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Left Ventricular Thrombus as Cause of Acute Ischemia of the Lower Limb and Ischemic Stroke: About A Case Report and A Review of the Literature

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ABSTRACT

The formation of an intraventricular thrombus (TIV) is a serious complication of acute myocardial infarction. The presence of a thrombus is associated with an increased risk of systemic embolization, risk of around 20% in the presence of a TIV of apical topography.

The importance of this risk makes it essential to detect this complication early, so as to initiate adequate anticogulant treatment without delay.

We report the case of 50 years old male presented with anterior myocardial infarction with left ventricular dysfonction and apical thrombosis which is complicated by acute ischemia of the lower left limb and ischemic stroke.

KEYWORDS: Acute Ischemia , Intracardiac Thrombosis , Left Ventricule, Myocardial Infarction

INTRODUCTION

The formation of a left ventricular thrombus is a known complication of myocardial infarction, mainly with elevation of anterior ST segments (STEMI). Its incidence is currently of the order of 10 to 20%.This is the reason why rapid reperfusion is desirable and curative anticoagulation is often recommended in post-infarction. Many strokes and embolisms have also been observed in patients with cardiac decompensation, regardless of its etiology, mainly when left ventricular ejection fraction (LVEF) is reduced.

CASE REPORT

A 50-year-old patient, with cardiovascular risk factors, active smoking, hypercholesterolemia. Who presented to the emergency department witha acute dyspnea, chest pain, on clinical examination we noted signs of heart failure, bood pressure at 100/60mmhg, tachycardia arterial oxygen saturation at 90% and pulmonary auscultation revelead crackling rales in both lund fields.

Electrocardiogram (EKG) showed a regular sinus rythm at 70 bpm, negative T waves in the anteroseptoapical. (figure1)

Echocardiogray transthoracic was performed showing normal sized heart chambers with a reduced ejection fraction at 43%, apical akinesia and the presence of apical thrombus measuring 24x7mm. Anticogulant treatment with LMWH and diuretic was initiated (figure 2)

MRI confirmed the diagnosis, showed 2 thrombus, the largest is appendu to the apex measuring 24x7mm and a second at the middle segment of the anteroseptal wall measuring 5 mm with a extended transmural late contrast of the anterosepto apical and infero apical wall (figure2)

Then, Coronary angiography has objectified bitroncular lesions, a tight, thrombotic lesion of the anterior interventricular artery with an aspect of small thrombuses upstream of the lesion, and significant stenosis of the proximal right coronary artery with ulcerated plaque, revascularized by angioplasty after prédilation and placement of an active stent in anterior interventricular artery and right coronary artery(figure3)

The patient was put on conventionel treatment of myocardial ofarction with antiplatlet aggregation, statin, beta blockers and blockers system renin angiotensin.

The evolution was marked by the installation of pain of the lower left limb, the leg became white, cold, without pulse, the clinic evokes acute ischemia of the lower limb. In emergency, the Doppler of the lower limb confirms the presence of an occlusive thrombus of the left popliteal artery with no flow in distality (figure 4). The patient has benefited a surgical embolectomy (Fogarty) 3 days later, he presented a transient hemiparesis of the right lower limb, Brain CT showed hypodensity of the left lenticular nucleus (figure5)

The patient is discharged on triple therapy (Aspirine+ Plavix+ Coumadine) for 1 month. One month later, the patient is seen again in consultation, we stopped the plavix and we maintained the dual therapy

DISCUSSION

The formation of an inverterverticular thrombus (TIV) is a serious complication of acute myocardial infarction [1]. Before the era of thrombolysis and percutaneous angioplasty, 40-60% of TIV was reported, depending on the series, in the course of a previous infarction [2] Through techniques aimed at preserving the myocardium, the incidence of this complication has decreased sharply. It is currently the order of 10 to 20% of cases in this type of necrosis, risk of around 20% in the presence of a TIV of apical topography.

Blood stasis in the heart, regardless of its etiology, increases the risk of thrombus formation. A large akinetic area of the left ventricle, especially at the apex, may be the cause of clot formation.

The three main risk factors for thrombi formation (within 15 days of STEMI) in the left ventricle are as follows:[3]

- Anterior topography of STEMI
- the existence of a dyskinetic zone or aneurysmal in the anterior wall
- low cardiac output,
- Occlusion of the anterior interventricular artery;
- Extended STEMI with LVEF< 30%

A left ventricular ejection fraction less than 35% is also a risk factor independent, but not a prerequisite, thrombus can form even when the ejection fraction is preserved [4]

The components of the Virchow triad (stasis, hypercoagulability and endothelial dysfunction) are considered essentially responsible for the phenomenon.

