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# Left Ventricular Apical Aneurysm Post Myocardial Infarction In MRI: About a Case

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**Abstract**

Left ventricular aneurysm is a dangerous mechanical complication of transmural myocardial infarction caused by left anterior descending coronary artery occlusion. Cardiac imaging, dominated by MRI plays a key role in characterization of this entity. Medications or surgery lower the risk of a ruptured ventricular aneurysm. We report the case of a left ventricular apical aneurysm post myocardial infarction in a 65-year-old patient after an anterior myocardial infraction.

***Keywords:*** MRI; myocardial infraction; aneurysm; leftventricular.

## 1. Introduction

True left ventricular aneurysm is a late and dangerous mechanical complication of myocardial infarction, and corresponds to a loss of the normal contour of the LV diastole, replaced by a thin fibrous, cicatricial, akinetic or dyskinetic wall. Its incidence has decreased significantly in recent years from 18.8% to 7.2% due to advances in the management of acute myocardial infarction [1]. Its incidence has decreased significantly in recent years from 18.8% to 7.2% due to advances in the management of acute myocardial infarction [1]. It is more common in the apical, anterior or anterolateral region and usually results from an infarction in the territory of the left anterior descending coronary artery [2].

Magnetic resonance imaging (MRI) plays a key role in the characterization of this entity while avoiding exposure to irradiation, it also makes it possible to study paracardiac structures in particular the mediastinum and large vessels.

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### *1.1. Observation*

We report the case of a 65-year-old patient, with type II diabetes as cardiovascular risk factors, and smoking weaned since 2018, who had presented in 2016 an extended anterior myocardial infraction treated in the acute phase by angioplasty with placement of 3 active stents on the left main coronary artery.

Transthoracic echocardiography has objectified an alteration of systolic function of the left ventricle at 20 % and an apical thrombus.

In 2020 he is hospitalized for the management of an acute pulmonary oedema and he was put an implantable cardioverter defibrillator (ICD) in primary prevention.

The patient is then referred to a rehabilitation center with initial heart transplant project secondarily abounded in front of the functional improvement: VO2 max increasing from 14 to 20ml / kg / min during rehabilitation with the ability to maintain 50 Watts for 25min at the end of rehabilitation.

3 months later he consults for dyspnea class II of NHYA, without angina pain , lipothymia, syncope or significant palpitation, and no sign of heart failure on clinical examination.

The Electrocardiogram is registered in regular sinus rhythm at 81 / min, with a sequela of antero-septo-apical necrosis and a related repolarization.

Transthoracic echocardiography objectified a very dilated ischemic heart disease with large apical aneurysm without visible thrombus but volute of spontaneous contrast. Systolic function of the left ventricle at 20%. Normal left atrial pressure and non-dilated inferior vena cava.

Cardiac MRI has objective a left ventricle massively dilated to more than 500ml, presenting a very severe alteration of its systolic function at 9%, in relation to a very large apical aneurysm [Figure 1,2] encompassing all the anterior, lateral, inferior and septal dyskinetic walls, lined with an extended planar thrombus but infra centimetric thickness (8 to 9mm) [Figure 3].

Hypertrophy and hyperkinesia of the basal segments, with a maximum anteroseptal parietal thickness and less than 16mm

At the late gadolinium enhancement, a very broad apical necrosis sequelae was found including the four adjacent apical segments, and the middle anterior segment [Figure 3]

In addition, subendocardial necrosis of about 50% transmurally of the infero basal segment.

Normal appearance of the VD.

No effusion or pathological enhancement of the pericardium.

### *1.2. Discussion*

Left ventricular anevrysm results from the phenomenon of remodeling which is secondary to various mechanical and neuro-hormonal factors. Activation of the catecholamines and renin angiotensin systems, secondary to a decrease in average blood pressure and systolic and diastolic parietal stresses, trigger ventricular remodeling and lead to stretching of necrotic and non-necrotic tissues.

Clinically, there is a variable symptomatology, made of anginal recurrences, dyspnea, palpitations, syncope, lipothymia, or thromboembolic complications. The clinical examination is poor, it may reveal a beating mass on palpation and / or a third heart sound on auscultation. The electrocardiogram generally objectifies the persistence of the ST segment elevation in the necrotic territory beyond the 4th week. While the front chest x-ray may show cardiomegaly with a focal deformity sometimes calcified of the lower left arch [3, 4]. Ventricular aneurysm is well seen in echocardiography and appears as a thin-walled pocket developed in the necrotic territory responsible for deformation of the left ventricular cavity diastole, and is dyskinetic in systole. This pocket communicates with the left ventricle through a wide collar in the case of true aneurysm.

