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Chronic Constrictive Pericarditis after Cardiac Surgery

Postoperative constrictive pericarditis (PCP) after cardiac surgery is a unique entity with distinctive characteristics from other etiologies of constrictive pericarditis. Since its first description in 1972, several isolated cases but few series have been published, as its real incidence is probably underestimated due to its nonspecific and subtle presentation, requiring high clinical suspicion. (1)

We report the case of a 61-year-old male patient with cardiovascular risk factors: overweight, ex-smoker, and with dyslipidemia, who developed PCP after cardiac surgery. He had undergone radiation therapy for Hodgkin’s lymphoma at the age of 23, a pacemaker implanted due to third-degree AV block in 2008, and mechanical aortic valve replacement for severe symptomatic aortic stenosis with functional class (FC) I-II dyspnea in 2013.

Progressive symptoms, FC III-IV dyspnea, asthenia, and hyporexia persisted for months after the cardiac surgery, and he was repeatedly hospitalized for decompensated heart failure, paracentesis for ascites, liver congestion, weight loss, jugular ingurgitation, and Kussmaul’s sign. The study of the disease etiology revealed hemolytic anemia leading to periprosthetic leak suspicion, which was confirmed and treated with 4-mm Amplatzer vascular plug implantation. Pacemaker-mediated tachycardia with AF-based rhythm of rapid ventricular response occurred simultaneously. Prosthetic aortic valve dysfunction was also ruled out.

After excluding the distractors mentioned above as the cause of the patient’s dyspnea and heart failure, and since his symptoms persisted, a multislice computed tomography was performed for suspected chronic constrictive pericarditis (CCP), revealing 12-mm pericardial thickening in the anterolateral wall (Figure 1). A second Doppler echocardiography showed abnormal respiratory variability in the left ventricular outflow tract, in the pulmonary vein diastolic velocity, and in the tricuspid flow. The right chambers were mildly enlarged with interatrial and interventricular septum displaced toward the left chambers. Pericardial thickening by homogeneous, non-calcified 8-mm thick material surrounding the ventricle caused constrictive physiology with marked signs of systemic congestion. The patient evidenced preserved systolic function and grade-III diastolic dysfunction.

A pericardiectomy was performed once PCP was confirmed. Pathological examination of the pericardium showed a chronic inflammatory process with fibrosis, calcification, and foreign body giant cell reaction (Figure 2). A few months after the pericardiectomy, the patient showed gradual and progressive improvement. At present, he is stable with functional class II dyspnea, reduced edema of the lower limbs and ascites, weight gain (due to increase in muscle mass), and improved general condition. He continues only with medical treatment for congestive heart failure.

Constrictive pericarditis is the final phase of a pericardial inflammatory process. The most common etiology is idiopathic (48%), followed in order of incidence by post-cardiac surgery, radiotherapy, and tuberculosis in developing countries.

Postoperative constrictive pericarditis is a rare complication, with an estimated incidence of 0.025-0.3%. (2) The timing of presentation can be extremely variable, ranging from 1 to 240 months after cardiac surgery. For that reason, PCP should be highly suspected during postoperative and long-term follow-up. The profile characterizing patients with PCP are old age, high prevalence of male patients, history of myocardial infarction, and diabetes. Other factors involved in PCP development include previous chest irradiation, postoperative wound infection, and osteomyelitis. (3)

Its pathophysiology remains unclear and is probably multifactorial. Most develop with the open pericardium at the end of surgery. The accumulation of blood is the initial stimulus to develop adhesions. An increased inflammatory response occurs with up-regulation of cytokines (II-6, II-1 and TNF-α) and increased oxidative stress, causing a postpericardiotomy syndrome that can progress to effusive pericarditis. In this process, the increase of growth factors leads to fi-
brosis and constriction. Another associated factor is a certain degree of pericardial bacterial contamination that would exacerbate the inflammatory process. The incomplete resolution of this condition is the first step to chronicity. The different inflammatory responses among patients (extension of fibrosis, inflammation and neovascularization) explain the variability of the time interval between surgery and diagnosis of PCP.

Although clinical signs, echocardiography, and CT scan can reinforce the suspicion of PCP, the diagnostic gold-standard remains the direct measurement of diastolic equalization of all intracavitary pressures and the “dip and plateau” pattern in the diastolic tracings. Cardiac magnetic resonance represents the most sensitive technique for diagnosing pericardial thickening or calcification. However, cases of PCP without pericardial thickening or pericardial thickening without pericardial constrictive physiology have been reported, resulting in incorrect diagnosis of this entity. (4)

Pericardiectomy is the only treatment for permanent constriction. The indications are based on symptoms, echocardiographic findings, magnetic resonance imaging/computed tomography, and cardiac catheterization. Pericardiectomy is a high-risk procedure, with a mortality rate of 5-21%. It may be of little benefit for patients who are not subjected to extensive pericardiectomy or for those with radiation-induced CCP. (5)

Recovery of the functional capacity (clinical improvement) may take weeks or months, and it is associated with persistence of altered ventricular filling in the echocardiography. Up to one third of the patients may show no improvement or continue with signs of heart failure. Chronic – and sometimes irreversible – changes like fibrosis and atrophy of the adjacent myocardium are often significant prognostic markers after pericardiectomy. Low ejection fraction, high diastolic pressure, kidney failure, moderate to severe tricuspid regurgitation, and old age are also predictors for increased mortality and poor long-term outcome. (6)

Postoperative constrictive pericarditis has a peculiar and distinctive pathophysiology, which in part differs from the other causes of constrictive pericarditis. Although there is still no conclusive explanation for its occurrence, PCP should be suspected especially when other causes of dyspnea and heart failure are ruled out during patient follow-up after cardiac surgery.

Conflicts of interest
None declared.
(See authors’ conflicts of interest forms on the website/Supplementary material).

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REFERENCES

Spontaneous Coronary Artery Dissection: A Rare Cause of Cardiogenic Shock in Young Women

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS). It usually occurs in young women, sometimes with ST-segment elevation, and is associated with fibromuscular dysplasia (FMD). (1-4) Spontaneous coronary artery dissection may manifest as dissection with intimal rupture or as intramural hematoma. In general, SCAD resolves spontaneously with good long-term prognosis (1, 2, 4, 5), but sometimes its outcome is more complex, as in the cases presented below.

Case 1: A 46-year-old female patient, without risk factors, presented at emergency complaining of 3-hour duration angina associated with vaginal symptoms. ECG showed no ischemic changes, ultrasonic troponin was 281 ng/L (NV <14 ng/L) and echocardiography revealed anterolateral and mid and apical hypokinesia. Coronary angiography (CAG) revealed severe proximal lesion at the inferior subdivision of the first diagonal (Dg) branch, and medical treatment was decided. Thirty minutes after CAG,