Anatomopathological Session

Case 5/2014 - 41-Year-Old Woman with Rheumatic Disease and Previous Mitral Valve Repair with Pulmonary Embolism and Cardiogenic and Septic Shock

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A 41-year-old female sought medical care due to severe dyspnea. The patient had had acute rheumatic disease in childhood. During evolution, she developed mitral stenosis. The symptoms became incapacitating and she underwent mitral commissurotomy at 36 years. She progressed well for a few years until dyspnea recurred and she was once again submitted to surgery, at 41 years, when she underwent mitral valve plasty (03/16/2005).

After the last surgery, she had dyspnea on great exertion for about three months, when it progressed and started to be triggered by middle, and finally by mild exertion, and at three days before hospitalization (10/10/2005), it had become present even at rest. The patient attributed the recent worsening to current medication discontinuation: captopril 25 mg, furosemide 80 mg, 0.25 mg digoxin and warfarin 2.5 mg daily.

Physical examination (10/10/2005) showed the patient was in good general health, dyspneic, with a marked increase in jugular venous pressure, pulse rate of 92 bpm, blood pressure of 100/60 mmHg. Lung examination was normal. Cardiac auscultation showed irregular rhythm without additional heart sounds. Systolic murmur +/4+ was diagnosed in the mitral valve area. There were no alterations at the abdominal examination, but slight edema of the lower limbs.

Laboratory tests (10/10/2005) showed hemoglobin 11.7 g/dL, hematocrit 35%, WBC, 3,900/ mm³, platelets, 12.9000 /mm³, creatinine 1.3 mg / dL, urea 31 mg / dL, sodium 135 mEq/L, potassium 3.6 mEq/L, INR 1.19 and activated partial thromboplastin time (patient / control) 1.09.

The electrocardiogram (10/10/2005) showed frequency of 90 bpm, atrial fibrillation, low voltage QRS complex, undetermined QRS axis in the frontal plane and presence of

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intraventricular stimulus conduction disturbance, of the right branch type and decreased left ventricular potential, suggesting right ventricular overload (Figure 1).

The patient was admitted for treatment. She remained in the emergency unit for five days and was admitted (on October 15, 2005). She received furosemide 120 mg intravenously, 40 mg of enalapril, 0.25 mg of digoxin, 50 mg of hydrochlorothiazide and 120 mg of enoxaparin daily by subcutaneous route, as well as dobutamine 10 μg/kg.min intravenously.

At hospitalization she had hypotension, increased edema and creatinine elevation (Table 1). After three days, the patient developed anuria, anasarca and finally shock with hypotension with 60 mmHg despite the use of 15 μg/kg.min of dobutamine.

The laboratory tests (10/20/2005) showed creatinine 3.2 mg/dL and then 5.9 mg / dL, Urea 75 mg / dL and, during evolution, 115 mg / dL (Table 1).

At physical examination (10/20/2005) the patient was in poor general condition, with blood pressure of 80/50 mmHg, heart rate 90 bpm, crackles in both lungs, arrhythmic heart sounds (atrial fibrillation), systolic murmur + / 4 + in the mitral area, ascites and edema + + + + / 4+.

The electrocardiogram (20/10/2005) showed atrial fibrillation, heart rate of 100 bpm, low QRS voltage, intraventricular conduction disturbance of the right bundle branch block type stimulus, decreased left ventricular strength (Figure 2).

The echocardiogram (on October 21) showed normal left ventricle, dilated and hypokinetic right ventricle, mitral valve calcification, commissural fusion, moderate stenosis and moderate tricuspid regurgitation (Table 2). Transesophageal echocardiogram (on October 21) showed pulmonary artery dilation with a large thrombus image (10 × 5.0 cm) extending to its left branch and the presence of autocontrast in the left atrium.

The diagnosis of pulmonary thromboembolism was made and 100 mg of r-TPA was administered intravenously in two hours. The patient went into shock, which required vasoactive drugs. Intravenous norepinephrine was administered, associated with ceftriaxone and metronidazole, as well as vancomycin for empiric treatment of the systemic infection. Mechanical ventilation was initiated with tracheal intubation for ventilatory support.

