



Endocarditis and spondylodiscitis as a complication of postinfarction ventricular aneurysm rupture

Endocarditis y espondilodiscitis como complicación de ruptura de aneurisma ventricular postinfarto

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ABSTRACT. Postinfarction ventricular aneurysm is defined as a dilatation of the left or right ventricle in a saccular form that can cause its rupture at the level of the ventricular wall or septum and can predispose to endocarditis due to the production of turbulence caused by short circuits that cause damage to the endocardial tissue. with the consequent bacterial colonization and systemic embolism. Once the diagnosis is confirmed, antibiotic treatment should be given and in case of complications, surgical treatment should be considered. We report the case of a male patient with a previous history of acute myocardial infarction, who attended the emergency room due to systemic inflammatory response data, showing endocarditis in the tricuspid valve and a ruptured ventricular aneurysm, causing embolic phenomena in the dorsal column, therefore which required surgical treatment.

Keywords: Ventricular aneurysm, infarction, endocarditis, spondylodiscitis.

RESUMEN. El aneurisma ventricular postinfarto se define como una dilatación del ventrículo izquierdo o derecho en forma sacular que puede provocar su ruptura a nivel de la pared o septum ventricular y puede predisponer a endocarditis debido a la producción de turbulencias ocasionadas por cortocircuitos que ocasionan daño del tejido endocárdico con la consecuente colonización bacteriana y embolismos sistémicos. Una vez confirmado el diagnóstico, debe otorgarse tratamiento antibiótico y, en caso de complicaciones, valorarse el tratamiento quirúrgico. Se reporta el caso de un paciente masculino con antecedente previo de infarto agudo de miocardio, el cual acudió a urgencias por presentar datos de respuesta inflamatoria sistémica, evidenciándose endocarditis en válvula tricúspide y un aneurisma ventricular roto, condicionando fenómenos embólicos a columna dorsal, por lo que requirió tratamiento quirúrgico.

Palabras clave: Aneurisma ventricular, infarto, endocarditis, espondilodiscitis.

INTRODUCTION

Post infarction ventricular aneurysm is defined as a non contractile and circumscribed dilation of the left or right ventricle in a saccular form, secondary to a large area of thinned necrotic tissue, as a consequence of an acute myocardial infarction (AMI). It is more common in males, between 50-60 years of age, predominating in the left ventricle in 95% of cases and

5% in the right ventricle.^{1,2} A review of the pathophysiology, diagnostic approach and treatment of this serious complication.

CASE PRESENTATION

57 years old male with a history of smoking and cocaine use, lower ST segment elevation AMI 15 years ago (Figure 1A), not revascularized, without treatment by own decision. He

