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Acute Renal Infarction as a Complication of Atrial Fibrillation: A Case Report

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Abstract **Case Report**

Background Atrial fibrillation is a common cause of thromboembolic events. Rarely, emboli can travel to the renal arteries and cause acute infarction.

Keywords: Acute renal infarction, atrial fibrillation, Thromboembolism.

INTRODUCTION

Atrial fibrillation (AF) is the most common arrythmia, with a global prevalence of 0,51%. It is associated with the development of cardiac thrombi and many thromboembolic events, mainly acute ischemic strokes [1]. Very rarely, AF can be complicated by acute renal infarction (ARI). Diagnosis of this condition can be challenging, and no treatment guidelines have been established so far. In this paper, we provide a case report of a man presenting with ARI as a complication of mitral stenosis-related AF.

CASE REPORT

A 59-year-old Moroccan man was admitted in the Cardiology Department for diagnosis assessment of an intracardiac mass. Cardiovascular risk factors were limited to chronic smoking and obstructive sleep apnea. His medical history included mitral stenosis complicated by atrial fibrillation. He underwent percutaneous commissurotomy twenty-five years ago and remained asymptomatic since then. The day before admission, he complained of acute right lumbalgia. The pain was very intense and persistent, prompting the realization of a computerized tomography (CT) urogram which showed acute renal infarction. In the thoracic slides, the presence of an intracardiac mass was noted. The patient presented no cardiovascular symptoms such as chest pain, dyspnea, or palpitations.

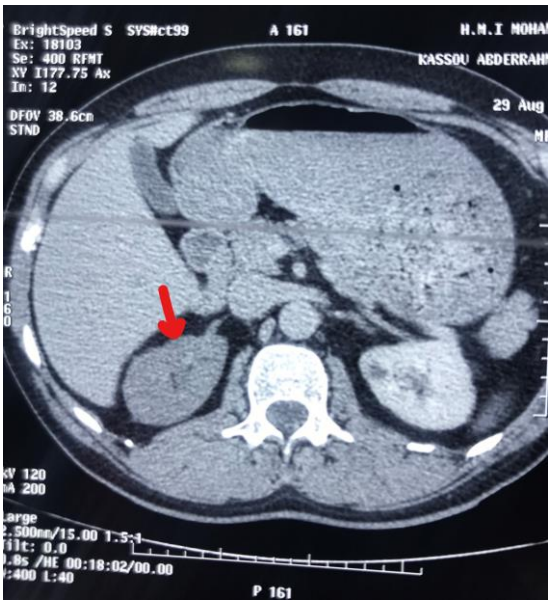

Fig-1: CT urogram showing right acute renal infarction

Physical examination found the patient was in a good general state. He was slightly overweight with a body mass index of 29.4 kg/m². Body temperature was normal. His heart rate was 94 beats per minute and blood pressure 125/80 mmHg. He had a normal respiration, with a rate of 18 breaths per minute at rest, and normal oxygen saturation at 100%. Auscultation found the classic diastolic murmur of mitral stenosis. There were no signs of heart failure and vascular examination was normal. Abdominal examination

revealed a tenderness of the right lumbar region, but no palpable mass, hepatomegaly, or ascites. Electrocardiogram at admission showed atrial fibrillation with low QRS voltage. Chest radiograph found an enlargement of the cardiac silhouette.

Biological findings included slight renal failure (eGFR = 57 mL/min/1.73 m²) as well as moderate inflammatory syndrome, with white blood cell count reaching 10.0×10^3/µL and increased levels of C-reactive protein (22.8 mg/L). High-sensitivity troponin was negative. Hepatic function was normal. Urine analysis found microscopic hematuria and an elevated white cell count.

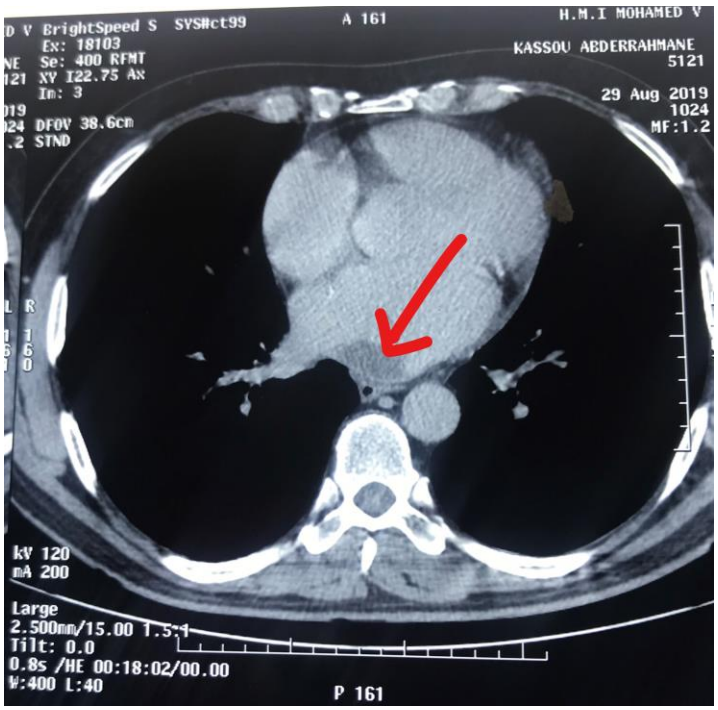

Fig-2: CT scan showing an intracardiac mass

Initial echocardiographic evaluation demonstrated severe mitral stenosis with a valve area of 0.9 cm² and mean transvalvular gradient of 9.67 mmHg, as well as moderate tricuspid regurgitation and a high probability of pulmonary hypertension. Both atria were dilated, and there was a large thrombus inside the left atrium. Left and right ventricular functions were normal.

