Case 2 - Seventy-Year-Old Man with Mitral Stenosis and Pulmonary Fibrosis. Who Developed Sepsis after Balloon Mitral Valvuloplasty

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The patient was a seventy-year-old male individual with mitral valvulopathy and dyspnea on slight exertion and pulmonary infiltrate. The patient came from the town of Rio Prado, state of Minas Gerais, Brazil and was a smoker until the age of 66 years.

He was first treated at *Instituto do Coracao* (InCor - The Heart Institute) at age 66 (March 21, 2001), due to dyspnea and palpitations for 6 years, initially triggered by heavy exertion, which progressively started to be triggered by slighter-than-usual exertion. The patient used 3.75 mg of Warfarin, 75 mg of captopril, 40 mg of furosemide and 0.25 mg of digoxin daily.

At physical examination (March 21, 2001), the patient presented irregular pulse, with a heart rate of 118 bpm, blood pressure of 160/90 mmHg, increased jugular venous pressure. Pulmonary assessment was normal. The heart assessment showed systolic shock, palpated at the 6th intercostal space, out of the left hemiclavicular line, limited by two digital pulps. The first heart sound was hyperphonetic in the mitral area and the second sound was hyperphonetic in the pulmonary area. There was systolic murmur +++/6 and a whirringrumbling diastolic murmur ++/6+ in the mitral area. The assessment of the abdominal area and limbs was normal.

The electrocardiogram (March 1, 2001) showed atrial fibrillation rhythm, mean HR of 100 bpm and indirect signs of right atrial overload (Peñaloza-Tranchesi) (Figure 1).

The laboratory assessment (June 2001) showed hemoglobin 16.9 g/dl, hematocrit 49%, leukocytes 8900/mm^3 with normal differential count; glycemia was 113 mg/dl, creatinine was 1.3 mg/dl, urea 41 mg/dl, sodium 139 mEq/l, o potassium 4.6 mEq/l and INR was 1.3.

The echocardiogram (Dec. 2001) showed a 10-mm interventricular septum and posterior wall, aortic diameter of 32 mm, left atrial diameter of 70 mm, left ventricular diastolic diameter of 49 mm and systolic of 36, with ejection fraction of 60%. The right ventricle was dilated and hypokinetic and the right atrium was also dilated. Signs of pulmonary arterial hypertension were identified; systolic pressure was 66 mmHg and the patient presented marked mitral stenosis (mean gradient estimated at 13 mmHg). The patient presented commissural fusion, thickening of mitral valve cusps, severe mitral regurgitation and moderate tricuspid regurgitation.

The patient was prescribed 0.25 mg of digoxin, 180 mg of diltiazem, 200 mg of acetylsalicylic acid (due to the impossibility of establishing anticoagulation control at the patient's hometown), 20 mg of enalapril and 40 mg de furosemide. The surgical correction of the mitral valvulopathy was indicated.

The patient developed dyspnea at moderate exertion. However, he also presented weight loss of 8 kg after 2001.

The abdominal and pelvic ultrasonography assessment was normal. The chest x-ray showed interstitial infiltrate and perihilar alveolar involvement in both lungs (October 2004). The test for alcohol-acid resistant bacilli (AARB) in sputum and the presence of malignant cells in the lungs were negative.

The high-resolution chest tomography (October 2004) disclosed normal vascular structures, upper and lower paratracheal, prevascular, subcarinal and bilateral perihilar lymphadenopathy. The trachea and bronchi were normal. There was retractile peribronchial opacity with diffuse distribution in both lungs, associated with pulmonary alveolar filling. The findings were considered compatible with pulmonary congestion or alveolar carcinoma.

The investigation also included bronchoalveolar lavage, which was negative for neoplasia, bacteria and fungi and showed 75% of neutrophils, 20% of macrophage, and 5% of other cells; the transbronchial biopsy disclosed the presence of an inflammatory process, with a predominance of mononuclear cells.

A new tomography disclosed decreased pulmonary volume without pleural effusion with symmetrical, peribronchial alterations and areas of parenchymal densification, associated with fine reticular opacities and frosted glass aspect, interlaced by bronchiectasis and bronchioloectasis. The findings were considered compatible with sarcoidosis, silicosis or chronic hypersensitivity pneumonitis (chronic allergic alveolitis).

