Case 6/2007 - Early Post-operative Cardiogenic Shock After Stenting of Thoracic Aorta Aneurysm

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At presentation, a 72-year-old woman complained of stabbing precordial pain radiating to the dorsum and not associated with physical exertion for one year. She was a smoker, had nighttime coughing and dyspnea, was hypertensive and used 75 mg captopril and 25 mg hydrochlorothiazide.

Medical investigation included angiotomography (May 23, 2005) which revealed enlargement of the mediastinum due to a bulky aneurysm in the thoracic aorta in its arch and descending portion, measuring 9 cm in transversal diameter and 7 cm in length, as well as mural thrombosis and an aneurysm at the thoracoabdominal junction with dimensions of 5 cm by 4 cm. Bilateral pleural effusions were detected, and the patient was referred to InCor for treatment of the aortic aneurysm.

Upon physical examination (June 10, 2005), her blood pressure was 120/80 mmHg, the heart rate was 100 bpm; lung auscultation was normal, and cardiac auscultation revealed a systolic murmur in the aortic and mitral areas; the abdominal examination showed no abnormalities. Limb pulse amplitude was reduced.

The electrocardiogram (ECG) (June 10, 2005) showed sinus rhythm, a heart rate of 95 bpm, left chamber overload, intraventricular stimulus conduction disorder (left bundle branch block-type), and alteration of ventricular repolarization (figure 1).

The patient was admitted to undergo urgent surgical treatment.

Chest X-Ray (June 11, 2005) revealed enlargement of the mediastinum, normal cardiac area and no abnormalities in the lung fields.

Laboratory test findings (June 11, 2005) were: hemoglobin 12.6 g/dl, hematocrit 40%, leukocytes 12,300/mm^3 (neutrophils 65%, eosinophils 8%, basophils 1%, lymphocytes 19% and monocytes 7%), platelets 224,000/mm^3, creatinine 1.8 mg/dl, and urea 83 mg/dl. Urine testing and coagulogram revealed no abnormalities.

The echocardiogram (June 16, 2005) showed a 25 mm aortic root diameter, 28 mm left atrium, 43 mm left ventricular diastolic, and 29 mm systolic diameters. The left ventricular ejection fraction was estimated to be 60%, with preserved wall motility. There was an aortic aneurysm with images suggestive of thrombosis in its lumen.

The coronary angiography showed stenosis of 50% of the right coronary artery and posterior descending branch of the right coronary, with no lesions in the left coronary trunk or its anterior interventricular branch, although there was a 40% lesion of the circumflex branch.

After coronary angiography, the levels of creatinine rose to 2.8 mg/dl on June 20, and then fell to 2.1 g/dl, and the levels of urea rose to 121 mg/dl and later dropped to 82 mg/dl. The patient also displayed progressive anemia with leukocytosis 10.9 g/dl (June 17, 2005), hematocrit 34% and a leukocyte count of 21,400mm^3 (neutrophils 84%, eosinophils 1%, lymphocytes 8% and monocytes 7%). Urine testing revealed proteinuria 0.4 g/l, leukocyturia 30,000/ml, and cylindruria (880/ml hyaline casts and 330/ml granular casts). Upper digestive endoscopy showed moderate erosive antral gastritis.

The patient developed pain in the right hypochondrium, which, along with fever, was attributed to possible acute cholecystitis.

An abdominal ultrasound (June 17, 2005) showed biliary and renal lithiasis. Diagnosis of acute cholecystitis was made and the patient was started on ceftriaxone plus metronidazol for ten days.

New laboratory tests performed on June 27, 2005 revealed: creatinine 1.6 mg/dl, urea 56 mg/dl, potassium 3.9 mEq/l, sodium 141 mEq/l, hemoglobin 10.3 g/dl, hematocrit 34%, leucocytes 16,900 (neutrophils 82%, eosinophils 4%, basophils 1%, lymphocytes 9%, monocytes 4%), and platelets/mm^3 427,000.

The patient underwent surgery on the morning of June 29, 2005 consisting of implantation of an endovascular prosthesis with a stent through the aortic arch. When the patient was taken off extracorporeal circulation, a complete atrioventricular block occurred followed by cardiac arrest; the heart rate was restored after 30 minutes of resuscitation maneuvers, although with a complete atrioventricular block requiring an atrioventricular pacemaker implantation and high doses of dopamine (20 μg/kg/min), amrinon (0.75 μg/kg/min), noradrenaline (2.1 μg/kg/min) and adrenaline (0.5 μg/kg/min).