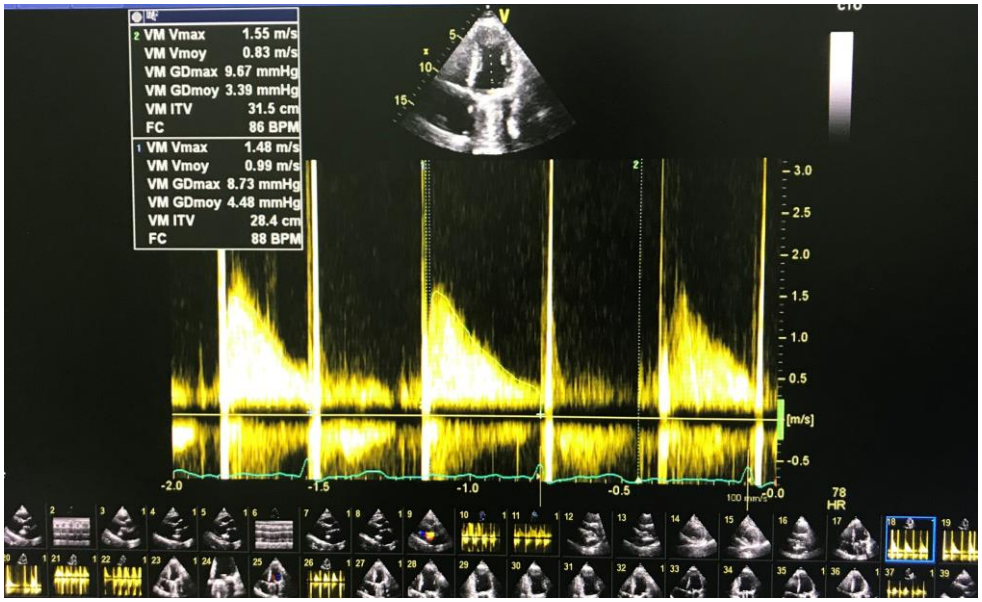

Fig-3: Echocardiography showing the severe mitral stenosis

The patient was admitted in the Intensive Care Unit. Anticoagulative therapy was prescribed, based on intravenous unfractionated heparin at the dose of 500 UI/kg/24h, with daily monitoring of aPTT (target: 1.5 – 2.5). His treatment regimen also included propranolol (100 mg o.i.d.) and tramadol (50 mg t.i.d.). After discussion, the Heart Team's decision was surgery. After preoperative evaluation, including a normal coronary angiography, the patient underwent mitral valve replacement as well as thrombectomy and tricuspid annuloplasty. The surgery was successful, and the patient quickly recovered. He was discharged with oral anticoagulants as well as nephrology and cardiology follow-ups.

DISCUSSION

ARI is a rare cause of renal injury, with an incidence of about 0,3% [2]. It is significantly underdiagnosed as other, more common disorders such as nephrolithiasis, acute pyelonephritis, appendicitis, and mesenteric ischemia could have a similar presentation [3].

Mechanisms of ARI are varied, with the most common one being thromboembolism, with blood or cholesterol clots occluding renal arteries or their branch vessels. AF is the underlying etiology is most cases and can, in turn, be idiopathic or caused by cardiopathies such as mitral stenosis, arterial hypertension, and ischemic heart disease. In a Danish study conducted by Frost et al. on 29,862 patients diagnosed with AF, 2% eventually developed renal artery thromboembolic events [4]. When ARI is confirmed, echocardiography should be performed to diagnose any cardiac source of emboli such as a large atrium, mural thrombus, valvular heart disease, atrial myxoma, infective endocarditis, or atrial septal defect. Less common etiologies include renal thrombosis, renal artery dissection, coagulation disorders, infective endocarditis, fibromuscular dysplasia, sickle cell disease, Marfan syndrome, and cocaine use [5, 6].

Clinical presentation is ambiguous. Abdominal or flank pain and hematuria are the most common symptoms found in literature. Other nonspecific signs may include fever, nausea, and vomiting. On the other hand, ARI can be asymptomatic and detected incidentally on abdominal CT scans [7].

Biology can be a valuable diagnostic tool. Many serum markers are elevated in ARI, such as C-reactive protein, LDH, alkaline phosphatase, and fibrinogen. White blood counts may be elevated. Urine analysis confirms hematuria and leukocyturia. Finally, renal function is often altered. Unfortunately, none of these findings are specific to ARI [7,8].

Imaging is the key to diagnose renal artery occlusion. Renal arteriography is the gold standard for diagnosis as it allows for the complete visualization of renal vessels. In practice however, the diagnostic modality of choice is contrast-enhanced abdominal CT scan. It is non-invasive, with a sensibility of about 85%, and capable of identifying segmental lesions [9].

Management of ARI remains unclear, as this pathology is rare and there are no well-defined guidelines. Conservative therapy, consisting of systemic anticoagulation with intravenous heparin followed by oral anticoagulants such as warfarin, is the generally accepted course of action [8]. Thrombolytic therapy is indicated patients presenting early [10]. Surgical embolectomy is also an option and is typically reserved for emboli that do not respond to conservative therapy [11]. Treatment of the underlying etiology (mitral stenosis in our case) is also essential. If left untreated, the patient is at risk of developing severe complications such as permanent renal failure or arterial hypertension [12].

CONCLUSION

ARI is a rare, fairy underdiagnosed entity that can cause severe complications if left untreated. In most cases, thromboembolism is the mechanism. Therefore, when ARI is confirmed, cardiac evaluation is absolutely essential, including electrocardiogram and transthoracic echocardiography. In patients with sinusal rhythm and a normal echocardiography, holter monitoring is essential for the diagnosis of paroxysmal AF.

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