Case 3/2019 – Young Male with Intense Dyspnea, Pulmonary Infiltrate, Normal Cardiac Area and Obliteration of the Apical Portion of the Left Ventricle

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A 25-year-old male patient was hospitalized for severe dyspnea, even at rest, and with productive cough. The patient had been seen a week before with a complaint of back pain that irradiated to the precordial region triggered by exertion, such as climbing stairs and walking a block, which had started two weeks before.

The patient was a smoker (8-pack-years) and reported having hypothyroidism after treatment with radioactive iodine. Previous examinations were carried out for him to be cleared for exercise practice.

The electrocardiogram (ECG) (November 24, 2010) showed left atrial overload and a left ventricular strain pattern. There was an accentuation of ST-depression in the stress test.

At admission (October 14, 2011), heart rate was 100 bpm, blood pressure was 100/70 mmHg, and the physical examination was normal.

The ECG (October 14, 2011) revealed sinus tachycardia (101 bpm), PR interval of 144 ms, QRS duration 103 ms, QTc of 451 ms, left chamber overload and secondary alterations in ventricular repolarization (Figure 1). The chest X-ray (October 14, 2011) was normal. (Figure 2)

The laboratory tests (October 14, 2011) showed: red blood cells, 4,600,000/mm^3; hemoglobin; 14 g/dL, hematocrit; 39%, MCV 85 fL, RDW-CV 12.9%; leukocytes, 11,110/mm^3 (72% neutrophils, 7% eosinophils, 17% lymphocytes and 4% monocytes); platelets, 185,000 / mm^3; sodium, 138 mEq/L; potassium, 4.5 mEq/L; PT, (INR) 1; APTT (rel), 0.93; D-dimer, 485 ng/mL; CK-MB, 0.94 ng/mL; troponin I, 0.535 ng/mL; urea, 35 mg/dL; and creatinine, 0.94 mg/dL. On the following day, CK-MB was 0.71 ng/mL; troponin I, 0.511 ng/mL; total cholesterol, 219 mg/dL, HDL-c, 25 mg/dL; LDL-c, 171 mg/dL; triglycerides, 116 mg/dL; glucose, 92 mg/dL.

The echocardiogram (October 17, 2011) disclosed diameters of the aorta 27 mm, of the left atrium 43 mm, septum thickness of 11 mm, posterior wall of 10 mm, normal right ventricular diameter, and left ventricular diameters (diastole/systole) of 54/34 mm, ejection fraction of 65%; marked hypertrophy in the mid-apical region, pseudonormal filling pattern. There was no left ventricular outflow tract obstruction or valve alterations.

The coronary angiotomography (October 18, 2011) did not disclose coronary calcifications or lesions. However, left atrial dilation and obliteration of the apical portion of the left ventricle were observed.

Diagnoses of hypertrophic cardiomyopathy and hypercholesterolemia were made. He was prescribed 50 mg of atenolol, 100 μg of levothyroxine and 20 mg of omeprazole and was referred for outpatient follow-up (October 18, 2011).

Four days after hospital discharge (October 22, 2011), the patient sought emergency medical care for severe dyspnea, even at rest and in decubitus, in addition to productive cough.

The physical examination (October 22, 2011) disclosed a heart rate of 101 bpm, blood pressure of 112/70 mmHg, pulmonary auscultation showing diffuse crackling rales, cardiac auscultation and abdomen without alterations, and mild edema of legs and feet, without any suggestive signs of deep venous thrombosis.

The ECG (October 22, 2011) was similar to the previous one, with sinus tachycardia, left chamber overload and ST-segment depression with a superior concavity and accentuation of T-wave negativity in leads V_3 to V_6. (Figure 3).

A bilateral diffuse alveolar infiltrate was observed on the chest X-ray (October 23, 2011). (Figure 4)

The laboratory tests (October 22, 2011) showed: erythrocytes, 3700000/mm^3; hemoglobin, 11.6 g/dL; hematocrit, 31%; MCV, 84 fL; RDW-CV 13.2%; leukocytes, 4100/mm^3 (18% band cells; 67% segmented; 2% eosinophils; 11% lymphocytes; and 2% monocytes), platelets, 207000/mm^3; creatinine, 1.06 mg/dL; urea, 35 mg/dL; BNP, 1296 pg/mL; potassium, 4 mEq/L; sodium, 132 mEq / L; arterial lactate, 7 mg/dL; urine type I: density 1.009, ph = 5.5, proteins 0.5 g/L, leukocytes 2000/mL, erythrocytes 79000/mL and presence of severe hemoglobinuria.

The pulmonary angiotomography (October 24, 2012) showed no alterations in vascular and mediastinal structures or signs of pulmonary thromboembolism; there was a confluent, diffuse, centrolobular micronodular infiltrate, in some areas with a tree-in-bud pattern, with a predominantly bronchocentric distribution, associated with areas of ground-glass opacity attenuation, more evident in the apices and posterior basal segments. These findings were considered compatible with an inflammatory or infectious process. There was bilateral pleural effusion, moderate to the right and small to left. (Figure 5)

The high-resolution chest tomography (November 1, 2011) disclosed lymphadenomegaly of para-aortic (2.7 x 1.3 cm)

Keywords

Young Adult; Heart Failure/physiopatology; Hypothyroidism; Cardiomyopathy, Hypertrophic Tobacco and Disorder; Hypercholesterolemia; Diagnostic Imaging; Inflammation; Pneumonia.

