Article / Autopsy Case Report
Artigo / Relato de Caso de Autópsia

Myocardial abscess as a complication of an infected arteriovenous fistula: autopsy report

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ABSTRACT

Myocardial abscess is a severe and life-threatening infectious complication that is commonly but not exclusively associated with infective endocarditis. It may also be developed in necrotic myocardial tissue, post trauma, in septic burn patients, in transplanted heart, in ventricular aneurysm and post angioplasty. Patients on hemodialysis are prone to bacteremia, and infectious complications occur in 48-73% of cases. Myocardial abscess is a rare complication of an infected arteriovenous fistula. We present an autopsy report of a hemodialysis patient who had an arteriovenous fistula with a polytetrafluoroethylene graft where a local infection developed. The patient presented with fever and toxemia. On post-admission day 2, he unexpectedly suffered sudden cardiopulmonary arrest and died. The autopsy revealed a myocardial abscess, near a branch of the left coronary artery, with septic embolism.

Keywords: Heart Diseases/pathology; Abscess/etiology; Arteriovenous Shunt, Surgical; Renal Dialysis; Staphylococcus aureus.

CASE REPORT

We report the case of a 57-year-old male patient, previously diagnosed with systemic arterial hypertension, diabetes mellitus, coronary heart disease, and chronic kidney disease. He had been on hemodialysis and consequently had an arteriovenous fistula (AVF) in the left arm. He had been under regular treatment with captopril and atenolol. Previously, the patient had been hospitalized for four days because he was bleeding from the AVF, which required surgical ligation. The polytetrafluoroethylene (PTFE) graft was left in place, and a Shiley catheter was placed in the right subclavian vein. During that first hospital stay, he had fever. The patient had returned to the hospital two days after discharge complaining of disorientation, hypoactivity, fever, nausea, vomiting, and abdominal distension. He also reported anuria. The initial physical examination revealed the following: regular pulse (80 bpm); blood pressure, 120 × 70 mmHg; respiratory rate, 20 breaths/min; temperature, 36.3 °C. Although the patient was conscious sometimes he presented some spatiotemporal disorientation. Hemodialysis was being performed through the Shiley catheter inserted into the right subclavian vein. On his left arm, there was a prominence at the site where the AVF had recently been ligated. In addition, the patient presented with the following: rhonchi in the

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lung bases; rhythmic heart sounds with systolic mitral murmur; distended, diffusely painful abdomen with signs of ascites; liver palpable 3 cm below the right costal margin; no rebound tenderness; and mild lower limb edema. He was hospitalized with the diagnosis of infection at the AVF site.

The initial laboratory test results are presented in Table 1. Urinalysis showed proteins, 731.8 mg.dL^{-1}; glucose, 169 mg.dL^{-1}; leukocytes, 82,000 cells.mm^{-3}; red blood cells, 50,000 cells.mm^{-3}; and presence of budding yeast. Serology was negative for antibody to human immunodeficiency virus (HIV) and antibody to hepatitis C virus, as well as for hepatitis B surface antigen, but positive to hepatitis B surface antibody. The chest X-ray showed normal lung parenchyma, enlarged heart size and the presence of a catheter into the topography of the right superior vena cava. The electrocardiogram revealed sinus rhythm, left ventricular overload, and diffuse changes in ventricular repolarization (Figura 1). An abdominal ultrasound showed signs of chronic parenchymal liver disease and portal hypertension, as well as splenomegaly and moderate ascites. A cerebral contrast-enhanced computed tomography scan revealed an enhancement of lateral fissures and cortical sulci as well as normal ventricular systems. Two blood cultures were performed, and both were positive for oxacillin-sensitive *Staphylococcus aureus* (time to detection, 17 hours).

The patient was treated with captopril, atenolol, aspirin, simvastatin, vancomycin, and ceftriaxone. He also underwent a hemodialysis session, during which he received a transfusion of packed red blood cells.

Probing of the surgical incision in the left arm showed purulent discharge, and the stitches were removed in order to allow spontaneous drainage of the remaining secretion. During hospitalization, the hemodynamic parameters and vital signs remained stable despite daily peaks of fever (38 °C). On postadmission day 2, the patient was unexpectedly found dead in his bed.

