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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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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 vagal symptoms. ECG showed no ischemic changes, ultrasensitive 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,
the patient developed polymorphic VT (PVT) and underwent cardiopulmonary arrest (CPA). ECG showed 3-mm ST-segment elevation in V2-V4. A second CAG disclosed progression of previous lesion. The attempt to perform percutaneous coronary intervention (PCI) was unsuccessful. Twenty minutes later, the patient repeated angina, PVT, and CPA. The third CAG evidenced severe vasospasm in the left main coronary artery (LMCA), proximal LDA occlusion and moderate ostial and proximal Cx lesion that seemed severe due to vasospasm. Intra-aortic balloon pump (IABP), mechanical respiratory assistance (MRA) and inotropes were used to assist the patient. Intracoronary nitroglycerin infusion improved LMCA and Cx vasospasm. Then, LAD PCI was performed.

Given the persistence of hemodynamic instability, an emergency coronary surgery (LIMA-LAD and VB-Cx) was performed. Cardiogenic shock persisted during the postoperative period, so mechanical ventricular assistance (MVA) with A-V ECMO was started as bridge-to-transplant. Cardiac transplantation was performed 48 hours later. The patient experienced no new cardiovascular events during the 8-month outpatient follow-up. CT angiography showed no evidence of the disease in other vascular beds.

Discussion: Spontaneous coronary artery dissection has a multifactorial etiology, and is associated with connective tissue disorders, systemic inflammatory conditions, peripartum period, and FMD. (2, 4, 5).

Angiography remains an essential diagnostic test, and for patients with intramural hematoma (1) (Figure 1) more sensitive tests such as intravascular ultrasound (IVUS) or optical coherence tomography are recommended.

Case 2: A 30-year-old woman, who presented with migraine headaches and consumed ergotamine three times a week, consulted for 10-minute duration angina at rest. The ECG revealed no ischemic changes and ultrasensitive troponin was 186 ng/L. The echocardiographic study showed apical hypokinesia. Within a few minutes, the patient experienced angina and ST-segment elevation in V2-V4. Coronary angiography revealed spontaneous coronary artery dissection in the proximal and mid third of the LAD, affecting the entire length of the artery; therefore, PCI with stent was performed. Angina, lateral ST-segment elevation and cardiogenic shock were repeated within two hours. Coronary angiography showed dissection of the distal LMCA and mid third Cx; PCI with stent to both arteries was performed using IABP mechanical ventilation and inotropes, with a positive reaction. The patient experienced no new cardiovascular events during the 8-month outpatient follow-up. CT angiography showed no evidence of the disease in other vascular beds.

Histology of the coronary arteries with hematoxylin and eosin stain. A. Right coronary artery with beading appearance. B. Anterior descending coronary artery with predominant intimal fibrotylosis. C. Anterior descending artery with predominant fibromuscular dysplasia. D. Part of the muscular coronary artery dissection is observed with Masson’s trichrome stain.
(OCT) (1, 4) are required to differentiate it from an atherosclerotic lesion. Due to its strong association with FMD, its presence should be routinely searched in case of SCAD, preferably by invasive angiography, as it is more sensitive than computed tomography angiography. (6)

No consensus has been reached on the treatment of SCAD. Its management depends on the hemodynamic state of the patient, his symptoms and the degree of dissection. A conservative approach is suggested in asymptomatic patients, based on observational data demonstrating that in most cases SCAD has a tendency to spontaneous cure and because revascularization is associated with high failure rates. It includes medications used for the treatment of ACS; their role in SCAD is unknown and discussed due to lack of randomized studies for this pathology. (1-3, 6) Aspirin and beta-blockers are the most widely accepted agents, while anticoagulants, angiotensin-converting enzyme inhibitors and statins are more controversial. Revascularization is recommended if the patient is hemodynamically unstable, if the dissection generates ischemia, ventricular arrhythmia or involves the LMCA. Thrombolitics should be avoided as they may worsen the dissection. Percutaneous coronary intervention should be performed in cases with adequate anatomy; otherwise, coronary artery bypass grafting (CABG) should be considered. Moreover, CABG is decided for cases with LMCA dissection, extensive dissections with proximal involvement of main arteries and failed PCI (1-3, 6) Long-term survival is above 90% and recurrence rate is variable (10-50%), depending on the follow-up period. (1, 3, 5, 6)

Conflicts of interest
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Cardiac Apical Pseudoaneurysm after Transcatheter Aortic Valve Replacement (TAVR)

Case description: This is the case of an 81-year-old female patient with progressive dyspnea even on minimal exertion during the past year. Clinical history: dyslipidemia, hypertension, pneumonia, and father’s death due to coronary artery disease. Physical examination revealed systolic hypertension (148/78 mm Hg), mild tachypnea (23/min), grade III/IV protosystolic murmur in the aortic area, with decreased vesicular murmur at the bases. The echocardiography revealed left ventricular ejection fraction (LVEF) of 60%, mild concentric hypertrophy (106 g/m2), mild aortic valve sclerosis with significant involvement in the opening of all its cusps, valve area of 0.48 cm², maximum velocity 4.25 m/sec, pressure gradient 72.3 mm Hg, moderate left atrial enlargement (48 ml/m2), enlarged right atrium (31 ml/m2), grade II diastolic dysfunction, grade B mitral regurgitation, and mild pulmonary hypertension (47 mm Hg). Coronary angiography showed no significant obstructions.

Under diagnosis of severe aortic stenosis, percutaneous iliofemoral transcatheter aortic valve replacement (TAVR) was performed and a 26-mm CoreValve was implanted. The patient continued with outpatient follow-up after a 5-day hospital stay. Seven months

Fig. 1. Two-dimensional echocardiography showing minimal mass effect on the right ventricular tip. RA: Right atrium. LA: Left atrium. RV: Right ventricle. LV: Left ventricle. PSA: Pseudoaneurysm.