Pulmonary arteriography (on October 24) showed pulmonary artery pressures of 30/15/22 (systolic/diastolic/mean) mmHg. No images suggestive of pulmonary thromboembolism were identified.
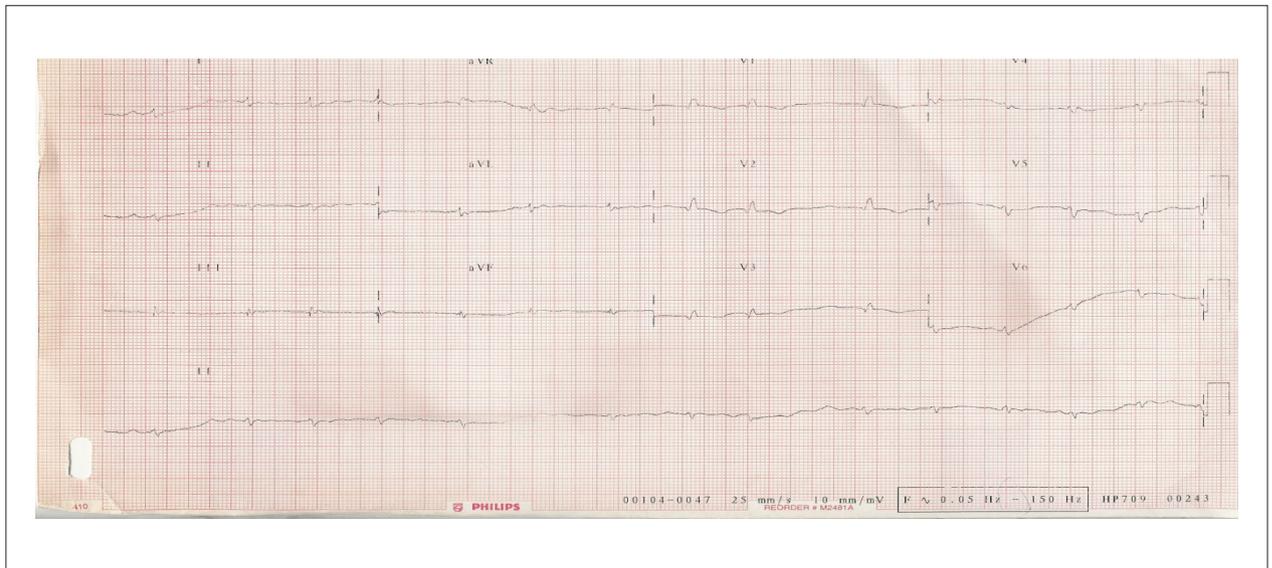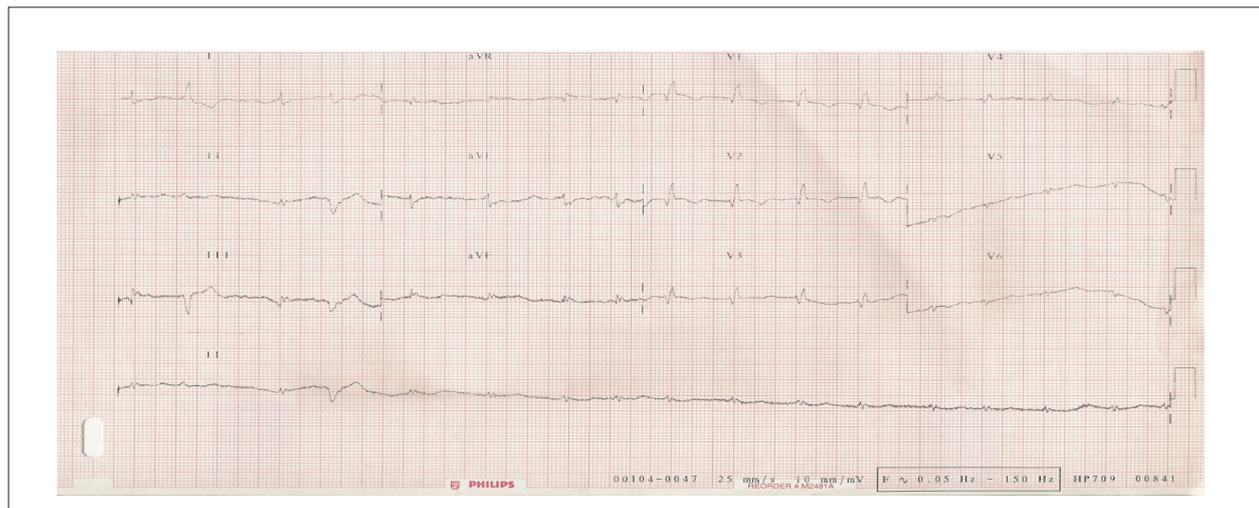
Figure 1 – Atrial Fibrillation, low voltage QRS complexes, right bundle branch block, low voltage of left QRS complexes, right ventricle hypertrophy.

Table 1- Laboratory assessment

	17 Oct	20 Oct	26 Oct
Urea (mg/dL)	75	115	97
Creatinine (mg/dL)	3.2	5.9	5.8
Sodium (mEq/L)	135	136	142
Potassium (mEq/L)	3.6	4.5	4.3
Hemoglobin %	12.2	11.3	8
Hematocrit (g/dL)	37	33	27
MCV (µm³)	97	97	108
Leukocytes/mm³	4.400	6.200	13.400
Neutrophils (%)	67	74	82
Eosinophils (%)	5	3	0
Basophils (%)	1	1	0
Lymphocytes (%)	11	8	13
Monocytes (%)	16	14	5
Platelets/mm³	186.000	17.500	22.8000
PT (INR)	1.1		2.41
aPPT (rel)	1.27		Incoagulable
Uric acid (mg/dL)		12.1	
AST (IU/L)		12	
ALT (IU/L)		12	
FAALP (N <120 U/L))		170	
Gama GT IUL (N <28)		55	
Phosphorus (mg/dL)			8.4

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Table 2 - Echocardiograms

	Oct 21	Oct 25
Interventricular septum (mm)	10	10
Posterior wall (mm)	9	9
Left Ventricle		
Diastole (mm)	52	52
Systole (mm)	35	37
EF (%)	60	55
LV Mass (g/m²)	150	120
LV segmental motility	Normal	Diffuse hypokinesis, worse in septal and anterior segments
Aorta (mm)	29	25
Left atrium (mm)	60	56
Right Ventricle (mm)	23	37
Segmental Motility	Moderate diffuse hypokinesis	Moderate diffuse hypokinesis
Right atrium	Normal	Increased
Mitral valve	Moderate stenosis	Moderate stenosis
Valve area (cm²)	1.4	1.4
Tricuspid valve	Moderate insufficiency	Marked insufficiency

Figure 2 – Atrial Fibrillation, low voltage QRS complexes, right bundle branch block, low voltage of left QRS complexes in horizontal plane, right ventricle hypertrophy.

The patient had an abundant epistaxis episode, which required transfusion of fresh plasma.

CT scan of the skull (on October 24) showed a hypoattenuating nodular area in the caudate nucleus head to the left, with no other alterations.

Blood cultures (10/25/2005) showed the presence of *A. baumannii* (sensitive only to imipenem). Hemodialysis (on October 26) was not tolerated by the patient, due to hypotension, and could not be performed.

The patient developed shock and died (10/26/2005).

