Giant Left Ventricular Pseudoaneurysm in a 79-Year-Old Female Patient: Diagnostic and Management

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ABSTRACT

Left ventricular pseudoaneurysm is a rare but dangerous complication, which occurs in the early post myocardial infarction period. Small pseudoaneurysms are not fatal, while large ones cause death due to sudden rupture and cardiac tamponade if surgery is not performed on time. As left ventricular pseudoaneurysm is uncommon in population, only few case reports were found in the published literature. In this article, we present a case of left ventricular pseudoaneurysm in a 79-year-old female patient after a silent posterolateral myocardial infarction, which increased to gigantic size for 3 months and was diagnosed accidentally by transthoracic echocardiography. Since the patient refused surgical treatment, the difficulties in deciding on the management of the patient based on a review of the literature is described. The main goal of this case is to describe the 6-month survival rate of a 79-year-old female patient with left ventricular pseudoaneurysm after silent posterolateral myocardial infarction despite refusal of surgical treatment and extremely low adherence to drug treatment due to cognitive impairment.

Key words: *left ventricular pseudoaneurysm, echocardiography, diagnosis, prognosis.*

INTRODUCTION

Myocardial ruptures include left ventricular free wall rupture and internal ruptures of interventricular septum and papillary muscles, the latter being the most severe and potentially fatal complications developing in the early post myocardial infarction (MI) period.[1-3] Some left ventricular free wall ruptures may be subacute, in this case the myocardial rupture is small and sometimes closed by partially thrombosed hematoma "sealed" to the pericardium. It means that the thrombus, hematoma, and pericardium cover the rupture, thus forming a false aneurism or pseudoaneurysm of the left ventricle.[2,3] Pathologically, in pseudoaneurysm, there is a small narrow canal (isthmus) which connects the left ventricular cavity with a massive aneurysmal sac containing blood, clots, and only pericardial fibrous elements – no myocardial tissue.[4] Macroscopically, the rupture is linear or arched, with transmural localization, edges are irregular, the site of rupture is jagged.[5]

Left ventricular pseudoaneurysm is a very rare complication of MI[2,3] and develop in less than 0.1% of all patients with MI.[6] Postmortem examination of 303 patients who died of MI revealed no left ventricular pseudoaneurysm-related deaths.[5] It is known, that only small pseudoaneurysms are compatible with life, but with large ones and absence of timely surgical

treatment patients die because of spontaneous rupture and tamponade development.[2,3] As a rule, reports in the existing literature describe small left ventricular pseudoaneurysms. In this article, we report a case of a giant left ventricular pseudoaneurysm in a 79-year-old female patient after posterolateral silent MI, who refused surgical treatment.

CASE ILLUSTRATION

A 79-year-old patient female was urgently admitted to a cardiology unit with breathlessness on mild exertion, increasing in supine position (at night the patient takes the sitting position), swelling of lower extremities (shins and feet), and weakness. History taking was complicated by patient's marked cognitive impairments. The patient lived alone and had no relatives. The medical records showed that she had been suffering from arterial hypertension for a long period of time, over the past 3 months her blood pressure persisted but was not higher than 110/70 mm Hg. The patient had paroxysmal atrial fibrillation for 5 years. Three years ago she suffered an acute cerebrovascular event which triggered progressive memory loss. Two years ago, the patient developed angina pectoris III functional class. Coronary angiography findings revealed circumflex artery stenosis, stenting was performed. Meanwhile echocardiography showed that local left ventricular contractility was normal. Adherence to drug regimen was poor (ramipril, bisoprolol, atorvastatin, clopidogrel, aspirin, warfarin were taken irregularly, dosage instructions were not followed). Blood pressure and heart rate were not monitored.

