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-

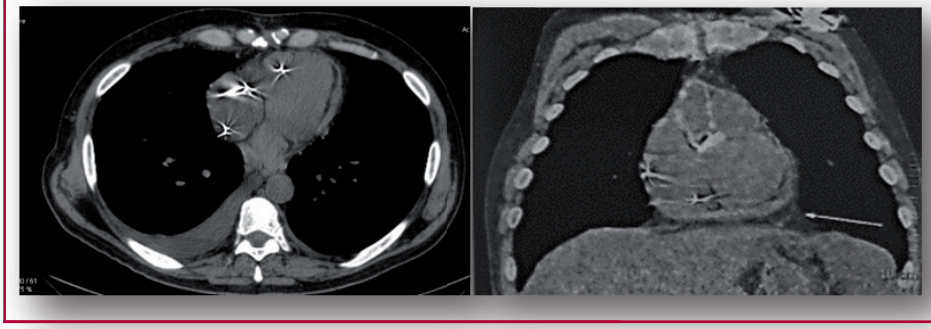

Fig. 1. Multislice computed tomography of the chest showing increased pericardial thickening.

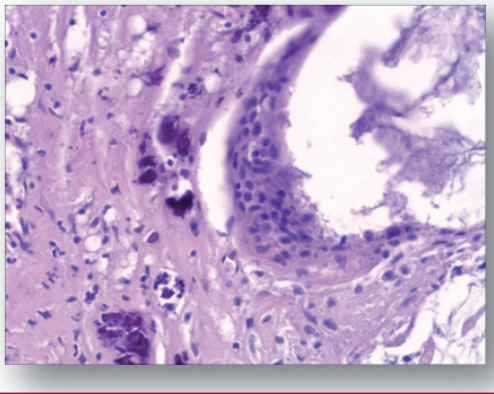

Fig. 2. Pathological anatomy of the pericardium with its inflammatory process.

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.

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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 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,

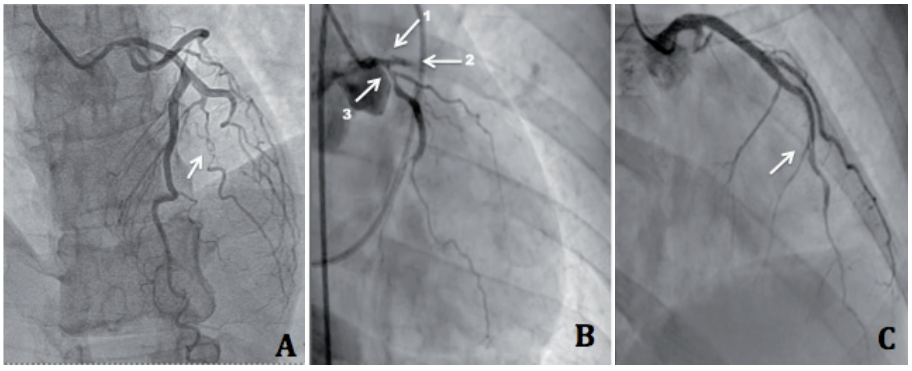

Fig. 1. A and **B**. Case 1 LMCA, LAD, and Cx angiography. **A.** First cardiac catheterization shows inferior subdivision of the Dg branch with severe proximal lesion (arrow). Arterial vessels exhibit tortuosity and distal smooth narrowing, angiographic characteristics of FMD. **B.** Third cardiac catheterization: severe vasospasm in the LMCA (arrow 1), proximal LAD occlusion (arrow 2), and moderate ostial and proximal Cx lesion that seems severe due to vasospasm (arrow 3). **C.** Case 2 LAD angiography. The dissection line is observed in the middle third of the artery, with angiographically normal proximal artery.

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. Cardiac pathological anatomy revealed fibromuscular dysplasia (FMD) of the coronary arteries with LAD and Cx dissection, ischemic scarring, and infarction at different evolutionary periods.

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.