The formation of the clot is most often early, occurring in the first 10 days after the infarction. 30 to 60% are formed during the first 48 hours . In the series of Keren et al, the average embolism rate was 55%, in case of mobile and protrusive thrombus, by 18% in case of non-mobile protruding thrombus and 10% in wall thrombus [5]

The incidence of left intraventricular thrombosis varies according to the series: from 5% to 25% after myocardial infarction and from 11% to 36% in case of dilated myocardiopathy [6]

Anatomical features of the thrombus (size, mobility, width of adhesion to the myocardium) are all variables that influence the embolic risk. large infarctions are associated with more myocardial damagepronounced, an inflammatory response and a state more severe hypercoagulability, which increases the risk of thrombus formation.

Transthoracic ultrasound is considered the 'gold standard' examination in the evaluation of heart function and the search for thrombus, which appear as defects, separated from the myocardium by contrast. The examination can be done quickly, but is dependent on the operator. Its sensitivity (23%) is considered very low[7]

If the echocardiography does not optimally visualize the endocardial wall, especially apical, a contrast ultrasound should be performed to exclude a thrombus, before any further investigation.

In case of doubt or poor echogenicity, cardiac magnetic resonance imaging (MRI) with gadolinium injection should be considered.

Plusieurs études, dont celle de Srichai, confirment que l'IRM semble avoir une meilleure spécificité et sensibilité (99 % et 88 %) que l'échographie transthoracique (96 % et 23 %) ou transoesophagienne (96 % et 40 %) pour la détection de thrombus cardiaque.[8]

Transesophageal echocardiography is not recommended from as a first line given its poor visualization of the apex of the VG. The injected CT scan, with an early phase and a late phase, could also be an alternative to looking for a thrombus in a cardiac cavity.

In case of intraventricular thrombus, the risk of embolization in non-anticoagulated patients is 10 to 15% and most of the time, embolizations take place within the first four months. There are two echocardiography risk factors for embolization: mobility and thrombus protrusion. In case of thrombus detected, it is imperative to follow the evolution of the size of the clot by transthoracic ultrasound or cardiac MRI [9]

It is recommended to start anticoagulant therapy early in patients who have suffered from extensive anterior STEMI to avoid intraventricular thrombus formation. However, no consensus has been established so far on whether or not to initiate anticoagulant therapy in other patients at high risk of thrombus formation, such as people with LVEF less than 30% and/or extensive apical akinesis.[10]

Anticoagulation established for a period of 3 to 6 months, remains the treatment of choice, reducing the risk by 33% embolic compared to untreated subjects. The risk of recrudescence, upon discontinuation of treatment, is present in patients with an aneurysm or apical dyskinesia, anticoagulation must then be continued ad vitam. It is exceptional in case of akinesia apical [11]

Unfortunately, antiplatelet drugs prevent very little from the formation of intraventricular thrombi and therefore cannot replace conventional anticoagulation with antivitamin K (AVK). In addition, no data have been published on the use of NOACs in the prevention of intracardiac clot formation.

Some authors recommend anticoagulation from the outset in cases of extensive anterior STEMI or non-ischemic cardiomyopathy with severe impairment of LVEF.

CONCLUSION

The formation of a left ventricular thrombus remains a frequent complication of acute myocardial infarction, especially in the absence or failure of early revascularization techniques. It is correlated with size of the infarction and its anterior localization. In 20% of cases, systemic embolic events, the main complication, can occur.

Echocardiography remains the first choice examination, supplemented if necessary by nuclear magnetic resonance imaging.

In patients at high risk of intraventricular thrombi development, it is recommended to weigh the benefit/risk balance of short- or medium-term anticoagulation.

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Figure légends

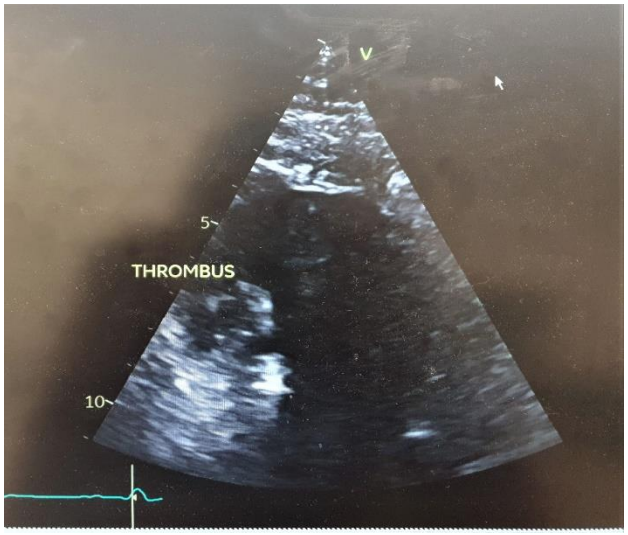

Figure 1: Transthoracic echocardiography apical 4 chamber view showing an intraventricular apical thrombus

