Pseudoaneurysm of the Left Ventricle in a Patient with Silent Myocardial Infarction

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Key words:
Left ventricle pseudoaneurysm, silent myocardial infarction, echocardiograhy.

We describe the case of a 63-year old man with a pseudoaneurysm of the left ventricle (PSA) after a silent myocardial infarction. The patient manifested symptoms of heart failure and the pseudoaneurysm was echocardiographicaly diagnosed. Coronary angiography revealed three-vessel coronary artery disease. The patient underwent successful surgical pseudoaneurysm resection and complete myocardium revascularization.

Manuscript received:
April 2, 2002;
Accepted:
November 4, 2002.

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Pseudoaneurysm of the left ventricle (PSALV) is an exceptionally rare cardiac complication, which is created after wall rupture of the left ventricle[1]. Myocardial infarction, is the most common cause that leads to cardiac rapture and formation of PSALV[2-3]. Other less common causes are cardiosurgical operations (replacement of cardiac valves, resection of aneurysm of the left ventricle, etc.) penetrating cardiac trauma and infectious endocarditis[4-6].

PSALV can develop a few days (3-6) after myocardial infarction or even much later (2 years)[7]. The most frequently reported clinical symptoms of PSALV are heart failure (36%), chest pain (30%), dyspnea (25%) and sudden death (3%). Although 10-13% of the reported cases of patients with PSALV are asymptomatic, the development of PSALV following silent myocardial infarction has never been reported in the literature[8].

We describe the case of a male patient with severe coronary artery disease, in whom development of PSALV presented with recent onset dyspnea, possibly after a silent myocardial infarction. The patient was admitted to the hospital immediately and underwent surgical resection of PSALV and complete revascularization of the myocardium.

Case report

A 63 year-old man was admitted to our clinic after 15 days of consistently worsening dyspnea during effort and at rest. The patient reported that his brother had undergone coronary artery by-pass while his personal medical history was clear. The clinical examination revealed rhythmical arterial pulse, cardiac frequency 80 beats/minute and arterial pressure 110/80 mmHg. The patient was hemodynamically stable, was not complaining of dyspnea and no distension of the jugular veins was observed at a sprawl angle of 45°. Lower limb edema or liver enlargement were also not observed.

No pathological findings were observed on heart and lung auscultation. The chest x-ray in the posteroanterior projection showed enlargement of cardiovascular shadow with lengthening and increased convexity of the left lower cardiac border and in the left anterior oblique position, the cardiac arc of the left ventricle protruded beyond the border of the spine (Figure 1).

The ECG showed sinus rhythm with QS complex morphology with T-wave inversion in leads II, III, aVF, counterclockwise rotation and T-wave inversion in leads V5 and V6 (Figure 2).

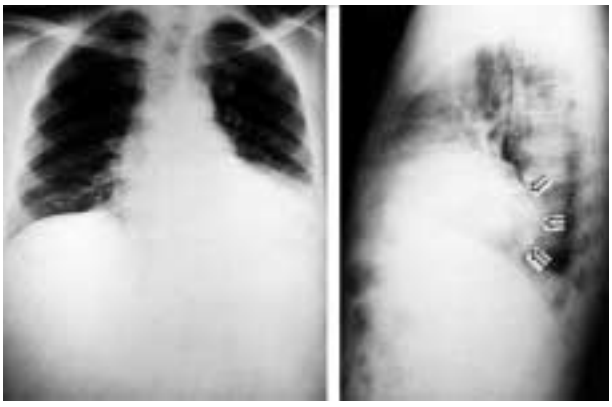


Figure 1. Enlargement of the cardiovascular shadow. (A) Lengthening and increased convexity of left lower cardiac border. (B) Protrusion of left ventricular segment beyond the border of the spine.

