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Case Report

Cardiocentro ERNESTO CHE GUEVARA SANTA CLARA CUBA

Interventricular septal rupture after acute myocardial infarction with intermittent opening and closing

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ARTICLE INFORMATION

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Acronyms
IAM: Infarto agudo de miocardio
SIV: Septo interventricular
ECG: Electrocardiograma

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ABSTRACT
Ventricular septum rupture is a serious complication in patients with acute myocardial infarction. It occurs in approximately 1% of heart attack patients; its mortality rate is high and surgical repair is the treatment of choice. The case of an elderly male patient who was admitted to the Intensive Care Unit for acute anterior myocardial infarction is reported. This patient received thrombolytic therapy with Cuban recombinant streptokinase and 24 hours later presented hemodynamic deterioration with electrocardiographic changes and appearance of systolic murmur at the apex. Echocardiography was performed which showed a ventricular septal defect with intermittent opening and closing. Despite treatment, the patient died of circulatory failure hours later. Echocardiographic images and the anatomical specimen are shown. What was unusual in this case was the intermittent opening and closing of the ventricular septal defect. No similar report was found in the bibliographic databases consulted.
Key words: Myocardial Infarction; Heart Rupture, Post-Infarction; *Shock*, Cardiogenic

Rotura del septo interventricular después de infarto agudo de miocardio con apertura y cierre intermitentes

RESUMEN
La rotura del septo interventricular es una grave complicación en pacientes que sufren infarto agudo de miocardio. Se presenta aproximadamente en el 1 % de los pacientes infartados, su mortalidad es elevada y el tratamiento de elección es la reparación quirúrgica. Se presenta un paciente anciano que ingresó en la Unidad de Cuidados Intensivos por infarto agudo de miocardio de cara anterior, que recibió tratamiento trombolítico con estreptokinasa recombinante cubana y 24 horas más tarde, presentó deterioro hemodinámico con cambios electrocardiográficos y aparición de soplo sistólico en la punta. Se realizó una ecocardiografía que mostró un defecto del septo interventricular con apertura y cierre intermitentes. Horas más tarde el paciente falleció por insuficiencia cardiocirculatoria, a pesar del tratamiento. Se presentan las imágenes ecocardiográficas y la pieza anatómica. Lo inusual del presente caso fue la apertura y el cierre intermitentes del defecto interventricular. No se

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encontró ningún informe similar a en las bases de datos bibliográficas consultadas.
Palabras clave: Infarto de miocardio; Rotura Cardíaca Postinfarto; Choque Cardiogénico

INTRODUCTION

Acute myocardial infarction (AMI) is a major health problem both in developed and developing countries. Currently, it affects more than 3,000,000 people each year[1]. Advances in preventive measures and diagnostic and therapeutic techniques have reduced hospital mortality of AMI from 25 and 30% in the 1960s to 6% nowadays[2].

The main causes of death in patients with AMI are cardiac arrhythmias and mechanical complications that lead to cardiogenic shock. The latter, in patients with AMI, cause a severe hemodynamic compromise with high mortality, even when the correct treatment is performed[3].

The case of an elderly patient with AMI who received thrombolytic treatment with Cuban recombinant streptokinase, and who developed a fatal cardiogenic shock after a rupture of the interventricular septum (IVS), is reported.

CASE REPORT

A male patient, 85 years old, with a history of apparent health, was admitted to the Intensive Care Unit referred from the health area by presenting a severe oppressive retrosternal pain, radiating to the jaw, with a sudden onset while he was going to perform his daily activities. It lasted about an hour and a half, and was accompanied by profuse sweating and feeling of death; relieved with opioid administration.

A 12-lead electrocardiogram (ECG) was performed. It showed sinus rhythm, heart rate of 100 bpm; PR, 113 ms; QRS, 95 ms; QT 320 ms; QRS axis, -35°; presence of Q waves in leads I, II, III, aVF, and from V2 to V6, with ST positive displacement from V2 to V6, and diffuse disorder of ventricular repolarization. The P wave in DII was 0.035 sec and 0.01 mV. An acute coronary syndrome with ST elevation was diagnosed, and thrombolysis with Cuban recombinant streptokinase was performed, according to the protocol of the service. There were no incidents during drug administration, except isolated premature ventricular beats. The ECG performed after thrombolysis showed no changes compared to the initial one, therefore, it was assumed that the treatment was not effective for the recanalization of the infarct-related artery.