Hemodynamic measurements showed cardiac index 1.9 l.kg^{-1}.min^{-1}, systemic vascular resistance 18 WU, pulmonary

Key words

Shock; postoperative period; stents; aortic aneurysm, thoracic.

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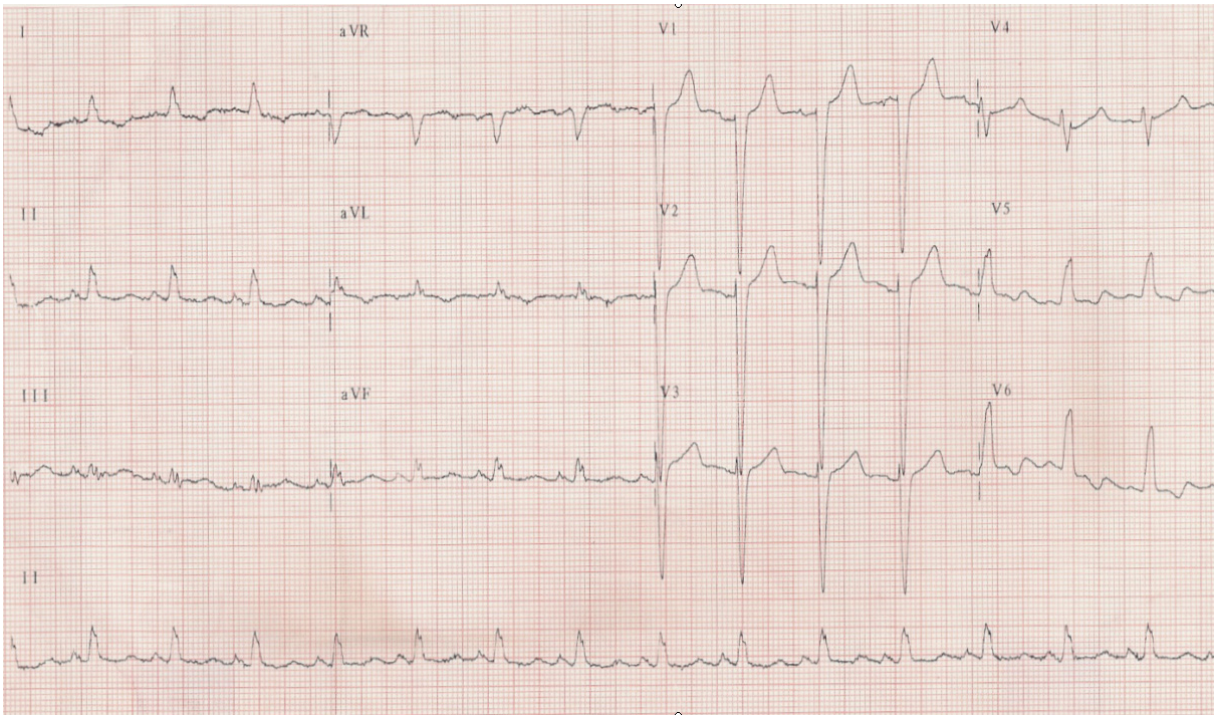

Fig. 1 - *Electrocardiogrm (ECG) – Left chamber overload, left bundle branch block .*

vascular resistance 3.6 WU, mean arterial blood pressure 61 mmHg, and central venous pressure 21 mmHg.

Arterial blood gases were: pH 7.20, pCO_2 55 mmHg, O_2 saturation 58.5%, sodium bicarbonate 33 mEq/l, and base excess (-) 6.7 mEq/l. Lactate was 128 mmol/l.

The electrocardiogram (June 29, 2005) performed after cardiac arrest and with the pacemaker turned off showed a rhythm with no P wave, varied waveforms of QRS complexes, 100 bpm heart rate, QS complexes in I, II and aVF, and in V_1 to V_3, with ST-segment elevation in I, II, aVF, V_5 and V_6 (figure 2).

