Case 6 - 80-year-old Woman, with Arterial Hypertension and Chronic Dissection of the Aorta, presenting Intense Dyspnea and Chest Pain

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An 80-year-old female patient sought medical help due to chest pain and dyspnea. At 72 years of age (April 1996) she had been submitted to a right mastectomy due to breast carcinoma. After the surgery, she was submitted to radiotherapy and subsequently, to long-term treatment with tamoxifen. During evolution, she presented endometrial thickening, diagnosed through ultrasonography, and a hysterectomy was indicated.

At the time, the patient also complained of dyspnea on exertion. Additionally, systemic arterial hypertension and aortic systolic murmur were also diagnosed.

The patient was then referred to preoperative assessment. She was evaluated at the hospital for the first time on January 3, 1997, when she was 73 years.

Physical examination disclosed a heart rate (HR) of 60 bpm and blood pressure (BP) of 140/95 mmHg. Pulmonary assessment was normal. The heart sounds were normal, with an ejection systolic murmur ++/4+ in the aortic area. Abdominal and extremity assessment was normal.

The electrocardiogram (ECG) performed on December 30 showed regular sinus rhythm, left-chamber overload and ventricular repolarization alterations: inverted and asymmetric T waves from V_4 to V_6 and also from I to aVL (Figure 1).

On December 30, 1996, the chest x-ray showed a slightly enlarged cardiac area, at the expense of the left ventricle (LV).

The laboratory assessment disclosed: 13 g/dl of hemoglobin, 39% of hematocrit, glycemia of 91 mg/dl, 0.8 mg/dl of creatinine, total cholesterol of 196 mg/dl, HDL-cholesterol of 49 mg/dl, LDL-cholesterol of 120 mg/dl, and 136 mg/dl of triglycerides.

The patient was submitted to a hysterectomy on April 19, 1997.

The echocardiogram, performed on September 19, 1997, disclosed LV hypertrophy with preserved function and aortic and left atrial dilatation, as well as mild aortic stenosis. The cardiac chamber dimensions are shown in Table 1.

Key Words

Hypertension; heart failure; pulmonary embolism; thoracic aorta/surgery.

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From 1997 on, she presented progressive worsening in dyspnea, which started to be triggered by mild exertion, such as walking a block on a level surface.

The perfusion myocardial scintigraphy performed with MIBI.99mTc and dipyridamole (January 1998) showed homogeneous radiotracer uptake and was not considered suggestive of myocardial ischemia.

As the symptoms persisted, the hemodynamic and angiographic studies were indicated. The hemodynamic study did not show aortic transvalvular gradient. The coronary angiography (July 1998) did not disclose obstructive lesions in the coronary arteries. The ventriculography disclosed normal LV wall motility.

The subsequent echocardiographic assessments were considered suggestive of increase in myocardial hypertrophy. Additionally, LV outflow tract pressure gradient was also diagnosed. The measurements obtained are shown in Table 1.

In 1999, the control chest x-ray showed a round hypotransparent image in the lung. A chest tomography was indicated. On April 28, 2000, the tomography disclosed mild left pleural effusion with pleural thickening and a hyperdense area in the left lower lobe, without adenomegaly. She also presented cardiomegaly and atheromatous aorta.

The management was assessed together with the thoracic surgical team. The expectant management was chosen, as well as evolution control tomographies, performed preferably every six months.

On January 2002, the patient complained of tachycardic palpitations and worsening of dyspnea. She sought medical help and a diagnosis of atrial fibrillation was made. She initiated treatment with warfarin.

On August 12, 2002, the control chest tomography disclosed a 57-mm aneurysm in the ascending aorta that extended to the aortic arch, where the diameter was 35 mm; the diameters in the descending aorta and the thoracoabdominal transition were 34 and 24 mm, respectively. There were signs of dissection in the ascending aorta that started at the aortic root and reached the aortic arch.

The abdominal ultrasonography performed on August 16, 2002, disclosed calculous cholecystitis, diffuse atheromatosis of the abdominal aorta and renal cysts.The Tc 99m -DTPA renal scintigraphy performed in August 2002 was normal.