After 24 hours of admission, the patient had clinical

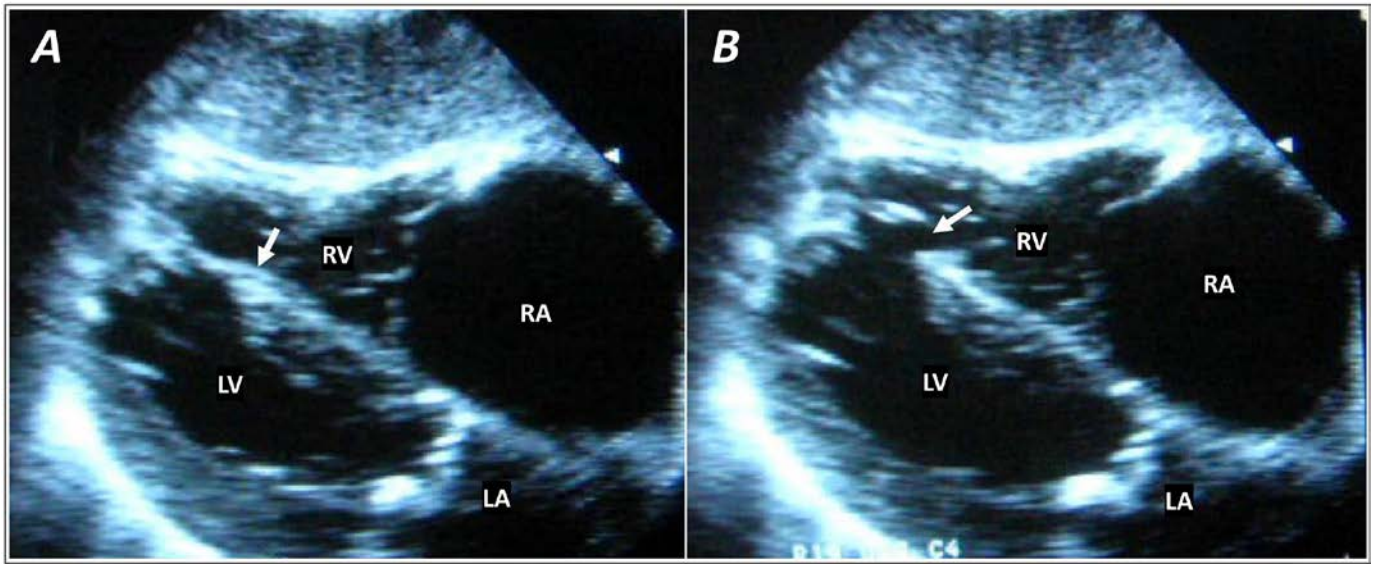

Figure 1. Interventricular septum rupture. The arrow indicates the ventricular septum defect. **A.** Closed in systole. **B.** Open in diastole. Caption: RV: right ventricle; RA: right atrium, LV: left ventricle; LA: left atrium.

symptoms characterized by malaise, increased heart and respiratory rates, and intense retrosternal pain. Severe hypotension (54/26 mmHg) and increased heart rate (135 bpm) were found. Physical examination revealed intense holosystolic murmur at the left sternal border with no signs of venous congestion and absence of pulmonary rales; delayed capillary refill and temperature gradient in the mid-third of the extremities. Cardiogenic shock was diagnosed and, from a clinical standpoint, it was considered that the cause could be a chordal rupture of the mitral valve or a rupture of the IVS.

An echocardiography was performed which revealed, in subxiphoid view, right chambers dilatation and thinning of the IVS in the apical region, with an image cut at that level that was reminiscent of valve movement with opening and closing of the defect (Figure 1). The valvular apparatus showed degenerative changes (slight calcification) and seemed to be competent according to the pulsed Doppler. Moreover, the absence of thrombus or pericardial effusion was established. Echocardiographic examination confirmed the clinical diagnosis of interventricular septum rupture.