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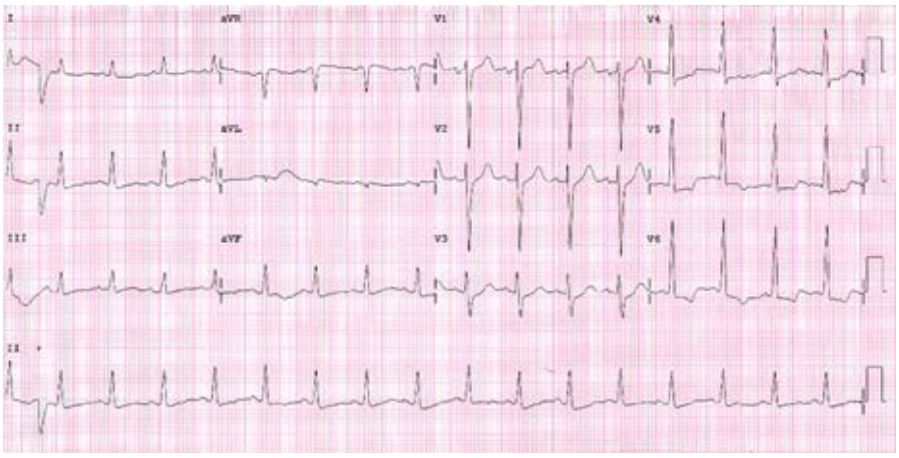

Figure 1 – *ECG: Sinus tachycardia, left chamber overload, intraventricular conduction disturbance of the stimulus, ventricular repolarization secondary alterations.*

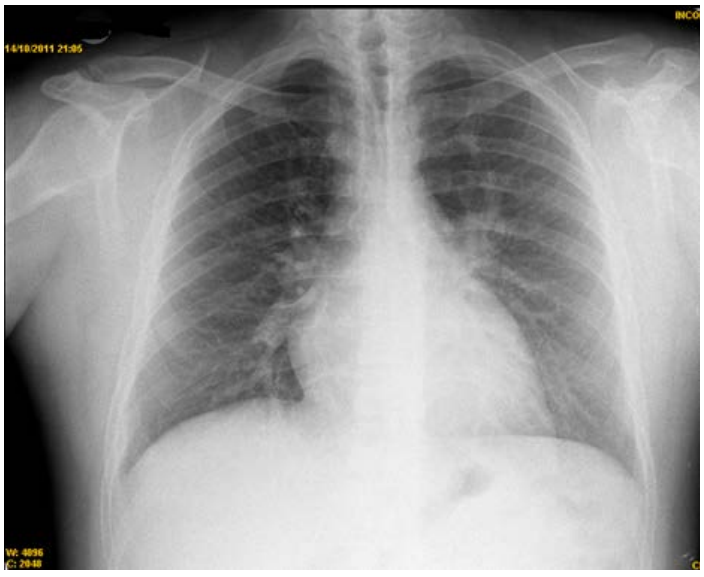

Figure 2 – *PA Chest X-ray: normal*

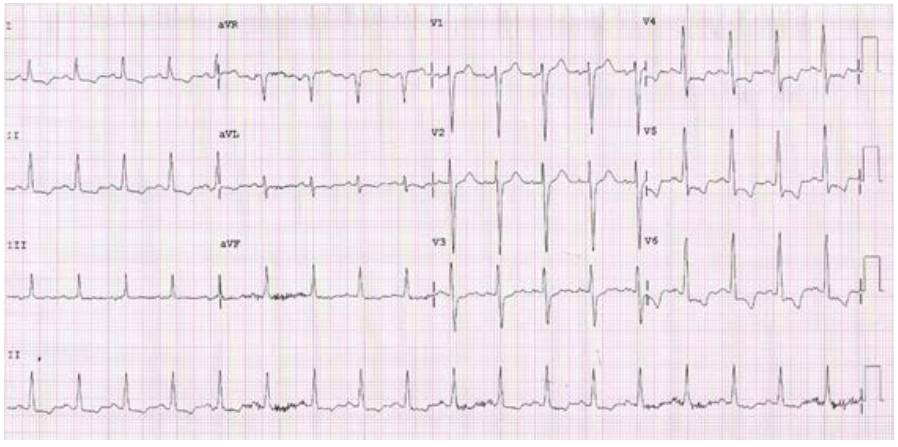

Figure 3 – *ECG: sinus tachycardia, left chamber overload, and ST-segment depression with superior concavity from V_3 to V_6.*