The cardiac catheterism showed pulmonary hypertension and capillary gradient and pulmonary artery pressure gradient between pulmonary artery and capillar (Table 1); the coronary angiography (October 2004) did not disclose luminal

Key Words

Mitral valve stenosis/surgery; pulmonary stenosis; sepsis; balloon dilatation

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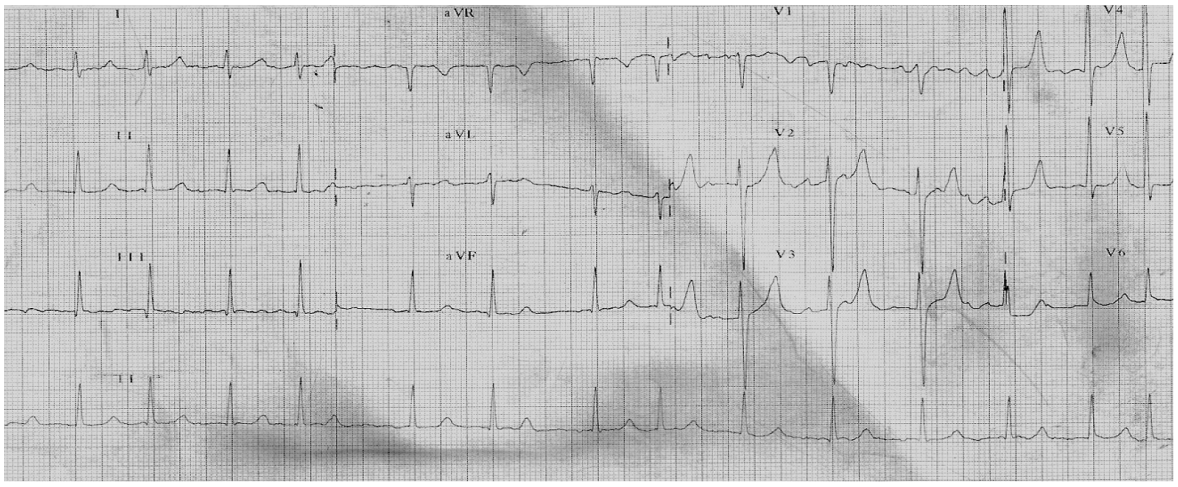

Figure 1 - Atrial fibrillation and indirect signs of right atrial enlargement (signal of Peñaloza-Tranchesi).

obstructions in the coronary arteries.

The echocardiographic reassessment (April 2005) disclosed a 62-mm left atrium, 29-mm aorta, 8-mm interventricular septum, 7-mm posterior wall, left ventricle: systole 33 and diastole 44 mm. The mitral valve area was estimated at 1.2 cm^2; the right ventricular systolic pressure was estimated at 35 mmHg. The patient presented severe mitral stenosis and moderate tricuspid regurgitation. The transesophageal echocardiogram showed a Block score of 10 (motility 3, calcification 3, thickening 2 and subvalvular apparatus 2). There were no images suggestive of intracavitary thrombus. The spirometry results suggested a restrictive lung pattern.

Considering the pulmonary condition, the balloon mitral valvuloplasty was indicated and carried out on April 28, 2005. A few hours after the procedure, the patient started to present intense dyspnea, with wheezes and diffuse rales, as well as elevated heart rate, hypoxemia and arterial hypotension, (85/65 mmHg). The patient was then transferred to the Intensive Care Unit.

At physical examination, the patient was cyanotic, tachypneic (35 incursions per minute), HR was 110 bpm, blood pressure was 96/65 mmHg; crackling rales in the lower 2/3 of both hemithoraces. The patient presented increased jugular venous pressure; cardiac auscultation disclosed hyperphonetic second heart sound in the pulmonary area and first sound in the mitral area, without murmurs. There were also signs of peripheral hypoperfusion. A diagnosis of acute pulmonary edema was made.

Table 1 - Cardiac chamber pressures (mmHg)

	Systolic	Initial diastolic	Final diastolic	Mean
Right atrium				5
Right ventricle	60	0	2	
Pulmonary artery	60	40		
Pulmonary occlusion				25
Left ventricle	120	0	5	
Aorta	120	80		96

The electrocardiogram (April 28, 2005) disclosed atrial fibrillation rhythm with 121 bpm, QRS axis at 0° backwards, with variation of beat frequency, indirect signs of right atrial overload (Peñaloza - Tranchesi) and ventricular repolarization alterations.

The echocardiogram (April 29 2005) showed left ventricular dimensions (45 mm in diastole/27 mm in systole), septum and posterior wall thickness of 8 mm, aortic diameter of 32 mm and left atrium of 63 mm; markedly enlarged right atrium; slight mitral valve stenosis (valvular area: 1.6 cm^2) and moderate mitral regurgitation, as well as the presence of a small atrial septal defect, with a predominant left-to-right shunt. There were no pericardial alterations.

The dyspnea improved with oxygen treatment and intravenous diuretics and nitrates. Two days later the patient presented worsening in the dyspnea and hemodynamic instability and was submitted to orotracheal intubation for respiratory support.

