

Case 1/2011 – Seventy-Four-year-old Female Patient with Sudden Apnea and Acute Cholecystitis, Five days after Acute Myocardial Infarction without Critical Coronary Lesions

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A 74-year-old female patient sought medical attention due to sudden dyspnea, five days after being discharged from a previous hospitalization due to acute myocardial infarction.

Five days before, the patient had sought medical attention, complaining of dyspnea triggered by minimal effort that had started three months before, accompanied by lower-limb edema, pain, paresthesia and weakness.

At physical examination (May 11, 2009) her heart rate (HR) was 85 bpm and blood pressure (BP) was 190/110 mmHg. Lung assessment showed pulmonary rales. Heart and abdominal examination were normal. There was no limb edema and the pulses were palpable and symmetric.

The electrocardiogram (ECG) (May 11, 2009) showed sinus rhythm, HR of 88 bpm, PR interval of 164 ms, SÂQRS –30° backward, QRS duration of 88 ms, QT interval of 383 ms. The patient had ST-segment elevation from V₁ to V₆, I and aVL (Figure 1).

The ECG performed on the following day (May 12, 2009) disclosed a HR of 54 bpm and a decrease in the ST-segment elevation (Figure 2). Chest x-ray was normal.

The laboratory assessment (May 11, 2009) showed hemoglobin, 12.9 g/dL; hematocrit, 41.0%; leukocytes, 17,200/mm³ (1.0% meta; 2.0% band cells and 84.0% segmented; 5.0% lymphocytes and 8.0% monocytes); platelets, 389,000/mm³; urea, 46 mg/dL; and creatinine, 1.07 mg/dL (glomerular filtration rate, 53 ml/min.1.73 m²).

Myocardial infarction was diagnosed and the patient received ASA 200 mg and nitrate. A coronary angiography was performed and disclosed 40% stenosis in the right coronary,

in the diagonal branch and in the first diagonal branch of the left coronary artery (Table 1).

The echocardiogram (May 13, 2009) disclosed diameter of aorta, 33 mm; of the left atrium, 37 mm; of the right ventricle, 20 mm; septal thickness, 13 mm, posterior wall, 12 mm, left ventricular diameters (diastole/systole) in 45/28, ejection fraction of 68.0% (Teicholz). The left ventricle was hypertrophic and did not have regional motility alterations. Heart valves and aortic arch were normal.

The patient developed atrial fibrillation and was discharged on May 13, 2009, having received a prescription for warfarin, acetylsalicylic acid 100 mg, atorvastatin 20 mg, captopril 75 mg, propranolol 80 mg and 20 mg of omeprazole.

At clinical consultation (May 15, 2009), the patient complained of sudden dyspnea and lower-limb pain. At physical examination, the patient presented regular general status, good skin color, was hydrated, acyanotic, anicteric, tachypneic (28 respiratory incursions per minute) and afebrile (BT 35.7°C). HR was 120 bpm and BP was 120/70 mmHg. Lung assessment showed decreased vesicular murmur in both hemithoraces. Heart assessment showed arrhythmic heart sounds, with no murmurs. The abdomen was globular, flaccid, and painful at superficial and deep palpation in the right hypochondriac region (+ Murphy's sign). The hydro-aerial noise was preserved and there was no visceral megaly. Extremity assessment showed thin distal pulses with ++/4+ edema.

The ECG showed atrial fibrillation with high ventricular response and the chest x-ray did not add any diagnostic information. The echocardiogram was similar to the previous one (May 13, 2009), except for the description of atypical motility in the interventricular septum.