Anatomopathological Correlation

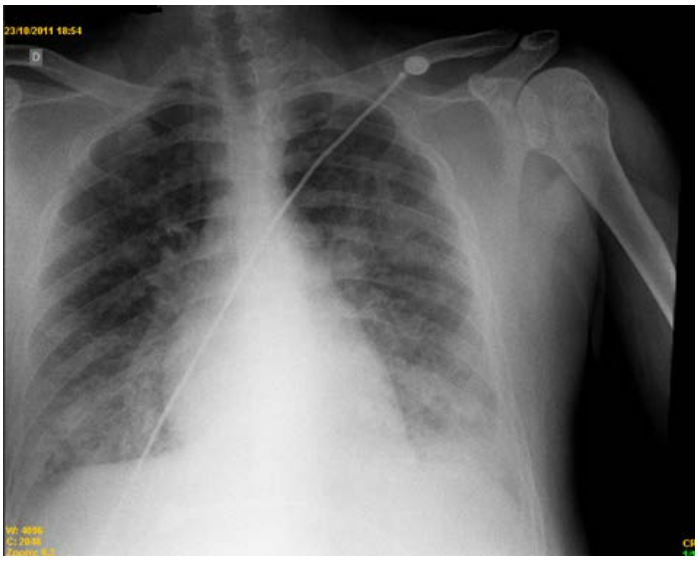

Figure 4 – *PA Chest X-ray PA: bilateral alveolar infiltrate.*

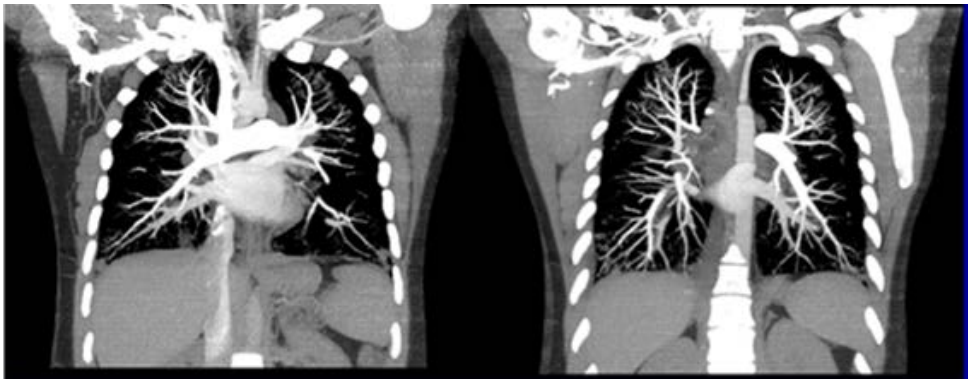

Figure 5 – *Pulmonary angiotomography showing no signs of thromboembolism.*

and right subcarinal (1.9 x 1.5 cm) chains; there were no tracheal and bronchial alterations; there was a predominantly centrolobular diffuse interstitial and alveolar infiltrate, at times confluent with interlobular thickening, more evident at the bases. There was also bilateral pleural effusion, moderate to the right. (Figure 6)

The echocardiogram (November 03, 2011) showed left atrial dilation and left ventricular mid-apical hypertrophy (18 mm), with intense trabeculation and obliteration of its tip, suggestive of endocardial fibrosis (Figure 7), and moderate mitral regurgitation, with signs of papillary muscle fibrosis and hypertrophy, with a left intraventricular gradient of 30 mmHg.

A new echocardiogram (November 11, 2011) disclosed left ventricular mid-apical hypertrophy, with intense trabeculation and obliteration of its tip, suggestive of endocardial fibrosis; ejection fraction subjectively estimated at 50% due to discrete apical hypokinesia. Mitral regurgitation was quantified as minimal in this echocardiogram and in subsequent ones (November 16 and 23, 2011).

The bronchial lavage (November 11, 2011) did not disclose the presence, by PCR, of *Pneumocystis carinii*, *Mycobacterium tuberculosis*, *Legionella sp*., Adenovirus, Herpes simplex or Cytomegalovirus. The cytology showed 115 cells/mm^3 (leukocytes 9% - 87% polymorphonuclear, 10% lymphocytes, 3% monocytes, 29% macrophages, 62% epithelial cells (7% flat, 21% cylindrical goblet, 72% cylindrical ciliated cells), and absence of bacteria and fungi.

The transesophageal echocardiography (November 23, 2011) did not disclose any new alterations (Table 1).

A new echocardiogram (December 07, 2011) disclosed a left ventricle with moderate to severe systolic function impairment (Table 1), and a hyperrefringent image was observed in the left ventricle, probably corresponding to the apical hypertrophy of the ventricular septum.

Due to lowering of consciousness level, a lumbar puncture was performed (June 29, 2004); the CSF analysis showed ADA (adenosine deaminase) of 2.5 U/L. Tests for Adenovirus, Cytomegalovirus, Herpes simplex, *Cryptococcus sp*,