From April 30 on, after blood cultures had been collected, the patient started to receive antibiotics: 2g of cefepime and 500 mg of azithromycin daily, by intravenous route. In spite of treatment, the patient started to present daily fever peaks from May 6, 2005 on and the cefepime was substituted by 1 g of vancomycin/day. The blood culture disclosed the presence of vancomycin-sensitive *S. aureus*.

The laboratory assessment results are shown in Table 2. Blood gas assessment showed progressive worsening of gas exchange and alveolar ventilation (Table 2). The hemodynamic measurements with Swan-Ganz catheter showed, initially, normal cardiac output and pulmonary

vascular resistance; however, later measurements showed decrease in the systemic resistance to values below the normal range (Table 3). In spite of the use of vasoactive drugs, volume and slow hemodialysis (May 13, 2006, the patient developed refractory shock and presented cardiac arrest in asystole and died on May 14, 2005.

Clinical aspects

The patient was a 70-year-old male individual, ex-smoker for 4 years with no description of the annual smoking load and occupational history, with a diagnosis of mitral valvulopathy (significant double mitral lesion, stenosis > regurgitation), chronic atrial fibrillation and diastolic heart failure, with a 10-year history of progressive dyspnea, possible consumptive syndrome (loss of 8 kg) in the preceding 4 years and chest x-ray that showed perihilar interstitial-alveolar infiltrate, with basal predominance, in addition to a chest tomography showing paratracheal, prevascular, subcarinal and bilateral perihilar lymphadenopathy, in addition to peribronchial retraction with diffuse distribution associated with pulmonary alveolar filling, suggesting pulmonary congestion or alveolar carcinoma. The test for alcohol acid-resistant bacilli (AARB) was negative in sputum, bronchic aspiration and transbronchial biopsy were negative for malignancy, bacterial and fungal diseases. A second chest tomography had no description of lymphadenopathy, with a frosted glass pattern, compatible with sarcoidosis, silicosis or even chronic hypersensitivity pneumonitis (chronic allergic alveolitis). The spirometry disclosed a restrictive pattern.

The clinical discussion will encompass the clinical and evolution aspects of the underlying diseases, as follows: mitral valvulopathy and pneumopathy and its complications.

The mitral stenosis, a disease that results from the thickening and decreased motility of the mitral valve leaflets, leading to its obstruction, has as the main etiology the rheumatic disease and current data suggest that 99% of the cases have this entity as the confirmed or probable cause[1]. However, of these cases, around 30% to 50% did not have previous symptoms of rheumatic disease[2,3].

In the case of rheumatic valvulopathy, young women are most often affected, with a 2:1 female:male ratio[2,4]. Other etiologies include congenital malformation, mucopolysaccharidosis disease, cardiac carcinoid disease, systemic lupus erythematosus, rheumatoid arthritis and calcification of the atrioventricular annulus, with the latter being more frequently observed in elderly individuals.

The symptoms of mitral stenosis result mainly from increased pulmonary capillary pressure and venous and capillary distension, which lead to pulmonary edema as soon as the venous pressure exceeds the oncotic plasma pressure. However, in some patients, the pulmonary edema might not occur due to the decreased vascular permeability, caused by the disease evolution.

The pulmonary hypertension, very common during the evolution of mitral stenosis, is caused by not only the passive

Table 2 - Laboratory assessment at hospital admission

	april 29	may 01	may 9	may 12	may 14
PH	7.42	7.28	7.39	7.24	6.66
PCO_2 (mmHg)	32	56	52	65	168
PO_2 (mmHg)	58	57	77	72	67
Sat O_2 (%)	88.8	89.7	59	83.5	79
HCO_3 (mEq/l)	20	25	39	32	18
Ex.B (mEq/l)	-2	-1.4	12.3	3.3	-21
Lactate (mmol/l)*	32			13	107
Sodium (mEq/l)	135	138	143	147	144
Potassium (mEq/l)	3.2	4.2	4.2	4.7	6.4
Urea (mg/dl)	30	43	63	98	157
Creatinine (mg/dl)	0.9	0.9	0.7	1.4	2.7
Hemoglobin (g/dl)	12.9	10.7	10.7	9.5	9.4
Hematocrit (%)	38	35	35	33	33
Leukocits/mm³	20.700	14.700	14.300	25.600	21.700
Neutrophils (%)	92	68	91	97	88
Platelets/mm³	327.000	256.000	255.000	82.000	104.000
PT(INR)	-	1.25	1.34	-	1.81
APTT (rel)	-	1.04	1.15	-	4.12

Lactate (mmol/L): < 2.5 = normal; 2.5 - 4.9 = slightly increased; 5.0 - 9.9 = moderately increased ; > 10 = markedly increased.

