L, *et al*. Utilidad clínica de los distintos marcadores biológicos CPK, CPK MB masa, mioglobina y troponina T en una unidad de dolor torácico. ¿Cuándo, cuáles y cómo pedirlos? Rev Esp Cardiol. 2002; 55(9):913-20.
8. Corbalán R, Dighero H, Florenzano F, Yovanovich J. Pautas de la Sociedad Chilena de Cardiología y Cirugía Cardiovascular: Síndrome coronario agudo sin elevación ST (SCA SEST) Rev Chil Cardiol. 2006; 25(3):339-49.
9. Valero-Elizondo G, Jáuregui-Aguilar R, Soto-Márquez P. Paciente joven con infarto del miocardio recurrente sin aterosclerosis coronaria. Gac Méd Méx. 2002;138 (5):473-6.
10. Mangariello BN, Payaslian M, Albornoz M, Miguelez P, Gos Re V, Biasco A, *et al*. Tromboembolia coronaria múltiple como causa de infarto agudo de miocardio. Rev Argent Cardiol. 2010;78(3):255-8.
11. Manzano MC, Vilacosta I, San Román JA, Aragoncillo P, Sarriá C, López D, *et al*. Síndrome coronario agudo en la endocarditis infecciosa. Rev Esp Cardiol. 2007;60(1):24-31.
12. Bermejo García J, Martínez Martínez P, Martín Rodríguez JF, De la Torre Carpente M, Bustamante Bustamante R, Guerrero Peral AB, *et al*. Inflamación e infección en la enfermedad coronaria estable y en el síndrome coronario agudo. Rev Esp Cardiol. 2001;54(4):453-9.
13. Chierchia SL. Inflamación y síndrome coronario agudo. Rev Esp Cardiol. 2001;54(10):1135-40.
14. Domínguez Franco AJ, Pérez Caravante M, Jiménez Navarro MF, De Teresa Galván E. Prevención y tratamiento del síndrome coronario agudo. Rev Esp Cardiol. 2006;6(Supl G):36-45.
15. Martin Rabasco J. Cardiopatía isquémica crónica. Fisiopatología y etiología, datos clínicos, síntomas, examen físico, elección de un procedimiento diagnóstico y tratamiento. [Internet] [citado 2012 Nov 26]. Disponible en: http://www.monografias.com/trabajos-pdf4/cardiopatia-isquemica-cronica/cardiopatia-isquemica-cronica.pdf
16. Jeanmet T, Maragnes P, Le Pennec V, Labombarda F. Origen anómalo de la coronaria derecha con un trayecto interarterial. Rev Esp Cardiol. 2010;63(4): 492-3.
17. Valera Martínez FJ, García-Sánchez F, Serrano Martínez F, Torregrosa Puerta S, Maroñas Andrade JM, Margarit Calabuig JA, *et al*. Origen anómalo de la arteria coronaria derecha del seno aórtico izquierdo. Cir Cardiov. 2005;12(2):141-4.
18. Sáez de Ibarra JI, Fernández-Tarrío R, Forteza JF, Bonnín O. Fístula coronaria gigante entre el tronco coronario izquierdo y vena cava superior complicada de disección coronaria. Rev Esp Cardiol. 2010; 63(6):743-4.
19. Serra CM, Pacheco G, Sala JP, Balestrini C. Fisiopatología: un aporte para el reconocimiento de la enfermedad coronaria en la mujer. Rev Fed Arg Cardiol. 2006;35(2):92-100.
20. Guimarães Victor E, De Oliveira Parente GB. Radioterapia mediastínica e lesão ostial de tronco de

coronária esquerda. Arq Bras Cardiol. 2004;82(3): 295-7.
21. Valeti US, Wilson RF, Vlodaver Z. Nonatherosclerotic Ischemic Heart Disease. In: Vlodaver Z, Wilson RF, Garry DJ. Coronary Heart Disease. Minneapolis: University of Minnesota; 2012. p. 365-88.
22. Rodríguez M, Solari S, León L, Ramírez J, Pérez O, Guarda E. Complicaciones cardiovasculares por uso de cocaína. Rev Chil Cardiol. 2007;26(4):459-66.
23. Osío LF, Restrepo G, Sánchez J, Olaya P. Mixoma atrial: múltiples formas de presentación. Reporte de casos y discusión del tema. Rev Bras Ecocardiogr Imagem Cardiovasc. 2011;24(1):97-102.
24. González EL, Pizzi MN, Caponi MG, Vigliano C, Varela Otero MD, Dulbecco E, *et al*. Mixomas cardíacos: presentación clínica, resultados quirúrgicos y pronóstico a largo plazo. Rev Argent Cardiol. 2010; 78(2):108-13.
25. Oliveira R, Branco LM, Galrinho A, Abreu A, Abreu J, Fiarresga A, *et al*. Mixomas cardíacos, 13 anos de experiencia no seu diagnóstico ecocardiográfico. Rev Port Cardiol. 2010;29(7-8):1087-100.
26. Vilela EP, Moura L, Pepe D, Nunes E, Erthal F, Campana E. Mixoma atrial gigante simulando estenosis mitral grave en paciente joven. Arq Bras Cardiol [Internet]. 2010 [citado 2012 Nov 27];95(5):e125-7. Disponible en: http://www.scielo.br/pdf/abc/v95n5/es_v95n5a23.pdf
27. Moreno-Martínez FL, Lagomasino Hidalgo A, Mirabal Rodríguez R, López Bermúdez FH, López Bernal OJ. Mixoma auricular izquierdo recidivante. Arch Cardiol Mex. 2003;73(4):280-3.
28. Medrano Plana Y, Vázquez Roque FJ, Lagomasino Roque AL, Puig Reyes I, Hernández Borroto CE, Bermúdez Yera G, *et al*. Mixomas cardiacos. Resultados del tratamiento quirúrgico en 23 años de trabajo. Rev Electrón PortalesMedicos.com [Internet]. 2010 [citado 2012 Nov 27];5(6) [aprox. 4 pp.]. Disponible en: http://www.portalesmedicos.com/publicaciones/articles/2081/1/Mixomas-cardiacos-Resultados-del-tratamiento-quirurgico-en-23-anos-de-trabajo.html
29. Aguilera B, Suárez-Mier MP, García García R. Muerte súbita por embolia cerebral de mixoma de la aurícula izquierda. Cuad Med Forense. 2011;17(3): 149-53.
30. Marchena Yglesias PJ, De Benito Cordón LP, Hervás Laguna MJ, Ruiz Climente M, Dimas Núñez JF. Mixoma auricular izquierdo como forma de presentación de una angina de esfuerzo. An Med Interna (Madrid). 2006;23(6):279-81.
31. Clavellina Rosas JM, Varela Carrillo M. Infarto agudo miocárdico inferior secundario asociado a un mixoma de la aurícula izquierda. An Med Asoc Med Hosp ABC. 2000;45(3):145-8.