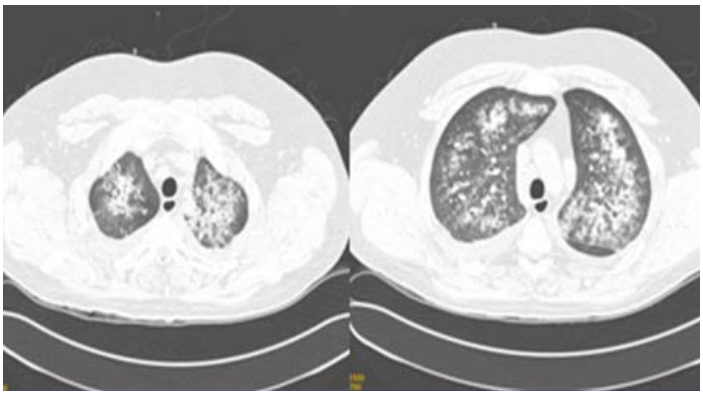

Figure 6 – *Chest CT: diffuse interstitial and alveolar infiltrate.*

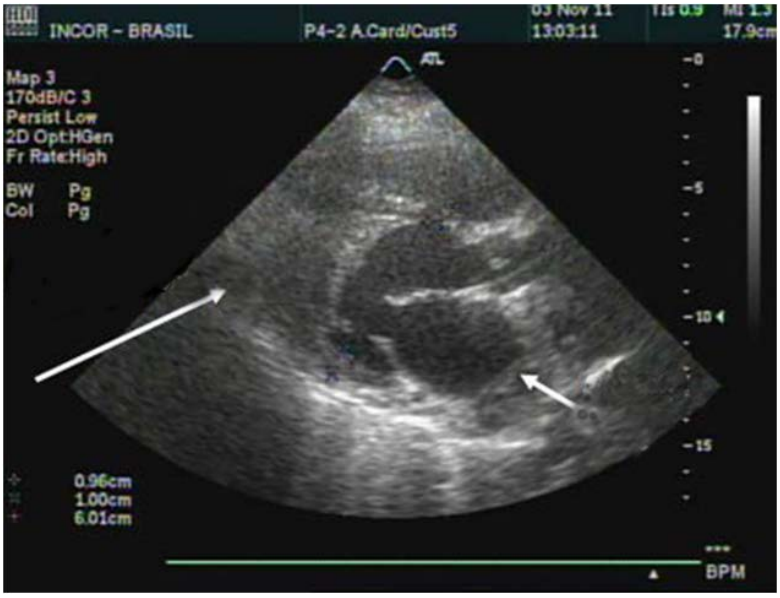

Figure 7 – *Parasternal, long-axis transthoracic echocardiogram: left atrium (short arrow) dilation and amputation of the left ventricular apex (long arrow).*

Table 1 – Echocardiograms at the last hospitalization

	Nov 3, 2011	Nov 11, 2011	Nov 23 (TE)	Dec 7, 2011
Aorta (mm)	25	-	32	
Left atrium (mm)	48	-	47	
Right ventricle (mm)	24	-	-	
Septum (mm)	10	-	12	
Posterior wall (mm)	10	-	10	
LV diastolic diameter (mm)	60	-	60	
LV systolic diameter (mm)	40	-	42	
LV ejection fraction VE (%)	61	50	55	35%
Pulmonary artery systolic pressure (mmHg)	-	30	40	

LV: left ventricle; TE: Transesophageal.

Anatomopathological Correlation

Table 2 – Laboratory evolution

	30 Oct	15 Nov	30 Nov	6 Dec
Red blood cells (millions/mm³)	4.3	2.8	2.2	3.1
Hemoglobin (g/dL)	12	7.9	6.4	9.3
Hematocrit (%)	37%	26	22	29
Leukocytes/mm³	26,210	9,650	9,210	26,970
Neutrophils (%)	91	97	87	78
Eosinophils (%)	0	0	0	0
Lymphocytes (%)	6	2	11	20
Monocytes (%)	3	1	2	2
Platelets/mm³	276,000	69,000	101,000	83,000
Creatinine (mg/dL)	1.36	2.98	4.94	2.64
Urea (mg/dL0	68	189	256	120
Sodium (mEq/L)	135	146	155	139
Potassium (mEq/L)	4.2	4.4	5.1	4.8
Lactate (mg/dL)	23	37	11	35
PT (INR)	1.2	1.1	1.3	1.2
APTT (rel)	0.86	0.85	1.04	0.98
CRP (mg/L)	15.90		124	109
AST (U/L)		90	37	
ALT (U/L)		211	359	

PT: prothrombin time; APPT: partially activated thromboplastin time; CRP: C-reactive protein; AST: aspartate aminotransferase; ALT: Alanine aminotransferase.

Toxoplasma sp and resistant acid-fast bacilli were negative and there was no growth of bacteria or fungi in the cultures. The cell count was 11 cells /mm³ - 31% lymphocytes, 66% monocytes and 3% macrophages; protein level was 475 mg/dL; glycorrhachia was 105 mg/dL.

Skull (November 29, 2011) and abdomen CT (November 30, 2011) showed normal results.

A new high-resolution chest tomography (November 30, 2011) disclosed persistence of the diffuse and symmetrical interstitial-alveolar infiltrate, characterized by diffuse ground-glass parenchymal attenuation and multiple nodular and micronodular opacities, predominantly centrilobular, sometimes confluent and delineating a tree-in-bud pattern, with a predominant distribution in the pulmonary medulla, compatible with alveolar filling. Further findings suggestive of inflammatory or infectious processes associated with edema or alveolar hemorrhage were described. There was no pleural effusion.

Blood culture was positive for *Staphylococcus haemolyticus*, sensitive to vancomycin and teicoplanin, and urine culture was positive for *Pseudomonas aeruginosa*, sensitive to piperacillin /tazobactam. A subsequent culture of urine disclosed growth of *Candida* non-albicans.

He was initially treated with vancomycin and piperacillin/tazobactam and, subsequently, imipenem and meropenem, teicoplanin, amphotericin, fluconazole, caspofungin, and acyclovir.

Due to the presence of signs suggestive of pulmonary alveolar hemorrhage and hematuria, there was a diagnostic suspicion of Goodpasture Syndrome and the investigation was initiated.

The search for neoplastic markers (July 10, 2004) disclosed - alpha-fetoprotein = 1.9 ng/mL, CA-125 = 401.4 U/mL, CA-15.3 = 14.9 U/mL, CA-19.9 = 20.1, carcinoembryonic antigen (CEA) = 2.7 ng/mL. The search for antinuclear and antimitochondrial antibodies, and antineutrophil cytoplasmic antigen (ANCA) was negative. The C3 fraction of the complement was 18 mg/dL, and the C4 fraction was 10 mg/dL. The evolutive results of the laboratory tests are shown in table 2.