The ECG performed on September 2002 disclosed sinus rhythm, HR of 51 bpm, intraventricular conduction

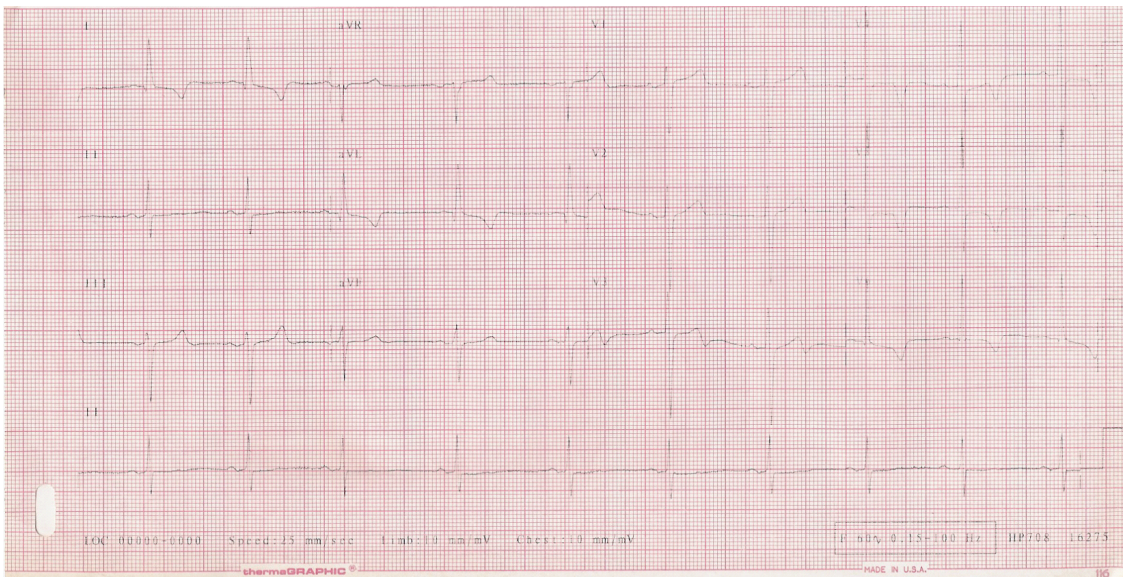

Fig. 1 – Electrocardiogram: sinus rhythm, left-chamber overload and secondary alterations of ventricular repolarization.

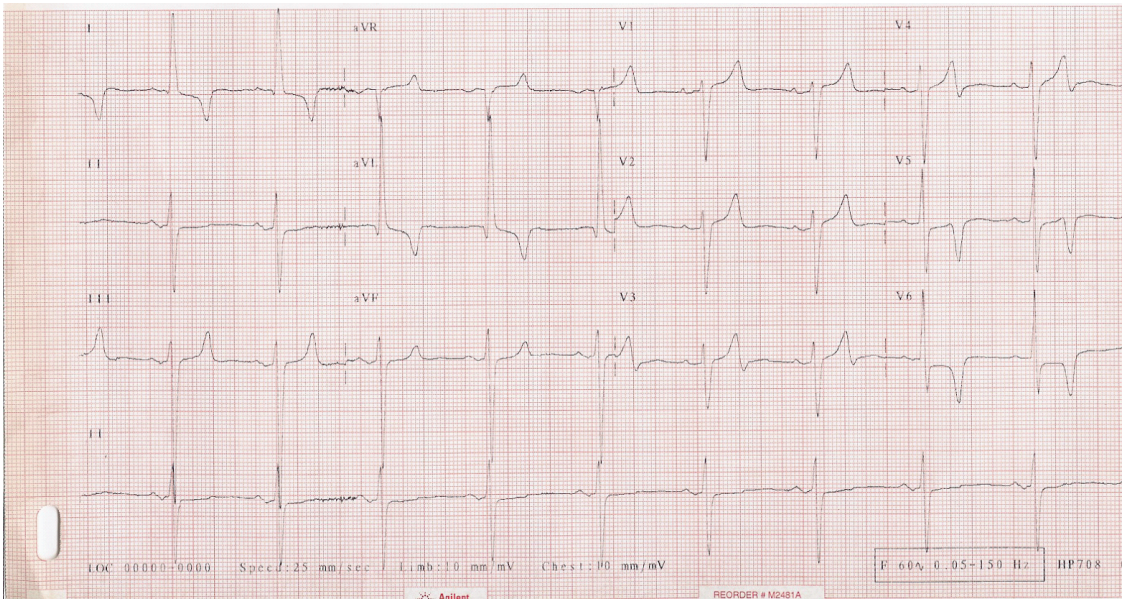

Fig. 2 – *Electrocardiogram: sinus rhythm, left-bundle branch block and of its anterosuperior division, left-chamber overload and secondary alterations of ventricular repolarization.*

disorder of the left-bundle branch block, as well as of the its anterosuperior division, overload of the left chambers, plus-minus T wave at V_4 and negative T wave at V_5, V_6, I and aVLl (Figure 2).