Clinical aspects

This is 41-year-old patient with a history of rheumatic disease who developed mitral stenosis and underwent mitral commissurotomy at age 36 due to the presence of incapacitating symptoms. In our country, the combination of mitral stenosis with rheumatic disease is quite common[1]. About 25% of all patients with rheumatic disease have isolated mitral stenosis; 40% have double mitral dysfunction[2]. The mean time interval between the initial acute onset and the appearance of symptoms can vary from a few to more than 20 years.

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The presence of symptoms of heart failure (classes III and IV of the New York Heart Association), together with echocardiographic data that confirm significant anatomic lesion, is crucial for intervention indication: balloon valvuloplasty or surgery (commissurotomy or valve replacement). Whenever possible, there is an attempt to correct the valve defect, keeping the patient's valve system, postponing prosthesis implantation. In this case, commissurotomy was performed, which maintained the patient well for approximately five years, when she started to present symptoms again, when mitral valve repair was performed. This evolution in the rheumatic patient can occur due to repeated episodes of valvulitis, hence the need to maintain secondary prophylaxis with benzathine penicillin in patients with cardiac involvement, preferably throughout life or up to the fifth decade, when it is not possible[1].

After the last surgical intervention, the patient remained asymptomatic for a short time, with dyspnea recurrence that developed into striking symptoms in about three months. The deterioration was attributed to drug discontinuation which, in our country, is a common cause of heart failure decompensation, regardless of the etiology.

On admission, the patient had respiratory distress with clean lungs, irregular heartbeat without incidental heart sounds, minor systolic murmur in the mitral area and mild lower-limb edema. These findings point to a syndromic diagnosis of right heart failure. The normal pulmonary symptomatology and the absence of additional heart sounds do not indicate left ventricular dysfunction the cause of decompensation. The irregular rhythm suggests uns atrial rhythm, which may be atrial fibrillation, a common association with mitral valve disease together with large atriums.

The patient's initial laboratory tests did not exhibit significant alterations. The electrocardiogram (ECG) confirmed the presence of atrial fibrillation and alterations compatible with right ventricular overload, corroborating the aforementioned physical examination. Moreover, it showed low voltage complexes. The so-called dielectric effect is defined by the presence of QRS complexes with an amplitude < 0.5 mV in the frontal plane leads and < 1 mV in the precordial plane. The etiology is varied, including extracardiac factors (obesity, chronic obstructive pulmonary disease, hypothyroidism), pericardial diseases (pericardial effusion, constrictive pericarditis) and intrinsic myocardial diseases (rheumatic myocarditis, restrictive cardiac syndromes, arrhythmogenic right ventricular dysplasia).

The patient's initial treatment was directed to heart failure due to systolic dysfunction, consisting of angiotensin-converting enzyme (ACE) inhibitors, diuretics, digitalis and full heparinization due to atrial fibrillation, considering the risk for thromboembolic events. After hospitalization, the patient developed low cardiac output syndrome with hypotension, convergent blood pressure and worsening of renal function, despite the use of inotropic agents (dobutamine). Moreover, there was worsening of the congestive symptoms, with worsening of edema and crackles in both lung. Given this clinical picture, the differential diagnosis includes diseases that present with predominantly right heart failure, leading to shock.

The most likely hypothesis is pulmonary thromboembolism (PTE). In the case of PTE, it would be possible to explain the clinical, electrocardiographic and evolution alterations ("shock with clean lungs"). It should be noted that the patient had risk factors for PTE, with heart failure, atrial fibrillation and valvular heart disease, plus the fact that this disease is responsible for approximately 15% of decompensated heart failure.

Echocardiography was crucial for the patient's diagnosis. The valvular dysfunction with an area of 1.4 cm^2 would hardly justify the patient's clinical picture alone, or her evolution, considering the undertaken measures. The clear signs of right ventricular dysfunction, with evidence of large thrombus in the pulmonary artery, corroborate the clinical picture, pointing to the diagnosis of PTE. The pulmonary hypertension in this case can be a consequence of mitral valve disease as well as the PTE.

The differential diagnosis for the image of a large thrombus located in the pulmonary artery is the pulmonary artery sarcoma, or metastatic squamous cell tumor. Of these, the most frequent diagnostic error of pulmonary embolism is the pulmonary artery sarcoma. It is a rare tumor of the cardiovascular system, originated from the dorsal area of the pulmonary artery trunk or the right or left pulmonary arteries. Due to the insidious growth and the rarity of presentation, it is often inappropriately treated as PTE[3]. However, this possibility becomes unlikely in this clinical case, due to failure in identifying the lesion on the pulmonary arteriography.

Another differential diagnosis, when evaluating the presence of atrial fibrillation with thromboembolic phenomena associated with the dielectric effect on ECG, is cardiac amyloidosis. However, it has low clinical suspicion when one analyzes the history of the disease, as well as the echocardiographic results and subsequent clinical course.

The use of thrombolytic therapy has consensual indication in this case, considering the clinical signs of thromboembolic event. It is classified as massive PTE when there is hemodynamic instability. The therapy rationale is the thrombus dissolution, decreasing the right ventricular overload and the pulmonary artery pressure levels. There are reports in the literature on the acute resolution of large thrombi, decreasing the mechanical obstruction of the right ventricle[2]. However, the migration of thrombus fragments distally can impair the success of thrombolysis and the expected outcome in relation to clinical evolution might not be attained. In fact, although indicated, there is no evidence of reduction in mortality with the use of thrombolytic agents in cases of massive PTE[4].

The patient's unfavorable evolution, although the arteriography did not disclose a thrombus in the pulmonary artery system, leads us to reflect on what else contributed to the poor outcome. Here we face some relevant points.