The patient noticed the deterioration of her condition 3 months prior to admission, when she suddenly felt weakness when she was at home. Then she developed signs of left heart failure (HF) (breathlessness on mild exertion increasing in supine position), and was hospitalized. Echocardiography showed the left ventricular wall with depressed myocardial contractility in posterolateral segment, the rupture of the basal segments with forming pseudoaneurysm with size 6.2x2.5 cm, and volume about 62 ml. Coronary angiography reveals 70% restenosis in the stent of the circumflex artery. Surgical treatment was recommended, but the patient refused it and was discharged with recommendation to add loop diuretic (furosemide, 20 mg and spironolactone, 50 mg on a once-daily basis) to her therapy.

Over the next three months, the patient's condition progressively deteriorated; manifestations of right ventricular heart failure (swollen lower legs and feet) supervened, and she had to be hospitalized. On admission, the patient's condition is of moderate severity (NYHA class III); due to shortness of breath, she is lying with the head of the bed raised. The respiratory rate is 24 per minute. Auscultation revealed weak breath sounds, moist rales over the lower lateral divisions of the lungs on both sides. Heart sounds are muffled; the rhythm is regular; systolic murmur is heard at the apex and at the base of the xiphoid process of the sternum, diastolic murmur at the left lower sternal border (3rd-4th intercostal space). The heart rate is 92 bpm. BP is 100/70 mmHg. The abdomen is soft and non-tender. The liver is enlarged and 3 cm below the costal margin, tender on palpation. Swollen thighs, shins, and feet.

Complete blood count and basic metabolic panel revealed no significant changes. However, NT-proBNP was increased to 2450 pg/mL, serum creatinine was increased to 140 μmol/L. Electrocardiogram (ECG) showed scarring of posterolateral wall. (**Figure 1**)

Chest X-ray showed signs of venous congestion and left-sided hydrothorax (**Figure 2 A,B**).

Two-dimensional Echocardiography findings revealed dyskinesia of the basal posterolateral and mid posterolateral left ventricular segments, hypokinesia of the lower basal and mid segments of the left ventricular, and hypokinesia of its anterolateral basal and mid segments. At the basal level of the posterolateral wall, there is a defect (wall rupture) up to 1.9 cm, communicating with a cavity with size 6.1x9.4 cm, volume about 315 ml. (pseudoaneurysm). The cavity of pseudoaneurysm demonstrates spontaneous echo contrast and mural thrombi (**Figure 3A, B**). Colour Doppler echocardiography showed blood flow pattern "to-and-fro" between two cavities: circulation from the left ventricular through the isthmus to the cavity of pseudoaneurysm and

