Acute Coronary Syndrome in Essential Thrombocythemia: Usefulness of Optical Coherence Tomography

Essential thrombocythemia is a rare myeloproliferative neoplasm, characterized by platelet proliferation with quantitative and qualitative alterations. Patients suffering from this condition are more likely to have thrombosis and hemorrhages. It has an incidence of 1 to 2.5 new cases per 100,000 inhabitants per year, (1, 2) and its frequency increases with age, with a 2:1 ratio in favor of women. (3)

One of the complications of essential thrombocythemia is coronary thrombosis, which can be potentially fatal. The incidence of acute coronary events with this hematology-oncology disease is 9.4%, with a rate of fatal and non-fatal thrombotic events of 1.9 per 100 patients/year. (4)

We report the case of a 35-year old female patient with thrombocythemia under study and no cardiovascular history, who presented with moderate, oppressive chest pain lasting 20 minutes, in functional class IV.

Physical examination showed the patient was hemodynamically stable, with blood pressure of 110/60, heart rate of 60 bpm, and no signs of heart failure.

The electrocardiogram revealed sinus rhythm without conduction disorders, narrow-QRS with 0.5 mm transient ST segment elevation (lasting < 20 minutes) from V1 to V3.

Lab tests reported hematocrit 37%, hemoglobin 13 mg/dl, white blood cells 5500/mm^3, and platelets 1,200,000/mm^3. Ultrasensitive troponin was requested, with negative value: 5 pg/ml (normal <14 pg/ml). JAK2 was negative, Leyden factor V was negative, lupus anticoagulants were negative, and bone marrow puncture revealed megakaryocytic hyperplasia consistent with chronic myeloproliferative neoplasm.

Coronary CT angiography showed lack of filling in proximal anterior descending artery causing 80% luminal obstruction, possibly indicative of thrombus or soft plaque (Figure 1A). The patient was started on anticoagulation with unfractionated heparin and aspirin 100 mg/day.

The patient coursed asymptomatic for angina, and a coronary angiography was performed at 72 hours of admission, which showed no significant obstructions (Figure 1B); the study was completed with an optical coherence tomography (OCT) that revealed atherosclerotic fibrolipid plaques in the anterior descending artery at the proximal and middle-third levels (Figure 2A), and an image consistent with plaque rupture (fissure) at the proximal third level in the origin site of the first diagonal branch (Figure 2B).

Due to findings in the catheterization study, atorvastatin 80 mg/day was added to the previous treatment.

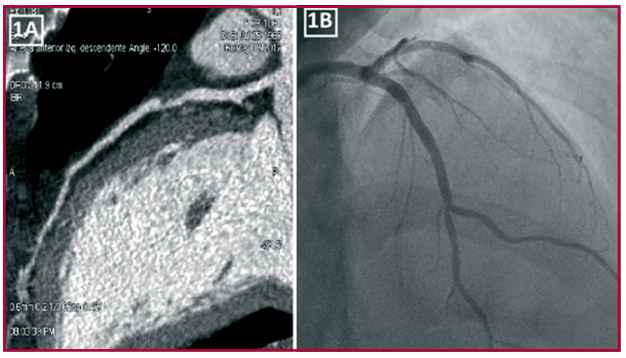

Fig. 1. A. Multislice CT-scan image showing 80% obstruction in the proximal third of the anterior descending artery. **B.** Coronary angiography image showing anterior descending artery with no significant lesions.

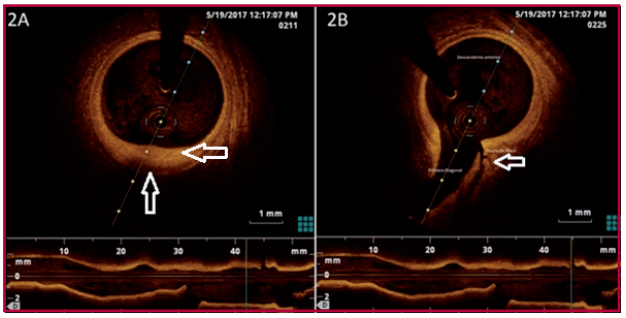

Fig. 2. A. Optical coherence tomography image showing at 6 clock the presence of fibrolipid plaque in the anterior descending artery. The lipid core is observed as an area of low refringence (*arrow*), and the fibrous capsule covering it as a more refringent layer in contact with the arterial lumen (*arrow*). **B.** Optical coherence tomography image showing plaque rupture at the origin of the first diagonal artery. Notice the fissure of the fibrous capsule causing the lipid core to come into direct contact with the arterial lumen.