The patient developed worsening of the pulmonary and hemodynamic picture, anemia (received packed red blood cell transfusion), thrombocytopenia and renal failure (was submitted to dialysis) and underwent a cardiorespiratory arrest with pulseless electrical activity; initially, he was successfully resuscitated, but had a recurrence and died (December 7, 2011).

Clinical aspects

This is the case of a young, male patient, smoker, who had hypothyroidism post-treatment with radioactive iodine, who sought medical care due to symptoms of dyspnea and cough for a week; these complaints had been preceded by chest pain at exertion two weeks before. The medical evaluation at the first consultation was notable for the presence of tachycardia and mild leukocytosis in the whole blood count.

The chest X-ray was normal despite the presence of dyspnea at rest. Interestingly, the ECG performed during a routine medical evaluation 11 months before symptom onset showed signs suggestive of heart disease, with left chamber overload.

If we evaluate the present case based on the analysis of the possible diagnostic hypotheses at the time of the first consultation, we can observe that the main clinical elements on this occasion are: chest pain and dyspnea. There are several causes of chest pain and dyspnea in young individuals, which include diseases of the cardiovascular system, as well as of other organs and systems such as the digestive and musculoskeletal systems.

In the present case, the following can be considered: as non-cardiac causes, spontaneous pneumothorax, pneumonia and pulmonary embolism. The clinical and radiological presentation was not compatible with the first two hypotheses, whereas pulmonary embolism is a hypothesis compatible with the initial clinical presentation, especially if we take into account the discrepancy between symptom intensity and the radiological findings and the presence of persistent tachycardia; in spite of the fact that pulmonary embolism is a rare event in young patients,[1] in this case ,there were risk factors such as history of smoking, as well as the presence of possible cardiopathy (as suggested by the ECG performed 11 months before symptom onset).

A complementary investigation was performed for the presence of pulmonary embolism through D-dimer measurement, and pulmonary artery angiotomography. However, after obtaining a D-dimer value < 500 in association with absence of suggestive radiological findings at the angiotomography made the diagnosis of pulmonary embolism very unlikely.[2] If we consider cardiac causes for chest pain and dyspnea in young individuals, myocarditis and non-atherosclerotic coronary disease should be seen as noteworthy. In spite of the fact that myocarditis is able to result in a clinical picture compatible with that of the patient's presentation, pre-existing electrocardiographic findings did not support these possibilities.

We can evaluate the present case based not only on the analysis of the symptoms that led the patient to the hospital, but also taking into consideration the interesting electrocardiographic findings recorded 11 months before symptom onset; thus, we are led to consider the case from the perspective of the diagnostic possibilities of asymptomatic cardiopathy in a young individual, and which may have left chamber overload as its electrocardiographic manifestation. We can presume the possibility of diseases with primary myocardial involvement (cardiomyopathies), as well as diseases that determine secondary myocardial involvement, such as arterial hypertension and valvular diseases. However, the blood pressure measurement on arrival, as well as the cardiac semiology, did not indicate these possibilities. Regarding the cardiomyopathies, those of familial origin should be considered mainly in this context and may include both hypertrophy (hypertrophic cardiomyopathy) and dilation (dilated cardiomyopathy) or restriction (restrictive cardiomyopathy) as the myocardial phenotypic expression.

In this respect, the morphological and functional findings provided by the echocardiogram are of interest. In the present case, the findings of October 17, 2011 indicate a slight increase in the interventricular septal thickness, with hypertrophy of the mid-apical portions of the left ventricle, determining LV filling impairment, but without determining ventricular dilatation or systolic function impairment. These findings might be compatible with the presence of hypertrophic cardiomyopathy, a genetic disease that affects young male and female patients; it is usually asymptomatic during the first decades of life and is commonly diagnosed during routine physical examinations.[3] Chest pain at exertion and dyspnea are common symptoms. Genetic studies indicate that the hypertrophy is caused by dominant mutations in more than 11 genes encoding sarcomere or adjacent Z-disk protein components. Of the patients who were successfully genotyped, approximately 70% had mutations in two genes: the myosin heavy chain (MYH7) gene and the myosin-binding protein C (MYBPC3) gene; more than 1,400 mutations have been described, most of them restricted to family groups. In patients with hypertrophic cardiomyopathy, the left ventricular wall thickness may vary in intensity, ranging from mild (13-15 mm) to very intense (> 50 mm).[4] Asymmetric patterns of left ventricular hypertrophy, including noncontiguous areas of hypertrophy, can occur. Although diffuse thickening of the left ventricular wall is evident in approximately 50% of patients, a minority (10-20%) may present hypertrophy confined to small portions of the left ventricle.[5] Moreover, patients with hypertrophic cardiomyopathy may have unusual patterns of hypertrophy (e.g., apical hypertrophy), which is associated with giant T-wave inversion on the ECG and is typically caused by sarcomeric mutations.[6]