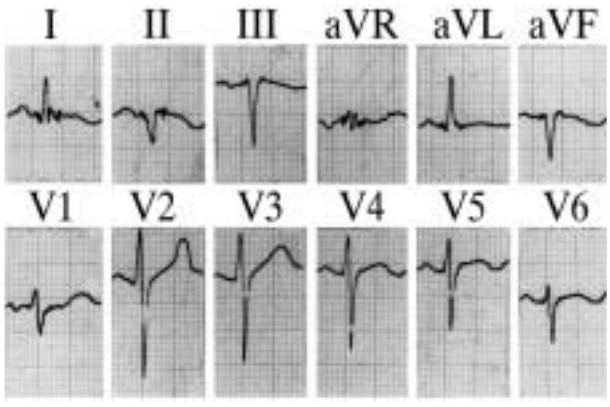
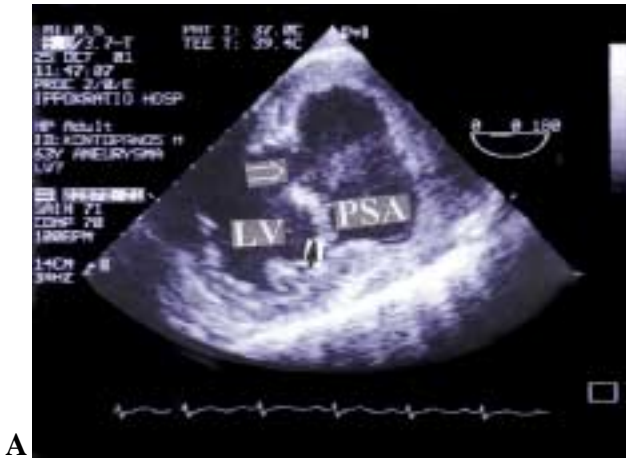


Figure 2. QS complex with T-wave inversion in leads II, III and aVF. Counterclockwise rotation and T-wave inversion in leads V5 and V6.

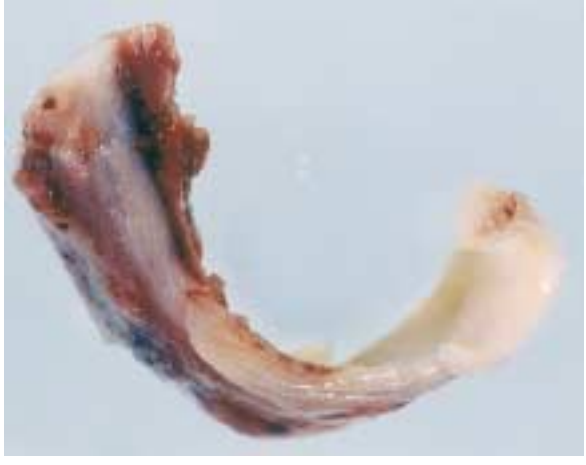
Hematological tests were normal and biochemical tests showed a slight increase in urea (74 mg/dl), uric acid (10.3 mg/dl) and total cholesterol (264 mg/dl). The levels of cardiac enzymes and troponine were normal.

Transthoracic echocardiography showed an enlarged left ventricle with decreased systolic performance (ejection fraction 30%), wall rupture in the posterolateral section of the left ventricle and imperceptible notation of a large aneurysm in the respective area, the internal surface of which was covered by thrombus. The site of cardiac rapture (width 3 cm) in the posterolateral section of the left ventricular wall, with thinness and increased echogenity of the corresponding area and a large aneurysmatic sac (oblong diameter 6 cm) the internal area of which was covered by a thick thrombotic layer, were recorded in transesophageal echocardiograph, mainly in transgastric view at the short axis (Figure 3A). In addition, contrast echo was observed within the aneurysmatic sac. Systolic flow to the cavity of the aneurysm and diastolic regurgitation to the cavity of the left ventricle were recorded with color Doppler.

Based on the echocardiography findings, diagnosis of pseudoaneurysm of the left ventricle was



A



B

Figure 3. (A). Transgastric view of transesophageal echocardiogram. Visible: the site of the rupture (big arrow) with increased thinness and echogenity of the posterolateral section of the left ventricle (black arrow), the large cavity with the thick thrombotic layer internally (white arrows) and the contrast echo inside the aneurysmatic sac (PSA). (B) The specimen that was removed during the operation. LV=Left ventricle. PSA= Pseudoaneurysm.

established and on the same day of his admission to the clinic, the patient underwent coronary angiography and left ventriculography. Coronary angiography showed severe stenosis of the three coronary arteries (anterior descending 90% in the 1° third part, circumflex 90% in the proximal section and right coronary artery 90% in 3° third part) and left ventriculography showed increased size of the left ventricle with decreased ejection fraction and large aneurysm in the inferior wall.