Echocardiography showed no motility disorders. Left ventricular systolic function was normal, with no relevant valve diseases.

The patient made good progress with no further chest pain, platelet reduction (450,000/mm^3 on discharge), and no changes suggestive of necrosis in the electrocardiogram. The patient was discharged 7 days after admission, continuing with aspirin 100 mg/day, anticoagulation with enoxaparin 80 mg/12 hours, atorvastatin 80 mg/day, and hydroxyurea 2000 mg/day.

Essential thrombocythemia is a condition that may present as an acute coronary syndrome, due to thrombotic events that affect the epicardial coronary arteries. It is a life-threatening complication. (5)

No cases have been described in the literature in which the pathophysiological mechanism of the acute coronary syndrome (in young patients with low risk

coronary pretest, and essential thrombocythemia) is the combination of plaque rupture and superimposed thrombosis.

Our patient was young and had no risk factors for coronary disease; however, the OCT revealed mild atherosclerosis and plaque rupture, accounting for the mechanism of the coronary event. Furthermore, this finding allowed treatment optimization at discharge, since it supported the continuation of high-dose statin therapy together with anticoagulation therapy.

Optical coherence tomography is a high-resolution intravascular diagnostic technique. Initially, it was developed for identifying plaque instability, but nowadays it is also used to identify periprocedural complications, correct stent implantation and, as in our case, plaque rupture detection. (6)

This case is reported to consider the possibility of using OCT in this type of patients, not only to get information about the mechanism of thrombosis but also to guide treatment on discharge.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/Supplementary material).

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Controversial Management of Severe Thrombocytopenia Induced by Abciximab

The three glycoprotein-IIb/IIIa inhibitors currently in clinical use, abciximab, eptifibatide and tirofiban, all share the same therapeutic target, namely blockade of the final common pathway of platelet aggregation and management of acute coronary syndromes. (1) Thrombocytopenia associated with glycoprotein-IIb/IIIa inhibitors occurs in about 1-2% of the patients exposed to this type of drugs. (2)

We report the case of a 50-year old, hypertensive, obese female patient, with type 2 diabetes. She was admitted with non ST-segment elevation acute coronary syndrome and maximum TnI of 0.55 ng/ml. Dual antiplatelet therapy was initiated with aspirin and clopidogrel, and anticoagulation with subcutaneous enoxaparin 1 mg/kg every 12 hours; a coronary angiography was performed. A drug-eluting stent was implanted in the mid-right coronary artery (RCA) with transient ST-segment elevation by microembilization of the acute marginal branch of the RCA. Intracoronary abciximab bolus application resulted in subsequent recanalization of the branch. Another drug-eluting stent was implanted in the most caudal branch of the obtuse marginal artery. Petechiae and ecchymosis in the upper limbs and body were targeted 24 hours after catheterization, progressing to the lower limbs. Control blood count revealed severe thrombocytopenia with 6,000 platelets/μl (199,000 platelets/μl before catheterization). Hematologists ruled out pseudothrombocytopenia, and recommended platelet transfusion if thrombocytopenia was suspected secondary to intracoronary abciximab administration.

The patient was transferred to the Intensive Care Unit (ICU) due to hypotension with SBP of 80 mmHg and anemia (hemoglobin 8.1 g/dl) (on admission, hemoglobin 12.8 g/dl), even though exteriorization of bleeding was not observed. During her stay in ICU, the patient had episodes of self-limited melena and increased cutaneous ecchymoses. Discontinuation of dual antiplatelet therapy was decided, informing her family of the high risk of severe hemorrhage and stent thrombosis. Three platelet concentrates were transfused while in ICU. Stabilization of platelets was achieved 4 days later. Antiplatelet therapy with aspirin was initiated on the 4th day after catheterization (46,000 platelets/μl), and on the 5th day, in view of hemoglobin stabilization and increased platelets (75,000/μl), clopidogrel therapy was restarted and was well tolerated, with no chest pain episodes or ECG abnormalities during follow-up. On discharge, 9 days after catheterization, the patient had platelet count of 230,000 platelets/μl and hemoglobin of 9.7 g/dl.