The first is the fact that the patient developed coagulopathy followed by evident bleeding. The normal coagulation at admission leads us to a diagnosis of acquired coagulopathy. The thrombolysis carried out in the PTE treatment certainly played a role in the etiology of the coagulation disorder. Moreover, as we will see below, the patient developed

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bacterial infection and there may have been disseminated intravascular coagulation secondary to sepsis. There are no reports of other documented bleeding, in addition to epistaxis, but the sharp decrease in hemoglobin levels associated with the incoagulable activated partial thromboplastin time (APTT) suggests active bleeding. Thus, hemorrhagic shock together with the clinical picture is among the possibilities and it might be related, in addition to the epistaxis, to the vascular access complication.

The second point is related to infection confirmed by blood cultures positive for *Acinetobacter baumannii*. A mixed shock (cardiogenic and septic) justifies the patient's poor prognosis and her refractoriness to the measures that were undertaken. In recent years, there has been an increase in the resistance of *Acinetobacter baumannii* to broad-spectrum antibiotics. This has coincided with the increased incidence of sepsis by this agent[5]. Risk factors associated with sepsis by *Acinetobacter* are: prior use of broad-spectrum antibiotics, use of urinary catheters, mechanical ventilation and previous surgery. The mortality in these cases is around 38%.

The main factors of poor prognosis related to sepsis by *Acinetobacter* are the use of inadequate antibiotics and mechanical ventilation[5]. This patient had both factors.

The third point that draws attention to this case is the worsening of left ventricular function, as demonstrated in the patient's last echocardiogram. Some possibilities can be suggested: myocardial depression in sepsis, rheumatic myocarditis and coronary thromboembolism. Rheumatic myocarditis results from an immune cellular process and therefore may occur without humoral manifestations, such as arthritis and chorea. It is usually associated with valvulitis and has a transitory character. It can be observed, in this case, that there is rheumatic disease activity, considering the early post-valvuloplasty dysfunction. Interleukin-4 appears to play a critical role in modulating local immune response due to its anti-inflammatory properties[1].

Myocardial depression in sepsis can be found in approximately 40% of septic patients due to several factors, including reduction of coronary flow, myocardial edema, direct action of cytokines (IL-1, TNF-alpha) and of nitric oxide, leading to reduced levels of intracellular calcium[6]. Both myocarditis and myocardial depression in sepsis usually involve the myocardium as a whole, not focusing on specific territories. There are cases, however, when these conditions may mimic myocardial infarction. This patient had diffuse left ventricular hypokinesis, but more pronounced in the anterior and septal regions.

Based on this information, one can consider myocardial ischemia as a possible diagnosis. Coronary lesions, even non-obstructive ones, may lead to myocardial ischemia due to hypoperfusion secondary to shock, sometimes culminating in myocardial infarction (currently classified as myocardial infarction type 2)[7]. Another possibility is coronary embolism as a result of systemic thromboembolic phenomenon secondary to atrial fibrillation.

In fact, there have been reports of this kind in the literature, involving both the right coronary artery as well as the anterior descending artery. It is noteworthy that, despite evidence

of spontaneous contrast in the left atrium, the presence of thrombus in the left atrium was not demonstrated. However, this fact does not exclude the hypothesis. Another possibility is coronary embolism resulting from paradoxical embolism. The present of patent foramen ovale is frequent, being estimated at about 15-20% of normal individuals. Thrombus in the venous system, right-left shunt, increased pressure in the right system and systemic embolism are conditions that make the diagnosis likely[8]. The patient had at least three such conditions. The fact that the echocardiogram did not identify the presence of patent foramen ovale can be a result of low sensitivity to identify this condition. However, this patient had already undergone two heart surgeries with valve manipulation, making this diagnosis unlikely.

As a last point, we have kidney failure, which progressed during patient evolution, with hemodialysis being indicated. This clinical picture can be easily explained by the mixed shock. However, one cannot rule out a possible thromboembolic etiology.

The patient died due to refractory shock, not tolerating dialysis. Considering what was discussed, we suppose that the patient did not adequately carry out the secondary prophylaxis of rheumatic fever, since the evolution of post-valvuloplasty. She had decompensated heart failure, related to medication discontinuation and pulmonary thromboembolism. She had an episode of massive PTE during hospitalization, which, despite adequate therapy, developed unfavorably. This evolution is due to sepsis by *Acinetobacter* and myocardial dysfunction, which may be related to sepsis and / or possibly acute myocardial infarction by coronary thromboembolism (Dr. Eduardo Gomes Lima, Dr. Ricardo D'Oliveira Vieira, Dr. Paula Bombonati).

Diagnostic hypothesis: Chronic rheumatic mitral valve disease, post-valvuloplasty mitral dysfunction, pulmonary thromboembolism and mixed shock (cardiogenic, septic) **(Dr. Eduardo Gomes Lima, Dr. Ricardo D'Oliveira Vieira, Dr. Paula Bombonati)**

Necropsy

The heart weighed 630 g (normal weight for women is between 250-300 g), with mild hypertrophy and moderate left atrium dilation, with marked thickening of the endocardium (Figure 3). Seen from the atrial side, the mitral valve had the "fish mouth" aspect with reduced opening, commissural fusion and severe thickening of the cusps (Figure 3). There was also mild multifocal calcification and evidence of previous valve surgery as shown by the presence of surgical stitches in almost the entire valve circumference, largely included in the valve tissue and surroundings (signs consistent with prior valvuloplasty - Figure 3). From the ventricular side, the valve apparatus showed marked deformity and shortening, represented by cords exhibiting severe thickening, fusion and retraction (Figure 4).