The laboratory assessment of May 16 showed D-dimer of fibrin, = 2,174 ng/mL, CK-MB = 49.7 ng/mL, troponin I = 1.46 ng/mL, glycemia = 117 mg/dL, cholesterol = 171 mg/dL, HDL-C = 28 mg/dL, LDL-C = 113 mg/dL, triglycerides = 151 mg/dL, hemoglobin = 12.89 mg/dL, hematocrit = 40.0%, leukocytes = 13,500/mm³ (neutrophils = 79.0%, 13.0% of lymphocytes and 8.0% of monocytes), platelets = 402,000/mm³, CK-MB = 44.6 ng/mL, troponin I = 1.46 ng/mL, creatinine = 1.29 mg/dL (GF = 42 ml/min.1.73 m²), urea = 119 mg/dL and INR = 1.3. Gasometry disclosed: pH = 7.34, pCO₂ = 40 mmHg, pO₂ = 152 mmHg, O₂ saturation = 99.7%, bicarbonate = 27.2 mEq/L, base excess = –3.7 mEq/L; lactate = 17.8 mg/dL. Sodium was 137 mEq/L and potassium was 3.9 mEq/L. A

Keywords

Dyspnea; cholecystitis, acute; infarction; renal insufficiency.

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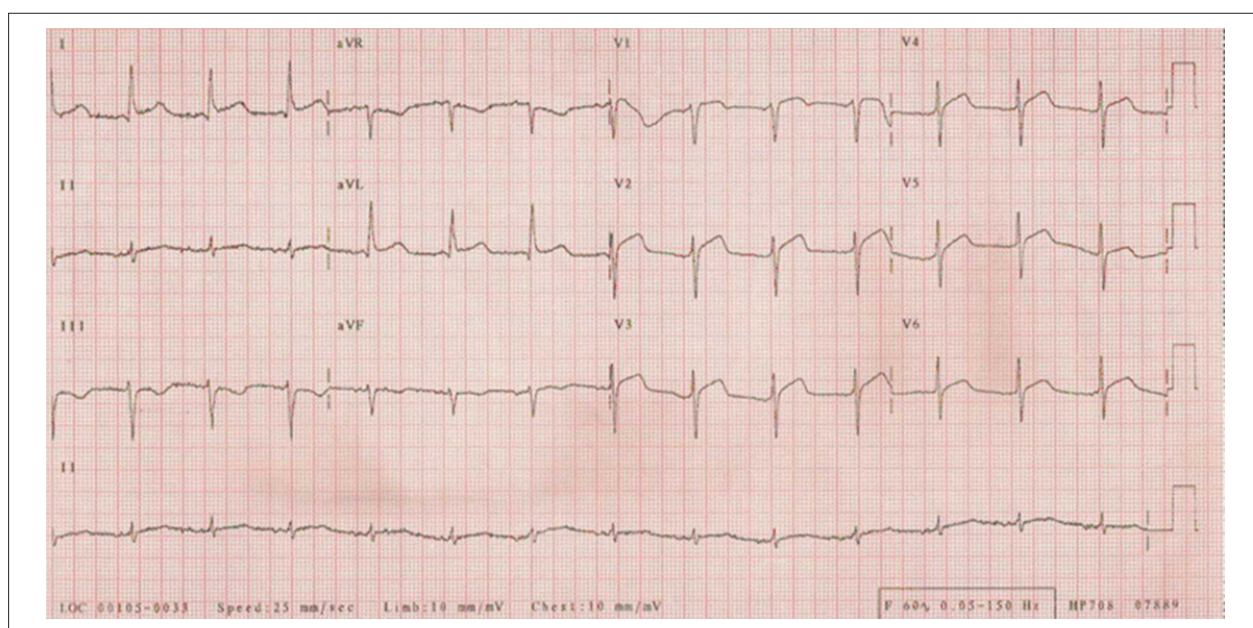


Figure 1 - ECG – sinus rhythm; ST-segment elevation from V₁ to V₆ and I and aVL, with positive T waves, suggestive of evolving extensive anterior acute infarction.

