Large infero-posterior wall pseudoaneurysm of the left ventricle: an unusual presentation

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Post-myocardial infarction left ventricular pseudoaneurysm resulting from free wall rupture is a rare finding and its recognition during life is uncommon. The diagnosis is difficult since symptoms, clinical evaluation and electrocardiographic and X-ray findings are usually non-specific. We herein present a case of a pseudoaneurysm manifesting after a silent myocardial infarction and diagnosed at echocardiography in a patient who, at the time of hospitalization, had a history and clinical and laboratory findings suggestive of pulmonary embolism. The patient was successfully operated. The present report underlines the diagnostic and prognostic value of two-dimensional transthoracic echocardiography. The clinical and laboratory findings are also discussed in the light of the recent literature.
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Case report

A 68-year-old male patient was admitted to the Medical Department with dyspnea and symptoms suggestive of congestive heart failure. The patient had stopped cigarette smoking 5 years previously when he had been submitted to resection of the upper lobe of the left lung due to the presence of a pulmonary neoplasm. Radiation therapy was subsequently performed and, at that time, electrocardiography and the overall cardiac evaluation were normal. Moreover, the patient's history also included chronic obstructive pulmonary disease and a fracture of the left ankle 4 months before hospitalization. The left lower limb was immobilized in a plaster cast for 45 days, without anticoagulation prophylaxis. During the last 6 weeks, exertion dyspnea increased, whereas in the last week symptoms of heart failure developed: dyspnea at rest, orthopnea, fatigue, dizziness and cough. No previous history of chest pain/myocardial infarction was reported. The patient was currently on beta$_2$-adrenergic agonist (salmeterol; aerosol) plus corticosteroid therapy (fluticasone propionate; aerosol). At the time of admission to the Medical Department, his blood pressure was 150/85 mmHg and physical findings included tachypnea (respiratory rate 28 breaths/min), tachycardia (110 b/min), a grade 2/6 systolic heart murmur, an elevated jugular venous pressure, inspiratory crackles and peripheral edema (particularly evident in the left leg).

Laboratory findings included elevated D-dimer (3812 ng/ml, normal value < 500 ng/ml) and troponin I (0.15 ng/ml, normal value < 0.1 ng/ml) serum levels. The serum concentrations of creatinephosphokinase-MB, glucose and total cholesterol and the blood cell count were within normal limits. Arterial blood gas analysis included: pH 7.42, pO_2 58 mmHg, pCO_2 41.5 mmHg, SO_2 91.9% (Tab. I).

The electrocardiogram revealed sinus tachycardia, premature supraventricular beats, left atrial enlargement, left ventricular hypertrophy, rSr' in V_1, poor R wave progression in the V_1-V_2 precordial leads and non-specific ST segment and T wave abnormalities (Fig. 1). Chest X-ray revealed a slightly enlarged heart and mild bilateral pleural effusion (Fig. 2). The patient's history and clinical and laboratory findings were suggestive of pulmonary embolism. Intravenous unfractionated heparin was started, and the patient was referred to the Cardiology Unit. At that time, a pseudoaneurysm was incidentally discovered during two-dimensional transthoracic echocardiography demonstrating no right ventricular dysfunction, akinetic motion of the infero-posterior segments of the left ventricle, the unexpected presence of a rupture in the infero-posterior wall of the left ventricle with a large echo-free space adjacent to the wall and a voluminous, stratified

Table I. Arterial blood gas analysis at the time of admission.

