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# Ruptured Sub-Annular Mitral Aneurysm in the Pericardium: Report of a Case

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## Abstract

The sub-mitral annular aneurysm is a rare cardiac pathology. The etiologies are diverse, ranging from the congenital form to the idiopathic form, including the acquired form. The clinical case we report is that of a 27-year-old young man, with no particular history, admitted with a picture of global heart failure. A cardiac ultrasound diagnosed a ruptured mitral sub-annular aneurysm in the pericardium. The difficulty lies in the surgical management of this condition, due to the lack of an adequate cardiac surgery service in most of the countries in Africa south of the Sahara.

## Keywords

Sub-Annular Aneurysm, Mitral Valve, Intrapericardial Rupture

## 1. Introduction

Mitral subannular aneurysm is a rare heart disease that can have many different forms and clinical presentations [1]. It was first described in 1962 in Nigeria on autopsy of 7 patients [2]. It has subsequently been described in other sub-Saharan African countries and also in other parts of the world including India [3], South Africa [4] and Senegal [1].

The basic pathology of these lesions has been described as a disjunction between the musculature of the left ventricle, the left atrial region and the mitral valve due to the complex disruption of embryogenesis [5]. In a recent study, Nayak *et al.* described that the submitral membranous curtain of the mitral valve seen in 45 of 75 open hearts is a potentially weak area, through which true con-

genital submitral aneurysms can occur [5].

The diagnostic means are transthoracic and transesophageal echocardiography, but also CT scan and magnetic resonance imaging [1] [2] [4] [5] [6] [7]. In this study, we report the case of a ruptured mitral sub-annular aneurysm in the pericardium, diagnosed in the cardiology department of the Ignace Deen National Hospital (Conakry).

## 2. Observation

The patient was a 27-year-old man with no known cardiovascular history who was seen for chest pain and NYHA class III dyspnea. The patient had presented 5 months earlier with an infectious syndrome of undetermined cause. The clinical examination revealed a preserved general condition. The hemodynamic state was stable: the blood pressure was 100/60 mmHg, oxygen saturation (room air) was 99%. On cardiac auscultation, the heart sounds were regular, muffled, with a heart rate of 87 beats/minute with a systolic breath, intensity 3/6, without radiation. There was no pericardial friction. The vesicular murmur was decreased at both lung bases. The rest of the clinical examination was normal except for a Hackett's stage 3 splenomegaly.

The ECG showed a regular sinus rhythm of 87 beats/minute with left ventricular hypertrophy (Sokolow index Sv1 + Rv5 = 65 mm) and a secondary repolarisation disorder (negative T waves in the left precordial region, inferior without necrosis Q wave) (**Figure 1**).

The frontal chest radiograph showed cardiomegaly (ICT = 0.59), at the expense of the left ventricle (**Figure 2**). Cardiac Doppler ultrasound (**Figure 3**)