Patients with drug-induced thrombocytopenia (DIT) typically present with petechiae, ecchymosis and epistaxis caused by acute, and often severe, decrease of platelet production. When thrombocytopenia

is severe (<20,000 platelets/μl), bleeding may occur in the gastrointestinal mucosa or genitourinary tract. In extreme cases, intracranial or pulmonary hemorrhages have been reported, with fatal outcome.

Drug-induced thrombocytopenia diagnosis is often a challenge, particularly in polymedicated patients. It is necessary to consider pseudothrombocytopenia and heparin-induced thrombocytopenia as part of the differential diagnosis, as we did with our patient, since it is an exclusion diagnosis. (3) Pseudothrombocytopenia is the result of in vitro platelet agglutination due to EDTA-dependent anti-platelet antibodies. If a different anticoagulant such as citrate is used, actual platelet count can be performed, as we did in our lab test, confirming that the actual count was 6000 platelets/μl. (3) Type-1 heparin-induced thrombocytopenia (HIT) results from direct interaction of heparin with the platelet membrane, and occurs in up to 10% of patients treated with heparin usually within the first 72 hours (rarely achieving <100,000 platelets/μl), and is not associated with bleeding or increased risk of thrombosis. (3)

Type-2 HIT is caused by the formation of antibodies against heparin-platelet factor 4 (PF4) complex. These antibodies activate platelets and cause release of prothrombotic microparticles, platelet consumption, and thrombocytopenia. The main symptom is the sudden onset of thrombocytopenia involving a drop of platelet count by 50% compared to baseline levels, and/or thrombotic complications some 5 to 14 days after the start of heparin therapy, although it is usually related to long-term, repeated heparin treatments, which did not occur in our case.

In general, venous or arterial thrombotic complications are more common, and platelet count is higher than in thrombocytopenia due to abciximab. Based on clinical criteria, hematologists ruled out HIT, since our patient was treated only with low-molecular weight heparin before and sodium heparin during catheterization, but not after the procedure. Lab demonstration of platelet activation using an antigen or functional assay confirms the clinical diagnosis if considered necessary by hematology. (3, 5)

Abciximab is a chimerical monoclonal antibody (human/mouse) that selectively inhibits GP IIb/IIIa receptor, causing prolonged platelet aggregation blockage. (4) Approximately 1-2% of patients treated with abciximab have acute thrombocytopenia within a few hours after starting treatment. (5) Although its pathophysiological mechanism is partly unknown, it seems that patient antibodies are produced against drugs that recognize murine sequences in the complementary determining region 3 (CDR 3), which is an abciximab region. (5, 6) Some patients with abciximab-induced immune thrombocytopenia have preexisting antibodies in serum, but many healthy people who have never had contact with the drug before have IgG antibodies that react with abciximab bound to platelets. (4, 6)

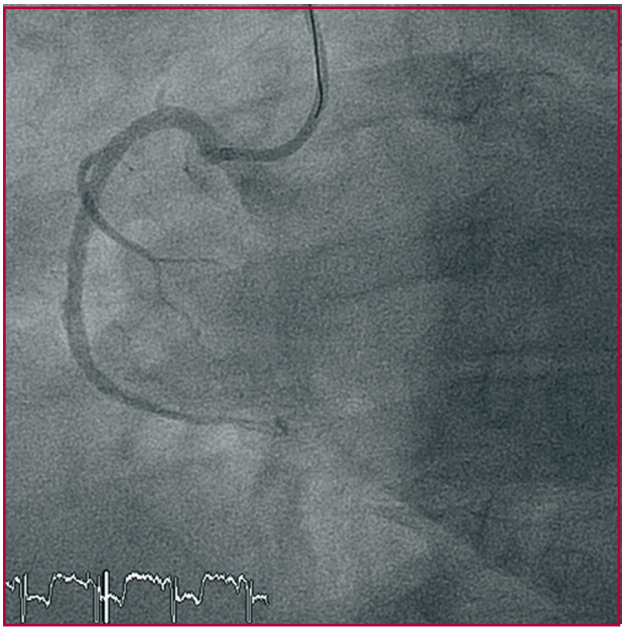

Fig. 1. Coronary angiography showing recanalization of the acute marginal branch of the right coronary artery after intracoronary abciximab bolus administration.