Blood gas parameters	
pH	7.424
pCO_2 (mmHg)	41.5
pO_2 (mmHg)	58.4
Acid-base status	
ABE_c (mmol/l)	2.5
SBE_c (mmol/l)	2.6
$cHCO_3$ (P,st)$_c$ (mmol/l)	26.5
Oximetry parameters	
ctHb (g/dl)	13.3
Hct_c (%)	40.9
FO_2Hb (%)	90.3
FCOHb (%)	1.7
FHHb (%)	8.0
FmetHb (%)	0
Electrolyte parameters	
cNa^+ (mmol/l)	136
cK^+ (mmol/l)	4.5
cCl (mmol/l)	102
Ca^{2+} (mmol/l)	1.22
$mOsm_c$ (mmol/kg)	279.1
Metabolite parameters	
Glucose (mg/dl)	112
Lactate (mg/dl)	10
Bilirubin (mg/dl)	0.1
Blood gases corrected for temperature	
pH(T)	7.424
pCO_2(T) (mmHg)	41.5
pO_2(T) (mmHg)	58.4
Oxygen status	
sO_2 (%)	91.9
ctO_{2c} (vol%)	16.9
$p50_c$ (mmHg)	24.07

thrombus (Fig. 3). The widest diameter of the orifice was 2.6 cm and the maximum internal diameters of the pseudoaneurysm were 6.3 × 7.9 cm (orifice/pseudoaneurysm diameter ratio 0.41). The left ventricular ejection fraction was 55%. A small pericardial effusion was documented. Color Doppler demonstrated flow from the left ventricle into the cavity (Fig. 4). Mild mitral regurgitation together with moderate tricuspid regurgitation associated with pulmonary artery hypertension (tricuspid regurgitant jet velocity 3.4 m/s) were also documented. Venous duplex scanning excluded deep thromboses; spiral computed tomography was negative for pulmonary embolism. Because of the propensity of the pseudoaneurysm to rupture, intravenous anticoagulation was stopped and the patient placed on loop diuretics, spironolactone, ACE-inhibitors, aspirin and beta$_2$-adrenergic agonists (aerosol). Coronary angiography confirmed the diagnosis of pseudoaneurysm (diameters 5.7 × 7.7 cm) and demonstrated the occlusion of the midportion of the left circumflex artery and a significant stenosis (95%) of the proximal right coronary artery. The patient underwent surgical patch repair and was discharged 2 weeks later. The postoperative course was uneventful. After a 5-month follow-up the patient is still asymptomatic.