Magnetic resonance imaging represents the most performed non-invasive investigation method in the study of left ventricle aneurysms and is superior to other imaging modalities frequently used in this field [5, 6] such as 2D echocardiography and SPECT imaging. It makes it possible to clarify the diagnosis and distinguish true aneurysm from pseudo aneurysm, and to provide valuable information, with excellent spatial resolution on the thickness of the ventricular wall, the precise location of the aneurysm, [7] the volumes of the heart chambers, the ventricular mass and the left ventricular ejection fraction, and also prognostic information concerning the size and transmurally of the infarction. Replacement of the myocardial infarcted wall with adipose tissue, calcifications and thrombi formation can be observed in chronic aneurysms

Thus cardiac MRI shows in true aneurysms:

-A thinning of the aneurysmal wall compared to the adjacent myocardium, with dilation of the left ventricular cavity on all sequences.

-Gently sloping connection of the aneurysmal wall to the healthy myocardium, with a collar is wide (unlike pseudo aneurysm)

-The ratio between the maximum diameter of the orifice and the maximum internal diameter of the cavity is 0.9 to 1.0 for true aneurysms [8] .

- the cinema sequences: akinetic or dyskinetic character

-On the sequences of late enhancement 10 min after injection of gadolinium: transmural contrast of the characteristic wall (Scar fibrous tissue) without pericardial enhancement.

The increase in volume is partly the result of simple thinning and dilation of the aneurysmal part of the LV wall.

However, the non-aneurysmal parts of the LV also increase in volume and thickness due to hemodynamic stress that aneurysm dyskinesia subjects them. As the size of the ventricle increases, it may eventually lose its systolic reserve which contributes to LV dysfunction. The true aneurysm has a lower risk of rupture than a pseudoaneurysm and is often treated medically with surgical repair usually reserved for cases associated with heart failure, systemic embolization, or refractory arrhythmia [11]. The treatment of left ventricular aneurysm is essentially surgical by removal of the aneurysmal pouch [12, 13, 14, 15]. Start with medical treatment first, as some authors suggest in the literature, and very controversial. Surgery involves a fairly heavy mortality, especially in the case of decompensated heart failure or arrhythmia, and the operative result is not always brilliant. Currently the use of surgery is limited to patients with heart failure refractory or with rhythm disorders rebellious to usual treatments. Thus our patient was entrusted to the surgeons after his agreement. The operatory suites were marked by a refractory cardiogenic shock followed by death.

### *1.3. Conclusion*

Our article suggests that magnetic resonance imaging plays a key role in the characterization of true ventricular aneurysms, and in the search for myocardial viability, thus improving diagnostic certainty and conditioning the management of these complications of myocardial infarction.