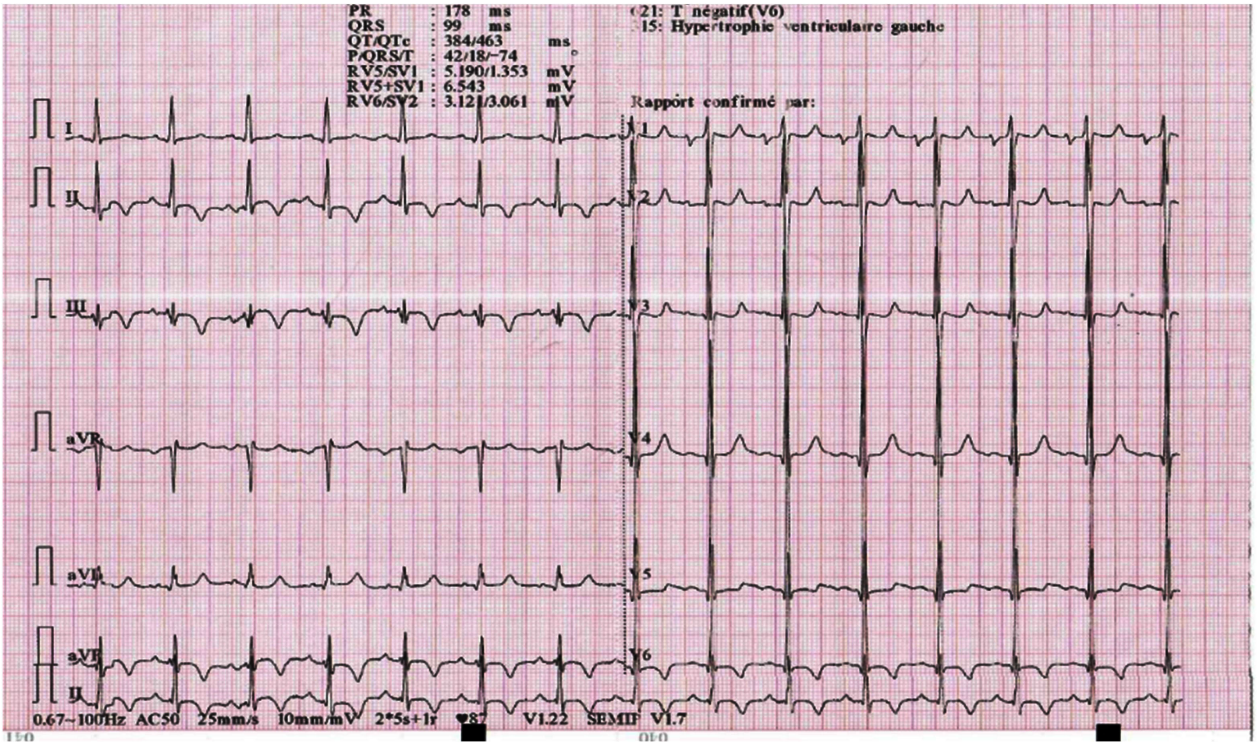



**Figure 1.** 12-Lead electrocardiogram showing left ventricular hypertrophy and negative T waves in II, III, aVF and V6.

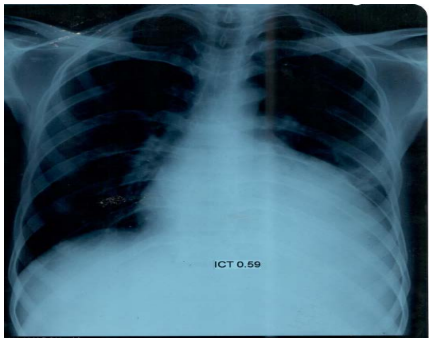
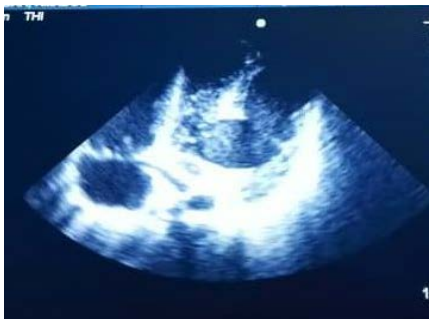
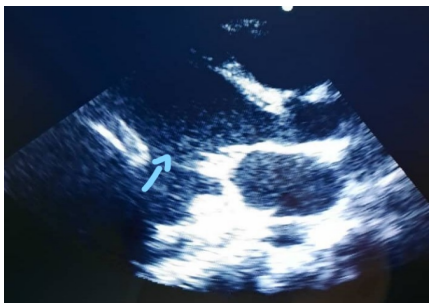


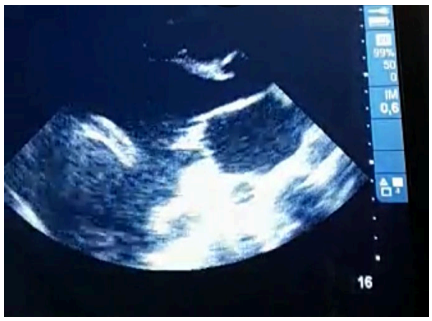

**Figure 2.** Chest X-ray showing cardiomegaly with a TIA of 0.59 at the expense of the LV. There is no evidence of pulmonary venous hypertension.