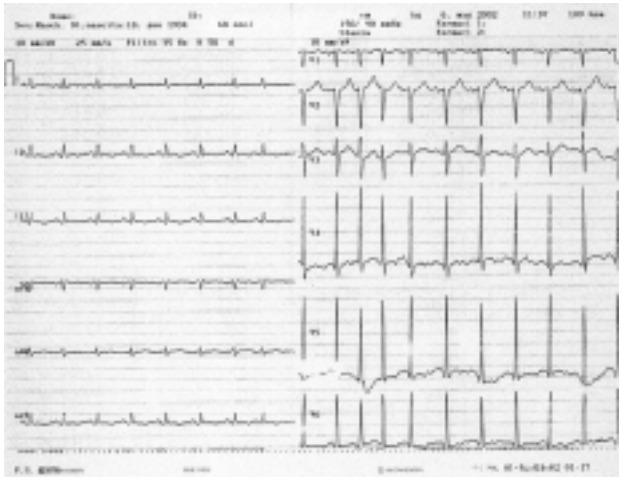


Figure 1. *Twelve-lead electrocardiogram at the time of hospitalization.*

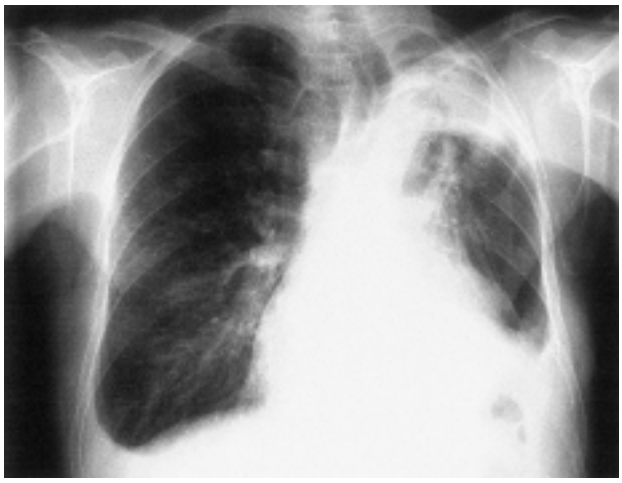
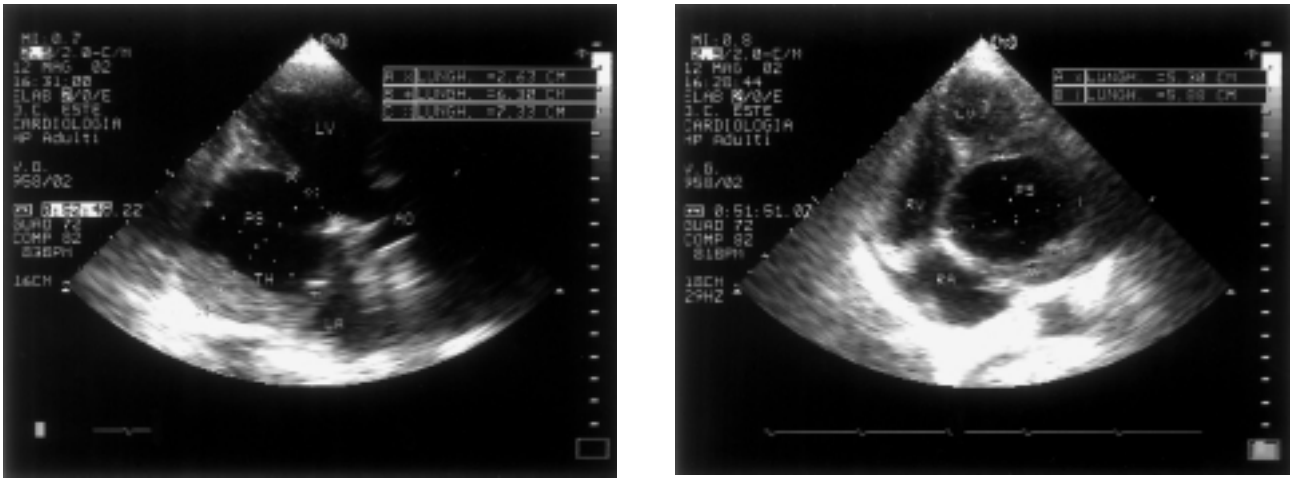


Figure 2. *Chest X-ray: a slightly enlarged heart and mild bilateral pleural effusion; no mass.*