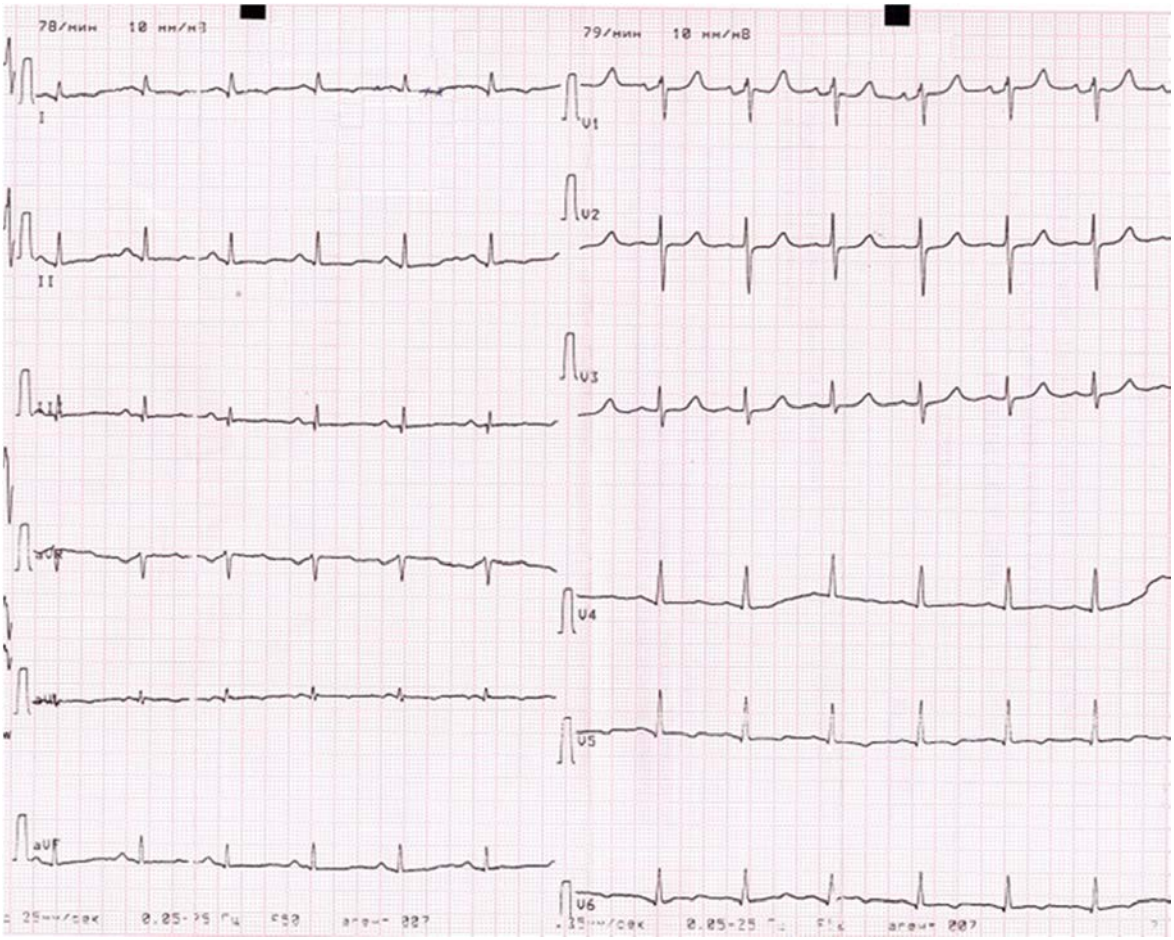

Figure 1. Electrocardiogram Sinus rhythm. qR waves in leads II, III, AVF, AVL, V5, V6, and symmetric inversion of T wave in leads I, aVL, V5 and V6.