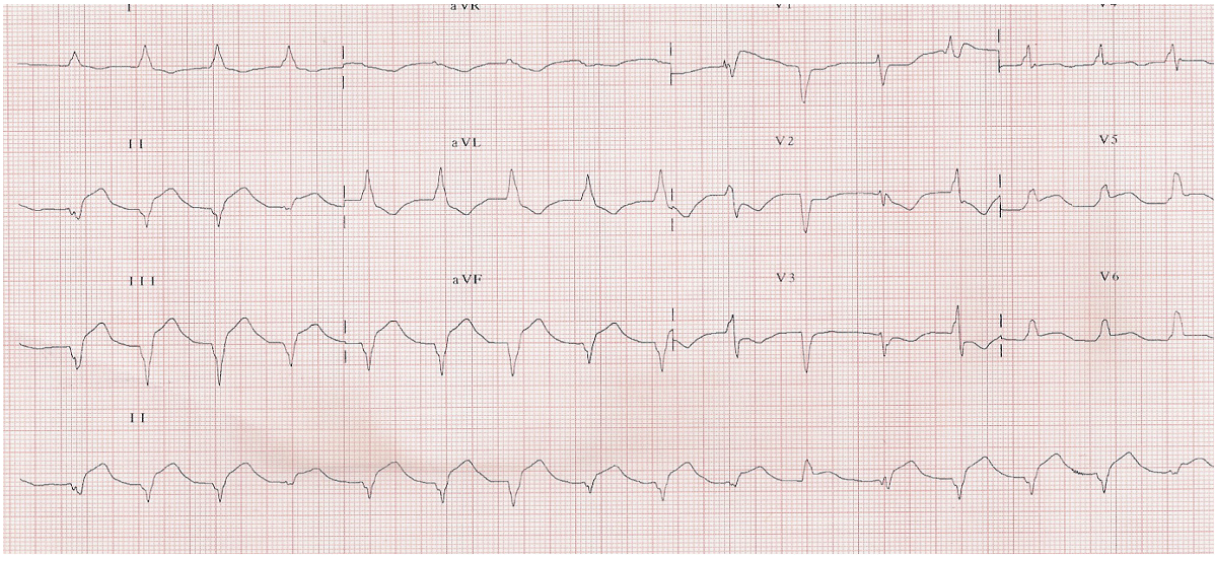

Fig. 2 - *Electrocardiogram (ECG) - Accelerated idioventricular rhythm with no P wave, ST–segment elevation in inferolateral walls.*

A few hours later, the patient developed sustained ventricular tachycardia refractory to xylocaine, amiodarone and electrical cardioversion; later, she went into cardiac arrest in the presence of pulseless electrical activity unresponsive to resuscitation maneuvers, and died.

Clinical aspects

This is the case of a 72-year-old female hypertensive patient, a smoker, who had been experiencing stabbing precordial pain radiating to the dorsum and unrelated to physical exertion, besides nighttime coughing and dyspnea for one year.

First of all, we should consider the origin and differential diagnoses of thoracic pain. Several disorders can present with this symptom from cardiac (angina), vascular (aneurysm, pulmonary hypertension), and pulmonary (pleuritis) causes to gastrointestinal diseases (gall bladder, gastropathies), musculoskeletal (costochondritis), and psychological (panic syndrome) disorders[1]. In this case, the patient reported stabbing pain radiating to the dorsum which was not associated with physical exertion, and such characteristic can be suggestive of aortic disease. We should keep in mind that at least half of patients with aneurysms are asymptomatic[2]. Pain in the chest or back occurs in 25% of the cases of non-dissecting aneurysms and may be caused by direct compression from other intrathoracic structures or the thoracic wall, or result from erosion into the adjacent bone. Other symptoms reflect vascular consequences (aortic regurgitation, heart failure, cerebrovascular accident, lower-limb ischemia) or local mass effect. Aneurysms of the aortic arch or the descending aorta may compress the trachea or a stem bronchus and cause tracheal deviation, wheezing, coughing, dyspnea, hemoptysis or recurrent pneumonia[2,3].

The patient was referred to our unit with a diagnosis of aneurysm in the arch and descending portions of the thoracic aorta and its thoracoabdominal junction.

The term "aortic aneurysm" refers to the dilation of the aortic lumen involving one or more segments. One of the criteria used defines it as a "localized and permanent dilation of the aorta to at least one-and-a-half time its normal diameter for that given segment[4].

Thoracic aorta aneurysms are classified according to the segment affected, i.e., the ascending, the arch or the descending portions. The term "thoracoabdominal aneurysm" refers to aneurysms of the descending thoracic aorta that extend distally to the abdominal aorta. Aneurysms of the ascending aorta are more common (60%), followed by those of the descending aorta (40%), whereas aortic arch and thoracoabdominal aneurysms are less frequent (10%)[5].