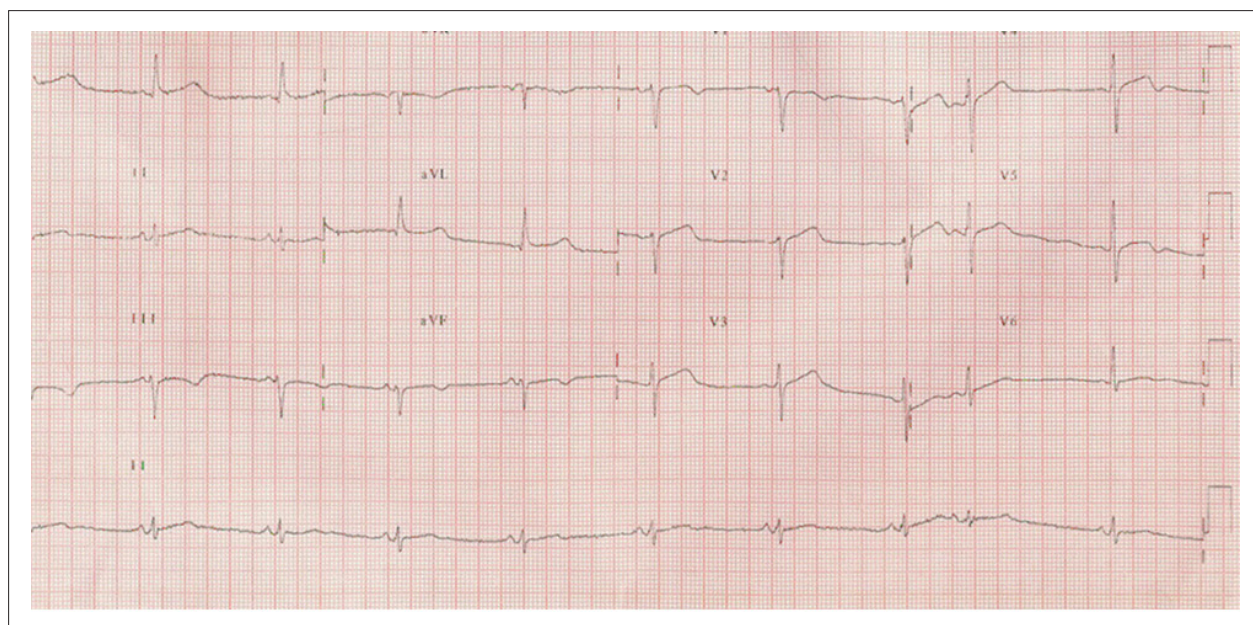


Figure 2 - ECG - decrease in ST-segment elevation.

Table 1 - Myocardial injury markers

Date	CK-MB (ng/ml)	Troponin (ng/ml)
May 11 2009	9.1	10.5
May 11 2009	7.29	9.66
May 12 2009	6.16	5.43
May 13 2009	8.60	5.7

diagnosis of acute cholecystitis was attained. Abdominal CT showed a distended gallbladder, with thickened and irregular walls and a thin perivesicular liquid lamina.

Arterial angiotomography of the pulmonary arteries was normal and surgical management was indicated.

The patient was submitted to a surgical intervention in the night of the same day (May 16, 2009). The surgical findings revealed gallbladder with a necrotic aspect, without perforations and presence of calculus inside it.

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The histopathological findings were chronic calculous cholecystitis, xanthogranulomatous, with cholesterosis and acute surge, represented by the accumulation of xanthomatous macrophages, extensive mucosal loss due to necrosis, covered by fibrin and neutrophils, with fibrosis, edema and neutrophilic and eosinophilic exudate permeating the wall.

The patient developed a septic-like condition, with kidney failure and need for vasoactive drugs, in spite of the antibiotic therapy with ceftriaxone and metronidazole.

Laboratory assessment (May 22, 2009) showed hemoglobin = 10.6 g/dL, hematocrit = 33.0%, leukocytes = 26.000/mm³ (2.0% of metamyelocytes, 18.0% of band cells, 75.0% of segmented cells, 1.0% of lymphocytes and 4.0% of monocytes), platelets = 249,000/mm³, urea = 169 mg/dL, creatinine = 3.3 mg/dL, lactate = 140 mg/dL, lipase = 439 U/L, potassium = 6.6 mEq/L and sodium = 142 mEq/L. Blood cultures were negative for aerobic and anaerobic agents, as well as fungi. Venous gasometry (7 PM, May 22), showed a pH = 6.98, pCO₂ = 55 mmHg, pO₂ = 43.7 mmHg, O₂ saturation = 57.3%, bicarbonate = 12.2 mEq/L and base excess = -18.6 mEq/L.