Another possibility to be considered – especially if we take into account the mild ventricular overload in the ECG, the presence of apical obliteration, the absence of hypertrophy greater than 14mm at the echocardiogram and the presence of atrial dilatation – is that of endomyocardial fibrosis. This is a cardiac disease of uncertain etiology. Its distinctive morphological characteristic is the obliteration of the ventricular apices, ventricular filling impairment and great dilation of the atria. However, its clinical presentation is usually that of a chronic disease, with signs of predominantly right heart failure and large dilation of the atria, often with intracavitary thrombi, findings that were absent in the present case. Although its etiology is still unknown, it is suggested to be associated with three basic conditions: eosinophilia and parasitic diseases, nutritional patterns (excess of vitamin D, toxic agents found in contaminated foods and magnesium deficiency have been reported) and genetic susceptibility.[7] In cases where there is an association with pulmonary cycle parasitic agents, there may be involvement of the lungs, with non-cardiogenic pulmonary edema, pneumonitis, and alveolar infiltrate.[8] A hallmark of this condition is hypereosinophilia in the peripheral blood, a manifestation that was absent in the present case. Cardiac involvement has also been described in other hypereosinophilic syndromes, such as Churg-Strauss Syndrome (characterized by asthma or allergic rhinitis and necrotizing vasculitis).[9] In the present case, the possibility of Goodpasture syndrome, a specific autoimmune disease of the lungs and kidneys, was considered and is characterized by the occurrence of antibodies against the basement membrane of these organs. Cardiac involvement has not been yet described in this disease.[10]

Anatomopathological Correlation

Despite the initial diagnostic and therapeutic approach, the patient's symptoms intensified, and he once again sought medical care due to dyspnea worsening and the appearance of productive cough. On physical examination, tachycardia persisted and pulmonary crepitations with discreet edema of the legs and feet appeared.

The chest X-ray, as well as the first chest tomography, suggested the presence of alveolar infiltrate. The laboratory evaluation is notable for the decrease in hemoglobin and sodium, and BNP elevation. Taken together, the clinical findings indicate that the patient had started a picture of heart failure (presence of lower limb edema, anemia – possibly dilutional – hyponatremia, elevation of BNP and distribution of infiltrate at the apices and posterior portions of the lungs, in association with right pleural effusion). Moreover, the finding of young leukocyte forms in peripheral blood, of leukocyturia, hematuria and hemoglobinuria indicate the existence of an inflammatory and/or infectious process.

The presence of the association of heart failure with inflammatory signs in a patient with underlying heart disease led us to consider the possibility of infective endocarditis. The diagnosis of endocarditis is based on the presence of predisposing heart disease (more commonly a valvulopathy), findings of an inflammatory process and persistent bacteremia; from the clinical-morphological viewpoint, the characteristic lesion is of vegetation detected by the echocardiogram. Despite the findings of underlying heart disease and progressive inflammatory/infectious process, there was no finding of vegetation by the echocardiography; additionally, the finding of *Staphylococcus haemolyticus in* blood culture only has diagnostic value when recovered in multiple cultures collected at different times, because it is a skin-colonizing agent.[11] Finally, it should be noted that the use of multiple antibiotic agents may reduce the chance of recovery of infectious agents in blood cultures.[12]

From a clinical and epidemiological point of view, one of the main causes of infection and septicemia in patients with heart disease is pneumonia, of which clinical and radiological characteristics are compatible with the clinical evolution of the present case. As there was recent hospital admission, it is possible to consider the possibility of acquired pneumonia caused by nosocomial bacterial flora. In this regard, a study of necropsies performed in patients with heart disease found pneumonia as the most commonly found infectious diagnosis.[13] Moreover, in a study of 1,989 patients hospitalized for heart failure, the presence of pneumonia was a factor related to a worse prognosis, as was the intensity of the inflammatory process measured by the Protein C level in peripheral blood.[14] **(Dr. Victor Sarli Issa)**

Diagnostic hypotheses: Restrictive cardiomyopathy, smoking, hypothyroidism, acute decompensated heart failure, pneumonia, multiple organ dysfunction. **(Dr. Victor Sarli Issa)**

Necropsy

The heart weighed 516 g. The cross-section of the ventricles showed marked concentric myocardial hypertrophy and fibrous obliteration of the left ventricular cavity apex (Figure 8). The hypertrophic process predominated in the apical portion, with extensive organized and organizing thrombosis, which impaired both the left ventricular inflow (Figure 9) and outflow tracts (Figure 3), leading to marked reduction in the cavity volume. There was no subaortic obstructive septal hypertrophy. In the basal portion of the inflow tract, the left ventricular free wall measured 2.0-cm thick and the ventricular septum, 2.5 cm. In the mid-apical portion the left ventricular free wall measured 2.5 cm. The mitral valve showed cusps and chordae tendineae of normal aspect, but its papillary muscles were surrounded by the ventricular cavity thrombosis (Figures 9 and 10). The histological analysis of the myocardium showed focal areas of cardiomyocyte disarray and thick-walled arterioles in the left ventricle (Figure 11). There was irregular fibrous thickening of the tip of the endocardium