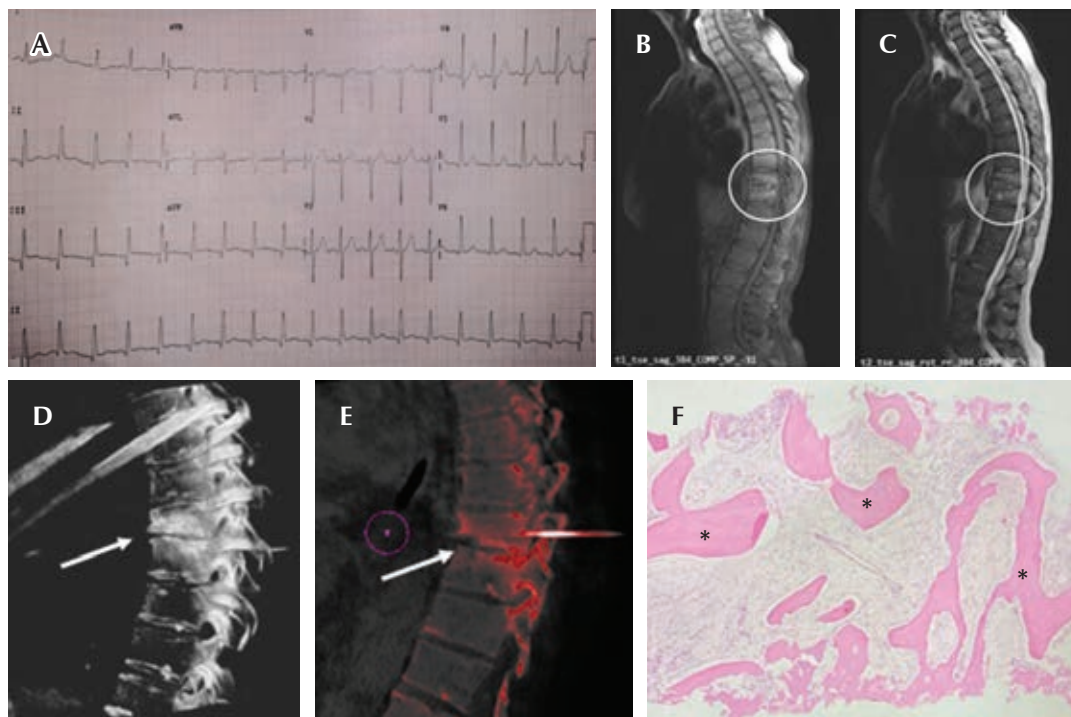


Figure 1: **A)** 12-lead electrocardiogram, HR 100 bpm, pathological Q waves in DII, DIII, aVF. **B, C)** MRI of the dorso lumbar spine showing destruction at the level of D9-D10 with T1, T2 and STIR sequences. **D, E)** Interventional-guided bone biopsy (arrows). **F)** Optical microscopy of bone marrow. Hematoxylin-eosin staining. Lymphocytes, plasma cells, macrophages, and myxoid degeneration are seen between the intertrabecular spaces (*).

began three months prior to admission with dyspnea with less effort than usual, fever of 38.5 °C predominantly at night, myalgias, arthralgias and pain in the thoracic spine, asthenia, for which he went to the emergency room, deciding to hospitalize him in Internal Medicine. Cardiovascular exploration in the mitral and tricuspid focus revealed a holosystolic, regurgitant murmur, both of intensity III/VI. In the left parasternal region, a holosystolic murmur was auscultated, intensity IV/VI, low tone, radiating to the right parasternal region, accompanied by a thrill. Blood cultures were performed, having positive isolation to *Streptococcus mitis*/*Streptococcus oralis*, starting treatment with ceftriaxone 1 g IV every 24 hours for seven days, with subsequent control of blood cultures which remained positive. It was assessed by stomatology finding pulp necrosis in upper left and right premolars, extracting said teeth. Given the persistence of pain in the spine, an MRI was

requested, finding destruction of the vertebral bodies at D9-D10 (*Figure 1B and 1C*), for which the Traumatology and Orthopedics service was consulted, indicating a percutaneous biopsy (*Figure 1D and 1E*), who reported bone marrow with hypocellularity and myxoid degeneration between the intertrabecular spaces (*Figure 1F*), reporting culture of said sample positive for *S. mitis*/*S. oralis*, adjusting treatment to vancomycin 1 g IV every 12 hours for six weeks. A transthoracic echocardiogram was performed, which reported dilated left cavities, LVEF 54%, inferior akinesia in its three segments, severe mitral regurgitation and moderate tricuspid regurgitation (*Figure 2A*), in addition to an 11 mm ventricular septal defect, in the lower border, with flow from left to right, Qp/Qs 2.1, and an inferoseptal aneurysm in the basal segment (*Figure 2B and 2C*). For persistence of fever and positive cultures, a transesophageal echocardiogram was performed, finding an oscillating mass of 10 × 8 mm, pedunculated,