When in the aortic arch, they are usually contiguous to aneurysms of the ascending or descending aorta. Aneurysms may result from atherosclerotic disease, cystic medial degeneration, syphilis and other infections. Atherosclerosis is the main cause of descending thoracic aorta aneurysms, contrary to those of the ascending aorta which result from cystic medial degeneration (or cystic medial necrosis)[3,5].

As a result of flow disorders along the aneurismatic segment, blood may stagnate in the vessel walls allowing the formation of mural thrombi. These thrombi may embolize and impair circulation of distal arteries. In some cases, it can be challenging to distinguish a mural thrombus within an aortic aneurysm from the so-called localized intramural hematoma, as this entity is considered by some authors to be a morphological variant of aortic dissection.

Rupture or dissection are the primary complications. Both the origin and location can affect the speed of growth of an aneurysm and its tendency to dissect or rupture. The initial size is an important predictor of speed of growth of a thoracic aneurysm[6,8]. Other studies have noted that the chances of rupture are greater in chronic obstructive pulmonary disease, advanced age, and when there is pain associated with the aneurysm[9].

In view of the previously stated factors, the patient was admitted to undergo surgery. Initial tests showed abnormal creatinine levels, left ventricular ejection fraction within normal limits, no segment contractility alterations, and no obstructive coronary lesions on coronary angiography.

After coronary angiography, the patient progressed with increased nitrogenous waste products, which may characterize nephropathy induced by contrast medium. This disorder is characterized by a $\geq$ 0.5mg/dl absolute increase in creatinine or a relative increase $\geq$ 25% above baseline levels, with a peak 48 hours after administration of the contrast medium. The main risk factors are: age $\geq$ 70 years, preexisting chronic renal disease and diabetes mellitus. Many patients recover their renal function in one to three weeks; however a small percentage experiences partial recovery or no recovery at all[10].

During hospitalization, the patient progressed with abdominal pain associated with fever, leukocytosis, and ultrasound findings consistent with acute cholecystopathy. Concomitantly, there was a drop in hemoglobin levels which could be explained by both the infectious condition and the erosive gastropathy evidenced by upper digestive endoscopy. The patient was started on antibiotics, the clinical symptoms receded, and she was released to undergo surgery.

After clinical stabilization, the patient underwent surgery which consisted of implantation of an endovascular prosthesis with a stent through the aortic arch. Thoracic aorta aneurysms are generally resected and replaced by a vascular graft of appropriate size. Aortic arch aneurysms can be successfully excised, but the procedure remains challenging. Aneurysm resection generally requires extracorporeal circulation, hypothermia and circulatory arrest, and is associated with significant morbidity and mortality[3]. An alternative approach is to employ endoprostheses, which have a significant role in treating patients who are at a risk of aortic rupture but are poor candidates for surgery[11].

After being taken off extracorporeal circulation, the patient progressed with CAVB followed by cardiorespiratory arrest (CRA) and return to spontaneous circulation after 30 minutes. Following cardiorespiratory arrest, the patient experienced circulatory shock with the use of high concentrations of vasoactive drugs. During hemodynamic monitoring, there was a reduction of the cardiac index and concomitant increase in vascular resistance with the use of vasopressor agents, characterizing a low cardiac output syndrome in the cardiac surgery postoperative period.

Low cardiac output syndrome after heart surgery can be defined as the incapacity of the heart to maintain adequate tissue perfusion to match metabolic demand. Clinically, it can be characterized as systolic systemic arterial hypotension < 90 mmHg or a value 30 mmHg lower than the baseline levels during a 30-minute period, alterations in the level of consciousness, agitation, confusion and coma, decreased limb temperature, cyanosis, reticular livedo and oliguria (diuresis <20ml/h). In some situations, this syndrome can occur when SAP is greater than 100 mmHg due to increased systemic vascular resistance[12].