Management of thrombocytopenia secondary to abciximab is controversial, and platelet transfusion and immediate discontinuation of the medication seem to be the most effective measures; (5) in our case, abciximab had been administered as intracoronary bolus and not as continuous perfusion.

Discontinuation of other antiplatelet treatments is also controversial, and there is no consensus among experts. Corticosteroid administration has been researched along with platelet transfusion, with no usefulness evidence. (5) In our experience, we decided to make a balance between the high risk of hemorrhage due to thrombocytopenia and the high thrombotic risk of drug-eluting stents implanted 24 hours before. We found that despite the discontinuation of dual antiplatelet therapy, no acute or subacute thrombosis of the coronary stents occurred (currently 10 months of follow-up free of events), reducing potential hemorrhagic complications. The resolution stage of thrombocytopenia has not been longer than that referred in the literature (3-7 days).

These cases should be reported to the pharmacovigilance committee in each hospital for a more realistic reference of the incidence of thrombocytopenia in daily practice. In some centers, performing a blood count 2 hours after the start of abciximab administration is part of the protocol for an early diagnosis of thrombocytopenia. (5)

All medical professionals should take into account this rare complication in patients undergoing catheterization and treated with glycoprotein-IIb/IIIa inhibitors, for better monitoring and to avoid severe complications in the first hours after the procedure.

Conflicts of interest
None declared.
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Pulmonary Vein Stenosis: A Rare Case of Heart Failure and Pulmonary Hypertension in a Cancer Patient

We report the case of a 55-year-old female patient with no cardiovascular risk factors or cardiac history. The disease began with recurrent facial palsy and left trigeminal neuralgia. Computed tomography scan revealed cervical lymphadenopathies and tumor of the left maxillary sinus. A biopsy of the maxillary sinus was performed, evidencing lymphoproliferative syndrome, which was sent for typification.

A few months later, the patient presented cough and fever together with progressive dyspnea to functional class III in the previous week.

Hospitalization was decided due to febrile syndrome and dyspnea. Presumptive diagnoses were B symptoms due to lymphoma versus right upper lobe pneumonia. The patient had leukocytosis (32,000 WBC) and poor general condition. Culture samples were collected and an empiric broad-spectrum antibiotic therapy was started. A computed tomography scan showed multiple supra- and infra-diaphragmatic enlarged lymph nodes at the mediastinal level, like a large mass that moved the trachea and compressed the superior vena cava entry into the right atrium, and was in close contact with the atria. Bilateral infiltrates in frosted glass (pulmonary edema), compression of the right upper lobe, moderate bilateral pleural effusion with passive atelectasis, and moderate pericardial effusion were also detected (Figure 1A).

Aspiration-biopsy of bone marrow was performed: flow cytometry revealed 37% large cells consistent with large B-cell non-Hodgkin lymphoma (LBCNHL). It was decided to start the pre-stage with glucocorticosteroid therapy.

The patient was transferred to the Coronary Care Unit two days after hospitalization due to FC III dyspnea and pericardial effusion. Given its semiology (bilateral crepitant rales) and X-ray images (venous-capillary hypertension pattern), it was interpreted as left heart failure (Figure 2).

The first transthoracic echocardiography (TTE) showed normal dimensions and thickness, left ventricular preserved ejection fraction (EF) and prolonged LV relaxation pattern (diastolic dysfunction grade I), moderate pericardial effusion, severe pulmonary hypertension (PHT) (60 mmHg), and no valve diseases (Figure 3).