The anticoagulant treatment was withdrawn.

On November 2002, the patient presented worsening of the dyspnea, which was then triggered by mild exertion and decubitus position.

At this time, tumor markers CEA, CA 125 and CA 19.9 were normal. A new coronary angiography did not disclose obstructive coronary lesions and the anti-hypertensive treatment improved symptoms.

On June 20 2003, the control tomography showed 1.2-cm nodules in the posterior basal segment of the right lower lobe and another nodule, < 1 cm, in the anterior segment of the right upper lobe. She also presented left posterior basal

fibroatelectasis striae, slight pleural effusion on the left side and cardiomegaly. The ascending aorta aneurysm was 57 mm in diameter and there was a lower-density area in the lumen, suggesting dissection, as in the previous assessment.

During evolution, the patient presented intense dyspnea, pleuritic chest pain and alterations in consciousness. Therefore, she was taken to the hospital.

On July 14, 2003, the physical examination showed the patient presented a poor general status, was moaning, had a HR of 60 bpm, inaudible blood pressure, cold and cyanotic extremities. Pulmonary auscultation showed basal crackling rales, systolic murmur ++/4+ in aortic area and systolic murmur +++/4+ in the tricuspid area. The abdominal assessment was normal and there were no signs of deep venous thrombosis in the lower limbs.

At this time, the ECG showed atrial fibrillation, mean HR of 80 bpm, right-bundle branch block as well as of the anterosuperior division of the left-bundle branch and diffuse alterations in ventricular repolarization (Figure 3).

Laboratory assessment showed:urea=123 mg/dl, creatinine = 1.6 mg/dl, potassium = 4.4 mEq/l, sodium = 141 mEq/l, prothrombin time (INR) = 1.69 and APTT ratio = 1.1. Arterial gasometry, with the patient using of a nasal catheter, showed a pH = 7.33, pCO_2 = 59 mmHg, pO_2 = 147 mmHg, hemoglobin saturation = 98%, bicarbonate = 31 mEq/l and base excess = 3.4 mEq/l. D-dimer was > 400 ng/ml.

On July 16 2003, the ultrasonography disclosed superior vena cava ectasia and calculous cholecystitis. She also presented mild pleural effusion on the left side, moderate pleural effusion on the right side and normal kidneys with a 1-mm caliceal calculus in the left kidney, with no signs of hydronephrosis.

During the hospitalization, the patient received noradrenaline, 120 mg of enoxaparin daily and sedatives.

On June 16 2004, the patient still presented confusion and hypotension and underwent a further decrease in the level of consciousness, which required orotracheal intubation for respiratory support. On the following days, she remained hypotensive and needed increasing doses of noradrenaline, presenting irreversible cardiac arrest (July 19, 2004).

Clinical aspects

The present case reports on a 72-year-old female patient who, after undergoing treatment of breast cancer with surgery, hormone therapy and radiotherapy, started to present dyspnea on exertion.

At this time, systemic arterial hypertension was also diagnosed and a systolic murmur in the aortic area was identified. The initial assessment showed signs of structural heart disease: enlarged cardiac area at the chest x-ray at the expense of the left ventricle and left-chamber overload at the ECG.

Considering these data, the main diagnostic hypothesis to explain the patient's symptoms is heart failure. The heart failure clinical syndrome is characterized by dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, lower-limb edema, hepatomegaly, fatigue and dizziness.

Several studies have been performed with the purpose of defining diagnostic criteria for heart failure. Using Framingham criteria, this patient presented a major criterion (enlarged cardiac area on chest x-ray) and a minor criterion (dyspnea on exertion). A definitive diagnosis requires two major or a major plus two minor criteria. The presence of signs of structural heart disease at the complementary examinations corroborates this diagnosis.