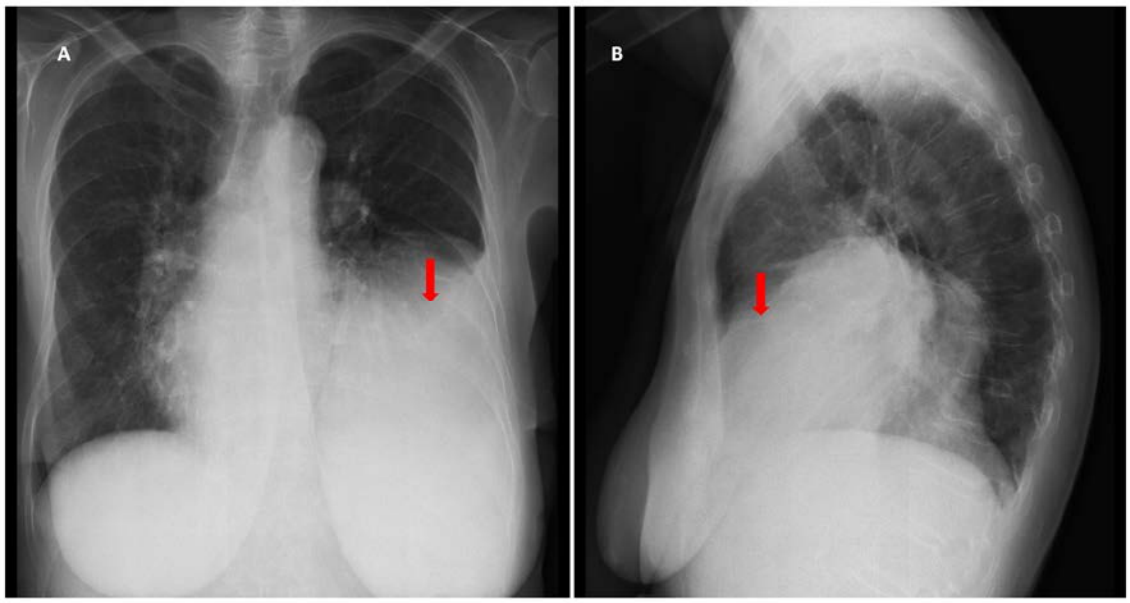

Figure 2. Chest x-ray. A. Frontal view. B. Left lateral view. On the left, there is a homogeneous shadow with an upper indistinct border at the level of rib IV anterior segment (arrow), due to effusion mainly in the anterior sinus.

backflow from the cavity of pseudoaneurysm through the isthmus to the left ventricular cavity (**Figure 4 A, B**). There were also revealed: diastolic dysfunction of the left ventricular of the restrictive type, a decrease in the ejection fraction up to 40%, an increase in pulmonary hypertension up to 55 mm Hg, dilation of the inferior vena cava up to 2.3 cm, which collapsed on inspiration less than 50%. Colour Doppler echocardiography showed moderately severe mitral, aortic and tricuspid regurgitations (**Figure 5A, B**).

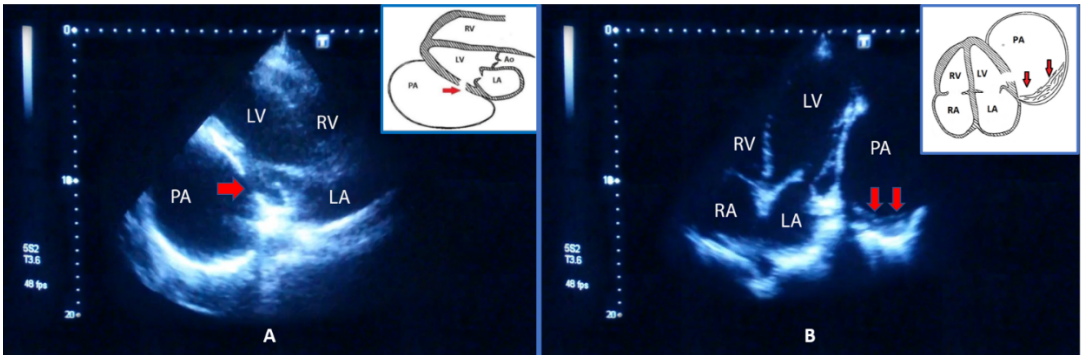

Figure 3. A. Echocardiogram in the parasternal long axis view and scheme. Thinning and wall rupture – a defect in the posterolateral left ventricular wall (arrow) through which the left ventricular communicates with the pseudoaneurysm. B. Echocardiogram in the apical four chamber position and scheme. Mural thrombi in the pseudoaneurysm cavity (arrows).