Table 3 - Hemodynamic measurements with Swan-Ganz catheter

Measurements	may 6	may 7	may 8	may 9
HR (bpm)	103	78	100	93
Systolic BP (mmHg)	114	85	94	119
Diastolic BP (mmHg)	57	50	58	67
CVP (mmHg)	13	24	36	8
Mean pulmonary (mmHg)	44	36	45	43
Pulmonary capillary (mmHg)	26	12	29	22
Cardiac index ($l.min^{-1}.m^{-2}$)	3.7	3.8	3.1	3.7
Syst. vasc. res. ($dina.s.cm^{-5}$) (Wood)*	1.153 (14)	548 (7)	600 (7.5)	1.252 (15.7)
Pulm vasc. res. ($dina.s.cm^{-5}$) (Wood)**	277 (3.5)	355 (4.4)	291 (3.6)	317 (4)
SVR index ($dina.s.cm^{-1}$)	1.621	779	851	1.794
PVR index ($dina.s.cm^{-1}$)	389	505	413	454

*Normal values: * systemic resistance : 900-1200 (11- 15 Wood); ** pulmonary resistance: 150-250 (2-3 Wood). Obs.: 1 Wood unit = 80 dina.s.cm-5. The patient's body surface was 1.42 m².*

component, which is simply the transmission of the increase in left atrial pressure, but also the reactive component, characterized by vasoconstriction, muscular hypertrophy and obliteration of the pulmonary vasculature. The degree of reversibility after the correction depends on the predominance between each component.

Another condition often observed is atrial fibrillation, which is present in around 40 to 50% of the cases[5] and is caused by the increased left atrial pressure and its dilatation. As a consequence, the patient presents hemodynamic worsening, caused by the loss of atrial contraction and the increase in ventricular frequency. There is also an additional increase of thrombotic risk, and in this case, the anticoagulation therapy is formally indicated.

The mitral stenosis is classified considering the valvular area as well as the atrioventricular gradient and the pulmonary artery pressure. Thus, it is classified as: Mild when the valvular area (VA) > 1.5 cm^2, gradient is < 5 mmHg and the pulmonary artery systolic pressure (PASP) < 30 mmHg; Moderate when the VA is between 1.0 and 1.5 cm^2, the gradient is 5 to 10 mmHg and PASP 30-50 mmHg; severe when the VA < 1.0 cm2, the gradient > 10 mmHg and PASP is > 50.

The mortality of untreated patients is 60% to 70% of the cases due to progressive pulmonary and systemic congestion, systemic embolism in 20% to 30% of the cases, pulmonary embolism in 10% and infections in 1% to 5% of the cases[4,6,7].

The clinical treatment, which includes diuretics, negative chronotropic agents and anticoagulation therapy, must be individualized for each case.

The correction treatment is indicated in cases of moderate and severe stenosis, when there are symptoms or marked pulmonary hypertension, and it can be carried out by percutaneous route through balloon valvotomy or surgically, by commissurotomy or valve exchange.

Regarding the balloon valvotomy, it has short-term outcomes that are similar to the surgical treatment[8] and can be used when the patient presents favorable anatomy, usually considering a Block score < 8, as this value presents better sensibility and specificity to predict the chance of complications.

It must be recalled as contraindications to the procedure the concomitance of moderate or significant mitral regurgitation, as well as the presence of intra-atrial thrombi at the echocardiogram.

The percutaneous treatment is considered successful when a residual valvular area ≥ 1.5 cm^2 and a decrease in the left atrial pressure to < 18 mmHg are achieved. The main complications of the procedure are mitral regurgitation in 2% to 10%, significant atrial septal defect in around 5%, and, less frequently, embolic events (0.5% to 3%), left ventricular perforation (0.5% to 4%) and acute myocardial infarction in 0.3% to 0.5% of the patients[9,10].

It is also important to discuss the mitral stenosis associated with mitral regurgitation, also called double mitral lesion. In this case, one of the lesions can be predominant or both can be equally important, resulting in different degrees of symptoms and alterations at the ECG, chest x-ray, as well as the presence and variable degree of left ventricular dilatation.

In this condition, dasimilarly to what occurs with the double aortic lesion, an increased gradient can be present in mitral lesions in which the valvular regurgitation predominates due to the increased transvalvular flow. Therefore, in this case, the measured gradient does not necessarily correlate with the degree of the stenosis[11].

Regarding the treatment, there are no guidelines on the precise moment for surgical indication in mixed lesions; however, the surgery is recommended for patients who present moderate to severe symptoms or signs of left ventricular dysfunction, with lesions that can justify them. The treatment in most of these cases is carried out through valve exchange, as the commissurotomy or plasty is seldom feasible[12].