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

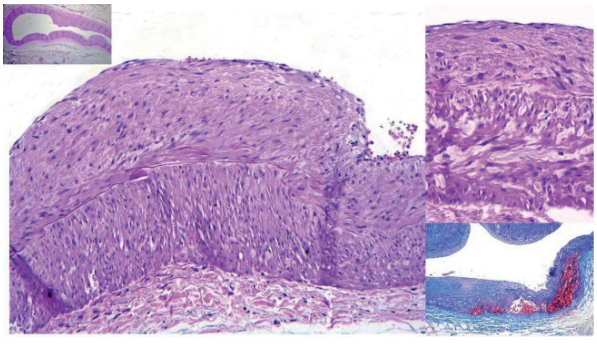

Fig. 2. Histology of the coronary arteries with hematoxylin and eosin stain. **A.** Right coronary artery with beading appearance. **B.** Anterior descending coronary artery with predominant intimal fibrodysplasia. **C.** Anterior descending artery with predominant fibromuscular dysplasia. **D.** Part 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. Thrombolytics 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

None declared.

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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 ileofemoral 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

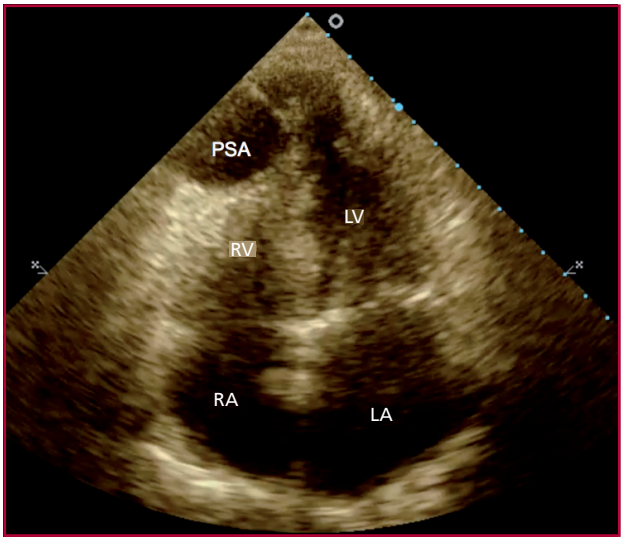

Fig. 1. Two-dimensional echocardiography showing minimal mass effect on the right ventricular tip. RA: Right atrium. LA: Left atrium. RV: Right ventricle. LV: Left ventricle. PSA: Pseudoaneurysm.

later, the patient presented with dyspnea. Chest x-ray showed several consolidations of the lung parenchyma. Antibiotic therapy was initiated due to diagnostic impression of pneumonia. Control echocardiography revealed an image suggestive of pseudoaneurysm at the cardiac apex. Contrast MRI reported a 70 x 40 x 53 mm multiseptated cystic lesion with lobulated contour (Figures 1 to 3). Ventricular pseudocyst was the final diagnosis and an expectant approach was decided.

Discussion: Transcatheter aortic valve replacement is a valuable option for patients who cannot undergo open procedures due to their comorbidities. The approach is usually via ileofemoral vessels, but the transapical access is a valid option for patients who also have tortuous vessels. In most medical centers, the apical access is the accepted protocol when the ileofemoral access is inadequate. (1)

Apical pseudoaneurysm is a TAVR complication of the transapical approach. (2) It is believed that the conditioning factors affecting this approach are old age, multiple comorbidities, fragile myocardium, bleeding, myocardial infarction, and localized tissue weakness. Other sequelae of the transapical access include bleeding with and without tamponade, stroke, mitral and aortic regurgitation and septal hematoma. (1)

The reported cases of apical pseudoaneurysm secondary to transapical TAVR have been scarce. In parallel, there is no report in the literature on cardiac apical pseudoaneurysm secondary to an ileofemoral approach. Therefore, this would be the first case reported. Schamroth (3) suggests that aortic aneurysms are caused by a linear tear that occurs in the ascending aorta at the time of implantation and then, over time, develops into an aneurysm. In our case, the reason would be an analogous ventricular apical tear due to one of the guidewires used during the procedure.

Some authors consider surgical repair necessary; (2, 4, 5) however, the reason why patients undergo TAVR instead of open surgery is precisely their high operative-risk with multiple comorbidities, increasing significantly the risk of surgical repair. (5) In this scenario, percutaneous closure should be taken into account as a possible treatment of choice.