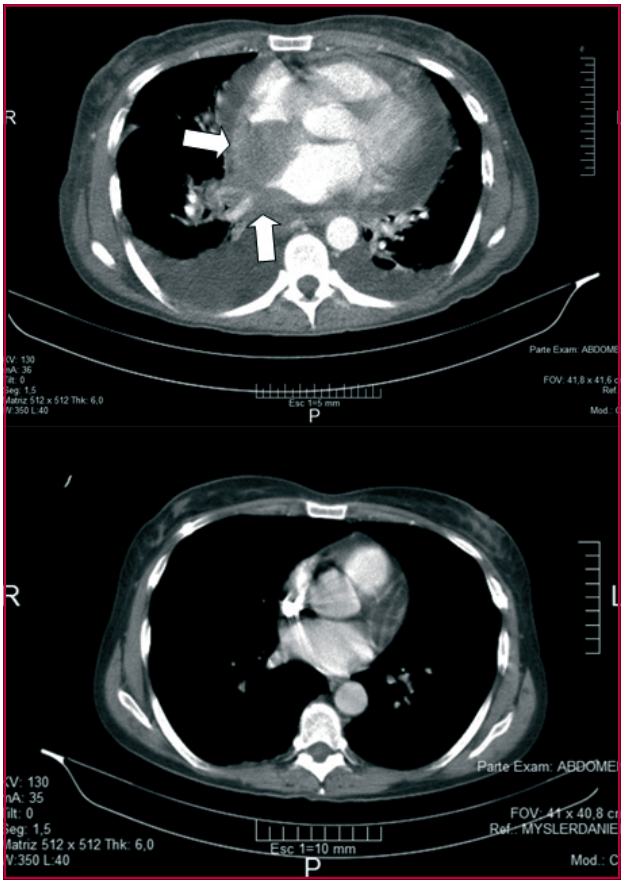

Fig. 1. Pretreatment chest computed tomography (CT) scan (1A top): mediastinal mass in close contact with right to left atria (*arrows*), bilateral pericardial and pleural effusion. Post-treatment chest CT scan (1B bottom): absence of enlarged lymph nodes, pleural or pericardial effusion.

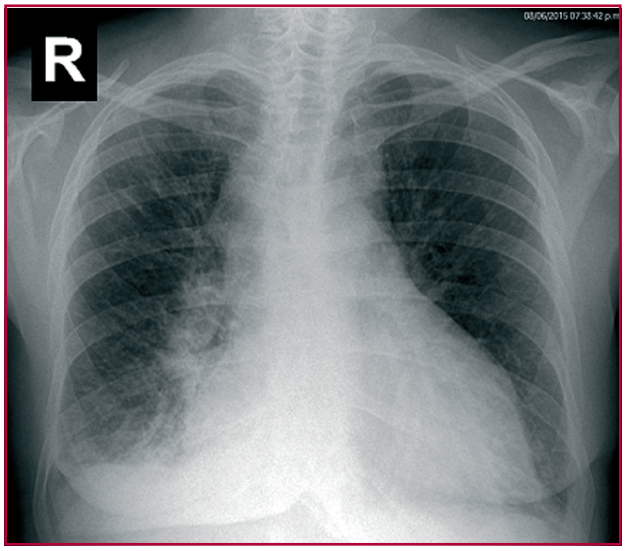

Fig. 2. Chest X-ray showing signs of bilateral venous capillary hypertension.

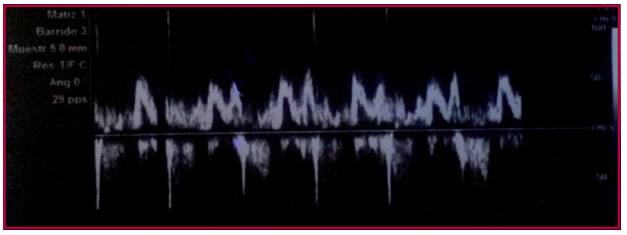

Fig. 3. First transthoracic echocardiography - Mitral flow.

The patient presented significant clinical improvement with intravenous furosemide and corticosteroids, and a second TTE performed 5 days after treatment initiation revealed moderate acute PHT (44 mmHg). At the end of the course of antibiotics, cultures were negative. Lumbar puncture was positive for neoplastic cells. A few days later, the patient was started on chemotherapy with cyclophosphamide, and then rituximab, doxorubicin, vincristine, and prednisone, to complete the R-CHOP regimen, adding methotrexate due to central nervous system (CNS) involvement. The patient made good progress. A third TTE performed 12 days after admission showed EF of 65%, without PHT (29 mmHg). The patient was discharged a week later, and made good progress in outpatient follow-up. A follow-up CT scan was performed two months after discharge (Figure 3B): no axillary or mediastinal lymph nodes were identified and no pleural or pericardial effusion was observed either.