The pulmonary involvement observed in this patient might be a direct consequence of the valvuloplasty or be caused by independent pulmonary disease. The pulmonary congestion and edema, secondary to the hemodynamic alterations determined by the mitral stenosis, occur when the pulmonary venous return is decreased, mainly in left ventricular failure and mitral stenosis. The increased venous pressure is transmitted to the pulmonary arteries because there are no valves in the pulmonary circulation. As the pulmonary vessels are also very compliant, there is a marked increase in intravascular volume. Later, an increase in capillary pressure induces a fluid transudation to the interstitium and the alveolar airways. The acute alveolar edema is reversible, but the chronic edema determines a degree of interstitial fibrosis. The interstitial edema is more distinct in the pulmonary region, due to a higher hydrostatic pressure in these areas. This unequal gravitational distribution of the edema also induces a redistribution of perfusion. At least two factors account for the flow redistribution to the upper zones: first, the basal edema reduces the compliance in the lower zones and the vessels cannot expand normally during inspiration. Second, the pulmonary elasticity induces hypoventilation, hypoxic vasoconstriction (Euler-Liljestrand reflex) and a decrease in perfusion to the lower zones. With higher increases in the left atrial venous and pulmonary venocapillary pressures, the pulmonary edema ensues. Initially, fluid-filled lobules are interspersed among normally aerated lung areas, producing a radiographic aspect of blurred opacity. As the edema becomes more diffuse, a homogenous opacity is seen[13].

At the chest CT, the pulmonary edema is described by the thickening of the interlobular septa and frosted-glass opacity in the lower areas. The alveolar edema appears as confluent acinar shadows, with air alveologram (juxtaposed acini containing air and fluid).

The diagnosis of pulmonary edema/congestion is quite attractive, as only one disease would be considered, which would explain all the clinical and radiological findings and the mitral stenosis; however, one cannot forget that the patient has a consumptive syndrome, a restrictive defect at the spirometry and significant lymphadenopathy, which would suggest interstitial diseases and lung malignancy.

The interstitial pulmonary disease comprehends a group of pulmonary diseases that diffusely and bilaterally affect the alveolar septa, presenting as inflammation at the initial phase

and fibrosis at more advanced phases. The characteristic lesion is the alveolitis, or pneumonitis, defined as the accumulation of inflammatory cells in the alveolar septa. The inflammatory cells also overflow into the lumen, forming alveolar exudates. The fibrosis occupies the alveolar septa and fills out the airways, leading to the progressive destruction of the parenchyma. Multiple cystic areas with fibrotic walls are formed, which can be seen as areas of honeycombing. They normally course with restrictive respiratory disorder at the spirometry[14,15].

The patient with pulmonary interstitial disease presents dyspnea on exertion and dry cough with an insidious or sudden course.

During the investigation, one must always look for signs of systemic disease, such as collagenoses and inquire about occupational history and use of medications. The presence or not of eosinophilia is very important for the diagnosis. The clinical investigation includes chest high-resolution tomography, bronchofibroscopy with bronchic aspiration and open lung biopsy or by videothoracoscopy.

Among the interstitial diseases of the lung, the hypotheses of sarcoidosis, silicosis or chronic hypersensitivity pneumonitis were considered. Silicosis is an occupational disease associated with the prolonged and intense inhalation of silica particles, forming small round diffuse opacities (silicotic nodules). The peripheral calcification of lymph nodes, "eggshell calcification", is an unusual finding that suggests silicosis. Normally, it courses asymptomatically and with no alterations in lung function tests; however simple chronic silicosis can be asymptomatic or present with progressive dyspnea on exertion, dry cough, few or no constitutional symptom and can have as complications large densities in pulmonary apex with dyspnea and mixed pulmonary dysfunction and is related with tuberculosis, if there are constitutional symptoms[16].

Sarcoidosis is a multisystem granulomatous disease that affects individuals aged 20 to 40 years, characterized by the accumulation of lymphocytes and mononuclear phagocytes in certain organs, in order to form non-caseous granulomas. The disease is of unknown etiology, but alterations in the immunological system arte clearly involved in its pathogeny. The chest x-ray usually discloses bilateral hilar adenopathy, frequently associated with paratracheal adenopathy and/or parenchymal reticulonodular infiltrates. The lung is the most affected organ, causing dyspnea on exertion, dry cough and wheezing; nasal granulomas can cause nasal congestion and, in the larynx, hoarseness. Hemoptysis and pleural effusion are rare. It can also affect the skin, eyes, central nervous system, and the heart. The diagnosis is made through the biopsy of the affected organ[17].

The hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis is a pulmonary disease of immunological origin, caused by the exposure to and repeated inhalation of organic dust or chemical substances. It has a diversified synonymy according to the antigen and/or sources that caused the disease, such as the farmer's lung, caused by the inhalation of dust from moldy plant material such as straw, hay, wheat, oats, barley, corn. Among the chemical substances implicated in this disease is the isocyanate, which is used in paints and polyurethane materials. In acute HP, the symptoms start hours after the exposure to the antigen and can be misdiagnosed as viral or bacterial respiratory infection. In subacute HP, the condition is intermittent and results from the non-continuous exposure to the antigen. The main symptoms are productive cough, dyspnea, fatigue, anorexia and weight loss. In chronic HP, the symptoms are similar to the subacute form, but they occur more insidiously and are caused by the continuous exposure to small amounts of the antigen[18].

Considering the history of the patient, one can rule out silicosis and chronic hypersensitivity pneumonitis due to the absence of occupational history, as well as sarcoidosis, considering that the patient was not at the most common age range for the disease, from 20 to 40 years and also due to the negative transbronchial biopsy. It must also be recalled that an important differential diagnosis for this disease would be idiopathic pulmonary fibrosis, a common disease in the 6th and 7th decades of life, of which evolution shows slowly progressive dyspnea, weight loss, reticular or reticulonodular infiltrate, more prominently in the chest x-ray; the high-resolution tomography shows sparse subpleural reticular infiltrates and cystic airways, mainly at the pulmonary bases; frosted glass pattern and thickened interlobular septa[14,15].

Other diagnoses that must be considered are pulmonary neoplasias. Considering the patient's age, history of smoking, progressive dyspnea history, weight loss in the last 4 years and taking into account the radiological assessment, it is necessary to include carcinomatous lymphangitis among the differential diagnoses, as it causes such radiological alterations, but does not produce distortion of the normal pulmonary architecture, that is, as mentioned in the first high-resolution chest tomography.

Another differential diagnosis that must be taken into account would be infectious endocarditis, as the patient presented valvulopathy and had an episode of fever at the ICU, in spite of antibiotic treatment; moreover, *Staphylococcus aureus* was isolated from blood cultures, which is a typical endocarditis microorganism. However, there have been few reports in the literature of a correlation between balloon valvotomy and posterior endocarditis, which could be associated to the central catheter passage.

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Diagnostic hypotheses: mitral stenosis of rheumatic etiology, sepsis, idiopathic pulmonary fibrosis

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Pneumologist's comments

The patient presents history, physical examination and image assessment that are compatible with associated pulmonary disease, contributing especially to the symptoms of dyspnea on exertion. The clinical findings suggest chronic pneumopathy, of which evolution presented acute episodes.

The radiological assessment shows interstitial (including the peribronchovascular interstitium) and alveolar involvement. As the patient's history and personal habits do not allow us

to perform an epidemiological correlation, the most probable diagnostic hypotheses are: sarcoidosis, hypersensitivity pneumonitis, carcinomatous lymphangitis and pulmonary edema. There is no radiological evolution that permits attaining the final diagnosis and its association with the cause of death. Among the possible diagnoses, findings such as mediastinal adenomegaly are missing, which would better characterize a sarcoidosis and even a pulmonary carcinoma. The bronchic aspiration and biopsy data are not pathognomonic, but they can suggest hypersensitivity pneumonitis. An open biopsy could elucidate the diagnosis.

Dr. Rafael Stelmach

Necropsyia

The necropsy disclosed double mitral lesion with predominance of stenosis (Figure 2) and mild aortic regurgitation; considering the morphological aspects - fibrosis, commissural fusion and areas of calcification in the mitral valve, as well as mainly the clinical history and the incidence in our country, the cause of such lesions must be rheumatic disease.

Next to a mitral valve commissure, where a tear probably occurred due to the balloon valvuloplasty procedure, there was a lesion with raw surface (Figure 3). The microscopic study disclosed endocarditis caused by Gram-positive cocci (Figure 4). This infection caused embolism to the coronary arteries, spleen (with presence of bacteria) and right kidney, as well as intramyocardial microabscesses (Figure 6). Finally, the patient had septicemia and septic shock, which was the final factor that triggered death.

As non-related disease, but which significantly contributed to the death, the patient presented pulmonary fibrosis (Figure 7), with small foci of mononuclear cell infiltrates. At this phase of fibrosis, it is frequently difficult to establish the cause of the process and that was the case with this patient, who also presented the additional difficulty of overlapping diffuse alveolar damage caused by the septic shock.