A new abdominal CT without contrast (May 22, 2009) showed a normal-sized liver, with regular architecture and homogenous parenchyma. The portal and hepatic veins were normal in caliber, with no dilatation of the intra- or extrahepatic biliary ducts. There were no abdominal collections. The spleen was of normal size, had regular architecture and homogenous parenchyma. The pancreas was of normal size, with normal dimensions and architecture. There was no Wirsung duct dilatation.

On the night of May 22 (9 PM), the patient presented bradycardia, followed by pulseless electrical activity. There was no response to the resuscitation efforts and she died on the 6th postoperative day.

Clinical aspects

The present case refers to a 74-year-old female patient, with no relevant clinical antecedents, who sought medical attention due to dyspnea triggered by minimal effort followed by lower-limb edema, pain, paresthesia and weakness. At this time the clinical assessment showed arterial hypertension and pulmonary rales.

Based on the history and the clinical examination, the first hypothesis to explain the patient's symptoms was heart failure (HF). When Framingham's criteria were applied to this patient, a major criterion (pulmonary rales) and two minor ones (effort dyspnea and lower-limb edema) were found¹.

It is noteworthy the fact that elderly female patients can present atypical manifestations of myocardial ischemia, such as dyspnea, weakness and fatigue. These patient's complaints, therefore, might correspond to an ischemic equivalent.

At the initial assessment, the patient was submitted to an electrocardiogram (ECG), chest x-ray and blood tests. The first showed ST-segment elevation; the chest x-ray was normal and the blood tests showed elevated myocardial necrosis markers.

Based on these data, the diagnostic hypothesis to be added is acute myocardial infarction (AMI). According to the universal

definition of AMI, the detection of increase or decrease of myocardial necrosis markers, preferably troponin, associated to the presence of myocardial ischemia are necessary for the diagnosis to be attained, with at least one of the following criteria: symptoms of ischemia, ECG alterations indicating new ischemia (new ST-T segment alteration or LBBB), development of pathological Q wave at the ECG, evidence of loss of viable myocardial mass or new alteration in segmental motility at imaging assessment².

The initial approach of the patient with AMI and ST-segment elevation includes platelet antiaggregants and immediate myocardial reperfusion therapy (thrombolysis or primary angioplasty)³. The patient underwent a coronary angiography, which did not disclose significant obstructive coronary lesions. The absence of significant lesions, however, does not rule out the diagnosis of AMI, which can occur in patients with no evidence of significant coronary atherosclerosis (Table 2)⁴.

At the subsequent assessment, the patient underwent an echocardiogram, which disclosed the presence of left ventricular hypertrophy (LVH), without systolic dysfunction or regional motility alterations. The presence of LVH can lead to heart failure (HF) with preserved ventricular systolic function, which is more common in elderly female patients with arterial hypertension⁵. Moreover, LVH can cause infarction without significant coronary atherosclerosis due to myocardial oxygen demand–supply disproportion. The absence of alterations in segmental motility, associated with the small elevation in biomarkers also suggests that the infarction area is a small one.

After the initial treatment, the patient presented atrial fibrillation (AF). This is the most common arrhythmia in patients

Table 2 – Causes of AMI in patients without coronary atherosclerosis.

Arterites
- Systemic Lupus Erythematosus
- Polyarteritis nodosa
- Syphilitic arteritis
- Takayasu's Disease
Trauma
Coronary spasm
Aortic dissection
Coronary dissection
Coronary embolism
Congenital anomaly
Myocardial Oxygen Demand–Supply Disproportion
- Aortic stenosis/failure
- Ventricular hypertrophy
- Thyrotoxicosis
Hematological diseases
- Thrombocytosis
- Polycythemia
- Hypercoagulability
Others
- Cocaine use

with HF and its treatment aims at controlling heart rate (HR), rhythm reversal and prevention of thromboembolic events.