At handling and maneuvering with water flow, valve mobility and cusp coaptation were significantly impaired, suggesting double valve lesion with stenosis greater than regurgitation as functional alterations. In the aortic valve, the semilunar showed diffuse thickening and mild collapse,
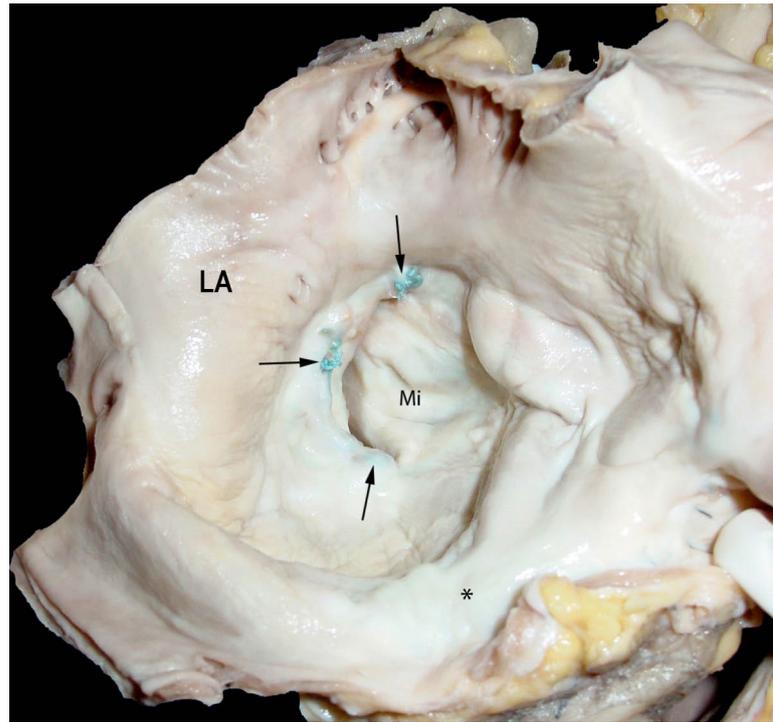
Figure 3 - *Photograph of the opened left atrium (LA), showing mild hypertrophy and moderate dilation. Note the endocardial thickening characterized by its whitish color (asterisk). The mitral valve (Mi), with double lesion, shows commissural fusion, characteristic of rheumatic disease, and the posteromedial one is evident in the photo. Note also the "fish mouth" opening, without adequate cusp coaptation. The arrows indicate the points of the previous valve surgery (valve repair).*
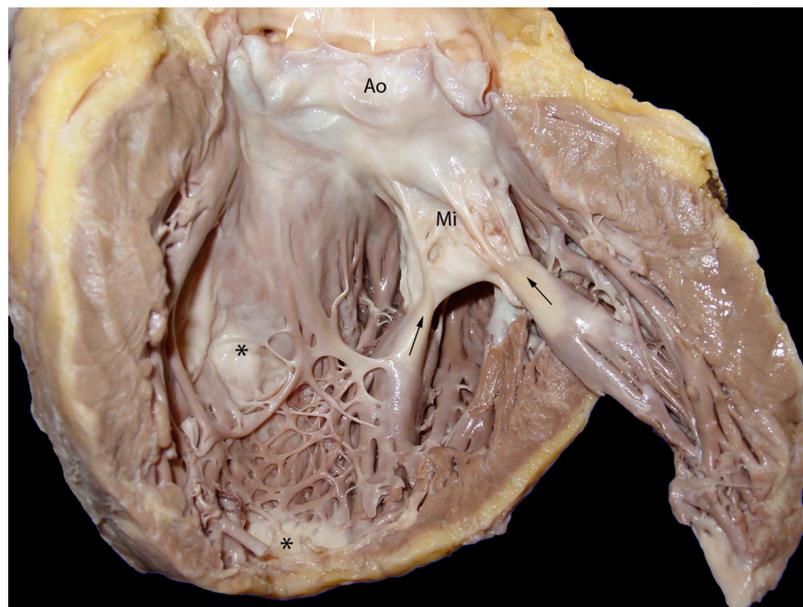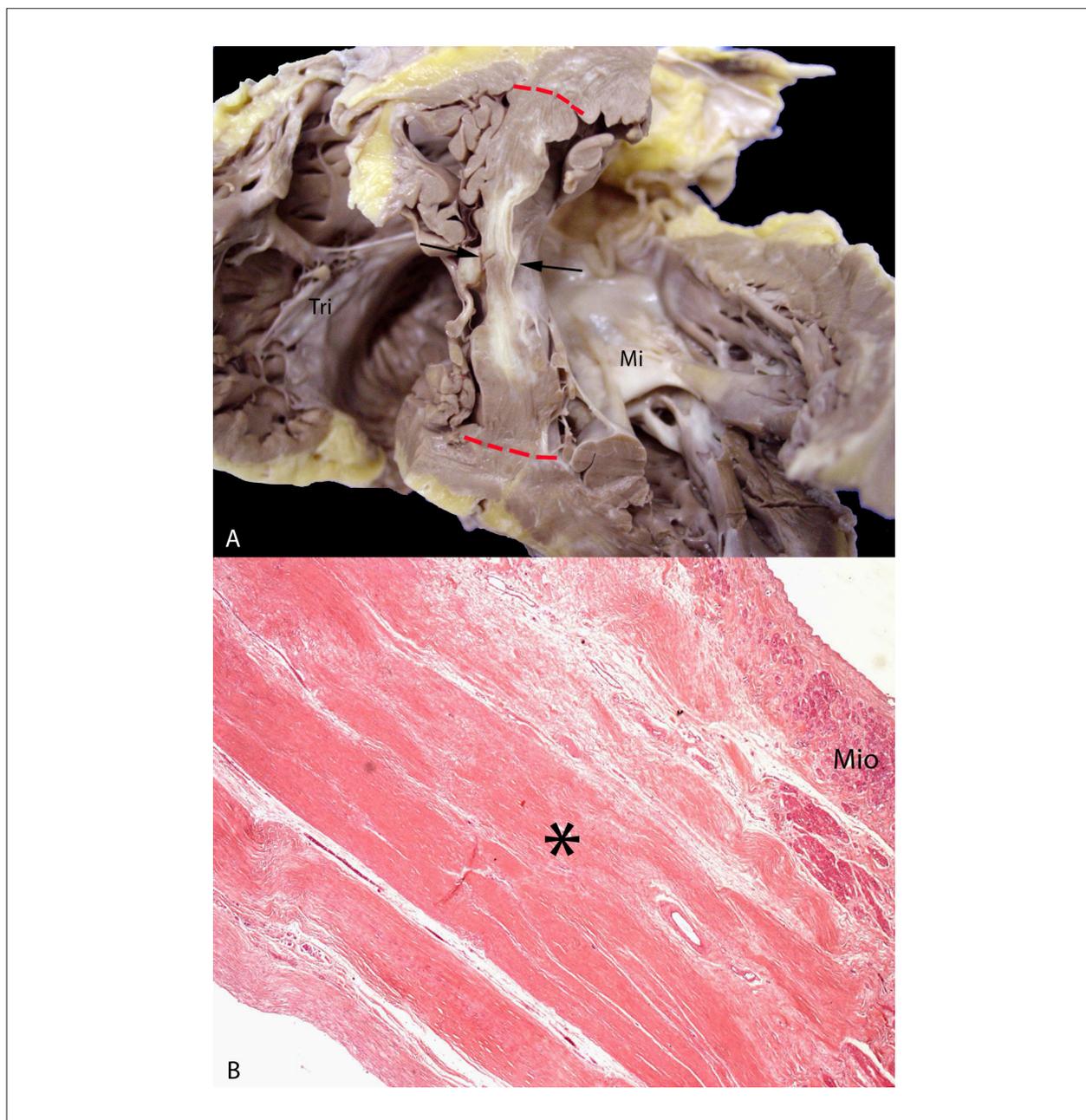