An autopsy was performed, and the findings were as follows*: heart weight, 627.0 g (normal range, 248-431 g) showing smooth epicardial surface with numerous petechiae. Left ventricular concentric hypertrophy and a yellowish-white area measuring 1.2 cm in its longest axis located in the basal topography of the antero lateral wall of the left ventricle, in close proximity to a branch of the left coronary artery (Figure 2), no other macroscopic lesion was observed in the rest of myocardial tissue, nor in the cardiac valves. It was also observed coronary atherosclerosis. Microscopic examination revealed acute arteritis of a branch of the left coronary artery, accompanied by septic thrombus and myocardial abscess in the adjacent tissue, with colonies of Gram-positive cocci (Figure 3). The lungs had two nodular, friable abscesses. One

* Moore GW. 227 - Adult autopsy weights and templates. http://www.netautopsy.org/axsop/axsop227.htm

Table 1 – Laboratory test results

Variable	Measure	Result	RV	Variable	Measure	Result	RV
Hemoglobin	g.dL^{-1}	8.4	12.3-15.3	AST	U.L^{-1}	21	10-35
Hematocrit	%	24	360-45.0	ALT	U.L^{-1}	14	9-43
Leukocytes	mm^3	3,200	4.4-11.3 × 10^3	ALP	U.L^{-1}	185	10-100
Metamyelocytes	%	2	0	GGT	U.L^{-1}	142	2-30
Band neutrophils	%	25	1-5	Total bilirubin	mg.dL^{-1}	2.6	0.3-1.2
Segmented neutrophils	%	60	45-70	K	mEq.L^{-1}	4.7	3.5-5
Eosinophils	%	0	1-4	Na	mEq.L^{-1}	133	135-146
Basophils	%	0	0-2.5	iCa	mmol.L^{-1}	1.4	1.11-1.14
Lymphocytes	%	5	18-40	Mg	mg.dL^{-1}	2.0	1.6-2.6
Monocytes	%	8	2-9	Creatinine	mg.dL^{-1}	4.6	0.4-1.3
Platelets	mm^3	218,000	150-400 × 10^3	Urea	mg.dL^{-1}	90	10-50
Prothrombin time	INR	1.62	1	Glycemia	mg.dL^{-1}	94	70-99
CRP	ng.mL^{-1}	344	<5				

RV = reference value; AST = aspartate aminotransferase; ALT = alanine aminotransferase; ALP = alkaline phosphatase; CRP = C-reactive protein; GGT = gamma glutamyltransferase; K = potassium; Na = sodium; iCa = ionized calcium; Mg = magnesium; INR = international normalized ratio.

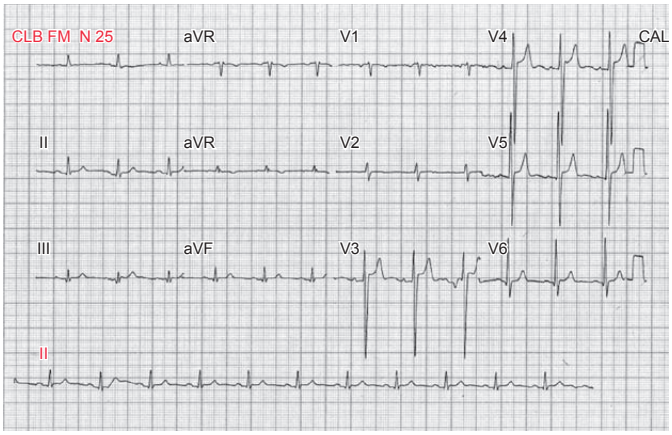

Figure 1 – Electrocardiogram showing sinus rhythm, PR interval of 0.16 ms, left heart overload, and diffuse changes in ventricular repolarization.