Final hematology-oncology diagnosis: large B-cell non-Hodgkin lymphoma (high histological grade and high proliferation rate) with CNS involvement. Cardiology diagnosis: left heart failure and PHT secondary to extrinsic pulmonary vein compression due to mediastinal mass.

Pulmonary vein stenosis is an uncommon entity. Until a few years ago, it was a condition appearing almost exclusively in children, associated to other forms of congenital heart disease, occurring as a primary condition or secondary to anomalous pulmonary venous return repair. (1) Its occurrence in adults is even rarer. The few cases reported in the literature are associated to mediastinal processes such as neoplasms, sarcoidosis, or fibrosing mediastinitis (a rare complication of tuberculosis or histoplasmosis). (2) However, its incidence has been increasing in recent years due to radiofrequency ablation procedures to treat atrial fibrillation, which may affect 1-3% of cases. (3-5) Symptoms in adult patients include dyspnea on exertion, cough, or even hemoptysis. Chest X-ray can show diffuse or localized pulmonary edema, depending on the veins involved. In most cases, echocardiography, particularly TTE, can evaluate pulmonary veins. Magnetic resonance imaging and computed tomography scan are very useful for the assessment of these patients. Treatment of cases due to extrinsic compression focuses on the underlying condition. Balloon angioplasty has shown positive outcomes in iatrogenic cases associated to radiofrequency ablation.

We present a case of heart failure and pulmonary hypertension in a female patient with mediastinal lymphoma, who first received diuretic therapy and, at the same time, corticosteroids and chemotherapy for her hematology-oncology condition. Images and clinical course supported the suspicion of extrinsic pulmonary vein compression as the pathophysiological mechanism of the process.

Conflicts of interest

None declared.

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Left Atrial Reservoir Function: Prevalence of Dysfunction in the General Population

The left atrium (LA) has three major roles: contractile pump that delivers 15% to 30% of the left ventricular filling; deposit or reservoir that collects pulmonary venous return during ventricular systole; and conduit for the passage of stored blood from the LA to the left ventricle (LV) during early ventricular diastole. Echocardiographic recommendations for chamber quantification stress the relevance of LA dimensions and its implications in clinical events, but do not mention the routine use of atrial function parameters. (1) The altered reservoir function (RF) can be an early marker of left ventricular diastolic dysfunction prior to ventricular hypertrophy or atrial dilatation, and it can even predict cardiovascular events. (2) No information is available about the normal RF value measured by two-dimensional echocardiography or how often it occurs in the general population. The purpose of our study was to determine the frequency of abnormal RF.

This was a retrospective, cross-sectional study conducted between May and October, 2016. Inclusion criteria was an echocardiography with adequate acoustic window to perform volumetric measures of the LA in 4 and 2 chamber apical views. In addition, patients had to be in sinus rhythm, with no significant valve diseases, and with left ventricular ejection fraction > 50% (Figure 1). Medical history and clinical background were reviewed.

The echocardiography was performed with a GE Vivid S5 or E9 ultrasound machine, and recorded in RAW format for offline analysis. Standard echocardiographic measurements were performed under existing regulations. (1) Maximum left atrial volume was obtained just before the opening of the mitral valve, and minimum left atrial volume just before its closure; both measurements were in 4- and 2- apical chamber views. The reservoir function was calculated with the following formula: Maximum left atrial volume - Minimum left atrial volume / Maximum left atrial volume × 100. (3)

Taking into account an abnormal RF prevalence of 4%, a sample of 59 patients was estimated for a 95% reliability level and 5% error margin. Categorical variables were expressed as percentage, and continuous variables as mean and standard deviation. Mean left atrial RF was obtained from all the study population. Kolmogorov-Smirnov goodness-of-fit test was performed to determine RF normal distribution. The RF limit value was considered as the mean minus two standard deviations. SPSS 17™ statistical package was used to perform the analyses.