Figure 4 - Photograph of the open heart through the left ventricular outflow tract (LVOT). Note the thickened anterior cusp of the mitral valve (Mi), with intense fusion and cord retraction, quite characteristic of rheumatic disease. At the top, the aortic valve shows mild thickening and retraction of the semilunar, exposing the aorta below the aortic bar (white arrows). Two whitish areas in the septum and LV tip (asterisk) correspond to septal infarction and endocardial fibrosis, respectively.

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indicating valve insufficiency. The left ventricle was moderately dilated and hypertrophied, showing an area of fibrous scar in the median septum and endocardial tip (Figure 4). The sections showed septal wall thinning with transmural replacement of heart muscle by fibrotic scarring (Figure 5), affecting approximately 10% of the left ventricular muscle mass. On the right chamber side, there was marked atrial dilation and in the ventricle, mild hypertrophy and moderate dilation (Figure 6).

The tricuspid valve suggested intense insufficiency, secondary to mild thickening of the cusps with discrete fusion and retraction of the cords, as well as annulus dilation (Figure 6). Macroscopic and microscopic examination revealed no obstructive coronary lesions, but only mild intimal thickening (Figure 7). The central pulmonary arteries and trunk showed no macroscopic alterations.

Upon examination of the other organs, we detected alterations of chronic passive congestion in the lungs (already

Figure 5 - *Photograph of the open heart cut transversally at the median height of the interventricular septum (between the dotted lines in red). Note, in A, the thinning of the wall (between arrows) with substitution by off-white fibrous tissue. B. Histology of the septum: there is little remain of the septal myocardium (Myo), which was largely replaced by fibrous scar tissue (asterisk), characterizing the healed transmural septal infarction. (Hematoxylin and eosin; 2.5 x magnification.)*
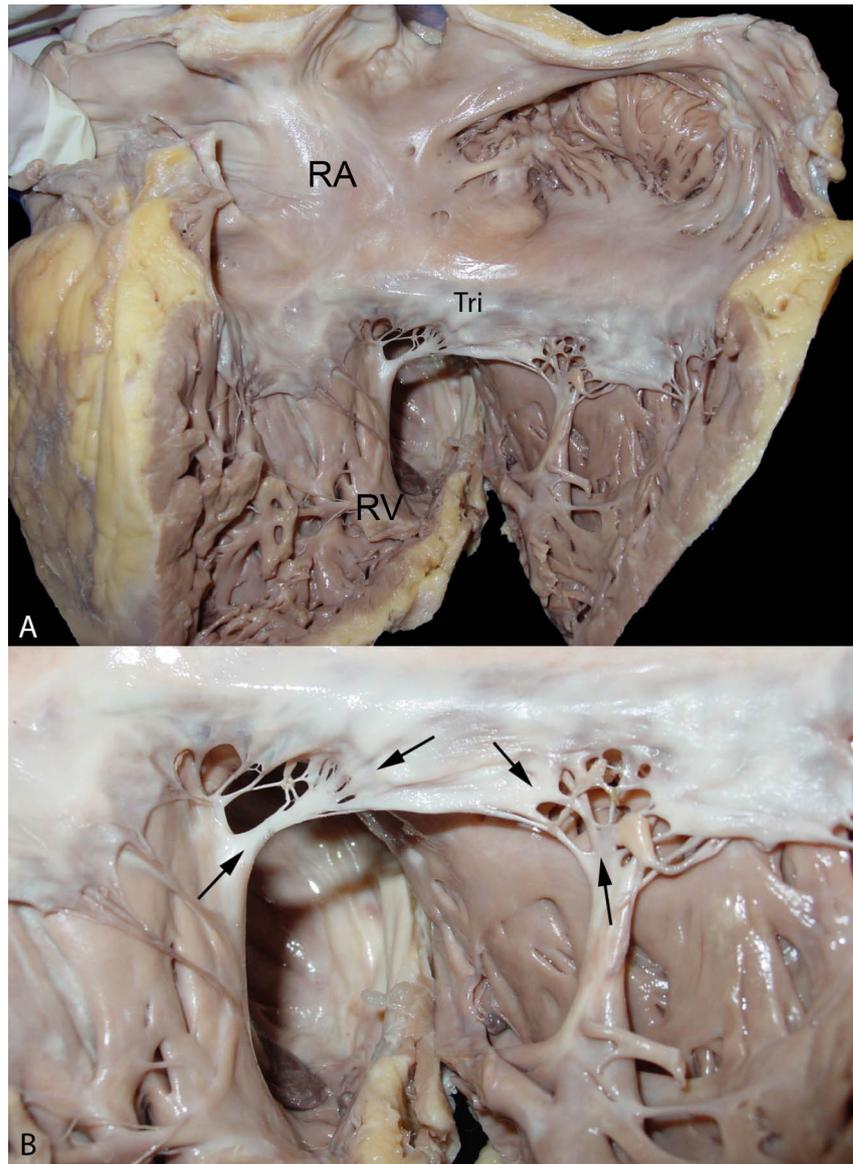
Figure 6 - *Photograph of the open heart through the right ventricle (RV) inflow tract. Note in A, dilation of the cavities of the right atrium (RA) and right ventricle (RV), the latter also showing hypertrophy. The tricuspid valve (Tri) shows intrinsic insufficiency (by focal thickening and shortening of the cusp and cords) and also by secondary annulus dilatation. B. Detail of the tricuspid valve where the arrows indicate the alterations described.*