In case of patients with preserved ventricular function, beta-blockers, calcium-channel blockers and digitalis can be used to control the HR. The rhythm reversal can be attained through electric or pharmacological cardioversion; however, when compared to the HR control, there is no proof of benefits regarding reduction in mortality⁶.

The prevention of thromboembolic events with oral anticoagulation or platelet antiaggregants must be carried out according to the risk stratification. Patients with previous thromboembolic event are considered high-risk and must receive oral anticoagulation. Moderate risk factors include age > 75 years, hypertension, HF, ejection fraction < 35% and diabetes; patients with two or more risk factors must receive oral anticoagulation. The patient was receiving warfarin at hospital discharge⁷.

At the hospital discharge, the diagnoses would be AMI (secondary to LVH), HF with preserved ventricular function, AF and arterial hypertension.

After a short period at home, the patient returned to the hospital complaining of sudden dyspnea and lower-limb pain. At physical examination, she was tachycardic and tachypneic, referred pain at abdominal palpation with positive Murphy's sign, in addition to lower-limb edema.

The main diagnosis to be ruled out in this situation is pulmonary thromboembolism (PTE), which can be secondary to deep venous thrombosis after immobilization during hospitalization due to clinical disease. Considering the patient's symptoms and using the risk score for PTE, the patient was considered to be high risk⁸.

The measurement of D-dimer has high sensitivity for the diagnosis of pulmonary embolism; however, it is not specific. The increase can be found in patients with cancer, AMI, sepsis or any other systemic disease. In patients with low probability of PTE, the finding of a negative D-dimer rules out the diagnosis⁹. In patients with high probability, such as the present case, its measurement does not allow confirming or ruling out PTE and, therefore, it is of little clinical usefulness. The patient underwent an angiotomography of the pulmonary arteries, which allowed ruling out PTE.

The patient underwent a new echocardiogram, which did not differ from the first one, except for the atypical movement of the interventricular septum, with a new elevation in myocardial necrosis markers. At this moment, another hypothesis to be mentioned is myocarditis, which is associated with the new segmental dysfunction, enzyme elevation and HF picture. However, the normal ventricular function would not explain the systemic findings in the present case. The cardiac involvement is probably secondary to the systemic disease.

The patient presented a positive Murphy's sign, suggesting the presence of biliary pathology, and after tomographic confirmation, she was submitted to a cholecystectomy. At that time, the laboratory assessment showed the presence of a new AMI, acute renal failure and signs of inadequate peripheral perfusion (BE = -3.7).

As for the renal involvement, the possibility of contrast-mediated injury caused by the previous use of iodinated contrast medium must be recalled. That, however, is a small possibility, due to the time since the procedure and the previously normal renal function.

One possibility to be considered, in view of the multisystemic involvement, is cholesterol embolization. This possibility results from the embolism of cholesterol crystals from severe aorta atherosclerosis and occurs after endovascular procedures in up to 2.0% of the patients¹⁰. Although controversial, the use of oral anticoagulation can increase the risk of embolization^{11,12}. Among the most common clinical manifestations are cyanosis of extremities (blue finger syndrome), livedo reticularis and kidney failure. Ischemic manifestations of other organs, such as bladder and digestive tract, can also occur. The laboratory assessment might disclose inflammatory marker elevation and eosinophilia. The result of the patient's coronary angiography, however, suggested that the patient did not have significant atherosclerosis and the clinical and laboratory assessment was absolutely not compatible with the diagnosis.

Considering all symptoms and signs and the examinations carried out since the patient first came to the hospital, one can identify the presence of neurologic symptoms (lower-limb weakness and paresthesia), heart disease, (AMI), kidney involvement (ARF) and gastrointestinal symptoms (cholecystitis). This clinical picture suggests the hypothesis of systemic vasculitis and, in this case, Polyarteritis nodosa (PAN).