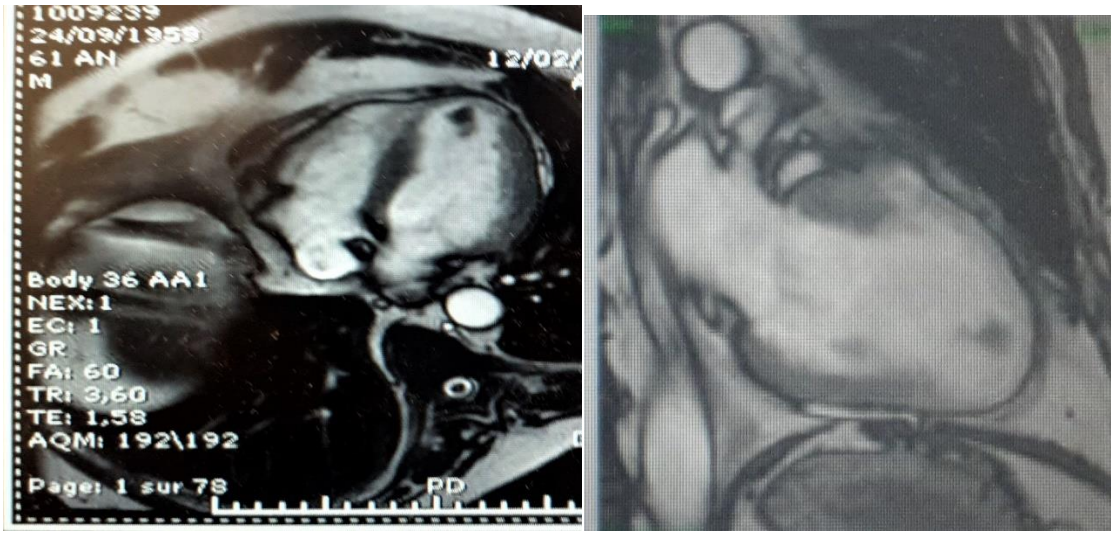

Figure 2: MRI showing two thrombus, A) apical thrombus , B) thrombus in the median anteroseptal wall

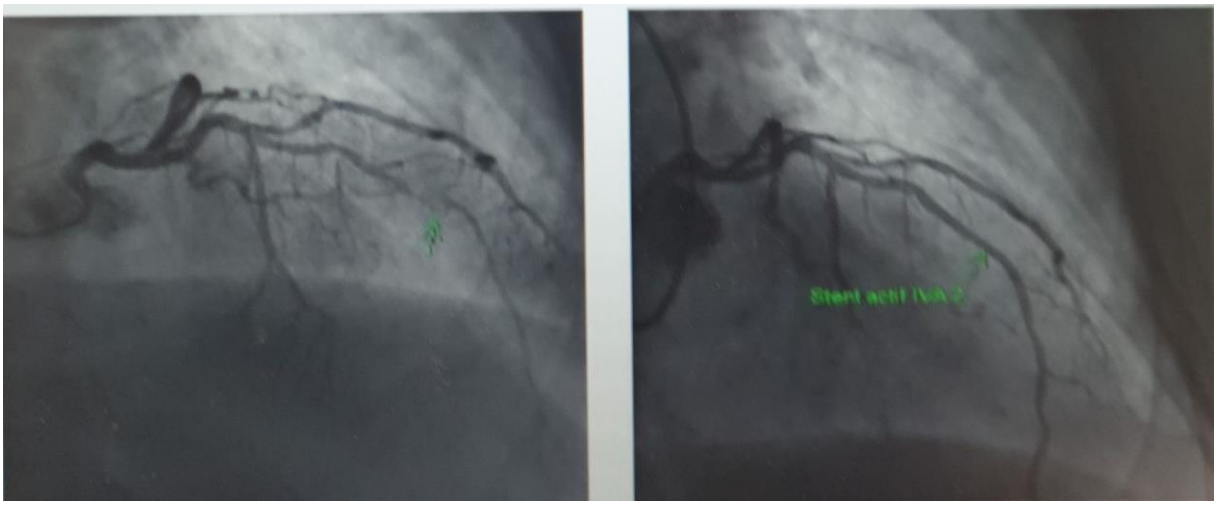

Figure 3: Coronary angiography showing a thrombotic lesion of the middle anterior interventricular artery with an aspect of small thrombuses upstream of the lesion, revascularized by angioplasty with an active stent .

