Acute myocardial infarction associated with hypercoagulability due to neoplastic lung disease

To the Editor

Abnormal activity of the coagulation system and endothelial dysfunction produced by neoplastic diseases generate a hypercoagulability state. The most frequent manifestation of these abnormalities is venous thromboembolism while arterial thrombosis is less common. We present the case of a patient with undiagnosed lung cancer who suffered an acute myocardial infarction without significant coronary lesions and a subsequent reinfarction with evidence of coronary thrombosis.

CASE REPORT

A 62-year-old woman,smoker and under study for cervical adenopathies attended the emergency department due to chest pain. The admission electrocardiogram (ECG) showed sinus rhythm with negative T waves from V4 to V6. A troponin I value of 1.67 ng/ml (< 0.1 ng/ml) indicated acute myocardial infarction (AMI) and treatment with nitroglycerin, aspirin, clopidogrel and anticoagulation therapy with low molecular weight heparin (LMWH) was initiated. Coronary angiography performed 12 hours after admission showed no significant coronary lesions.

A transthoracic echocardiogram revealing preserved left ventricular diameters with apical hypokinesia (with apical ballooning) and preserved systolic function was interpreted as a possible Takotsubo syndrome.

On the sixth day the chest pain recurred. The ECG showed lead II, lead III, and V3 to V6 ST-segment elevation and a new angiographic study revealed a thrombotic image with100% lesion in the distal third of the anterior descending coronary artery. The patient underwent thromboaspiration and balloon angioplasty due to the small vessel caliper (Figure 1). Six hours after the procedure, she presented acute neurologic deficit with dysarthria, left facio-brachiocrural hemiparesis and homolateral hemineglect. Brain magnetic resonance imaging showed multiple acute/subacute bilateral frontoparietal and cerebellar ischemic lesions. A transesophageal echocardiography revealed no embolic source and an angiotomography showed left pulmonary thromboembolism, splenic and bilateral renal infarcts, and a tumor at the base of the left lung (Figure 2). Venous Doppler was compatible with bilateral femoral vein thrombosis.

As these findings suggested a prothrombotic condition, a possible neoplastic origin was investigated with a mediastinal ganglion biopsy identified at the tomographic study. Histopathological exam was compatible with lung epidermoid carcinoma. In the following weeks the patient recovered from her neurological deficit without further events,and continued with her anticoagulation therapy with LMWH and ambulatory studies.

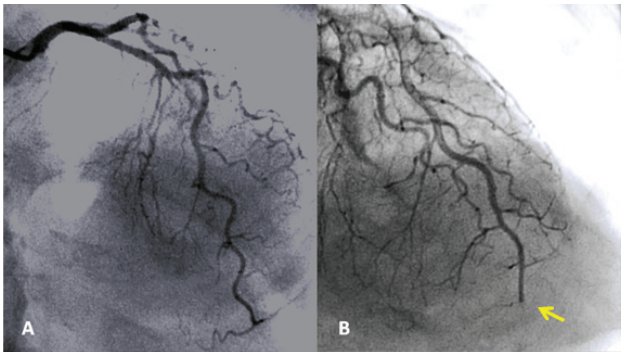

Fig. 1. A. First coronary angiography, without lesions in the anterior descending coronary artery. **B.** Second angiography, showing occlusion in the distal third of the anterior descending coronary artery.

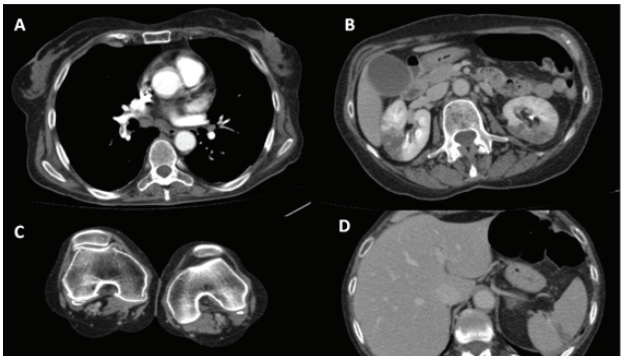

Fig. 2. Chest computed tomography scan with endovenous contrast showing bilateral pulmonary embolism on the right inferior pulmonary branch **(A)**, bilateral splenic infarcts **(B)**, right popliteal thrombosis **(C)** and splenic infarct **(D)**.