A total of 64 patients were included in the study. Mean age was 55.7 years (±15.2 years), 57.8% were women, and 56.2% had hypertension. All the patients had preserved left ventricular ejection fraction, with normal ventricular diameters and left atrial indexed volume (Table 1). The RF was 55.7 ± 9.7% with normal distribution (p=0.94); therefore, the limit RF was determined as ≥36.3%, and with this normal limit, 3 patients had abnormal RF (4.69% p5% CI 95 0.98%-13.09%).

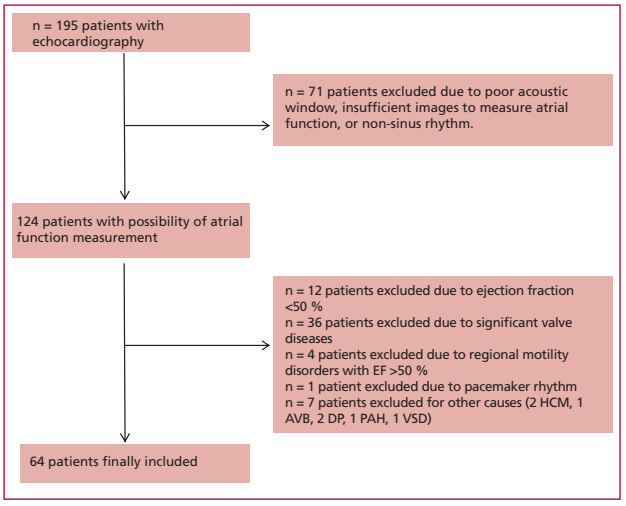

Fig. 1. HCM: Hypertrophic cardiomyopathy. AVB: Atrio-ventricular block. DP: Dual pacemaker. PAH: Pulmonary artery hypertension. VSD: Ventricular septal defect.

The LA plays an important role in the physiology of the cardiovascular system, acting as a reservoir during ventricular systole, receiving blood from the pulmonary veins. During early diastole, when the mitral valve is open, the LA acts as a conduit allowing the passage of blood volume from the LA to the LV due to pressure and aspiration differences of the LV; then atrial contraction determines the third function of the LA, that is, as a booster pump during late diastole. Left atrial function has a prognostic value in different clinical scenarios, even adding prognostic value to the measurement of atrial volume. (3) These functions can be determined by echocardiography, measuring atrial volumes in different phases of the cardiac cycle, and they can also be assessed with new technologies (for example, strain). (4)

Echocardiographic recommendations do not determine the normal values of atrial functions. The limit value we found is similar to that reported by other researchers in Argentina, measured with atrial strain. (5) A recent meta-analysis of 40 articles published normal atrial function reference values and revealed a normal reference range for reservoir strain of 39% (95% CI, 38%-41%). (6)

In our work, we did not use atrial strain to measure RF; however, since we used two-dimensional echocardiography, it broadens its use to any echocardiography laboratory that does not have the technology to measure longitudinal atrial strain.

Therefore, we conclude that the limit value of RF is >36.3%, and that this value is altered in 1-13% of the general population, despite normal atrial volumes.

Table 1. Population characteristics

History	
Age (years±SD)	55.7±15.2
Hypertension (%)	56.2
Diabetes (%)	10.9
Hypercholesterolemia (%)	50.7
Previous atrial fibrillation (%)	4.7
Coronary heart disease (%)	7.8
Glomerular filtration (ml/min±SD)	86.5±18.7
ECG criteria of LVH (%)	6.3
Echocardiography	
LV diastolic diameter (cm±SD)	4.62±0.42
LV systolic diameter (cm±SD)	2.92 ± 0.42
LV ejection fraction (%±SD)	66.2 ± 6.6
LV mass index (g/m2±SD)	62.6 ± 12.8
LA volume index (ml/m2)	25.5 ± 6
LA reservoir function (%±SD)	55.7 ± 9.7
E wave (m/sec±SD)	0.76 ± 0.15
A wave (m/sec±SD)	0.74 ± 0.15
E-wave deceleration	204 ± 37.9
E´ wave (cm/sec±SD)	0.12 ± 0.04
E/E´ ratio	6.87 ± 2.47

LV: Left ventricular. LA: Left atrial. LVH: Left ventricular hypertrophy according to Cornell's and/or Sokolov's criteria.

Conflicts of interest

None declared.

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