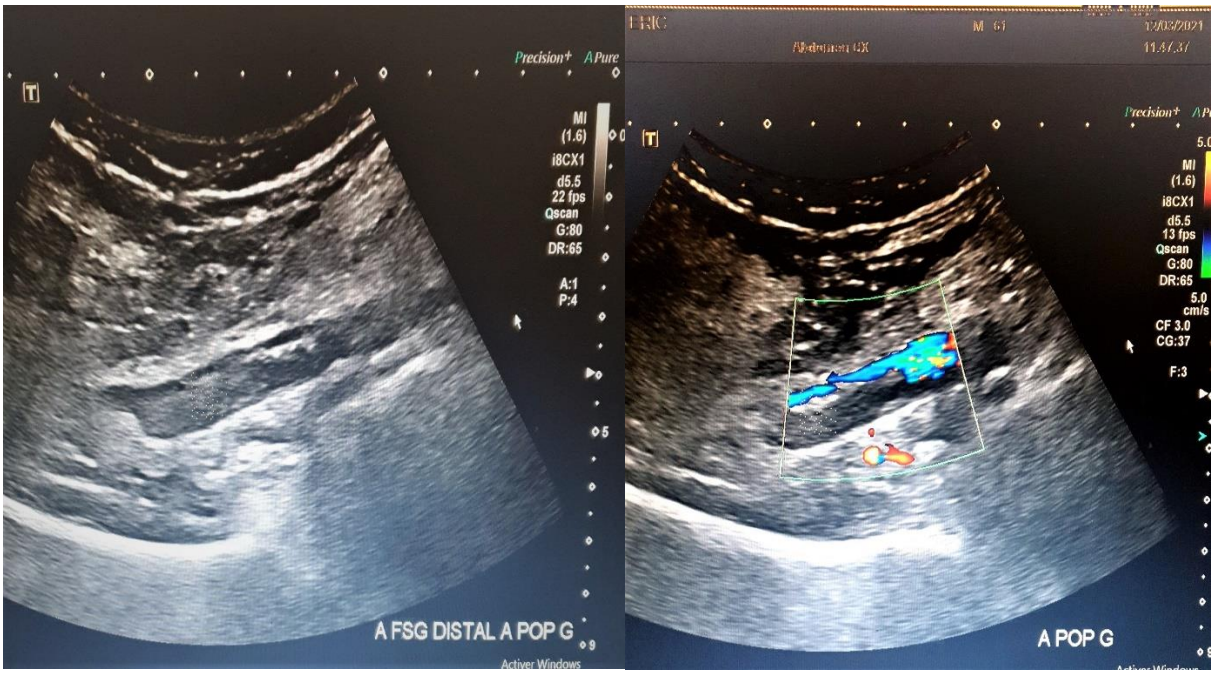

Figure 4: vascular doppler shows thrombus of the left popliteal artere

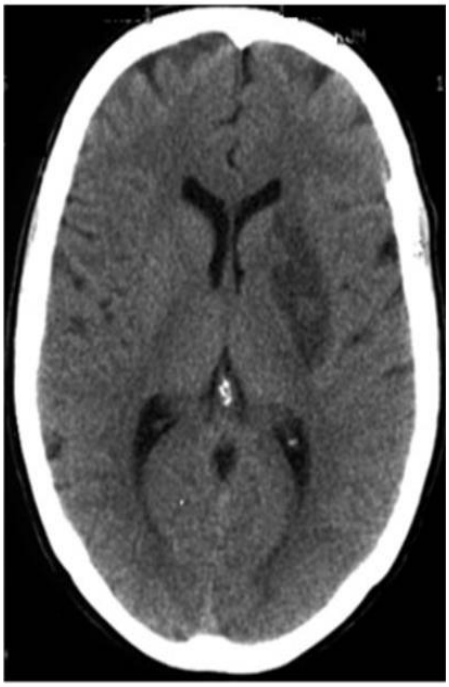

Figure 5: Brain CT showing hypodensity of the left lenticular nucleus