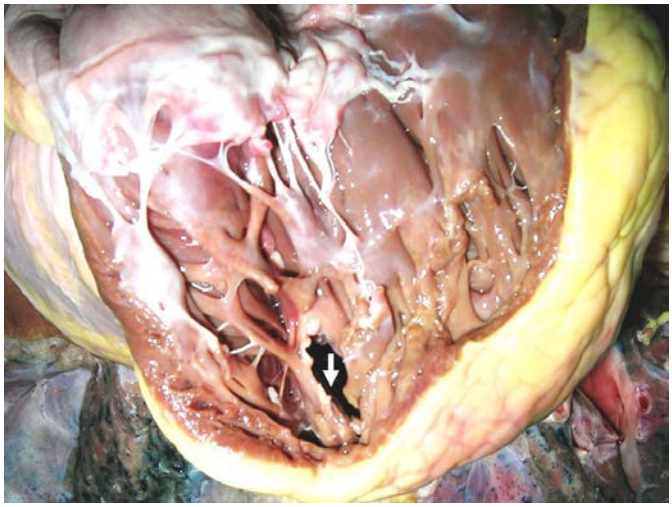

Figure 2. Anatomical specimen of the heart. The arrow is at the site of the rupture of the septum.

The patient died the following day. The autopsy showed a severe atherosis of the aorta and the coronary arteries, signs of acute myocardial infarction in the anterior region of the left ventricle, and a rupture of 1.5 cm^2 in the apical region of the interventricular septum (Figure 2).

COMMENT

IVS rupture occurs approximately in 1% of all patients with AMI and in 0.2% of patients receiving thrombolytic treatment; in the latter, it occurs in the early hours of the coronary accident, however, it is often seen later (between two to five days)[4] in patients where this strategy is not used. Its frequency is higher in women, especially at older ages and with anterior wall location.

In patients with anterior wall AMI, the rupture is observed in the anteroapical region, while in those with inferior wall AMI, the ventricular septum defect is observed in the basal region of IVS[5].

The mortality of this complication is very high, and the surgical closure is the treatment of choice. The operative mortality of these patients is between 25%, in anterior wall acute myocardial infarction, and 58%, in inferior wall infarctions[6]. Survival without short term surgery is exceptional; however, aspects of the treatment of ventricular septum defect closure are currently discussed, as well as the optimal time for surgery[7]. Some patients with severe hemodynamic compromise and a large left to right shunt, benefit from the temporary transcutaneous closure by catheter[8] or by left ventricular assist devices, with the aim of improving the patient's conditions for subsequent surgical repair[8]. Notwithstanding the above mentioned, there have been reports of spontaneous closures of ventricular septum defects that presented as complications of AMI [9].

Clinical diagnosis is made by detecting a murmur of recent onset that is intense, pansystolic, more audible in the lower left sternal area, with presence of thrill, and accompanied by a significant clinical deterioration, often with biventricular dysfunction. Hemodynamic manifestations in these patients are due to the acute overload of pressure in the heart's right chambers, caused by the defect of the IVS. The sudden onset or worsening of cardiogenic shock, increasing dyspnea and signs of pulmonary and splanchnic engorgement have also been described[10].

ECG traces taken at the time of the rupture of the IVS have shown increased heart rate, ST segment elevation, decrease in the depth of the Q wave and increase of the voltage and duration of the P wave[11].

Echocardiography is very useful for the diagnosis of functional and mechanical complications in AMI pa-

tients, as it can be performed immediately at the bedside, and has high sensitivity and specificity[12].

Other imaging tests for functional and perfusion testing and multislice computed tomography are useful for diagnosing IVS rupture in patients with AMI[13]. Similarly, the nuclear magnetic resonance studies have been used for the diagnosis and surgical repair of mechanical complications in patients with AMI [14].

Elevated levels of brain natriuretic peptide (BNP) have been documented in patients with AMI who have shown IVS rupture. This marker has been considered a predictor of left ventricular dysfunction, mechanical complications and death in patients with AMI[15].