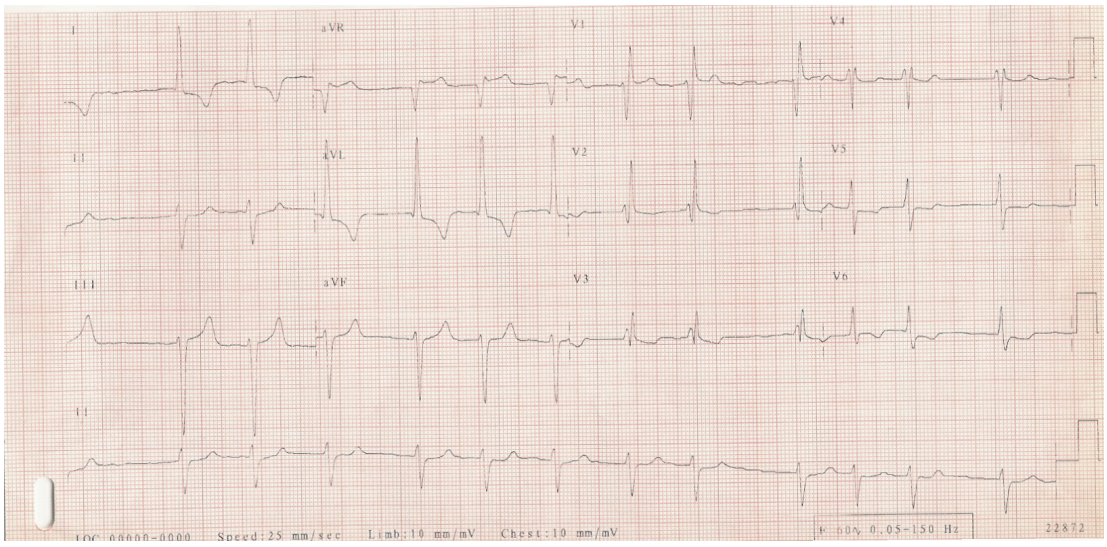

Fig. 3 – Electrocardiogram: atrial fibrillation, right-bundle branch block as well as of the anterosuperior division of the left-bundle branch and secondary alterations of ventricular repolarization.

Table 1 – **Echocardiographic Evolution**

	09.19. 1997	12.08. 2000	08.21. 2001	11.14. 2001	07.14. 2003
Left ventricle (mm)					
Interventricular septum	13	15	16	15	15
Posterior wall	13	14	15	14	12
Diastolic diameter	50	52	45	53	46
Systolic diameter	33	33	30	30	28
Δ diameter (%)	34	36.5	33.3	43.4	39.1
Aorta (mm)	44	44	34	27	-
Left atrium (mm)	51	46	50	47	42
Right Ventricle (mm)	20	Normal	Normal	Normal	Dilated
Aortic transvalvular gradient (mm Hg)	33	-	-	-	-
Left ventricular outflow tract pressure gradient (mm Hg)	-	50	113	67	
Ascending aorta aneurysm (mm)	-	-	51	Aneurysm and mural thrombus	58
Mitral regurgitation	Mild	Moderate	Moderate	Mild	Mild
Tricuspid regurgitation			Mild	Moderate	Severe
Right Ventricular Systolic Pressure (mm Hg)	-	-	40	53	-

The echocardiogram is a very important evaluation tool in the assessment of patients with heart failure, as it can disclose: size and volume of cavities, ventricular wall thickness, systolic and diastolic ventricular function, as well as valve morphology and function. In the present case, the echocardiogram showed: enlarged left atrium, normal- sized left ventricular (LV) cavity, but significant LV-wall hypertrophy, normal global LV systolic function (shortening fraction 34%) and a low aortic transvalvular gradient.

The LV hypertrophy (LVH) can lead to the decrease in compliance and, consequently cause diastolic ventricular dysfunction and heart failure. It is considered secondary when it is caused by an identified disorder. Systemic arterial hypertension is the most common cause and LVH is a marker of increased risk of cardiovascular events in the hypertensive patient[1]. Another cause of secondary LVH is aortic stenosis. In the present case, the degree of stenosis was mild and not likely the cause of the cardiomiopathy. Additionally, the aortic stenosis was not confirmed at the hemodynamic study, performed subsequently (in 1998), which did not show the presence of aortic transvalvular gradient.

Although the echocardiogram did not show signs of ischemic cardiomyopathy, such as alterations in segmental contractions, the examination is essential to assess the coronary failure. The present case was an elderly and hypertensive patient, who therefore presented risk factors for coronary artery disease, and who started to present dyspnea on exertion, which can correspond to an ischemic equivalent. This evidence of coronary failure is more common in female, elderly and diabetic patients. In the present case, a functional assessment was initially performed (dipyridamole myocardial perfusion scintigraphy), which did not disclose the presence of ischemia. Subsequently, a coronary angiography did not disclose the presence of obstructive lesions, ruling out the diagnosis.

Due to the fact that the patient was submitted to radiotherapy on the chest during the treatment of a breast carcinoma, the cardiomyopathy secondary to radiotherapy is a diagnosis that must be recalled. The irradiation of heart can cause damage to practically all its components, including the pericardium, myocardium, valves, coronary arteries and conduction system. Pericarditis is the typical acute manifestation of radiation injury, whereas chronic pericardial disease, cardiomyopathy, coronary artery disease, valvulopathies and conduction disorders can occur years after radiation exposure.The cardiomyopathy secondary to radiotherapy is a cause of restrictive cardiomyopathy, characterized by a rigid and non-compliant ventricle, preventing its adequate filling, which, therefore, leads to diastolic dysfunction. Typically, the left ventricular cavity is normal and there is no myocardial hypertrophy.