DISCUSSION

Our patient has a hypercoagulability condition, with multiple thromboses in arterial and venous territories, probably as consequence of her neoplastic disease. We believe that the two episodes of acute myocardial infarction are explained by this condition, with spontaneous lysis in the first case and not in the second. (1-3) Neoplastic cells promote a hypercoagulability condition by secreting proinflammatory cytokines which activate the coagulation cascade and platelets. Moreover, they favor maturation of monocytes to macrophages and secretevascular endothelial growth factor, stimulating tumor growth and spreading of cancer cells to distant organs. (1-3)

Regarding treatment, numerous studies have demonstrated that LMWH decrease the rate of new thromboembolic events compared to dicumarinic anticoagulants, probably because the latter present many pharmacological interactions and a narrow therapeutic range. (4, 5)

According to anatomopathological studies, myocardial infarction represents the cause of death of up to 6% of cancer patients. As there are no trials on arterial thrombotic events and cancer, information obtained from venous processes is used for its treatment.

Our case reminds us that AMI represents the end of a cascade of prothrombotic events which is not always triggered by atherosclerotic disease.

Dr. Luciano Battioni, Dr. Juan P. Costabel, Dr. Alberto Alves de Lima[MTSAC]
e-mail: juancostabel@yahoo.com.ar
Blanco Encalada 1543 (1428) CABA

REFERENCES

1. Caine GJ, Stonelake PS, Lip GYH, Kehoe ST. The hypercoagulable state of malignancy: pathogenesis and current debate. Neoplasia 2002;4:465-73. http://doi.org/dwwds9

2. Zuffa M, Devecka D, Rusnak I, Ujcova B, Dorociakova E. Myocardial infarction paraneoplastically facilitated? Bratisl lek list 2005;106:324-6.

3. Green K, Silverstein R. Hypercoagulability in cancer. Hematol Oncol Clin North Am 1996;10:499-530. http://doi.org/d3gg24

4. Robert F. The potential benefits of low-molecular-weight heparins in cancer patients. J Hematol Oncol 2010;3:3.

5. Lee AYY, Levine MN, Baker RI, Bowden C, Kakkar AK, Prins M, et al. Low-molecular-weight heparin versus a coumarin for the prevention of recurrent venous thromboembolism in patients with cancer. N Engl J Med 2003;349:146-53. http://doi.org/dnnp7g

Rev Argent Cardiol 2013;81:180-181 - http://dx.doi.org/10.7775/rac.v81.i2.1729

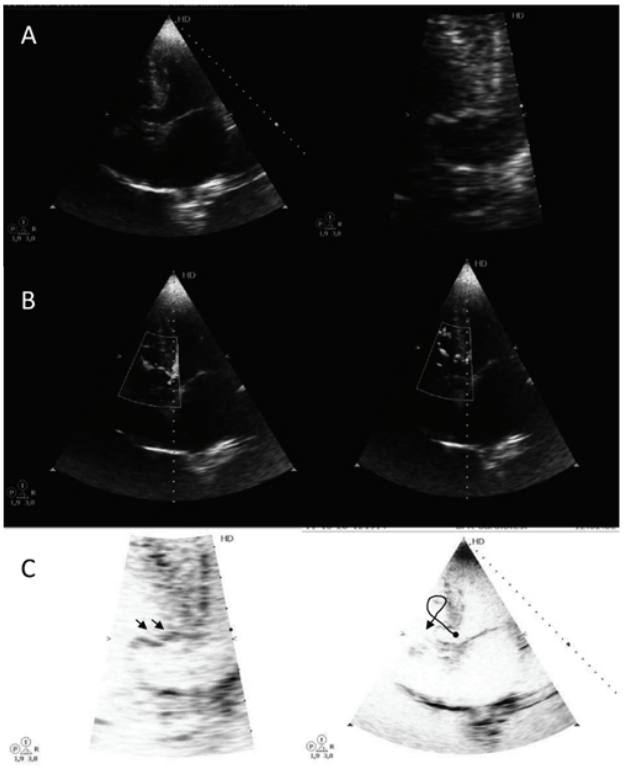

Fig. 1.

Aneurysm of the membranous ventricular septum with ventricular septal defect

To the Editor

We report the case of an aneurysm of the membranous septum in a patient with ventricular septal defect.