showing passive pulmonary hypertension) and liver, as morphological substrate of overall congestive heart failure associated with valvular heart disease. The right lung showed extensive lobar area with hardening and hemorrhage, macroscopically indicating a heart attack or "red-gray hepatization", with lobar pneumonia being subsequently characterized through histology. This infectious picture was also associated with the presence of acute pyelonephritis, represented by multiple cylinders of polymorphonuclear neutrophils in renal pyramids, which also infiltrated the interstitium. The spleen was enlarged (250 g, normal weight

is approximately 150 g) at the expense of the red pulp, showing an acute splenitis pattern (Figure 8). Morphological changes related to shock, such as acute renal tubular necrosis, hepatic centrilobular necrosis and cerebral edema with herniation of the cerebellar tonsils were also observed and thus, septic shock was considered as the immediate cause of death. **(Dr. Jussara Bianchi Castelli)**

Anatomopathological diagnoses: Chronic rheumatic mitral-aortic-tricuspid valve disease; valvular heart disease

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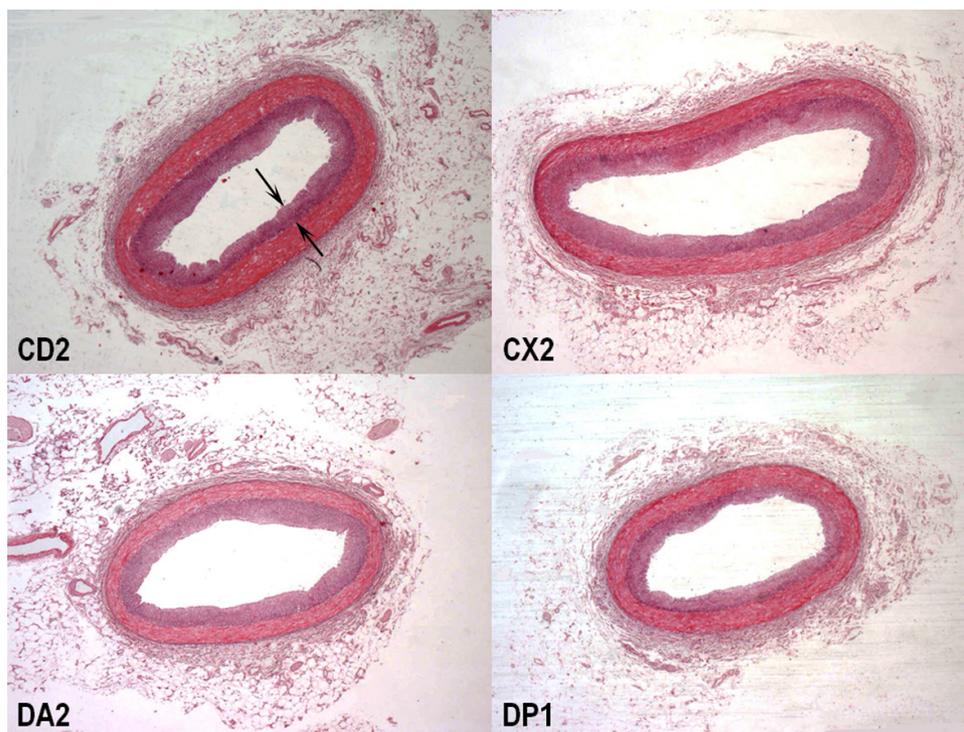
Figure 7 - *Histology of the major epicardial coronary arteries: right coronary (RC2), circumflex branch (CX2) and anterior descending branch (AD2) of the left coronary in the second centimeters, and posterior descending branch (PD1) of the right coronary artery, the first centimeter. Note the mild intimal thickening (between the arrows), resulting in a reduction of approximately 12.5% of the arterial lumen, similar to the other segments. (Hematoxylin and eosin; 2.5x magnification.)*

with overall congestive heart failure; healed transmural septal myocardial infarction; lobar pneumonia; acute pyelonephritis; septic shock. **(Dr. Jussara Bianchi Castelli)**

Comment

This case shows typical aspects of valvular heart disease by chronic rheumatic heart disease, due to the age and macroscopic and microscopic aspects of the heart. What seems unusual is the presence of myocardial infarction associated with rheumatic disease.