The subsequent echocardiographic assessments of the patient showed an increase in myocardial hypertrophy and the presence of a left ventricular outflow tract gradient. The ventricular hypertrophy, when it is secondary, is diffuse, which does not justify the demonstrated intraventricular

pressure gradient. Thus, the diagnostic hypothesis of primary hypertrophic cardiomyopathy must be highlighted.

The hypertrophic cardiomyopathy (HCM) is a genetic disease characterized by a disproportional LV hypertrophy of the left ventricle and, occasionally, of the right ventricle, caused by the disarray and hypertrophy of myocytes.The interventricular septum is the most commonly affected site; however, other forms of presentation can occur, with concentric or predominantly apical hypertrophy.

The ventricular volume is normal or decreased, and diastolic dysfunction usually occurs. The hypertrophy can cause obstruction of the LV outflow tract, generating an intraventricular systolic pressure gradient in up to 25% of the patients[2].

The symptoms can be induced by several mechanisms: LV outflow tract obstruction at rest or on exertion, diastolic dysfunction, arrhythmias, conduction disorders and systolic dysfunction due to extensive myocardial involvement.

Dyspnea on exertion is the most common symptom and occurs in up to 90% of the symptomatic patients[3]. Orthopnea and paroxysmal nocturnal dyspnea are less frequent symptoms. Chest pain on exertion can occur in 30% of the patients, generally with normal coronary angiography[4].

Syncope or presyncope is another important manifestation – approximately 25% of the patients with HCM report at least one episode of syncope. It can be caused by: LV outflow tract obstruction, myocardial ischemia during exercise, arrhythmias and baroreflex activation with inadequate vasodilation[5]. The arrhythmias can be either supraventricular or ventricular and due to the systolic and diastolic abnormalities, they might not be well tolerated. Atrial fibrillation is the most common sustained arrhythmia and can occur in up to 25% of the patients, considering that its incidence increases with age and is associated with an enlarged left atrium[6].

Although most HCM cases develop in childhood or adolescence, the late-onset disease is recognized. In elderly patients, the HCM is initially associated to mutations in the alpha-cardiac myosin heavy chain, troponin I and myosin-binding protein C genes[7]; however, the probability of identifying a mutation is lower. In general, the late-onset HCM has a better prognosis than the disease diagnosed in young patients.

New findings appeared at the patient's evolution. In 1999, the chest x-ray showed a round hypotransparent area in the lung, which was confirmed by the tomography (hyperdense image in the lower left lobe). The patient presented associated pleural effusion and thickening, without adenomegaly.A hyperattenuating image associated to pleural effusion and thickening in a chest tomography can correspond to an infectious, neoplastic or inflammatory process or pulmonary embolization.

When secondary to infection, it is generally associated with a more significant clinical picture, such as fever, worsening of the general status, weight loss and more prominent pulmonary symptoms (coughing and sputum). Considering that the patient had had breast cancer, the hypotheses of pulmonary thromboembolism (PTE) and active neoplastic process cannot be ruled out.

PTE is a severe disease, characterized by the partial or complete obstruction of the pulmonary arteries anywhere along their trajectory. It is generally associated with the triad: venous stasis, hypercoagulability and endothelial injury. Factors such as neoplastic processes, postoperative periods – mainly orthopedic ones – and systemic diseases that result in hypercoagulability can predispose to the onset of PTE.

Pulmonary metastases secondary to breast cancer are not uncommon. They usually affect more peripheral lung regions due to the preferential irrigation to the lung base.

In August 2002, a control tomography diagnosed an ascending aorta aneurysm with signs of dissection. The aortic dissection can be divided, according to the Stanford classification, as type A – when the ascending aorta is affected – and type B – when there is no proximal involvement. This differentiation is important for prognostic and therapeutic decisions, considering that the acute type A dissection presents higher mortality and thus, an emergency surgical intervention is indicated, whereas the type B dissection has better prognosis and must be preferably treated with medication. The chronic type A dissection is not common, as most patients die during its initial phase, when it is not surgically treated.

In 2003, the patient presented severe dyspnea, pleuritic chest pain and alteration of consciousness, associated to shock and the onset of murmur in the tricuspid area. There were no signs of deep venous thrombosis (DVT) in the lower limbs.