Postoperative low cardiac output may be related to decreased LV preload (cardiac tamponade, RV dysfunction), reduced contractility (ischemia or infarction due to poor intraoperative protection), tachycardia/bradycardia, etc. In the case here described, the patient was elderly and had risk factors for atherosclerosis (age, SABP, smoking), with a prior coronary angiography showing no obstructive lesions, progressing to CAVB followed by CRA. The post-CRA ECG can be analyzed from two different perspectives: the presence of an accelerated idioventricular rhythm (AIVR) identified by ventricular fusion complexes (highly suggestive of cardiac ischemia) or the presence of a nonsinus supraventricular rhythm with a left branch ventricular conduction disorder and presence of ST-segment elevation in the ínferolaterodorsal wall. Regardless of the form, the suspicion of an ischemic lesion has to be strongly considered. The causes of ischemia can be many, including: an atherosclerotic process (in which nonobstructive lesions may have a greater tendency to rupture, and resulting intracoronary acute thrombosis), aorta dissection, with retrograde dissection to the coronary system, and lesions due to ischemia and reperfusion after extracorporeal circulation.

The presence of lactic metabolic acidosis can be explained by the low cardiac output syndrome culminating in severe circulatory shock and significant tissue ischemia, mainly in splanchnic territory**.** CO_2 retention and significant hypoxemia can signal the presence of chronic obstructive pulmonary disease and/or alveolar gas exchange alterations due to the collection of interstitial liquid.

According to invasive monitoring data, we can conclude only that the patient had a reduced cardiac index concomitant with elevated systemic and pulmonary resistance levels and increased CVP. Volume optimization data (through dynamic variables, pulse pressure variation) or more accurate ventricular contractility data from parameters that better reflect inotropic function (LV and RV work indices), analysis of indirect data that would facilitate differential diagnoses (e.g., giant "V" wave in mitral regurgitation), a pulse oximetry spike in the presence of interventricular communication, and equalization of diastolic pressures in cardiac tamponade, could be very valuable for the clinical and hemodynamic evaluation of the patient and yield more precise interpretations, a more accurate assessment of differential diagnoses, and more adequate therapeutic measures. We should not forget that data on resistance are indirect measurements that may not represent important variations.

The main complications of thoracic aortic aneurysm repair include cerebrovascular accidents and hemorrhage. Myocardial infarction and renal failure frequently occur due to the massive nonphysiological stress from the aortic surgery. The most common causes of early death following surgery are infarction, heart failure, cerebrovascular accident, renal failure, hemorrhage, respiratory failure and septicemia. Advanced age, emergency surgery, prolonged time of transversal clamping, diabetes mellitus, previous aortic surgery and intraoperative hypotension are the most important factors that determine perioperative morbidity and mortality[3]. Complications associated with stent implantation are: blood flow leak around the prosthesis, known as "endoleak", displacement of the prosthesis from its site of implantation and, less frequently, stent fracture and intravascular infection[13-15].

During the immediate postoperative period, the patient progressed with sustained ventricular tachycardia refractory to drug treatment and electrical cardioversion. Potentially responsible factors could be ischemia, hydroelectrolytic disorders, acid-base disorders, hypoxemia and pro-arrhythmia effects, all of which should be identified and repaired. Cardiorespiratory arrest associated with pulseless electric activity followed, which could have resulted from myocardial ischemia, cardiac tamponade, pulmonary thromboembolism, drugs, hypertensive pneumothorax, acidosis, hyper or hypokalemia, hypovolemia, hypothermia, hypoxy or hypoglycemia. The arrest was refractory to cardiorespiratory and cerebral resuscitation maneuvers.

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Diagnostic hypothesis

Cause of death: low cardiac output syndrome or perioperative myocardial infarction.

Baseline disease: thoracic aortic aneurysm and arterial hypertension; associated diseases: nephropathy induced by contrast medium and acute cholecystitis

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Autopsy

At autopsy, the main finding was a large aneurysm originating at the root of the aorta coursing to the aortic arch (figure 3). An endoprosthesis (which was patent) had been implanted from the ascending aorta to the initial portion of the descending aorta. The portion of the descending aorta downstream of the endoprosthesis had semi-occlusive thrombosis, probably hindering blood flow (figure 4).

Along its full extension, the aorta was affected by severe atherosclerosis which was responsible not only for this aneurysm, but also for two others in the abdominal area, as well as for the thrombosis. The microscopic study showed the presence of atherosclerotic plaques, superimposed thrombi, and massive destruction of elastic fibers (figure 5). There is strong evidence that this destruction plays an important role in the genesis of aneurysms[16] as most patients with aortic atherosclerosis, even when severe, do not develop dilation which seems to be restricted to the cases in which elastolysis occur.