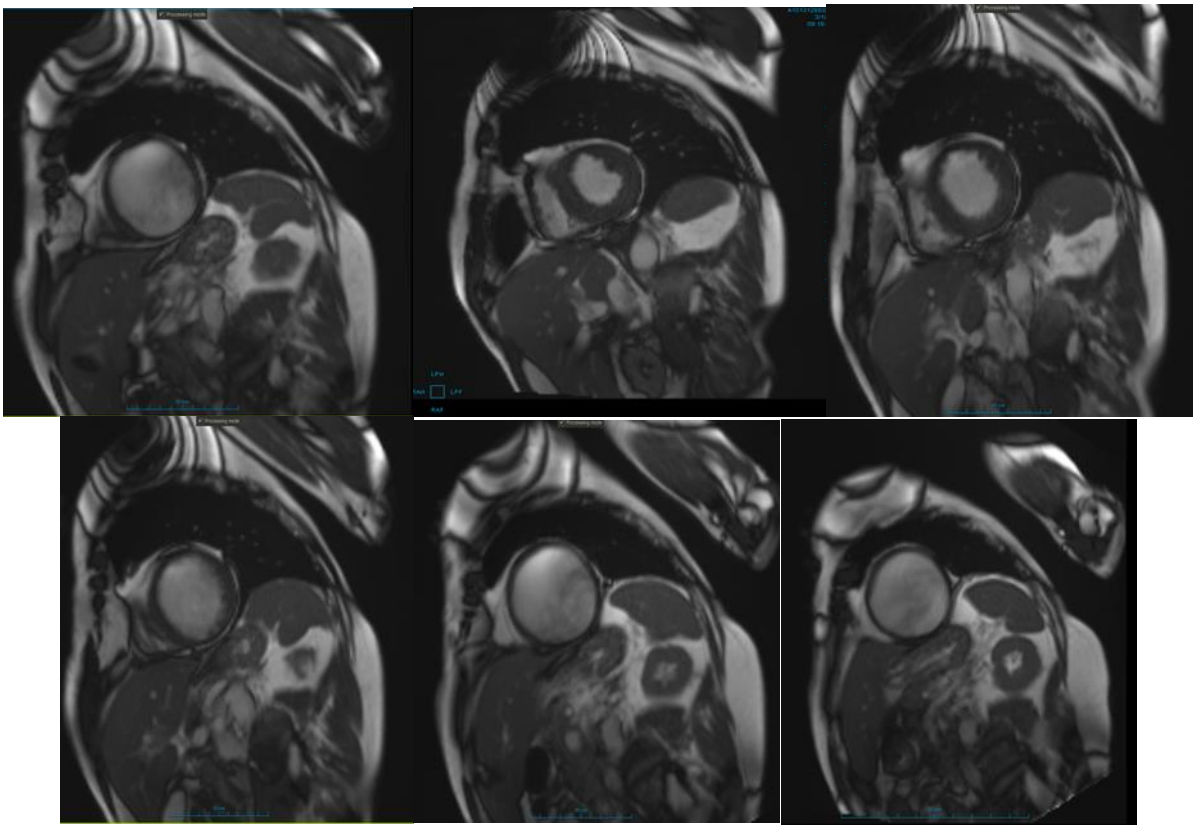

**Figure1 :** Series of images acquired in the short-axis plane in cine-MRI mode showing a large apical aneurysm of the left ventricle.

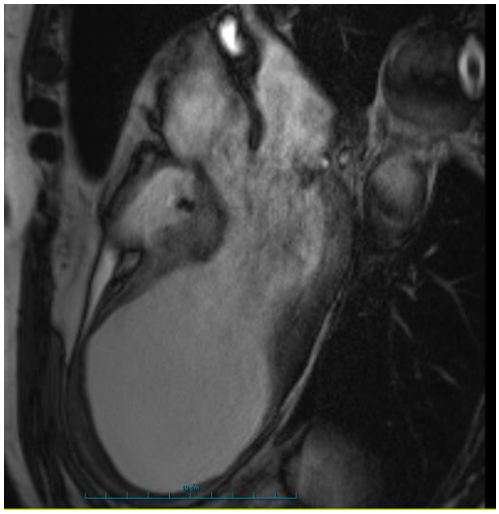

**Figure2:** Apical 3 chambers in MRI cine mode showing a large aneurysm with planner thrombus in the apical left ventricle.

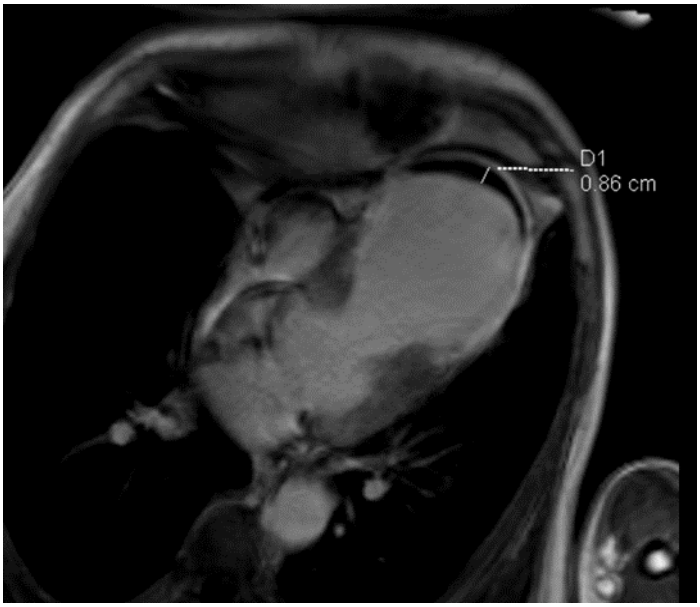


**Figure3:** Acquisition in Cine-MRI mode in the long axis-4 chambers, showing a large apical aneurysm of the Left ventricle lined with a thrombus of infra-centimetric thickness, with a sequelae of apical necrosis and the 4 apical segments.

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