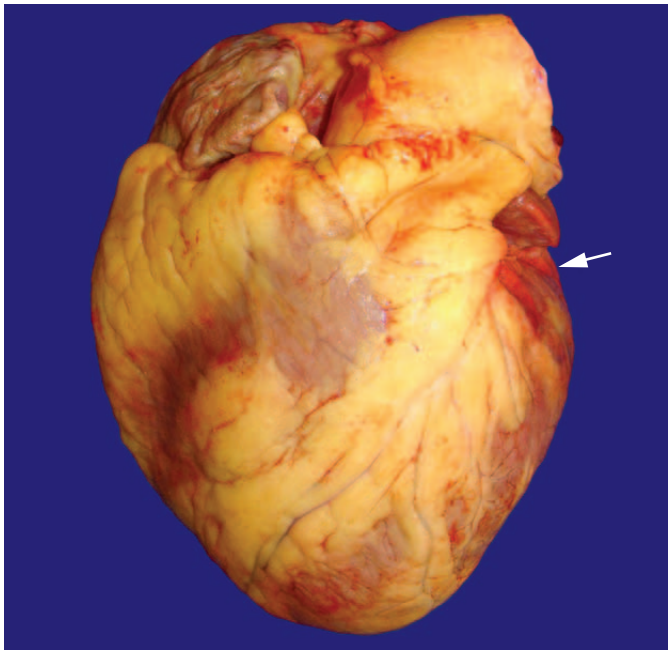

Figure 2 – Panoramic picture of the heart showing epicardial surface with petechiae. Hyperemic area in the topography of the upper portion of the left ventricle (arrow) that corresponds to the area of the abscess.

of the abscesses was in the lower lobe of the left lung and measured 0.6 cm, whereas the other was in the right upper lobe and measured 1.5 cm. Both abscesses had colonies of Gram-positive cocci (Figure 4). In the topography of the ligated AVF, there was a skin abscess, in the reticular dermis and underlying subcutaneous tissue, consistent with cellulitis and colonies of Gram-positive cocci suggestive of *Staphylococcus* sp. (Figure 5). Other autopsy findings included reactive splenomegaly (spleen weight = 626 g; normal weight = 112 g) with congestion of the red pulp, both of which were secondary to infection. In the lungs we found pulmonary congestion with siderophores (the so-called "heart failure cells") without diffuse alveolar damage (Figure 6A). The liver was enlarged (liver weight = 2,570 g; normal weight = 1,330-2,100 g) with chronic passive congestion. In addition, we found an end-stage renal disease (Figure 6B). The kidneys were atrophic with acquired cystic disease weighting: right kidney 85 g and left kidney 88 g (normal value for both = 230-440 g). The macroscopic examination of the central nervous system was unremarkable. Furthermore, there was generalized atherosclerosis in the aorta, renal arteries, and the circle of Willis.

DISCUSSION

A rare but serious clinical entity, myocardial abscess is characterized by suppurative infection of the myocardium, perivalvular structures, or cardiac conduction system. Myocardial abscess is found in 0.18-1.52% of all autopsies of adults.[1-3]

We described the case of a chronic kidney disease patient who had been on hemodialysis and presented with myocardial abscess and pulmonary abscesses following the infection of a AVF. One of the first cases of myocardial abscess was described by Cossio and Berconsky in 1933, in an autopsy report describing a pneumococcal abscess in infarcted myocardial tissue.[4] Since then, autopsy studies have revealed that myocardial abscess is common in cases in which abscesses are found in other organs following septicemia.[5] Another autopsy study reported that 21 of 23 patients with myocardial abscess had abscesses in other organs, most commonly in the kidneys, lungs, and brain.[1] In that study, the myocardial abscesses were small, raising the hypothesis that myocardial abscess is so severe that patients do not survive long enough for the abscess to increase in size.[1] A review of 63 autopsy reports showed the concomitant presence of myocardial abscess and abscesses in other organs in 81% of the cases.[3]

Myocardial abscess can be caused by bacteremia, by fungemia, or by direct extension of mural or valvular infective endocarditis[1,3] of native or prosthetic valves. An autopsy case series, evaluating 40 cases of infective endocarditis with myocardial abscess, Gonzalez-Vilchez showed that myocardial abscess is associated with infective endocarditis of native valves in 67.5% of cases;