(a)



(b)




(c)

**Figure 3.** Ruptured sub-mitral annular aneurysm in the pericardium. (a) Two-dimensional transthoracic echocardiography, apical 4-chamber slice; (b) AND; (c) Two-dimensional transthoracic echocardiography, long-axis parasternal incidence.

showed moderate dilatation of the left heart chambers. LV: telediastolic diameter = 61 mm; OG: diameter (anteroposterior = 44 mm, OG surface = 29 $cm^2$); volume = 33 $ml/m^2$. LV systolic function was preserved (EF = 55% Simpson biplane). There was a grade 1 mitral leak (SOR = 12 $mm^2$, RV = 13 ml) due to annulus dilatation. There was a ruptured mitral sub-annular aneurysm in the pericardium with spontaneous contrast, without signs of compression. The right ventricle had good function. Tricuspid leakage was moderate with a pulmonary arterial pressure of 56 mmHg (DO pressure was estimated at 10 mmHg).

The biology showed a CRP of 38.40 mg/l, without hyperleukocytosis. The haemoglobin was 11.8 g/dl.

The evolution was marked by the regression of the signs of cardiac failure after having benefited from a treatment based on a beta-blocker (bisoprolol 1.25 mg per day), a diuretic (furosemide 40 mg per day) and a conversion enzyme inhibitor (perindopril 5 mg per day).

The patient remained haemodynamically stable for 2 to 3 weeks on this treatment before being evacuated abroad where he underwent heart surgery. The post-operative effects are good and since his return to Guinea, we have been monitoring him. He is doing very well.

## 3. Discussion

Submitral annular aneurysm of the left ventricle is a characteristic anatomical lesion, recognised at autopsy or diagnosed clinically in the absence of coronary thrombosis [2]. Its true incidence is certainly underestimated, especially in the early stages when there are no clinical symptoms [8]. It is mostly seen in young people, particularly Africans living in the tropics [9]. Although a genetic cause has been suggested due to racial predilection, cases have been described in patients of other races from different parts of the world, including India [3]. The role of infection and inflammation in the pathogenesis of this disease including Takayasu's disease [10], tuberculous pericarditis [11], chlamydia pneumoniae infection [9] [12], rheumatic fever [13] among others have been reported. On the other hand, cases of non-infectious and non-traumatic aneurysms support the idea that aneurysms result from a congenital defect of the mitral valve annulus [3] [14]. Patients with annular aneurysms may be asymptomatic or have mitral insufficiency with or without left ventricular dysfunction. They may also present with myocardial ischaemia secondary to left coronary artery compression [15], heart failure, palpitations and lower limb oedema [16], or be found on assessment of a systolic murmur or cardiomegaly [6]. Ventricular arrhythmias (ventricular tachycardia) were another form of presentation of the disease as reported by Rao M *et al.* [17]. In our patient, the mode of presentation was the occurrence of chest pain associated with signs of heart failure.

The diagnosis of annular aneurysm was made by transthoracic echocardiography (TTE) showing the rupture (localized dilatation of the posterior wall) communicating the left ventricular cavity, below the posterior mitral leaflet, with

the pericardium. Doppler examination showed a grade 1 mitral leak. TTE also allows assessment of left ventricular function, the presence or absence of wall thrombus or pericardial effusion, and the status of other valves and pulmonary arterial pressure [18]. Transesophageal echocardiography is used to confirm the diagnosis and to better define the lesions [1] [18]. Several authors have not used CT or MRI scans [1] [4] [9] [15] as they are not essential for diagnosis. However, they are used to further explore the findings of TTE [19], provide information about the coronary arteries and thus avoid the need for coronary angiography as in the study by J Skularigis *et al.* [15]. Management involves initial medical stabilisation with diuretics and afterload reducing agents.

Open heart surgery is the gold standard in the treatment of sub-annular mitral aneurysm. Two techniques are commonly used: an extracardiac technique to treat the aneurysm via the epicardial route, *i.e.* under the wall of the aneurysm, and the intrapericardial route described by Antunes, which consists of crossing the atrioventricular groove through the left atrium [8]. Our patient benefited from the latter technique.

The evolution in the absence of surgery is most often marked by a rupture in a cardiac cavity or in the pericardium [1] like this clinical case.

## 4. Conclusion

Sub-annular mitral aneurysm is a rare cardiac condition of varying etiology and clinical presentation. In most cases, echocardiography allows a reliable and precise diagnosis. Treatment is surgical and should be done before rupture, which worsens the prognosis.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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