In spite of all the developments related to the diagnosis and treatment of IVS rupture, its incidence and mortality remain high in patients suffering from AMI.

REFERENCES

1. Lloyd-Jones D, Adams R, Carnethon M, De Simone G, Ferguson TB, Flegal K, *et al*. Heart Disease and Stroke Statistics 2009 Update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2009;119:e21-181.
2. Van de Werf F, Bax J, Betriu A, Blomstrom-Lundqvist C, Crea F, Falk V, *et al*. Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation: the Task Force on the Management of ST-Segment Elevation Acute Myocardial Infarction of the European Society of Cardiology. Eur Heart J 2008;29(23): 2909-45.
3. Birnbaum Y, Fishbein MC, Blanche C, Siegel RJ. Ventricular septal rupture after acute myocardial infarction. N Engl J Med. 2002;347(18):1426-32.
4. Crenshaw B, Granger C, Birnbaum Y, Pieper K, Morris DC, Kleiman NS, *et al*. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) trial investigators. Circulation. 2000:101(1):27-32.
5. Cummings RG, Reimer KA, Califf R, Hackel D, Boswick J, Lowe JE. Quantitative analysis of right and left ventricular infarction in the presence of postinfarction ventricular septal defect. Circulation. 1988;77(1):33-42.
6. Jones MT, Schofield PM, Dark JF, Moussalli H, Deiraniya AK, Lawson RA, *et al*. Surgical repair of acquired ventricular septal defect. Determinants of early and late outcome. J Thorac Cardiovasc Surg. 1987;93(5):680-6.
7. Curcio A, Martín J, Wilhelmi M, Soria JL. Doble complicación postinfarto agudo de miocardio: rotura del septo interventricular e insuficiencia mitral aguda. Rev Esp Cardiol. 1997;50(2):129-32.
8. Gregoric ID, Bieniarz MC, Arora H, Frazier OH, Kar B, Loyalka P. Percutaneous ventricular assist device support in a patient with a postinfarction ventricular septal defect. Tex Heart Inst J. 2008;35(1):46-9.
9. Williams RI, Ramsey MW. Spontaneous closure of an acquired ventricular septal defect. Postgrad Med J. 2002;78(921):425-6.
10. Coskun KO, Coskun ST, Popov AF, Hinz J, Schmitto JD, Bockhorst K, *et al*. Experiences with surgical treatment of ventricle septal defect as a post infarction complication. J Cardiothorac Surg. 2009; 4:3. Available at: http://www.cardiothoracicsurgery.org/content/4/1/3
11. Kerr F, Haywood LJ. Electrocardiographic changes produced by interventricular septal rupture. Br Heart J. 1976;38(10):1098-100.
12. Soriano CJ, Pérez-Boscá JL, Canovas S, Ridocci F, Federico P, Echanove I, *et al*. Septal rupture with right ventricular wall dissection after myocardial infarction. Cardiovasc Ultrasound. 2005;3:33. Available at: http://www.cardiovascularultrasound.com/content/3/1/33
13. Ghersin E, Lessick J, Abadi S, Agmon Y, Adler Z, Engel A, *et al*. Ventricular septal rupture complicating myocardial infarction: comprehensive assessment of cardiac coronary arteries, anatomy, perfusion and function by multidetector computed tomography. Can J Cardiol. 2008;24(3):e21-2.
14. Vogel-Claussen J, Skrok J, Fishman EK, Lima JA, Shah AS, Bluemke DA. Cardiac CT and MRI guide surgery in impending left ventricular rupture after acute myocardial infarction. J Cardiothorac Surg. 2009;4:42. Available at: http://www.biomedcentral.com/content/pdf/1749-8090-4-42.pdf
15. Fazlinezhad A, Rezaeian MK, Yousefzadeh H, Ghaffarzadegan K, Khajedaluee M. Plasma Brain Natriuretic Peptide (BNP) as an Indicator of Left

Ventricular Function, Early Outcome and Mechanical Complications after Acute Myocardial Infarction. Clin Med Insights Cardiol. 2011;5:77-83.