CASE REPORT

A 63-year old male patient diagnosed with ventricular septal defect (VSD) since childhood was referred for evaluation. The patient was in functional class I with slight dyspnea on maximum exertion and the physical examination revealed a loud holosystolic murmur with thrill. His medical history showed that he had been hospitalized two years before for suspected (unconfirmed) endocarditis due to prolonged fever, and in the past year for an isolated episode of atrial fibrillation that reversed with amiodarone in the emergency room. The transthoracic echocardiogram revealed an aneurysm of the membranous interventricular septum (AMS) completely prolapsed into the right ventricle in the portion adjacent to the tricuspid septal leaflet (Figure 1A). The AMS had a thin pierced wall with left to right shunt (Figure 1B) with 8 mm vena contracta and a spiral jet impacting on the free wall and returning to the right ventricular inflow tract (Figure 1C).

DISCUSSION

Interventricular AMS is an occasional finding in patients with known subaortic VSD. It probably represents an adaptive phenomenon in uncorrected VSD in infancy. (1) Because it is the weak area of the interventricular septum, the pressure tends to prolapse it to the right with probable complications (2-4) including pulmonary outflow tract obstruction, endocarditis, rupture, arrhythmias as in this case, prolapse and aortic regurgitation. Even though the shunt defect is restrictive, which has been historically considered an indicator of benign evolution, there is increased indication for surgical resolution.

Dr. Guillermo A. Ortíz, Dr. Maria I. Acuña Rodriguez, Dr. Pablo F. Boskis
Dr. Guillermo A. Ortiz
Cardiotest
Paraguay 2302 - 2° Piso (1121) CABA
e-mail: gortiz@intramed.net

REFERENCES

1. Jain AC, Rosenthal R. Aneurysm of the membranous ventricular septum. Br Heart J 1967; 29:60-3. http://doi.org/fhsbn8

2. Langer C, D Horstkotte, Piper C. Aneurysm of the membranous septum causes pre-syncopes and transient bilateral blindness. Eur Heart J 2007, 28:784. http://doi.org/bv2m65

3. 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, 5:391-3. http://doi.org/fr3bc9

April. Yilmaz AT, Ozal E, Arslan M, Tatar H, Oztürk OY. Aneurysm of the membranous septum in adult patients with ventricular septal defect perimembranosus. Eur J Cardiothorac Surg 1997, 11:307-11. http://doi.org/chswqd

Rev Argent Cardiol 2013;81:181 - http://dx.doi.org/10.7775/rac.v81.i2.1142

Late Bioprosthetic Mitral Structural Failure

To the Editor

We present the case of a female patient submitted to mitral valve replacement with a biological prosthesis who 7 years later exhibited prosthetic failure.

CASE REPORT

An 81-year old woman was admitted to the hospital due to dyspnea and signs of acute congestive heart failure. Seven years earlier she had undergone mitral valve replacement with a biological prosthesis (HPV 29 mm – FOC Medical SA) for rheumatic disease and concomitant Cox-Maze procedure. Previous to this condition, the patient had been asymptomatic with stable anticoagulation range and uneventful periodic echocardiographic evaluations. A transesophageal echocardiography revealed severe mitral regurgitation murmur and moderate ventricular dysfunction, with left atrial dilation and severe mitral regurgitation. The bioprosthesis had coaptation deficit of one of its leaflets with evidence of eversion and flail and clearly increased asymmetric motility (Figure 1; see also Video 1 in the on line version). The patient evolved with cardiogenic shock secondary to acute valve rupture. Following mechanical intubation, inotropic treatment and ventricular support with intra-aortic balloon pump counterpulsation, she was submitted to surgical intervention. Bioprosthesis exposure, resection and inspection showed relatively soft leaflets and tear in one of the veils that was not calcified (Figure 2). Despite host tissue proliferation, veil movement was not restricted. A 29 Hancock II mitral bioprosthesis was implanted in the same position and a semicircular tricuspid annuloplasty was performed (modified De Vega). Regardless this intervention, the patient died in the immediate postoperative period.