In many segments of the left ventricle, the microscopic study

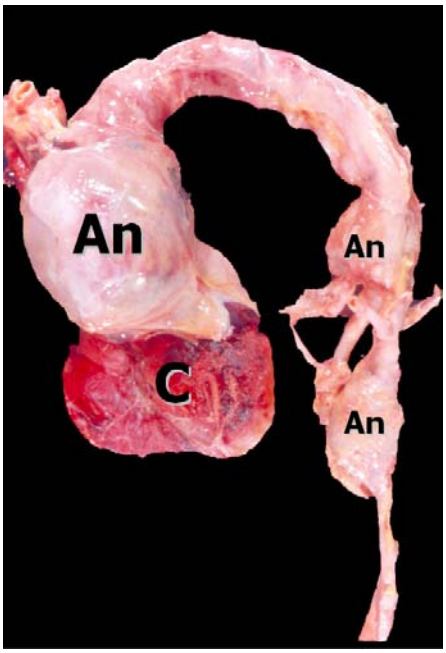

Fig. 3 - *Aorta showing aneurysm (An) of the ascending aorta and arch with dimensions similar to those of the heart itself (C). Aneurysms of the ascending and abdominal aorta.*

Fig. 4 - *Cross-section of the open aorta showing a semi-occlusive thrombus located close to the tip of the endoprosthesis.*

of the myocardium detected ischemic necrosis in contraction bands (figure 6). The extension of the infarction could not be adequately evaluated due to its short progression time, but this lesion was the final death-triggering factor by causing complete atrioventricular block and ventricular dysfunction.

The infarction was probably the result of a sum of factors: moderate coronary atherosclerosis with microangiopathy;

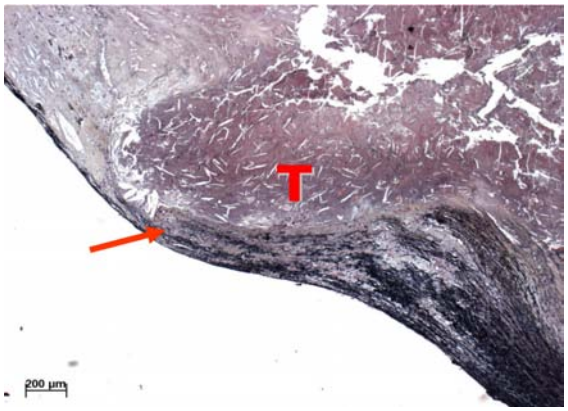

Fig. 5 - *Histological section of the aneurismatic aorta showing thrombus (T) containing crystals of cholesterol (nonstained clefts) and marked destruction of elastic fibers stained in black (arrow). Movat pentachromic staining, objective magnification = 5x .*

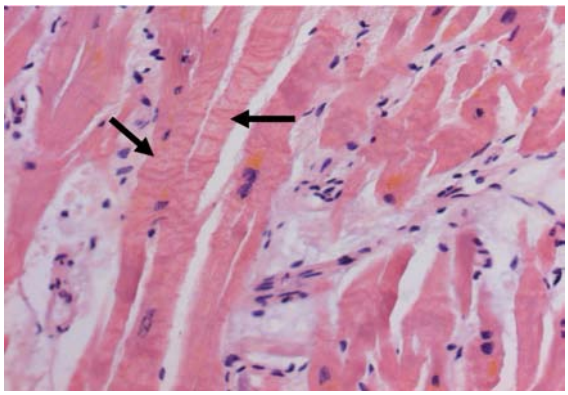

Fig. 6 - *Histological section of myocardium with ischemic necrosis in contraction bands (some necrotic cells are indicated by arrows). Hematoxylin and eosin stain, objective magnification = 20x.*

occasional extrinsic compression of the coronary arteries by the aneurysm; and abrupt circulatory difficulty due to the large incoming blood flow to the area of the descending aorta semi-occluded by a thrombus.

Moreover, the patient's overall status was complicated by the presence of several comorbidities: systemic arterial hypertension, chronic interstitial nephritis, renal lithiasis, chronic cholecystitis with calculi (which became more acute shortly before death), chronic obstructive pulmonary disease, and focal bronchopneumonia areas.

Anatomopathologic diagnosis: Atherosclerotic aortic aneurysms

Causa mortis: Myocardial infarction with a few hours of progression

(Dr. Paulo Sampaio Gutierrez)

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