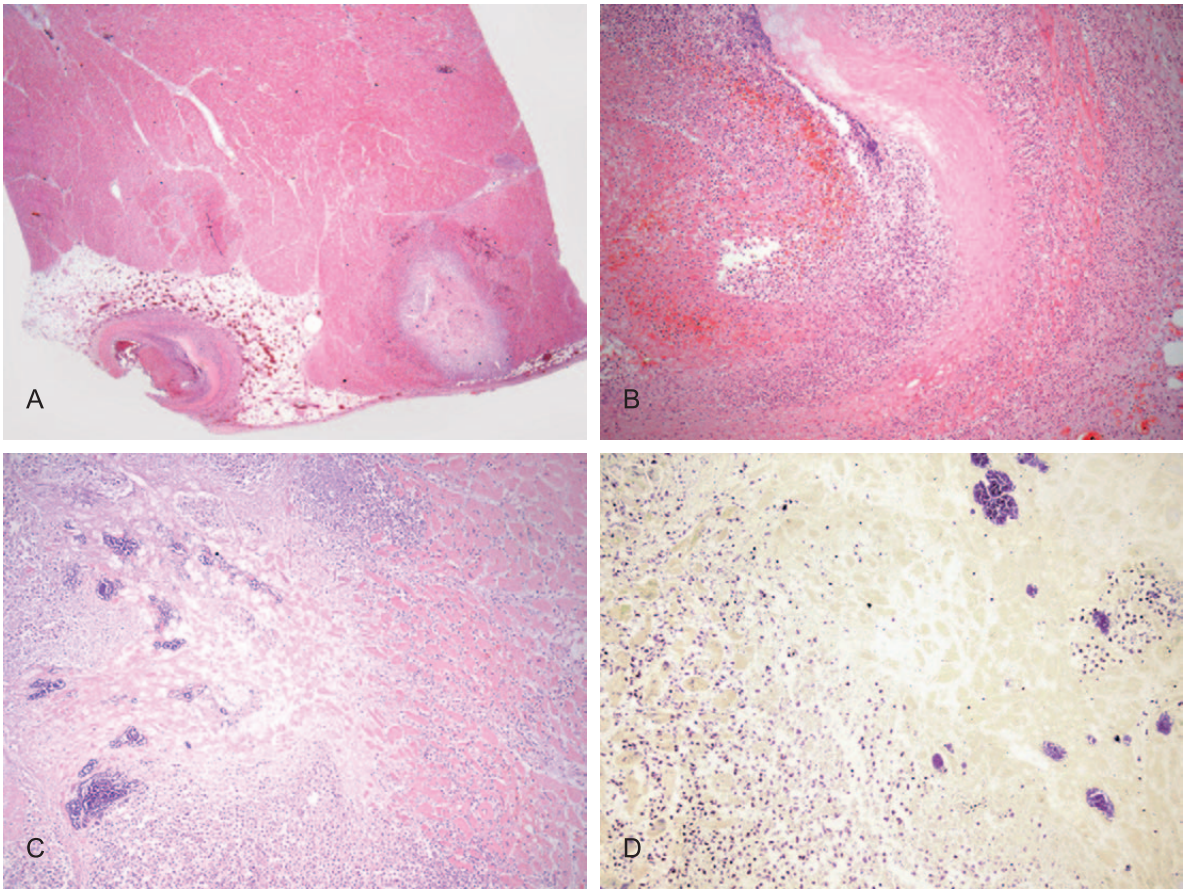

Figure 3 – Photomicrographs of the myocardium: **A** - (HE-25X) branch of a coronary artery with thrombus and an adjacent myocardial abscess; **B** - (HE-100X) detail of the branch of the coronary artery with septic thrombus; **C** - (HE-100X) detail of the myocardial abscess with bacterial colonies; **D** - (Brown-Hopps-200X) detail of the abscess showing gram-positive bacterial colonies.

in the remaining 32.5%, myocardial abscess is associated with infective endocarditis of prosthetic valves. In that case series, *Staphylococci* sp. accounted for more than 30% of cases, and the most commonly affected site was the aortic valve.[6] In another autopsy case series, involving 46 cases of myocardial abscess with infective endocarditis, the most commonly affected site was also found to be the aortic valve (in 30% of the cases), followed by the interventricular septum, mitral valve apparatus, and papillary muscles.[7]