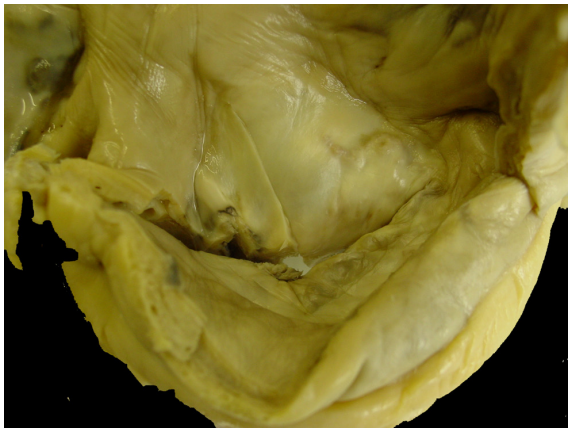

Figure 2 - *Atrial view of the mitral valve, showing irregularities mainly in the closing borders, with commissural fusion leading to the double lesion with stenosis predominance.*

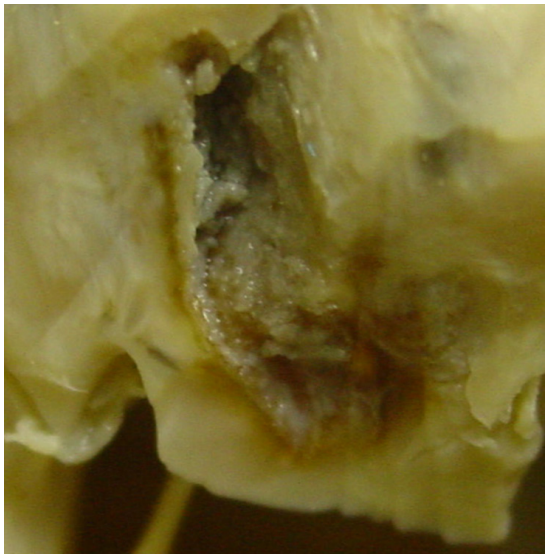

Figure 3 - *Cross-section of valve commissure, showing lesion with raw surface.*

The worsening in the hemodynamic and respiratory patterns must be a direct consequence of the shock, the diffuse alveolar damage caused by it and the adjunct role of the reactional myocarditis and the myocardial microabscesses.

Dr. Paulo Sampaio Gutierrez

Anatomopathological diagnoses - main disease: bacterial endocarditis in the mitral valve with previous rheumatic lesion; cause of death: septic shock; concurrent cause: idiopathic pulmonary fibrosis.

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Comments

There have been, in the Laboratory of Pathological Anatomy - *Instituto do Coração* (the Heart Institute) - *Hospital das Clínicas* - School of Medicine of the University of São Paulo, only two other cases of necropsy in adult individuals submitted to balloon mitral valvuloplasty (in addition to one whose procedure was carried out in the aortic valve). This was the first case that presented endocarditis after the valvuloplasty. In the literature, however, cases with this type of complication have been described[19-24] and data were obtained from 8 of them. The onset of infection occurred between 2 weeks and 6 months after the valvuloplasty, with a mean of 2.75 and a median of 2.5 months. The present case, therefore, is among the ones with the earlier onset. Considering the small number of reports, the estimate of the complication incidence must assessed with restrictions. In three groups of patients submitted to the procedure, one with 294 patients[21], another with 113[22] and another with 13[23] (the latter consisting only of children with rheumatic disease, aged 12 years or younger) there was one case of endocarditis in each, but there have been other patient follow-up series in which the infection did not occur[24].