The PTE is a potentially severe disease, of which real incidence is underestimated due to the broad variations in possible clinical manifestations. It may present with a dramatic picture of sudden dyspnea associated with hypoxia and cardiogenic shock, which may even be a cause of sudden death. However, it can also present with less clear and/or unspecific symptoms, such as mild dyspnea, pleuritic chest pain, malaise and fatigue, coughing, hemoptoic sputum, low fever[8,9]. The latter, most of time, do not receive a diagnosis of PTE when treatment is sought for the first time and, therefore, no appropriate therapeutics is provided. When compared to those patients that receive treatment, the untreated ones present higher mortality, mainly due to disease recurrence[10,11].

Some risk factors are clearly associated with the occurrence of PTE, such as prolonged immobilization, history of previous DVT and/or PTE, recent surgical intervention (mainly orthopedic ones), history of cancer, acquired or inherited thrombophilia, previous cardiac or pulmonary disease and the association of cigarette smoking and oral contraceptives in women[12]. Specifically in the case of breast cancer, PTE usually occurs at more advanced stages of the disease.

Unspecific findings such as sinus tachycardia or increase in the basal rhythm (such as high-frequency atrial fibrillation) and right-bundle branch block are common at the ECG. The D-dimer testing by ELISA is also a low-specificity assessment, but with a high negative predictive value, i.e., when it is negative, it practically rules out the diagnosis of PTE.

The treatment is initially based on the patient's stabilization, with ventilatory and hemodynamic support, as necessary. In case of no contraindication, full anticoagulation

must be initiated with non-fractionated or low-molecular weight heparin. Other approaches, such as thrombolysis and embolectomy, are reserved for a small number of patients with PTE, who present higher severity signs and risk of death.

The presence of hemodynamic instability is the most important clinical criterion regarding the risk of death. The presence of hemodynamic instability constitutes the most precise indication of thrombolysis in PTE[13]. Some other indications are acceptable, such as significant hypoxia, very large PTE and significant right ventricular dysfunction at the transthoracic echocardiogram.

In patients that present contraindication to thrombolysis or the ones that do not respond to the fibrinolytic treatment, the surgical embolectomy can be attempted.

The patient developed signs of poor tissue perfusion, with decrease in the level of consciousness, worsening of kidney function and hypotension, characterizing a condition of shock. She needed orotracheal intubation due to respiratory failure and presented hypotension that was refractory to vasopressors.

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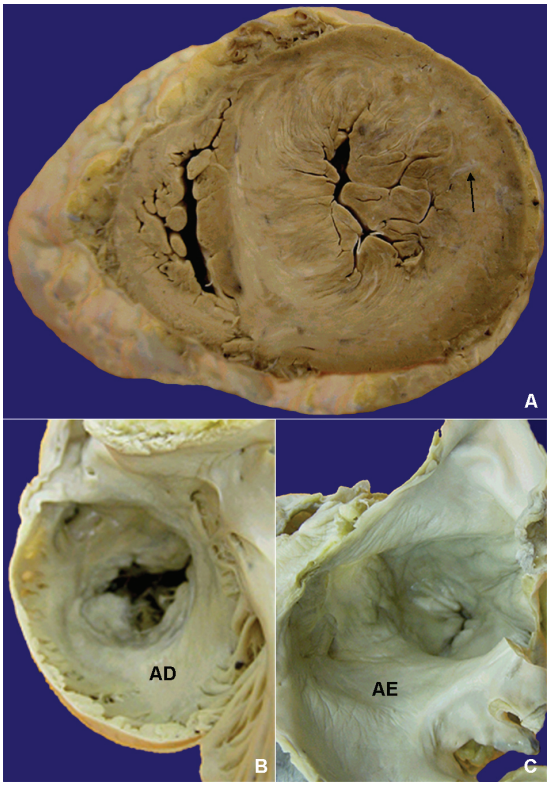

Fig. 4 – Heart macroscopy, in (A) cross-sectional view, showing the marked LV concentric hypertrophy, of which lumen is almost nonexistent. The black arrow shows many of the whitish striae present on the sectioned surface, which characterize myocardiosclerosis. (B) and (C) show the marked dilatation of the right atrium (RA) and left atrium (LA).

Diagnostic hypothesis

In summary, this patient had been previously treated for breast cancer and developed heart failure, probably secondary to hypertrophic cardiomyopathy. She presented acute decompensation caused by pulmonary thromboembolism, which caused refractory shock and death.