In the case reported here, there was no evidence of infective endocarditis, lending credence to our hypothesis that the myocardial abscess was due to septic embolism. The bacteremia and the fact that the blood cultures were positive for *S. aureus* bolstered that hypothesis. Albeit rarer, septic embolism to the coronary arteries constitutes another cause of myocardial abscess. Septic embolism can occur as a complication of infective endocarditis[8] or of distant foci of infection.[9] In such cases, multiple microabscesses can be seen in the vicinity of the myocardial capillaries, far from the mitral valve apparatus. It has been demonstrated that even such microabscesses can be fatal when accompanied by ventricular arrhythmias.[9] Other studies have reported that 74% of coronary embolisms occur in the left coronary artery, the anterior descending artery being the most susceptible because of the disparity between the vascular diameter and blood flow.[8,10,11] In the case reported here, we found myocardial abscess near a branch of the left coronary artery, on the anterior wall of the left ventricle. We also found acute arteritis with septic thrombus with colonies of Gram-positive cocci.

Myocardial abscess can develop in association with other clinical entities, including penetrating trauma, septicemia in burn patients, infected pseudoaneurysm, transplanted heart infection following sternotomy, HIV-associated myocarditis, and infection of a left ventricular aneurysm.[5] Even though rare, myocardial abscess