On the 5th day after his admission to the clinic, the patient was transferred to the cardiosurgical clinic where he underwent successful complete revascularization and PSALV resection. The section of aneurysm that was removed was sent for histological examination. The morphology of the specimen was elastic, its dimensions were 5×2 cm, its thickness 0.2 cm and its internal surface was covered by a thrombus of up to 1 cm thickness (Figure 3B). Histological examination showed that the specimen consisted of fibromyxomatous connective tissue with thin layer of inflammatory cells and infrequent deposits of hemosiderin. This tissue was connected to a thrombotic layer internally and with connective fatty tissue (pericardiac fat) externally (Figure 4A). The connective fatty tissue showed a mild fibroblastic reaction with focal inflammatory filtrations with mainly perivascular distribution (constituting of lymphocytes, plasmocytes, mastocytes, a few eosinophils and histiocytes), (Figure 4B). With histochemical color marker, three-color Masson and immunohistochemical stain desmin, no myocardial fibres were identified. *Conclusion*: Pseudoaneurysm of the left ventricle.

The postoperative course of the patient was normal and approximately 4 months after the operation, the clinical status of the patient was excellent.

Discussion

The PSALV is described as a located cavity which is formatted following rupture of the wall of the left ventricle and withholding of the blood from the solid fibrous symphysis of the pericardium layers[9]. The PSALV is a rare complication of myocardial infarction[1].

The timely diagnosis of PSALV and its immediate surgical treatment are of great clinical importance for rescuing the patient, because of the increased danger of acute rupture of aneurysm and sudden death[10]. In a recent review article, Frances et al describe the etiology, clinical picture, diagnostic value of various techniques, prognosis and the treatment of this serious post infarction complication, in detail[8].

The probable events that lead to the creation of PSALV are: Establishment of transmural myocardial infarction causes local reactive pericarditis at the site of infarction and consequently the growth of fibrous symphysis between the visceral and parietal layer of pericardium. Rupture of the myocardial wall at the necrosis site and escape of blood, which is entrenched by the symphysis of pericardium shaping the aneurysmatic sac. This sac progressively enlarge and its internal surface is covered by thrombus[10].

Davidson and other authors reported that cardiac rupture may occur even two years after myocardial infarction[7,11]. On the contrary, Slater et al sug-

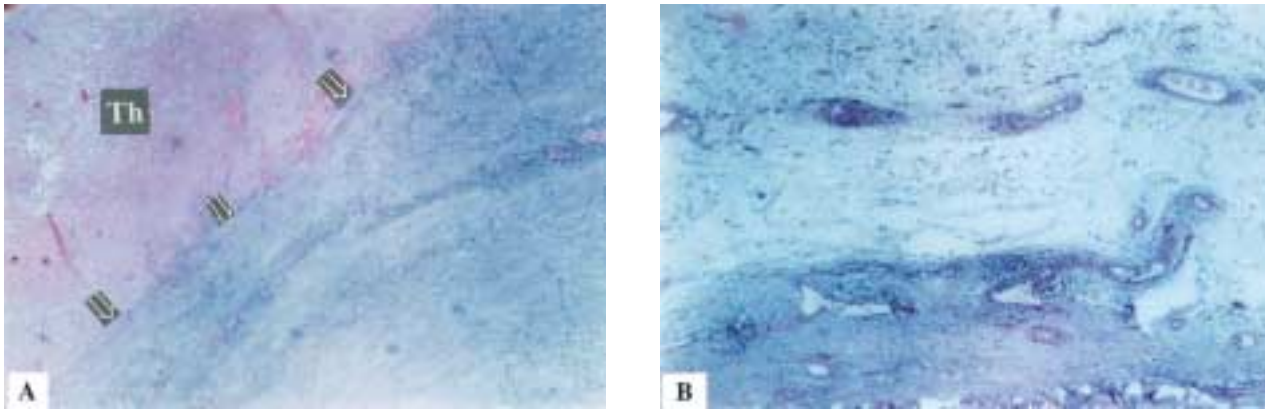


Figure 4. (A) The wall of the aneurysm consisted of fibromyxomatous connective tissue connected internally (arrows) with the thrombotic layer (Th) and externally with connective-fatty tissue. (B) The connective-fatty tissue shows a mild fibroblastic reaction and topical inflammatory filtration.