PAN is a necrotizing systemic vasculitis that typically affects medium-caliber arteries, with the occasional involvement of small arteries. In contrast with other vasculites (for instance, microscopic polyarteritis, Wegener's granulomatosis), PAN is not associated with the presence of antineutrophil cytoplasmic antibodies (ANCA)¹³. The clinical presentation involves systemic symptoms and kidneys, skin, joints, muscles, nerves and gastrointestinal tract are commonly affected. PAN can potentially affect any organ; however, it classically spares the lungs.

AMI is an unusual manifestation in PAN; however, myocardial ischemia can occur, secondary to coronary involvement¹⁴. HF can result from ischemic cardiopathy or be secondary to arterial hypertension caused by kidney disease.

The most common gastrointestinal manifestations include abdominal pain due to vasculitis of the mesenteric arteries. Nausea, vomiting, melena, diarrhea and gastrointestinal bleeding can be found. Rarer clinical presentations can occur, mimicking acute cholecystitis, caused by acute vasculitis limited to the cystic artery¹⁵.

According to the criteria published by the American College of Rheumatology¹⁶, the patient described here met three of these criteria, including lower-limb weakness and pain, recent increase in diastolic blood pressure (> 90 mmHg) and elevated levels of urea and creatinine. The diagnostic confirmation, however, requires the documentation of the presence of vasculitis

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Diagnostic hypothesis

PAN with cardiac, renal and gastrointestinal tract involvement.

Necropsy

The heart weighed 474 grams. There was left ventricular concentric hypertrophy and areas of transmural infarction undergoing organization in the anterolateral wall of the LV (Figure 3) and the anterior wall of the right ventricle (RV), where the occluded epicardial coronary branch was observed (Figure 4). Histological assessment of the myocardium showed an infarction with approximately 7-14 days of evolution. The epicardial coronary arteries were dissected and submitted to histological assessment, disclosing an acute inflammatory process affecting all arterial layers, with fibrinoid necrosis of the wall and acute thrombosis of the distal segments of the anterior interventricular artery and the right anterior ventricular arteries (Figure 5). There were no granulomatous lesions or a significant number of eosinophils in the inflammatory infiltrate. The histochemical analysis of the affected segments was negative for infectious agents (bacteria and fungi). Concentric fibrointimal thickening in the 4th centimeter of the anterior interventricular artery and organized occlusive thrombosis of the epicardial coronary branch of the RV anterior wall were observed. The lungs weighed 1,706 g together and showed extensive edema and alveolar hemorrhage.

There was concentric fibrointimal thickening of arteries in focal areas of the stomach wall, pancreas and peribronchial region (bronchial artery branches). The kidneys had rare isolated foci of glomerular sclerosis, with no glomerular nephritis or histological evidence of vasculitis in the parenchymatous branches. However, the renal artery was not histologically evaluated. There was no peritonitis and the surgical bed of the gallbladder had no abnormalities. The previous anatomopathological examination of the gallbladder, removed during surgery, had shown a chronic inflammatory process with an acute surge, mucosal ulceration and presence of calculus in the lumen. However, additional histological sections, carried out after the patient's death, showed arterial segments with concentric fibrointimal thickening and acute vasculitis foci with intense neutrophilic inflammatory process, fibrinoid necrosis of the arterial wall and acute thrombosis (Figure 6).

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Anatomopathological diagnoses

Systemic arterial hypertension with LV concentric hypertrophy; chronic calculous cholecystitis; polyarteritis nodosa with acute necrotizing vasculitis and thrombosis of coronary branches and arteries of the gallbladder wall; acute necrotizing cholecystitis; acute myocardial infarction, multifocal, with a 10-day evolution; pulmonary edema and alveolar hemorrhage (cause of death).

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Figure 3 - Cross-sectional view of ventricles. Concentric left ventricular hypertrophy and small intramural infarction undergoing organization (arrow) in its anterolateral wall.



Figure 4 - Macroscopic detail of the right ventricular anterior wall. Ventricular wall thinning due to transmural infarction undergoing organization (arrow) and occlusion of epicardial coronary lumen (arrowhead).