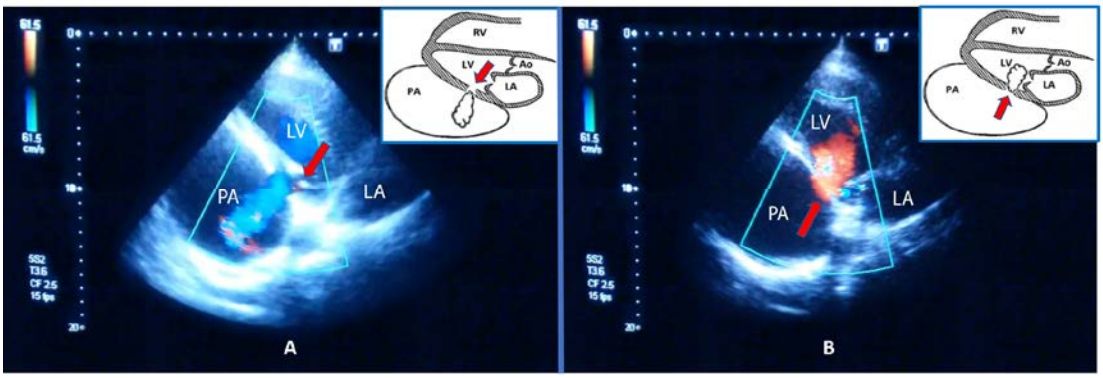

Figure 4. A. Color Doppler echocardiography and scheme. In the projection of the defect, the blue flow indicates cardiac shunt from the left ventricular through the isthmus into the pseudoaneurysm cavity (arrow). B. Color Doppler mapping and scheme. In the projection of the defect, the red flow indicates blood return from the pseudoaneurysm cavity through the isthmus into the left ventricular cavity (arrow).

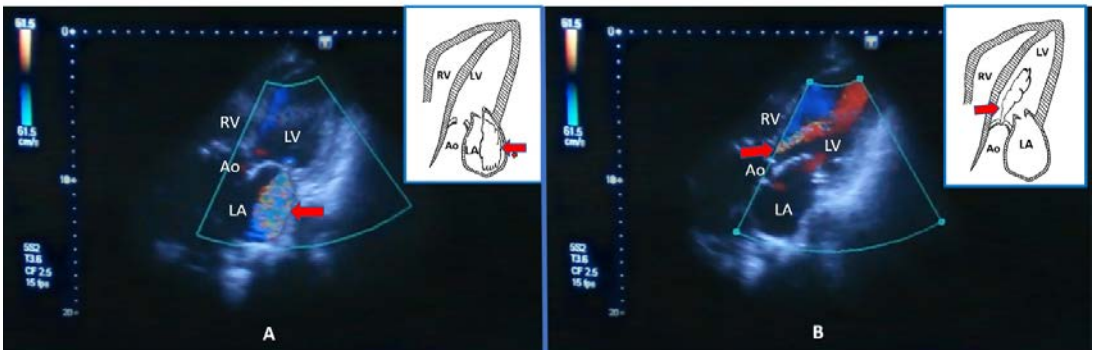

Figure 5. A. Color Doppler echocardiography and scheme. Apical five-chamber position. Turbulent flow in the left atrium during systole reaching the upper wall of the left atrium. The stream of mitral regurgitation is variegated (arrow). B. Colour Doppler echocardiography and scheme. Apical five-chamber position. Turbulent flow in the left ventricular outflow tract and cavity. The stream of aortic regurgitation is colored red (arrow).

Abbreviations: pseudoaneurysm (PA), left ventricle (LV), right ventricle (RV), left atrium (LA), right atrium (RA).

The patient flatly refused to be further examined and operated and was discharged to be followed up by a cardiologist with recommendations to continue drug therapy: ramipril 2.5 mg daily, bisoprolol 5 mg/day, atorvastatin 20 mg QD, rivaroxaban 20 mg, furosemide 20 mg and veroshpiron 50 mg orally on a once-daily basis. However, three months later, the patient died with symptoms of progressive HF up to functional class IY (NYHA) in another hospital.

DISCUSSION

In left ventricular pseudoaneurysm, myocardial rupture develops slowly for several hours or even days[7] and progresses from endocardium to pericardium.[8] Blood continually oozes to the pericardium through a small defect in the left ventricular wall causing local inflammation and "de novo" pericardial adhesions.[8] In addition, myocardial rupture may be contained by existing pericardial adhesions.[2] The above described mechanism clearly explains how the patient's posterolateral silent MI resulted in left ventricular pseudoaneurysm, which prevented rapid cardiac tamponade followed by patient's death.