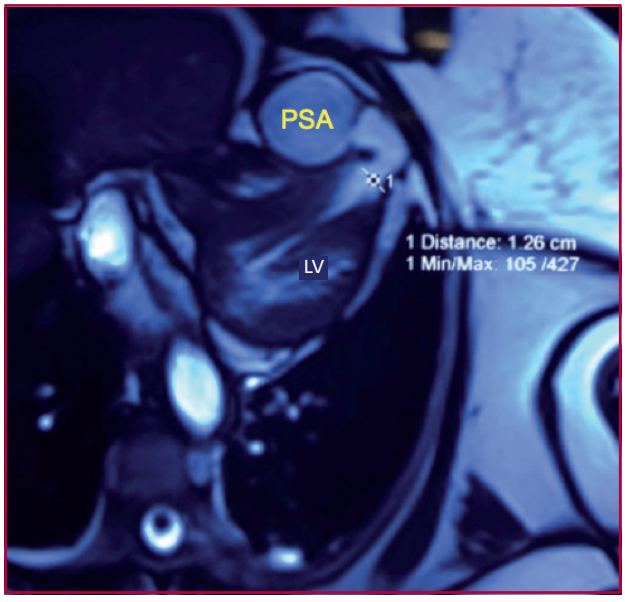

Fig. 2. Magnetic resonance imaging axial plane. LV: Left ventricle. PSA: Pseudoaneurysm.

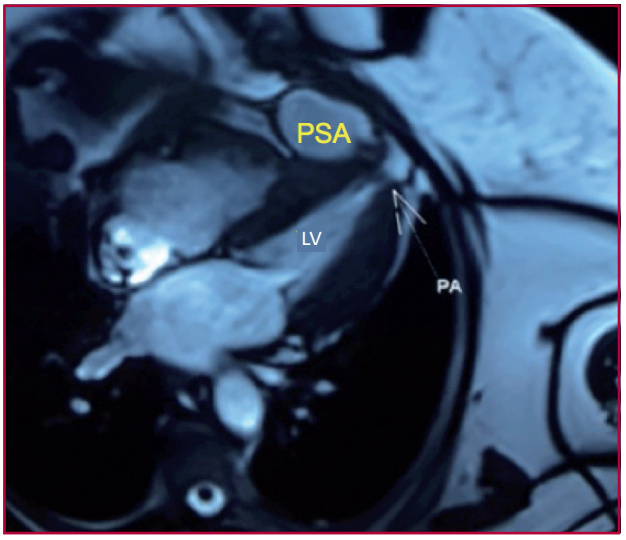

Fig. 3. Magnetic resonance imaging axial plane emphasizing the neck of the defect. LV: Left ventricle. PSA: Pseudoaneurysm.

Conflicts of interest

None declared.

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Influenza and Pneumococcal Vaccination in Patients with Cardiovascular Disease: Pilot Project

Infections caused by the influenza virus and by Streptococcus pneumoniae are associated with high risk of morbidity and mortality in patients with cardiovascular diseases. International and local government agencies recommend the use of vaccines; however, there is underutilization in our setting. (1) Several barriers are related to this issue, such as physician's agreement, patients' myths and beliefs, as well as economic limitations, or provision by the medical coverage system. We postulate that consultation with a specialist or hospitalization in the coronary care unit (CCU) is an important opportunity to improve adherence to vaccine prescription. The purpose of this study was to evaluate patient immunization rate after systematic prescription of influenza and pneumococcal vaccines in the CCU or at the doctor's office, and to describe the possible barriers to vaccine implementation.

We carried out a prospective study in a medical center in Buenos Aires, for 30 consecutive days during the epidemic season (May 15 - June 15). Once the informed consent form was signed, identification data, information about previous vaccines and reason for immunization were recorded. Within 1 to 2 months after immunization, telephone calls were made to collect data about effective vaccination, type of vaccines, adverse events, and reasons for non-vaccination (Figure 1). A conventional statistical analysis was carried out, comparing the immunization rate at discharge from CCU versus doctor's office, using the chi-square test. Based on 2012 data from the National Ministry of Health, in the City of Buenos Aires, influenza vaccination rate was 57% for patients between 2-64 years of age with risk factors. (1) Assuming an accepted error of 20%, a level of confidence of 95%, and 10% losses, about 80 patients should be recruited, and therefore a month for recruitment was estimated. The EpiInfo 3.5.3 software was used, and a value of $p < 0.05$ was considered significant.