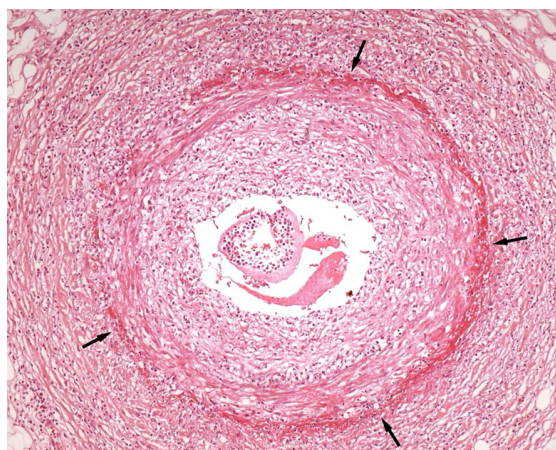


Figure 5 - Histological section of the first centimeter of the right anterior ventricular coronary. Inflammatory process affecting all layers of the arterial wall (pan-arteritis), with a circumferential area of fibrinoid necrosis at the external border of the middle layer (arrows). Hematoxylin-eosin, X 100.

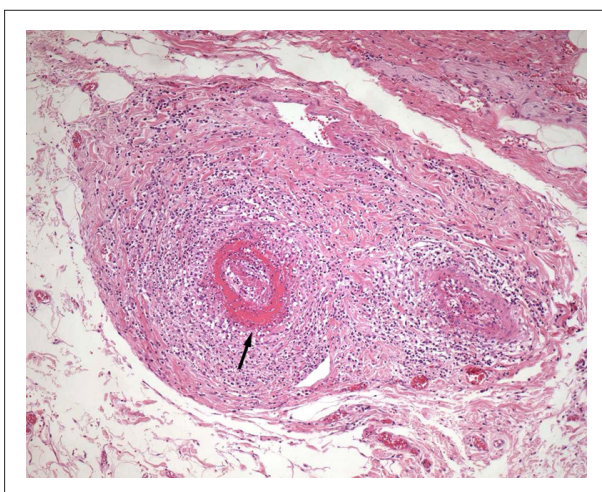


Figure 6 - Histological section of the gallbladder wall. Intense mixed inflammatory process affecting all layers of the small arteries (pan-arteritis), with thrombosis and vascular occlusion, in addition to fibrinoid necrosis of the wall (arrow). Hematoxylin-eosin, X 50.

Comments

The present case describes an elderly female patient, with systemic arterial hypertension, without significant coronary atherosclerosis, who had acute cholecystitis five days after an episode of acute myocardial infarction and died on the 6th postoperative day after the cholecystectomy. The necropsy disclosed acute vasculitis of the coronary branches and arteries of the gallbladder wall, non-infectious, and involvement of

vascular segments that presented intense mixed inflammatory process, affecting all layers of the small muscular arteries, with areas of fibrinoid necrosis and acute thrombosis. The finding of coronary segment organized thrombosis and fibrointimal thickening of small-caliber artery segments in several organs is compatible with cicatricial lesions of previous vasculitis.

The anatomopathological classification of the systemic non-infectious vasculites depends basically on the type of vessel involved and the histopathological characteristics of the observed lesions¹⁷. PAN classically affects segments of small- and medium-sized muscular arteries and can result in the formation of aneurysms at later phases. The inflammatory infiltrate is of the mixed type, affecting all arterial layers, with the presence of fibrinoid necrosis in the acute phase. The presence of vascular lesions is characteristic in different phases of the evolution, with areas of acute necrotizing arteritis, areas undergoing organization and cicatricial areas¹⁸. Gastrointestinal tract involvement is frequent in this disease, as well as in other systemic necrotizing vasculites and can have a negative impact on patient survival.

In the present case, although there was no clinical and laboratory characterization of the disease, the observed anatomopathological lesions are indicative of polyarteritis nodosa, according to the aforementioned description. The coronary involvement with consequent acute myocardial infarction has been described before¹⁴, with a case report somewhat similar to the present one, in which the patient presented acute cholecystitis and several subsequent complications attributed to PAN, including acute myocardial infarction¹⁹.

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