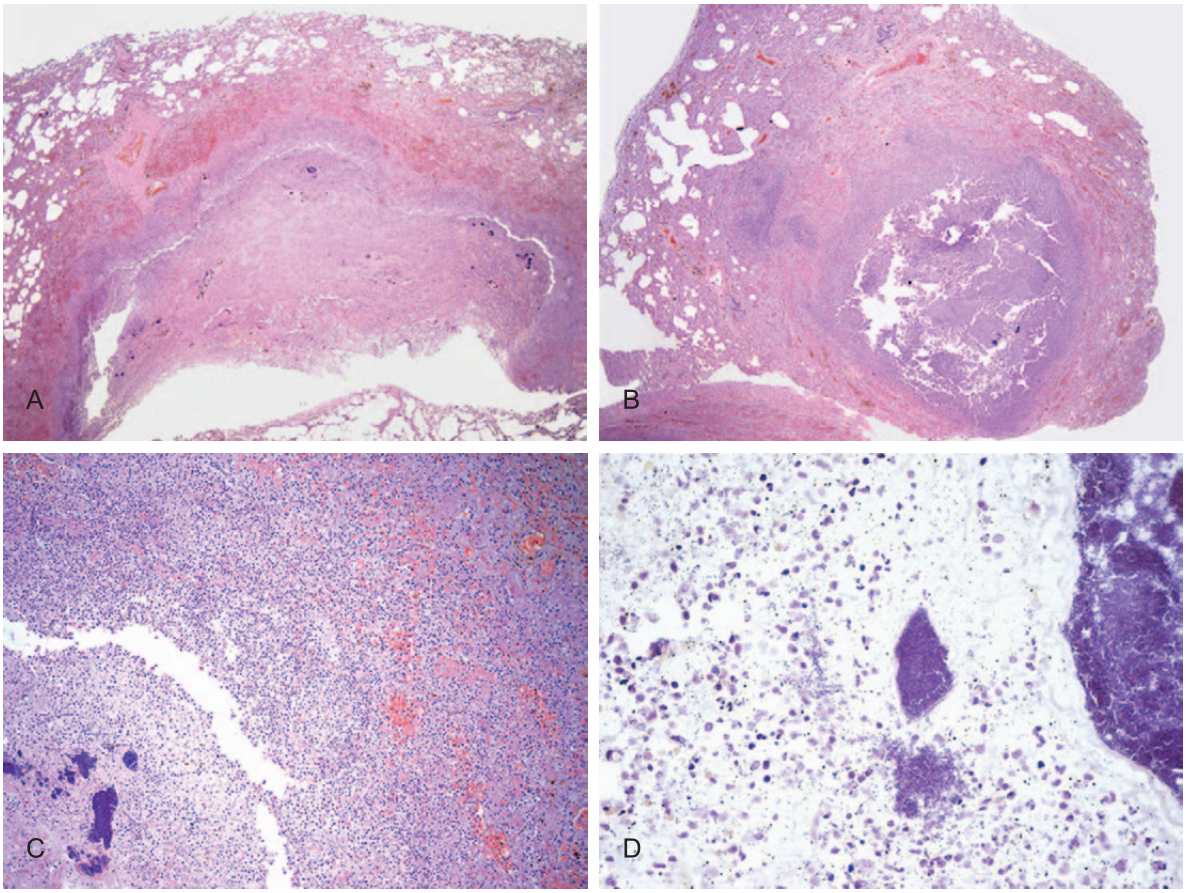

Figure 4 – Photomicrographs of the lung. **A** - (HE-25X) abscess in the upper lobe of the right lung; **B** - (HE-25X) abscess in the lower lobe of the left lung; **C** - (HE-100x) detail of the abscess in the right lung with bacterial colonies; **D** - (Brown-Hopps-200X) detail of the right lung abscess with gram-positive bacterial colonies.

has also been reported in association with acute myocardial infarction.[4] Weisz and Young reviewed 13 cases of this association.[12] In that study, bronchopneumonia was the focus of infection in 4 of the cases; in 5, no source of infection was identified; and in the remaining 4 cases, various foci were identified, including the gastrointestinal tract, kidneys, and extremities. The cause of the association between acute myocardial infarction and myocardial abscess is unknown. However, it has been postulated that the necrotic heart muscle and the inflammatory exudate act as a culture medium that facilitates bacterial growth.[12] The lack of vascularization in that region protects the bacterial colony from the host cellular immune response.[12] More recently, repeated percutaneous transluminal angioplasty has been identified as a risk factor for developing myocardial abscess secondary to intimal dissection.[13,14]

Patients who have chronic kidney disease on hemodialysis are prone to frequent bacteremia and systemic infection.[15] Infectious complications of vascular access constitute the major cause of morbidity and mortality among hemodialysis patients in the United States, the incidence of bacteremia in those patients ranges from 48 to 73%.[16] Most of those cases of bacteremia are caused by *S. aureus*, rates of mortality, recurrence, and remote complications being high (8-25, 14.5-44, and 14.5-44%, respectively).[17] Native AVFs are the least likely to develop infectious complications, whereas prosthetic AVFs and thrombosed AVFs have been associated with the highest number of infectious complications.[15] The infectious complications of the AVF graft observed in our patient were consistent with those reported in the literature.

Although infectious complications are common in hemodialysis patients, there have been few reports of myocardial abscess associated with vascular access for hemodialysis. By the year 2007, only 3 cases had been described in the literature; of those, 2 were related to hemodialysis catheter[18,19] and 1 was related to AVF infection.[20]