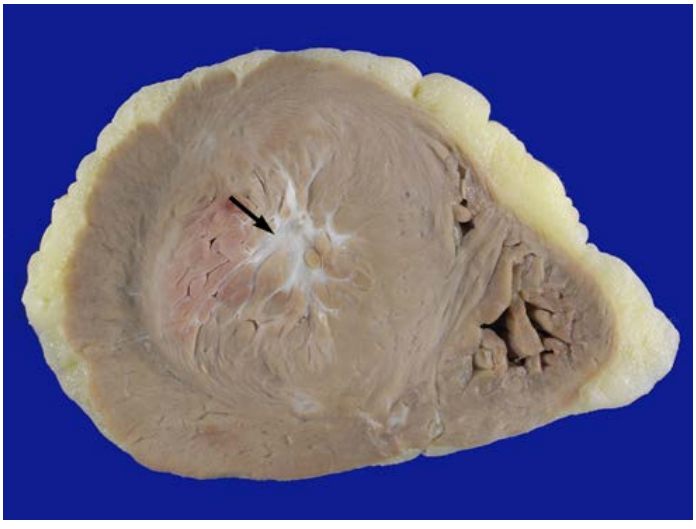

Figure 8 – *Cross section of the ventricles, showing the evident left ventricular concentric hypertrophy and the fibrous obliteration of the cavity apex (arrow).*

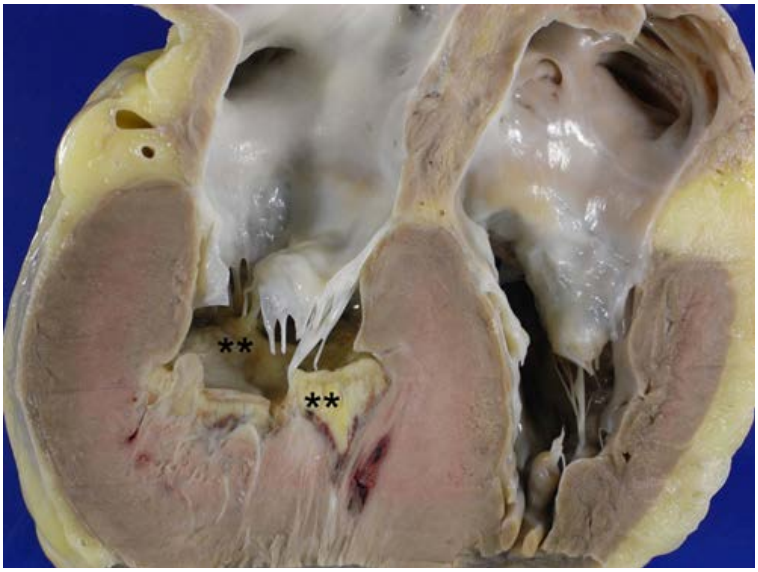

Figure 9 – *Longitudinal section of the heart, disclosing the ventricular inflow tract. The left cavity has decreased volume due to extensive organizing thrombosis, which surrounds the papillary muscles (asterisks).*

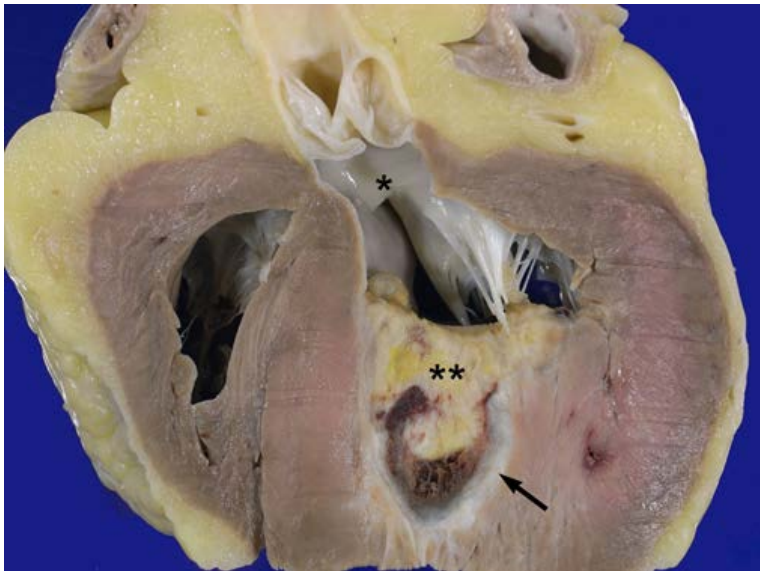

Figure 10 – *Extensive thrombosis in the left ventricular cavity (double asterisk) extending to the outflow tract. Note the whitish, fibrous thickening of the endocardium (arrow) and absence of hypertrophic obstruction of the subaortic region (asterisk).*

and mid-apical left ventricular region, with underlying myocardial penetration, as well as an extensive organized and organizing thrombosis (Figure 12). The endocardium close to the myocardium consisted of looser collagen, with neovascularization foci, hemosiderin deposition, and discrete mononuclear inflammatory infiltrate. Eosinophils were not detected. The lungs weighed 1,134 g together and showed marked chronic passive congestion, with septal thickening and hemosiderin deposition, as well as foci of fibrin extravasation into the alveolar spaces, rare fibrin thrombi in the parenchymal arterioles and extensive recent bilateral alveolar hemorrhage. There was no vasculitis. The kidneys showed acute tubular necrosis, with no glomerular lesions, thrombi or vasculitis. The thyroid weighed 8g, showing extensive atrophy with fibrous replacement of the parenchyma; the remaining follicles showed variable sizes and there were rare foci of lymphohistiocytic inflammatory infiltrate. Other necropsy findings were steatonecrosis of abdominal fat and centrilobular hepatic necrosis with cholestasis. **(Dr. Luiz Alberto Benvenuti)**