Due to lack of medical documentation, the date of MI and how long pseudoaneurysm developed are not known. Having studied incomplete medical record of the patient, we suppose that it happened 3 months prior to admission, when being in stable condition, the patient felt worse and developed acute left HF and paroxysmal dyspnea with no previous attacks of angina pectoris, which could prompt her to seek medical attention earlier. The literature describes a case of left ventricular pseudoaneurysm development as a result of transmural MI without preceding angina within 5 months, with HF also being the main clinical manifestation.[9] According to the literature, the time interval between MI and the diagnosis of pseudoaneurysm is from 1 to 11 months,[8] and the median time from MI to diagnosis of pseudoaneurysm is 3.9 months.[10]

We also suppose that the initial rupture of the left ventricular wall was small. However, later due to the continuous blood flow to the cavity of pseudoaneurysm, its volume increased to 315 ml. It significantly exceeded volume of the left ventricular, which cavity, due to the constant flow of blood into the pseudoaneurysm cavity decreased to 80 ml. Decreased blood flow resulted in spontaneous echo contrast and massive thrombus formations on the aneurysmal sac walls which are the major risks for a thromboembolic event.[1,11]

Based on literature, the most common localization of both left ventricular free wall ruptures, and true left ventricular aneurysms is the anterior wall of the left ventricular.[5] This results from frequent atherosclerosis of the left anterior descending artery,[1,8] while 78.6 % of left ventricular pseudoaneurysms[8] develop in posterolateral MI as a result of circumflex artery thrombosis.[7,8,12] An explanation for the greater prevalence of pseudoaneurysms in the posterolateral left ventricular wall, as compared to the anterior wall, is that anterior wall ruptures always have a vivid clinical picture, a fulminant course with the development of tamponade and do not have time to confine themselves to pericardial adhesions.[8]

Furthermore, the patient had the majority of the known risk factors for both, left ventricular rupture – female gender, age over 65, uncontrolled arterial hypertension, history of prior cerebrovascular event, and for pseudoaneurysm – silent posterolateral MI, transmural lesion and the wall thinning at the site of MI.[1,7,13,14] Clinical manifestations following pseudoaneurysm development were the signs of congestive HF, i.e. were not specific and could not alert the clinicians to possible left ventricular pseudoaneurysm. Other authors also pay attention to the non-specificity of clinical manifestations and physical examination data.[8,15,16] Chest X-ray in terms of exclusion or confirmation of pseudoaneurysm, as in this case, is often not informative (a non-informative method).[1,16]

It should be noted that the ECG was not very informative either, since with such a gross damage to the left ventricular wall, instead of qR waves in leads II, III, AVF, AVL, V5, and V6, one should expect the presence of QS or Qrs, which would confirm transmural damage to the myocardium of the posterolateral left ventricular

wall. Other authors also draw attention to the lack of marked ECG changes in the development of left ventricular pseudoaneurysm due to posterolateral MI,[12,17] in contrast to anterior left ventricular wall ruptures, which make the ECG highly informative.[14]

It is obvious that in this case, like in the most cases described in literature, left ventricular pseudoaneurysm was diagnosed accidentally by echocardiography in MI, congestive HF, rhythm disturbances, and thromboembolism.[2,8,9,12] Therefore nowadays, echocardiography as a widely available, noninvasive and informative diagnostic method has become the method of choice for early diagnosing of left ventricular pseudoaneurysm.[1,2,12,13,16,18]

Of course, in diagnostically complex cases, as well as in preparation for surgery may require the additional carrying out of other, but more expensive, imaging techniques: magnetic resonance imaging, multispiral computed tomography or ventriculography.

Since left ventricular pseudoaneurysm is a very rare condition, there is no randomized controlled study, to guide a management strategy. The information on surgically treated and untreated left ventricular pseudoaneurysms is not structured, and based on retrospective analysis of single cases.[13] No large studies comparing the results of surgical and drug treatment of left ventricular pseudoaneurysms have been conducted.

According to the current management strategy, patients with left ventricular pseudoaneurysm require surgical intervention if pseudoaneurysm is large, acute (less than 3 months), and localized in the anterior wall of the left ventricular.[1,8,13] Unlike true aneurysms, in which the wall is intact but fibrous and thin, the wall of pseudoaneurysms contains only pericardium and thrombus[3,4] and no endocardial or myocardial tissue.[1,13] It explains a high risk of rupture and tamponade formation, which accounts for 30-45%.[3] Such ruptures are very unpredictable in terms of onset and development.