In chronic rheumatic valvular heart disease, imposing a situation of increased myocardial work and therefore, greater oxygen consumption, it is unlikely that significant coronary disease would remain asymptomatic. In this case, the coronary artery lesions were very mild, with only fibrous intimal thickening without plaque or occlusive lesions or lesions recognized as being a risk for rupture and thrombosis. Therefore, it was considered unlikely that the detected healed infarction was related to atherosclerotic arterial disease and such events. In fact, the prevalence of chronic arterial disease is low among patients with rheumatic valvular heart disease and this is not a protective effect. This is associated with clinical and demographic differences and risk factors of these diseases, which has been shown in several studies, some discussed below.

A Brazilian study showed that the prevalence of coronary artery disease was lower among patients with rheumatic heart disease (4%) and high among patients with valvular heart disease of non-rheumatic etiology (33%)[9]. In another study of 77 necropsies of patients who died after surgery for valve dysfunction treatment in rheumatic disease, a rate of 13% of significant coronary artery disease was observed and that was more common after the age of 40, also in those patients with isolated aortic or mitral-aortic lesions, rather than with isolated mitral valve lesion[10].

Therefore, for these reasons, the cause suggested for the occurrence of myocardial infarction was a previous perioperative event. Epidemiological data record myocardial infarction as a complication of cardiac surgery in less than 1% (34 cases in 11,210) and point to a statistically significant association with mitral, aortic or double valve procedure. Only 33.3% of the 34 cases studied showed coronaries free of obstruction at the necropsy[11].

Apart from the handling and the trauma of the heart, in addition to surgical technical difficulties, for these cases without significant coronary disease, some other
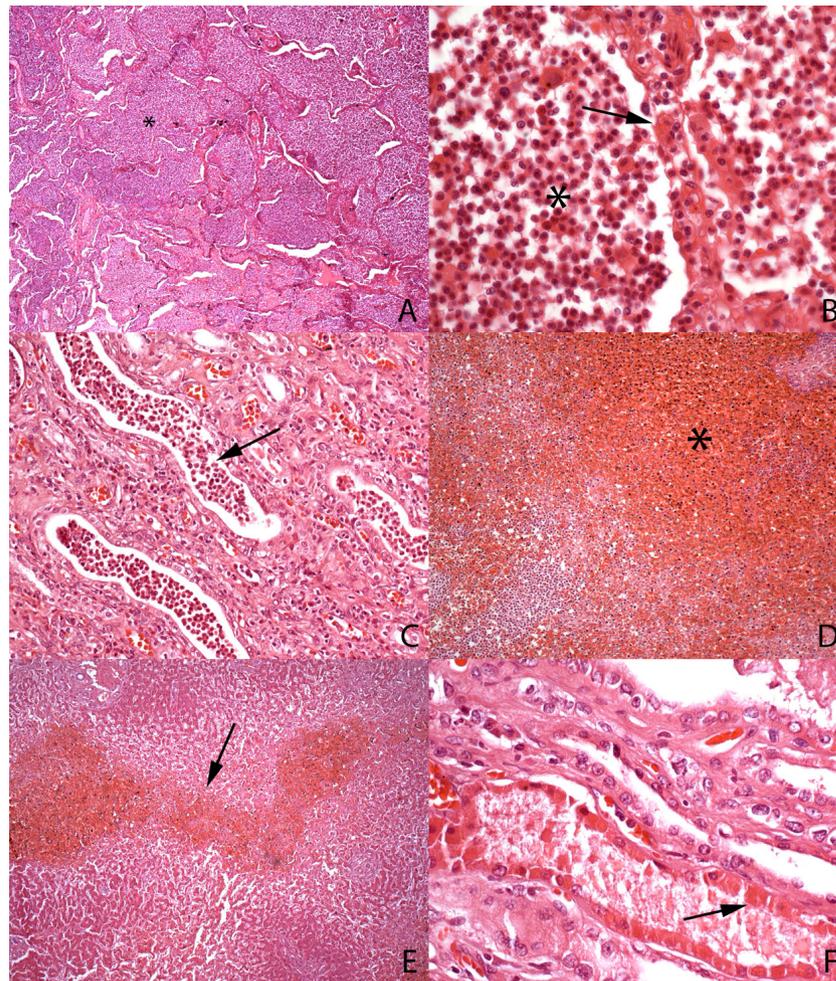
Figure 8 - *Histology of infectious alterations and secondary to septic shock observed in major organs. A. Lobar pneumonia characterized by dense alveolar filling with neutrophilic infiltration (asterisk). B. Detail of A at higher magnifications: Note the cellular composition, exclusively neutrophilic (asterisk) and the interalveolar septum (arrow). C. Acute pyelonephritis: kidney showing tubules filled with neutrophilic infiltration (arrow). D. Spleen showing acute splenitis with increased red pulp (asterisk). E. Liver with recent necrosis of zone 3 (arrow) observed in large areas. F. Rim, in another region, showing acute tubular necrosis: see more intense red color of the tubular lining cells (arrow). (Hematoxylin and eosin; magnification 2.5x, 40x, 20x, 10x, 5x and 40x, respectively.)*

etiopathological mechanisms are considered as a cause of perioperative infarctions in cardiac surgery for valve replacement, such as coronary embolization (personal communication: e.g., we observed once, in a necropsy, calcium emboli to the coronaries in a case of mitral valve replacement that presented with severe dystrophic calcification), coronary gas embolism, coronary vasospasm, topic hypothermia or inappropriate cardioplegia, among others. The prognosis of perioperative myocardial infarction is not necessarily bad, but its occurrence should warrant appropriate measures and prevention in surgical valve replacement[6,12]. **(Dr. Jussara Bianchi Castelli)**

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