hypermobile, at the base of the anterior valve of the tricuspid, which caused moderate tricuspid regurgitation, in addition to mitral valve with tenting that caused regurgitation severe mitral valve (Figure 2D to 2F). Thoracoabdominal CT angiography was performed, finding data of multisegmental pneumonia (Figure 3A and 3B), observing saccular calcification in the posterior region of the left ventricle (Figure 3C and 3D). Coronary angiography was performed, which showed 75% stenosis in the anterior descending artery and total occlusion in the middle segment of the right coronary artery (Figure 4A and 4B), corroborating skull and caudal LAO projection with saccular calcification suggestive of aneurysm (Figure 4C). He was scheduled for surgical treatment where a left ventricular aneurysm was observed. Tricuspid valve with vegetation and perforation of the anterior leaflet was observed, performing bicuspidization with resection of the anterior leaflet plus vegetectomy, identifying a basal septal defect of 20 mm, deciding to close with a pericardial patch. Revascularization was performed with

a bypass of the anterior descending artery distal to the aorta, observing mitral valve with myxomatous degeneration, which was replaced with a mechanical prosthetic valve, concluding the procedure without complications. The patient was subsequently discharged home, with follow up in outpatient Traumatology and Orthopedics, Cardiothoracic Surgery and Cardiology. A biopsy of the mitral valve was subsequently obtained, reporting data of myxomatous degeneration, and tricuspid anterior leaflet with fibrinoid necrosis and the presence of bacterial colonies (Figure 4D and 4E).

DISCUSSION

The presence of a true ventricular aneurysm in the inferoposterior wall is rare (3%), since those located in this region are usually pseudoaneurysms.³ Diagnosis is achieved with an echocardiographic study or at the time of cardiac catheterization, by means of left ventriculography, with CT and cardiac MRI

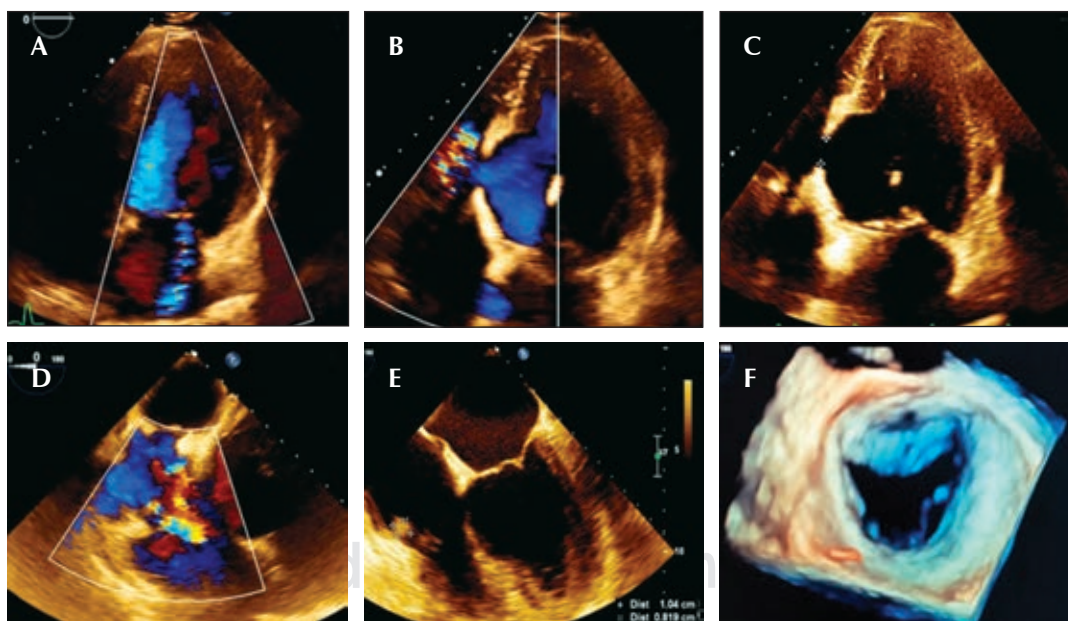


Figure 2: **A)** Transthoracic echocardiogram. Apical 4 chambers with Doppler color. Severe mitral regurgitation jet is observed. **B)** Apical 4 chamber Doppler color. Passage of flow through the ventricular septal defect is observed. **C)** Apical 4 chambers. Evidence of ventricular septal defect of 11 mm. **D)** Transesophageal echocardiogram with Doppler color. Ventricular septal defect is evident. **E)** Vegetation in the anterior tricuspid valve of 1.04 × 0.8 cm. **F)** 3D reconstruction of the mitral valve with tenting.