However, postoperative mortality is also high (35.7%)[8] because postoperative outcome is influenced by such factors as stage of HF, MI size, patient's overall heath and comorbidities. Moreover, surgical treatment is not always limited to pseudoaneurysm resection and closure of ventricular defect. Some cases require mitral valve replacement or coronary bypass surgery,[8] which makes risks of poor outcome even higher.

Meanwhile, some authors[1,10,11,13] believe that poor prognosis for such patients is more likely to be linked to progressive HF and thromboembolic complications, but not to a possible fatal rupture. Having observed the patients with pseudoaneurysm for four years, T.C. Yeo et al.[10] show that not all pseudoaneurysms have a high risk of rupture. Varvarigos N. et al.[1] suggest that small (up to 3 cm) chronic asymptomatic aneurysms which are not prone to increase may be treated only medically. Moreno R. et al.[11] think that the long-term prognosis for patients with post MI left ventricular pseudoaneurysms is relatively good, with low risk of fatal ruptures, which should be considered while choosing surgical intervention as the main treatment strategy. Díaz-Navarro R. and Nihoyannopoulos P.[18] also it's believed that despite the fact that surgical treatment is the method of choice to avoid the risk of fatal myocardial rupture, the long-term results of medical treatment of patients with left ventricular pseudoaneurysm appear to be relatively favorable. However, when surgery is an absolute indication but the patient's prognosis is quite poor, decision-making process for a clinician is very difficult and even counter-intuitive, especially if a patient refuses surgery. In published literature, there are cases which describe patients who refused surgical intervention in spite of having absolute indications and bad prognosis, medical treatment remained the only option for them.[1,2] It is necessary to note that after diagnosis, such patients were successfully receiving medical treatment for 4[1] and 2 years,[2] their condition being stable. Roa-Castro V.H. et al.[2] suggest that the long survival of such patients is due to dense pericardial adhesions. Varvarigos N. et al.[1] believe medical therapy to be the only optimal treatment for high-risk patients refusing surgery. Hulten E.A. et al.[13] also think that for patients with chronic pseudoaneurysm (more than 3 months) who have high risk for surgical intervention "conservative management may be

prudent".

Taking into account the above information, the opinions of cardiologists and cardiac surgeons on the management of this patient were divided. Those who insisted on surgical treatment argued their position with high risk of pseudoaneurysm rupture and an equally high risk of thromboembolic complications, and those who proposed conservative management of the patient, with extremely high risk of surgical intervention (advanced age, low ejection fraction, presence of severe concomitant pathology: a previous stroke). Nevertheless, the patient was offered surgery which she denied (twice, including previous hospitalization). Next 3 months, as well as 3 previous months, the patient treatment was conservative.

CONCLUSION

The case we report proves that left ventricular pseudoaneurysms have non-specific clinical presentation even if they are giant and have a rough wall defect. They are often diagnosed accidentally by echocardiography after the pseudoaneurysm has already been formed. Therefore, when examining patients with echocardiography, primarily with congestive heart failure, who have had posterolateral MI, it should be recommended to be alert to exclude left ventricular pseudoaneurysm, especially if the patient is a female, older adults and senile, with arterial hypertension and a history of acute cerebrovascular accident.

As the patient with the giant left ventricular pseudoaneurysm, massive thrombi formation, and unsatisfactory compliance to treatment did not develop the two major complications – thromboembolism and wall rupture which could have inevitably led to sudden and rapid death, this case proves that medical treatment can be the method of choice in patients with left ventricular pseudoaneurysm. Although the survival period was limited to only 6 months, it could definitely have increased with the patient's better compliance to treatment. This is especially important for older patients who refuse surgical treatment or for patients with an extremely high risk of surgical treatment, when conservative treatment is forced to become the only method.

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ETHICAL STATEMENT

The research work done for preparing rare the clinical case was approved by the Interuniversity Ethics Committee.

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