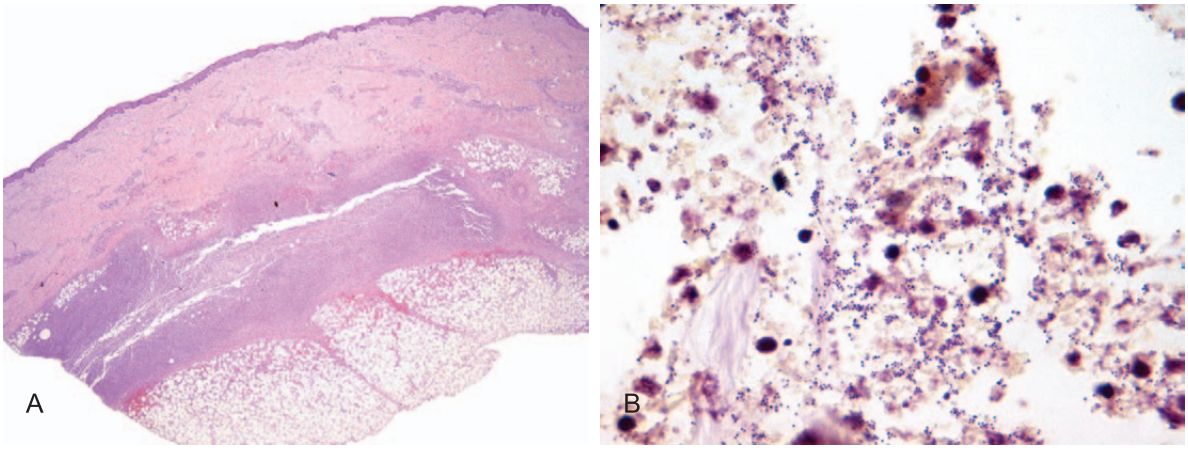

Figure 5 – Photomicrograph of skin. **A** - (HE-25X) skin and subcutaneous tissue abscess on the region of the arteriovenous fistula; **B** - (Brown-Hopps-1000X) detail of skin abscess with gram-positive bacterial colonies.

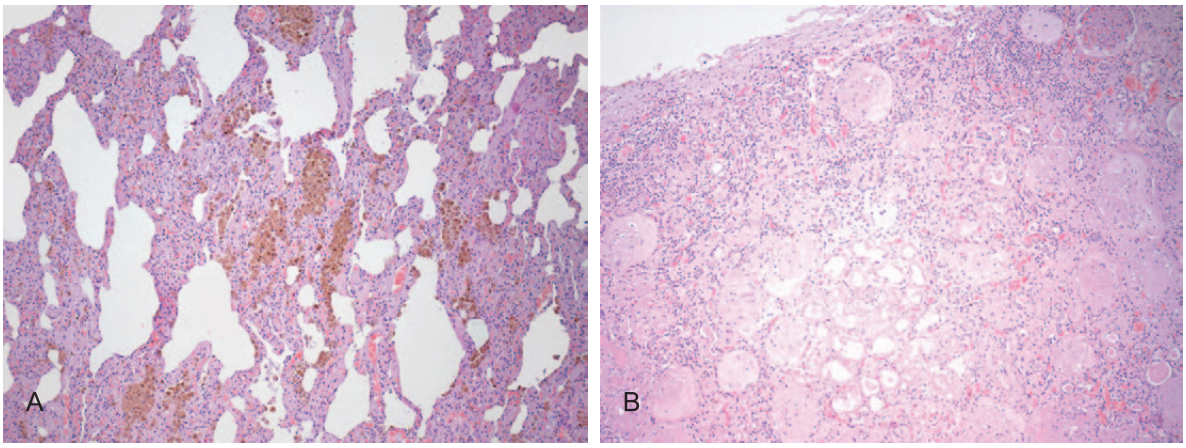

Figure 6 – Photomicrographs of the lung and kidney. **A** - (HE-100x) pulmonary tissue showing the heart failure cells; **B** - (HE-100x) glomerulosclerosis and chronic pyelonephritis of the end-stage renal disease.

The complications of myocardial abscess can be dramatic, with a seven-fold increase in the frequency of myocardial wall rupture at the site of the abscess.[12,21,22] The literature shows that the clinical features of myocardial abscess are varied and include congestive heart failure, cardiac arrhythmias, pericarditis, valvular regurgitation, and poor response to antimicrobial therapy, as well as an absence of cardiovascular symptoms.[20] Cardiac arrhythmias, including serious ventricular arrhythmias, have been reported in association with myocardial abscess.[23]

In the case reported here, the patient was already receiving proper antibiotic regimen, his vital signs were stable during the hospital stay, no hemodynamic instability was detected on the hemodialysis session, reasons why we believed the infection was getting under control. Finding him dead on the bed was considered unexpected. Besides that no autopsy findings other than the myocardial abscess was found to explain the death. Khan et al.[20] reported the case of a patient with myocardial abscess and hypothesized that arrhythmia was the cause of death, as we did in the case reported here even in the absence of an ECG record at the time of the cardiopulmonary arrest.

This case report draws attention to the possibility that the myocardium is a site where metastatic abscesses arise as a complication of an infected AVF in hemodialysis patients. Such a complication is serious and can lead to arrhythmias and death. A strong clinical suspicion of myocardial abscess is important in order to establish an early diagnosis and adopt the required therapeutic measures immediately.

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