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Necropsy

At the necropsy, the heart presented marked LV concentric hypertrophy, with multifocal myocardiosclerosis (Figures 4A, 5A and 5B). There were also recent ischemic lesion sites, represented by randomly distributed myocardial microinfarctions, presenting an evolution of approximately two weeks (Fig. 5C). The left atrium showed moderate dilatation and endocardial thickening (Figure 4C). The mitral valve presented no alterations and the aortic valve was apparently normal. The right cardiac chambers showed marked ventricular hypertrophy and atrial dilatation (Fig. 4B). The coronary arteries showed mild atherosclerotic lesions.

Externally, the ascending aorta showed a prominence on the anterolateral side (Figure 6A and 6B). When cut, one can observe the presence of chronic dissection of the wall restricted to the ascending aorta, ending before the subclavian artery. The false lumen had a short retrograde trajectory and was filled with thrombi. The dissection resulted in a prominent wall, generating mild stenosis due to extrinsic compression of the true aortic lumen (Fig. 6C). The entrance orifice was approximately 2 cm, with blunt borders and 3.5 cm from the aortic valve plane (Fig. 6D). The histological analysis of the aorta showed lesions that are usually present in dissection, such as decrease in the number of smooth muscle cells, accumulation of mucoid material and elastic fiber rupture.

The other organs showed signs of congestive heart failure, such as liver and lung chronic passive congestion, with passive pulmonary hypertension, in addition to lower-limb edema. There was generalized atherosclerosis, from mild to moderate, affecting the aorta and other branches. In the kidneys, the findings of renal arteriolosclerosis with retention cysts and other aspects of benign nephrosclerosis were also correlated with the arterial hypertension diagnosis. Systemic thromboembolism was also detected in the brain basilar and right femoral arteries, apparently with no effects on sites that were distal to them, of which probable source would be represented by thrombi in the dissection.

Other autopsy diagnoses were: obesity, chronic calculous cholecystitis, previous right radical mastectomy and hysterectomy, chronic pneumopathy with pulmonary emphysema and condensation foci with septal fibrous thickening and acute tracheobronchitis. No neoplastic processes were identified in the assessed organs.

The immediate cause of death was bilateral pulmonary thromboembolism with localized pulmonary infarctions in the left upper and lower lobes and in the right lower lobe, which measured approximately 5 cm each. Moreover, collaborating to the final event, initial signs of shock were observed in the

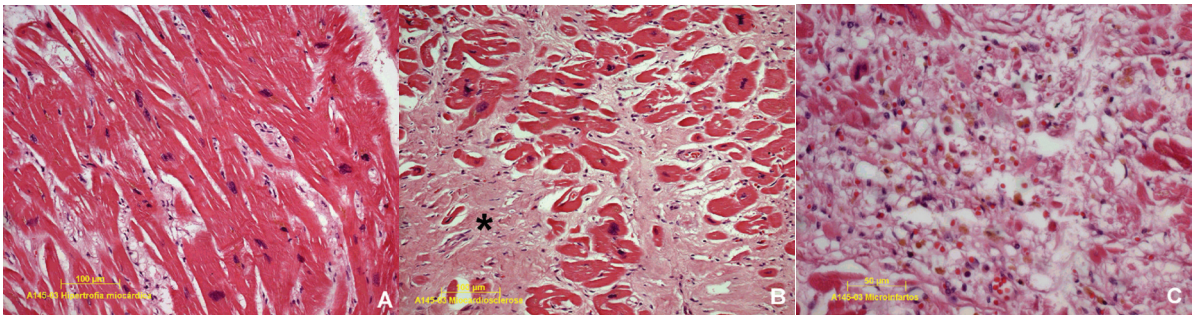

Fig. 5 – Photomicrograph of the myocardium. (A) shows the marked hypertrophy of cardiomyocytes, characterized by large-volume nuclei and increased fiber diameter. (B) shows fibrosis (asterisk) replacing and permeating the remaining cardiomyocytes in this area of myocardiosclerosis. (C) Microfoci of recent intermedio-mural infarction, characterized by cardiomyocyte replacement by granulation tissue, containing macrophages with hemosiderin, brown pigment in the cytoplasm of several cells present in this field. (Hematoxylin & eosin; 20X, 20X and 40X magnification, respectively).

myocardium (hypereosinophilia and nuclear pyknosis in groups of subendocardial cardiomyocytes), liver (necrosis in groups of hepatocytes in the centro-lobular region) and kidneys (initial focal acute tubular necrosis).