Anatomopathological diagnoses: Hypertrophic cardiomyopathy/endomyocardial fibrosis; chronic passive congestion of the lungs; thyroid fibrosis and atrophy; alterations secondary to cardiogenic hemodynamic shock (cause of death). **(Dr. Luiz Alberto Benvenuti)**

Anatomopathological Correlation

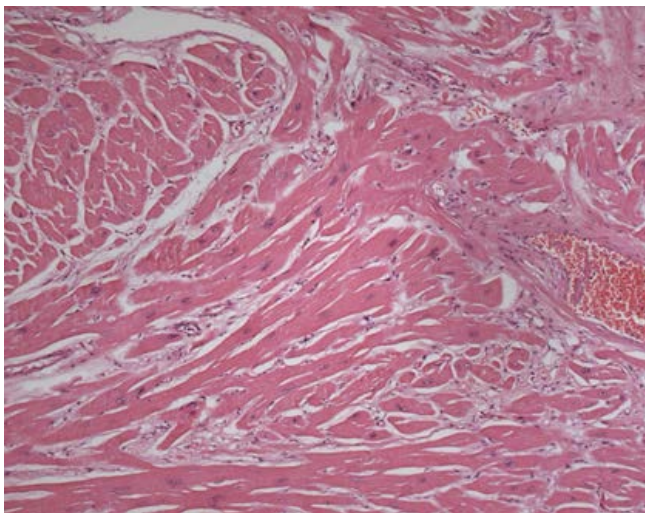

Figure 11 – *Left ventricular cardiomyocyte disarray. Hematoxylin-eosin, X100.*

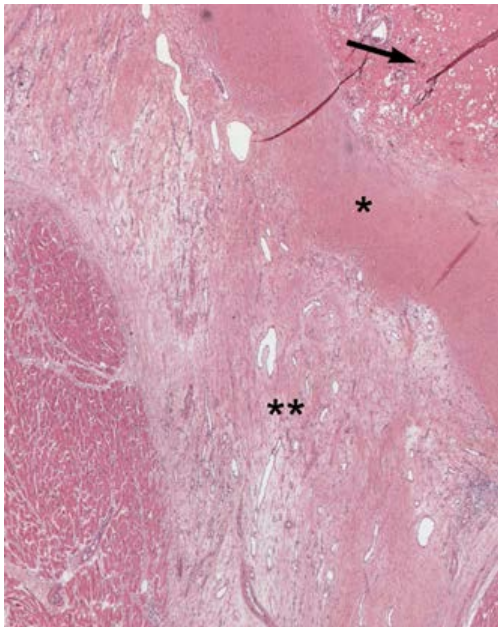

Figure 12 – *Histological constitution of the affected endocardium, compatible with endomyocardial fibrosis. There is luminal thrombosis (arrow), superficial area of dense fibrosis (asterisk) and underlying area of loose fibrosis with neoformed vessels and foci of discrete inflammatory infiltrate (double asterisk). Hematoxylin-eosin, X 25*

Comments

This is an interesting case of a 25-year-old male patient with a history of hypothyroidism after treatment with radioactive iodine at an unspecified date, who developed congestive heart failure and died after a year of clinical follow-up. Imaging tests disclosed significant hypertrophy of the left ventricular mid-apical region, with obliteration of the tip of the cavity, and the hypotheses of hypertrophic cardiomyopathy and endocardial fibrosis were suggested. The presence of suggestive signs of pulmonary hemorrhage and hematuria raised the suspicion of Goodpasture syndrome, but the complementary exams were not suggestive of this entity.

The necropsy showed it was a case of cardiomyopathy of unusual pattern, characterized by the superposition of findings of hypertrophic cardiomyopathy and endomyocardial fibrosis. On the other hand, the findings were not typical of any of these diseases alone. Although there was marked left ventricular wall concentric hypertrophy, with apical and mid-mural predominance related to the hypertrophic cardiomyopathy, the areas of cardiomyocyte disarray, which constitute the most significant finding of the disease, did not occur in extensive areas, as usual.[15] Regarding the endomyocardial fibrosis, there was fibrous obliteration of the left ventricular apex, the typical histopathological constitution of the affected endocardium, and

cavity thrombosis at different development stages, both the left ventricular inflow and outflow tracts were affected, which is not described in endomyocardial fibrosis and typically affects only the ventricular apex and inflow tract.[16] It is noteworthy that we have previously reported the simultaneous occurrence of both cardiomyopathies, with typical findings, in a patient who underwent surgical resection of endomyocardial fibrosis and subsequently died.[17] The present case illustrates the difficulty to classify the cardiomyopathy into one of the four traditional basic types, namely: dilated, hypertrophic, restrictive, and arrhythmogenic,[18] and there is a current trend towards a purely descriptive classification, i.e., the MOGE(S) classification.[19]

Regarding the suspected Goodpasture syndrome, the necropsy did not show lesions in the renal glomeruli or evidence of vasculitis in the lungs or other organs, and the alveolar pulmonary hemorrhage can be explained by the heart failure and terminal cardiogenic shock, which is the cause of death. Therefore, there are no anatomopathological elements to allow the diagnosis of Goodpasture syndrome, which is in line with the results of the performed complementary tests. The thyroid atrophy corresponds to the sequela of the radioactive iodine treatment of the patient's hyperthyroidism. **(Dr. Luiz Alberto Benvenuti)**

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