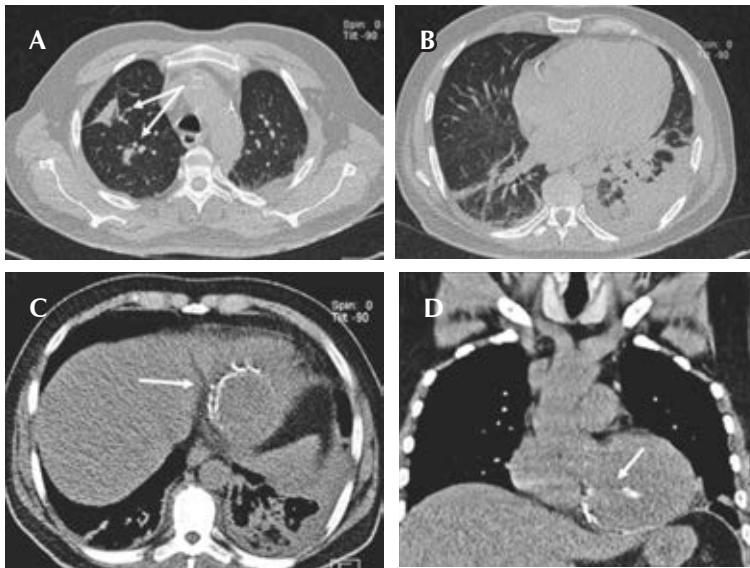


Figure 3: Chest CT. **A, B**) Multisegmental pneumonia data (arrows). **C, D**) Axial and coronal section. A calcified left ventricular aneurysm is observed at the basal level (arrows).

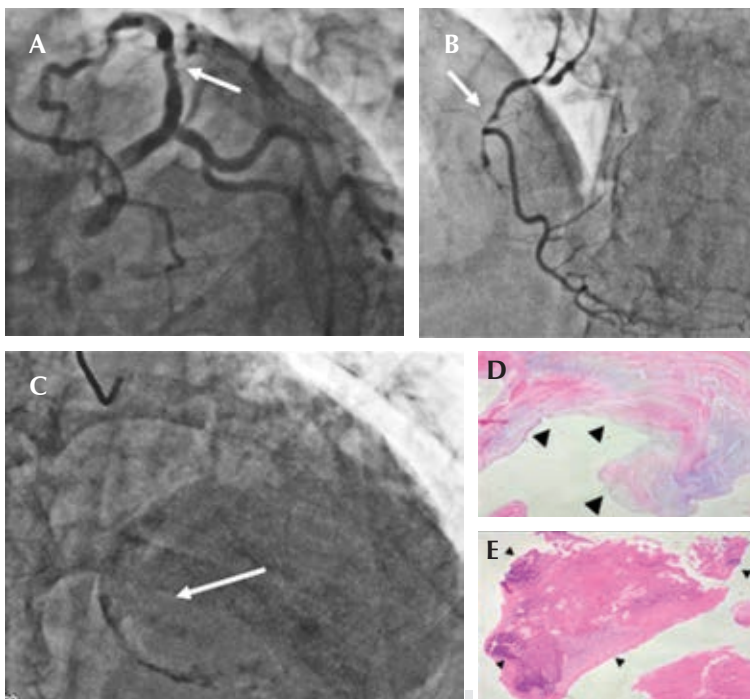


Figure 4: **A, B**) Angiography with coronary lesions in LAD and RCA arteries (arrows). **C**) Coronary angiography showing posterior saccular calcification of the left ventricle (arrow). **D**) Optical microscopy of the mitral valve. Hematoxylin-eosin staining. Myxomatous infiltration and disruption of elastic and collagen fibers are observed. **E**) Anterior tricuspid valve with fibrinoid necrosis and presence of hematoxylin-stained bacterial colonies (purple).