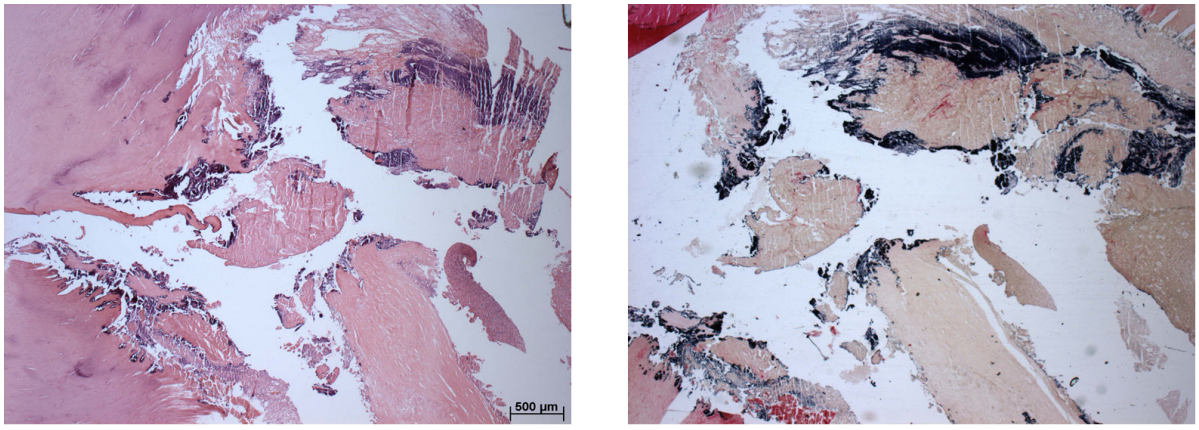

Figure 4 - Histological section of the mitral valve, showing abundant Gram-positive cocci, stained blue by hematoxylin and eosin (A) and dark purple by the Brown-Hopps method (Gram modified for tissue, (B). Magnification: 2.5 x.

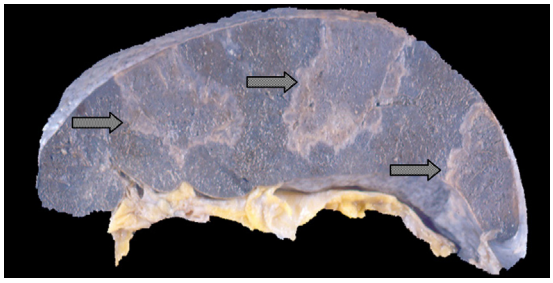

Figure 5 - Longitudinal section of the spleen showing infarction areas (shown by arrows).

Regarding the lungs, the necropsy ruled out some diagnostic possibilities, such as malignancy and sarcoidosis; however, the cause of the alterations was not clarified and the pattern was classified as idiopathic pulmonary fibrosis. Such degree of involvement is not commonly seen only as a consequence of passive congestion.

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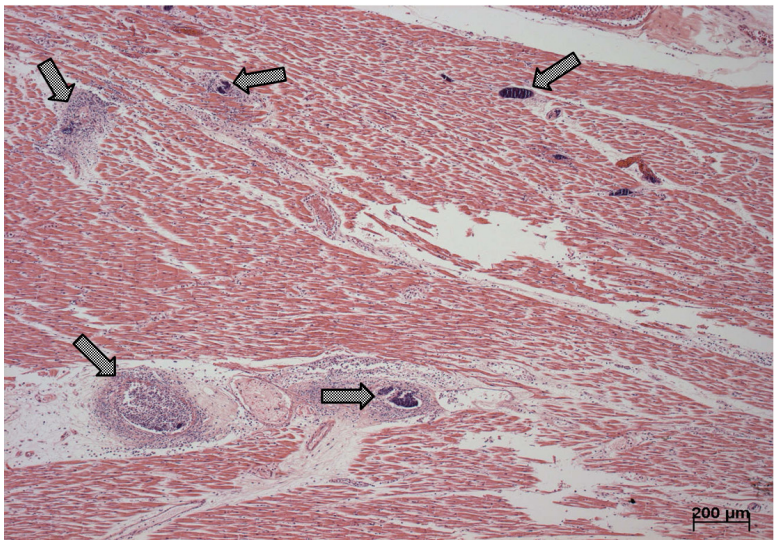

Figure 6 - Histological section of the myocardium, showing vessels filled by septic thrombi, consisting of bacteria and inflammatory cells. Hematoxylin & eosin stain; magnification: 5x.

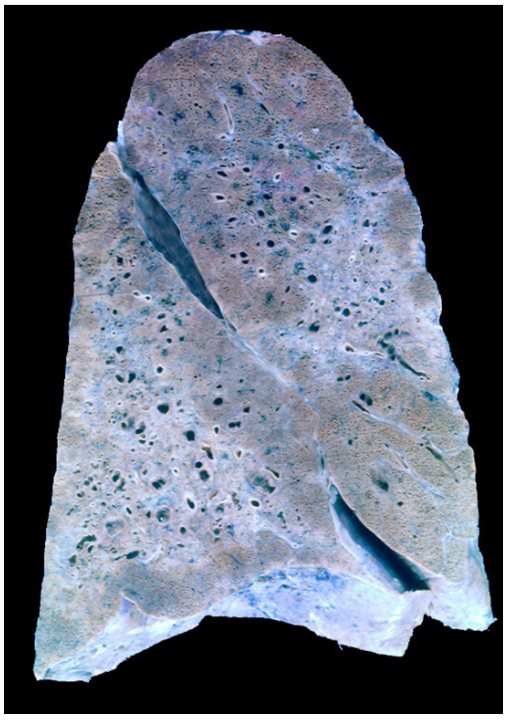

Figure 7 - Longitudinal section of the left lung, disclosing extensive areas of fibrosis, of whitish color, in addition to the chronic passive congestion, which gives the organ the brownish color.

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