A total of 80 patients were included (68% were male) and median age was 65 years (IQR 59-72 years); 19% of the patients belonged to prepaid medical care and the rest to medical insurance plans. Vaccination was indicated at discharge from CCU or the cardiology ward in 25 patients (31%), and at the outpatient clinic for the rest of the patients. The most common indications were acute coronary syndrome (59%), age >65 years (50%), active smoker (16%), diabetes (12%),

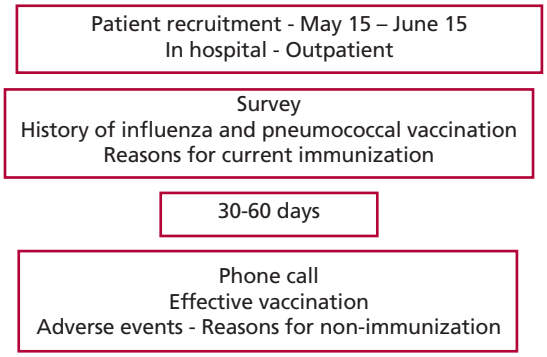

Fig. 1. Study flow diagram.

and heart failure (10%), several indications overlapping in some cases. Thirty-eight percent of patients received annual influenza vaccination. Influenza vaccine was indicated to 99% of the patients, but at 30 days, only 73% were effectively immunized. Patients referred that the main reason for non-immunization was 'a personal decision'. Three patients presented with influenza-like syndrome following vaccination. Regarding pneumococcal vaccination, 15 patients (12%) had been previously immunized. When the phone calls were made, it was found out that only 46% (32 patients) had received the vaccine. The three reasons why vaccination had not been carried out were personal decision (59%), lack of provision by the medical coverage (24%), and high cost of vaccines (18%). No adverse events after vaccination were registered. Vaccine indication from the CCU was associated with a trend to higher vaccination rate (52% vs. 42%), although the difference was not significant due to low power of the sample ($p=0.2$).

In Argentina, the main cause of death is cardiovascular disease, (2) which presents a seasonal pattern with cardiovascular mortality peaks in the winter months. An English study has demonstrated a significant correlation between seasonal outpatient consultations for influenza syndrome and mortality due to acute myocardial infarction, (3) evidencing correlation between infection, systemic inflammation and plaque accident. Today, there are ecological, case-control, and randomized studies demonstrating the effectiveness of influenza vaccination in reducing mortality and hospitalizations not only for pneumonia but also for cardiovascular reasons. (4) This association is also observed with pneumococcal vaccination. A recent randomized trial in Holland, involving more than 80,000 adults over 65 years of age, (5) demonstrated that the use of a more modern pneumococcal conjugate vaccine presented 75% efficacy in preventing the first episode of invasive pneumococcal disease. In a prospective cohort study, the effect of dual vaccination (pneumococcal and influenza vaccines) in more than 36,000 adults over 65 years of age was associated with a significant reduction of pneumonia, stroke, myocardial infarction, heart failure, hospitalizations, and

overall mortality. (6) Among the barriers described for immunization, the medical knowledge barrier was overcome in this study, since vaccines were systematically indicated. We have observed that the main reason for non-immunization was personal decision. We believe this is mainly due to myths and popular beliefs about the lack of effectiveness and the risk of infections caused by vaccines, but it may also be due to lack of adherence to the physicians' indications, as is the case with other pharmacological treatments. In this regard, large-scale population studies suggest that patient adherence to therapeutic strategies in high-mortality diseases –such as acute coronary syndrome– is almost total at discharge from CCU but dramatically falls during follow-up, both in cities and rural areas of industralized and developing countries. The trend to a higher vaccination rate at discharge from CCU compared to the doctor's office is a potential opportunity for intervention to improve adherence, which should be demonstrated in larger-scale studies.

Conflicts of interest

None declared.

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