also being useful.^{4,5} Surgical indications are the presence of angina, heart failure, arrhythmias or complications such as rupture, endocarditis or embolic phenomena as in the reported case.^{6,7}

The patient in the reported case has a history of a non revascularized inferior AMI, in addition to not being under pharmacological treatment to reduce the complications of ventricular remodeling, having evidence of the formation of an inferoseptal left ventricular aneurysm.⁸ Said ventricular aneurysm was complicated by rupture of the interventricular septum, despite its late presentation after AMI being infrequent.^{9,10}

CONCLUSIONS

The previous case presents exceptionally as a late complication, in which, thanks to an adequate history, cardiovascular physical examination and imaging studies, a timely diagnosis and treatment was obtained.

REFERENCES

1. Arnaoutakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database. *Ann Thorac Surg.* 2012; 94 (2): 436-443; discussion 443-444.
2. Cabrera-Rego J, Castañeda O, Valiente-Mustelier J, Llerena-Rojas R, López-Ferrero L, Mendoza-Rodríguez V. Presentación tardía de aneurisma ventricular post-infarto. Caracterización por múltiples modalidades de imagen. *Revista Finlay [Internet].* 2011; 1 (3). Disponible en: <http://www.revfinlay.sld.cu/index.php/finlay/article/view/57>
3. Romero TL, Gutiérrez JL, Vasquez TG. Aneurisma ventricular calcificado en un paciente con cardiopatía isquémica y antecedente de trauma cerrado de tórax. *Rev Costarric Cardiol.* 2006; 8 (1): 27-29.
4. Walpot J, Peerenboom P, van Wylick A, Klazen C. Aneurysm of the membranous septum with ventricular septal defect and infective endocarditis. *Eur J Echocardiogr.* 2004; 5 (5): 391-393.
5. Alvarado Sánchez E, Lacayo González H, Siles Varela MA. Ruptura miocárdica y formación de aneurisma después de un infarto agudo de miocardio silente: Reporte de un caso. *Rev Costarric Cardiol.* 2011; 13 (2): 33-36.
6. Egbe AC, Poterucha JT, Rihal CS, Taggart NW, Cetta F, Cabalka AK, et al. Transcatheter closure of postmyocardial infarction, iatrogenic, and postoperative ventricular septal defects: The Mayo Clinic experience. *Catheter Cardiovasc Interv.* 2015; 86 (7): 1264-1270. doi: 10.1002/ccd.25989.

7. Ronco D, Matteucci M, Kowalewski M, De Bonis M, Formica F, Jiritano F, et al. Surgical treatment of postinfarction ventricular septal rupture. *JAMA Netw Open*. 2021; 4 (10): e2128309. doi: 10.1001/jamanetworkopen.2021.28309.
8. Blázquez González JA, Cortina JM, Centeno J, López MJ, Forteza A, Pérez de la Sota E, et al. Corrección quirúrgica de la comunicación interventricular postinfarto. *Cir. Cardio*. 2009; 16 (2): 197-205.
9. Prior-Español A, Mateo L, Martínez-Morillo M, Riveros-Frutos A. Espondilodiscitis sin endocarditis causada por *Streptococcus mitis*. *Reumatol Clin*. 2016; 12 (6): 356-364.
10. Friedman BM, Dunn MI. Postinfarction ventricular aneurysms. *Clin Cardiol*. 1995; 18 (9): 505-511. doi: 10.1002/clc.4960180905.

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