gest that previous infarction with the appearance of fibrosis or pericardiac inflammation, provide protection against cardiac rupture during the next myocardial infarction[12]. This probably implies that cardiac rupture mainly occurs during the first days of infarction. In our case, although the time of acute necrosis or the moment of cardiac rupture cannot be precisely determined, the increased thickness of the specimen excised during the operation, the existence of a thick layer (one centimeter) of thrombus, the presence of fibromyxomatous tissue with deposit of hemosiderin and the thin layer of inflammatory cells speculate that acute necrosis and cardiac rupture occurred a long time before the appearance of symptoms of heart failure. Moreover, the ECG changes did not reveal recent acute transmural necrosis. Therefore, despite the predominance of opposing opinions concerning the time of cardiac rupture, it seems that acute transmural necrosis and cardiac rupture may occur long before the onset of heart failure symptoms. It is even possible, as in our case, that transmural necrosis may appear as silent myocardial infarction.

The clinical manifestation of PSALV development in our patient, was the appearance of left heart failure symptoms. This is in agreement with the clinical observations of Frances et al who found that most patients (36%) with PSALV had symptoms of heart failure[8].

Echocardiography is the method of choice for prompt diagnosis of PSALV and immediate clinical decision for rescuing the patient[13-15]. In our case, although diagnosis was established by transthoracic echocardiography, the transesophageal echocardiograph (transgastric view), showed a more distinct depiction of the site of the cardiac rupture, the extent of rupture, the size and localization of PSALV (Figure 3A). The small diameter of the rupture as compared to the length of the large diameter (ratio <0.5) and the presence of thrombus in the internal wall of the aneurysmatic sac constitute the most characteristic echocardiographic criteria of PSALV. In addition, in the transesophageal imaging, apart from the thick layer of thrombus which covered the internal surface of the aneurysm, an intense echo contrast was also recorded inside the cavity of the aneurysm. Moreover, the systolic velocity of the blood flow towards the cavity of the aneurysmatic sac and the regurgitant diastolic flow velocity towards the cavity of the left ventricle were recorded with color Doppler. This change of blood flow velocity, during the phases of cardiac cycle, between the left ventricle and the aneurysmatic sac, constitutes an additional diagnostic echocardiographic criterion of PSA[14].

PSALV, as observed in our case, is more usually located in the posterior and less often in the anterior surface of left ventricle[8,16]. The interpretation for the rare appearance of aneurysm in the anterior surface in relation to the posterior surface of the left ventricle is that the rupture of the anterior wall more often leads to hemopericardium and death[17]. Also, the sprawl of the patient at the hospital phase of infarction encourages the induction of inflammatory reaction in the posterior pericardium and the development of PSALV at this site of the left ventricle[8].

Despite the undeniable value of echocardiography for the diagnosis of PSALV, histological examination is necessary to confirm that the wall of the aneurysmatic sac does not contain myocardial fibres[7]. The direct macroscopic examination does not provide appreciable information on the presence or not of myocardial fibers. In our case the diagnosis of PSALV was confirmed by histochemical three-color Masson marker and immunohistochemical marker, desmin, that showed complete absence of myocardial fibers in the wall of the aneurysmatic sac (Figure 4).

Our case is of interest because it confirms the value of echocardiography in the diagnosis of PSALV for immediate clinical decision and prompt surgical treatment, it proves that silent myocardial infarction may precede cardiac rupture and it speculates that transmural necrosis and cardiac rupture may occur long before the onset of heart failure symptoms.

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