DISCUSSION

Both leaflet commissure and base are the most damaged sites due to the tension they bear. The processes of glutaraldehyde fixation, cell death and collagen/elastin matrix degeneration to which bioprosthesis are submitted during manufacturesuggest they might be intrinsic factors favoring later deterioration and calcification. Support dehiscences which were identified in valve substitutes during the eighties are no longer seen (1) and valve tears are less frequent. (2, 3) In porcine substitutes, the association with tears is better documented. (4) Differences in failure patterns and clinico-pathological presentation allow for a great variety of clinical presentations. Commissure dehiscences from the support or stent have also been reported in various porcine valve models. Depending on the different bioprosthesis used, between 45% and 84% of mitral valve implantations at 10 years have revealed prosthetic structural failure and higher reoperation rates at 15-year follow-up (29% for AVR, 50% for MVR). (5, 6) Multicentric studies refer actuarial and actual freedom from explant due to structural valve deterioration at 14 years of 68.8% and 83.4%, respectively. (2). No evidence of explants due to structural deterioration at 10 years was reported. (6)

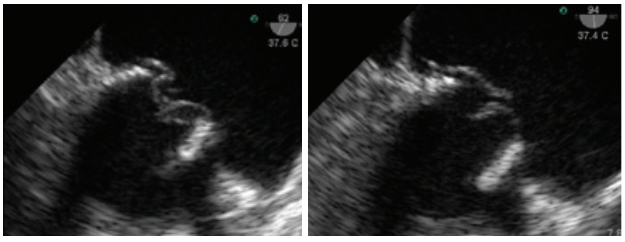

Fig. 1. Transesophageal echo showing bioprosthesis coaptation deficit with eversion and flail of one leaflet over the opposing one, and prolapse of a deformed leaflet (90° view).

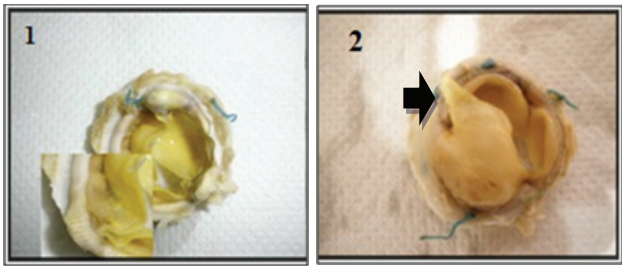

Fig. 2. TExplanted HPV prosthesis. 1. Left ventricular view. 2. Left atrial view showing the characteristic leaflet tear and rupture with detachment from the commissural fixation. No major leaflet degenerative alterations, thickening or calcifications were detected.

In conclusion, degenerative and structural failure or deterioration is an exclusive phenomenon that should have careful and thorough clinical and echocardiographic follow-up to establish changes that might give rise to a possible future reoperation. Although the time of presentation varies, the probability of reoperation still persists. Future anti-calcification or substrate modifying component treatments to ensure delay of biological deterioration could score the differences and future strategies in the choice of bioprosthesis.

Dr. Javier E. Ferrari Ayarragaray,
Dr. Víctor Darú[MTSAC], Dr. Matías Failo

REFERENCES

1. Jamieson WRE, Ling H, Burr LH, et al. Carpentier-Edwards supra-annular porcine bioprosthesis: evaluation over 15 years. Ann Thorac Surg 1998;66:S49-52. http://doi.org/dbbd5q

2. Marchand MA, Aupart MR, Norton R, et al. Fifteen-year experience with the mitral Carpentier-Edwards PERIMOUNT pericardial bioprosthesis. Ann Thorac Surg 2001;71:S236-9. http://doi.org/ccssz8

3. Ishihara T, Ferrans VJ, Boyce SW, et al. Structure and classification of cuspal tears and perforations in porcine bioprosthetic cardiac valves implanted in patients. Am J Cardiol 1981;48:665-77. http://doi.org/bgn5tf

4. Butany J, Feng T, Luk A, Law K, Suri R, Nair V. Modes of failure in explanted mitroflow pericardial valves. Ann Thorac Surg 2011;92:1621-7. http://doi.org/cgccg9

5. Yamak B, Kener E, Kiziltepe U, Mavitas B, Tasdemir O, Beyazit K. Late results of mitral valve replacement with Carpentier-Edwards high profile bioprosthesis in young adults. Eur J Cardiothorac Surg 1995;9:335-41. http://doi.org/c9tf5j

6. Riess FC, Bader R, Cramer E, Hansen L, Kleijnen B, Wahl G, et al. Hemodynamic performance of the Medtronic Mosaic porcine bioprosthesis up to ten years.Ann Thorac Surg 2007;83(4):1310-8. http://doi.org/djg75c

Rev Argent Cardiol 2013;81:182-183 - http://dx.doi.org/10.7775/rac.v81.i2.1579