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Anatomopathological diagnosis

Obesity; generalized atherosclerosis; hypertensive cardiopathy, with marked hypertrophy and multifocal myocardiosclerosis; global congestive heart failure; chronic dissection of the ascending aorta, type A of Stanford classification; systemic and bilateral pulmonary thromboembolism, in the brain basilar and right femoral arteries; benign nephrosclerosis; chronic calculous cholecystitis; chronic pneumopathy with pulmonary emphysema; previous right radical mastectomy and hysterectomy; cardiogenic shock.

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Comment

The recent foci of ischemic necrosis were correlated with the relative myocardial ischemia due to the marked concentric hypertrophy, which is a well-known fact in cases with a previous clinical history of systemic arterial hypertension, being one of the etiopathogenic mechanisms responsible for myocardial remodeling, myocardiosclerosis and myocardial dysfunction[14,15].

In the case described here, in addition to the arterial hypertension, the mild stenosis in the ascending aorta, caused by the extrinsic compression of the aortic lumen by the dissection, also collaborated to some extent to the pressure overload.

In this hemodynamic context are the secondary dilatation of the left atrium, with endocardial thickening and the chronic passive congestion observed in the lungs, as a morphologically expressed component of left heart failure. The findings of liver chronic passive congestion and lower-limb edema also characterized the right component of heart failure.

The clinical manifestation of aortic dissection presents a wide diversity, varying according to the site of anatomical

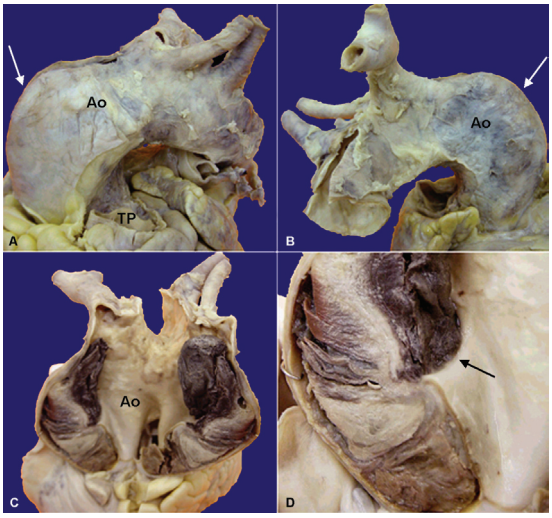

Fig. 6 – Macroscopy of the cardiac base vessels, frontal view in (A) and posterolateral view in (B). In (A) and (B), the aorta (Ao) presents a right anterolateral prominence, indicated by the white arrows; observe the position of the pulmonary trunk (PT). In (C), the aorta was cut longitudinally, exposing the dissection restricted to its ascending segment. In (D), the close-up view shows the entrance orifice (black arrow), with the blunt borders and the short retrograde trajectory of the dissection.

involvement. The presence of congestive heart failure is usually associated with aortic valve regurgitation due to the collapse of the supporting tissue[16]. However, in the present case, the aortic valve was competent and the left ventricular cavity was almost nonexistent due to the hypertrophy, with probable filling restriction and consequent congestion.

This case was classified as type II of DeBakey classification (affects only the ascending aorta) or type A of Stanford classification (affects the ascending aorta, extending or not to the descending aorta). The latter classification has currently been more frequently accepted, due to its simplicity in establishing prognosis and management[16]. The involvement of the ascending aorta occurs in 60% to 70% of the cases; of the

descending aorta, in 20%; of the aortic arch, in 10% and of the abdominal aorta, in 5%. The type A dissections are considered much more severe and of immediate surgical indication, with almost no controversies in the literature. However, the highest mortality occurs in the two first weeks (the time limit considered in the definition of acute dissection), with the mortality curve subsequently stabilizing. In the chronic phase, the risk of death is 5% a month in the first year and 1% a month between the first and the third years[16].

There are several well-known risk factors for aortic dissection. Its association with systemic arterial hypertension is around 80% to 90% in most case series. It is more common in male individuals older than 60 years, although it also occurs at other age ranges, often affecting young individuals with diseases of connective tissues such as Marfan and Ehler-Danlos syndromes, Turner or Noonan syndrome or giant-cell arteritis. Coarctation of the aorta and bicuspid aortic valve can also be associated, as well as iatrogenic factors such as catheterism, intra-aortic balloon and aortic manipulation during cardiac surgery. There have also been reports of dissection in cocaine users and in individuals that suffered closed chest trauma[16].

In the present case, the pulmonary thromboembolism, although it resulted in relatively small infarctions of the lungs, associated with the global congestive heart failure condition, seems to have been enough to cause the patient to develop cardiogenic shock, even though the cardiac decompensation was not morphologically expressed as dilatation of the ventricular chambers.

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