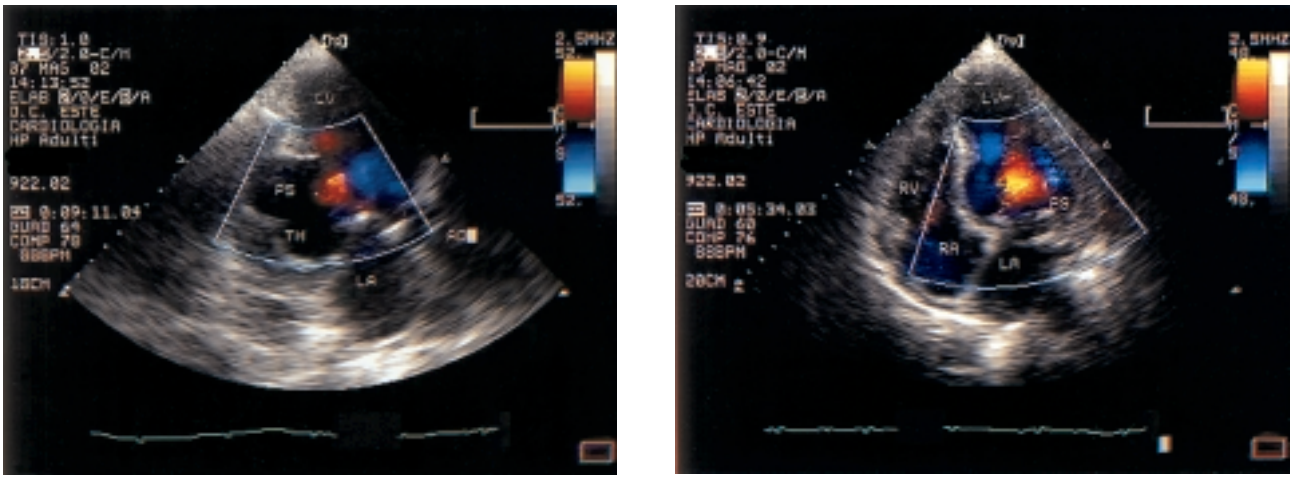
Discussion

Left ventricular pseudoaneurysm is clinically uncommon. In contrast to those of a true aneurysm, the walls of a pseudoaneurysm are composed of fibrous tissue devoid of the structural elements found in the normal wall. The rupture often leads to death[1-5]. The most common etiology of left ventricular pseudoaneurysm is myocardial infarction, especially in the infero-posterolateral walls. The reason for the prevalent occurrence at this site is not completely understood, but this finding may help to distinguish a pseudoaneurysm from a true aneurysm which occurs predominantly at the apex or at the anterior wall[3-5]. The right coronary and the left circumflex are the most involved arteries. The diagnosis of pseudoaneurysm is generally difficult since symptoms, clinical evaluation and electrocardiographic and X-ray findings are non-specific. Congestive heart failure, dyspnea and chest pain are the most frequently re-



A B

Figure 3. A: *3-chamber apical echocardiographic view of the pseudoaneurysm (PS) of the infero-posterior wall, showing the off-line measurements of the widest diameter of the orifice and of the maximum internal diameters.* B: *two-dimensional 4-chamber apical scan showing a large PS with a stratified thrombus (TH). Ao = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle.*



A B

Figure 4. *Three-chamber apical* (A) *and 4-chamber apical* (B) *echocardiograms showing flow from the left ventricle into the cavity of the pseudoaneurysm. Abbreviations as in figure 3.*

ported symptoms. A heart murmur is audible in more than two thirds of patients, and may be indistinguishable from mitral regurgitation. Almost all patients present with electrocardiographic abnormalities (95%): ST segment elevation is present in 20% of patients and non-specific ST segment changes in 75%. Chest X-ray abnormalities are present in 95% of patients: 65% have cardiomegaly and more than 50% have evidence of a mass. The patient described in the present report exhibited symptoms of cardiac failure and dyspnea, whereas the electrocardiographic changes and chest X-ray findings were not specific. Moreover, the history of lung cancer, the recent fracture of the left ankle, the sudden increase in dyspnea, the edema of the left leg, the elevated D-dimer value, and the arterial hypoxemia could have further supported the hypothesis of pulmonary embolism. Even though the patient with suspected pulmonary embolism was not in critical clinical conditions, we performed two-dimensional transthoracic echocardiography before venous duplex scanning and spiral computed tomography[6]. This technique excluded right ventricular dysfunction and allowed a conclusive, unexpected diagnosis of pseudoaneurysm, clearly delineating the location, size and shape of the site of rupture. Pulmonary artery hypertension was not related to pulmonary embolism, but, probably, to chronic obstructive pulmonary disease and to the previous resection of the upper lobe of the left lung. Our data highlight the importance of two-dimensional transthoracic echocardiography as a diagnostic as well as a prognos-

tic procedure in a patient with a pseudoaneurysm and, more generally, in the evaluation of right ventricular dysfunction and pulmonary hypertension in patients with suspected pulmonary embolism. Angiography of the left ventricle and of the coronary arteries confirmed the diagnosis of pseudoaneurysm and was a useful aid in the planning of surgery. The size of the rupture site as measured at echocardiography nicely correlated with the angiographic and surgical measurements. It cannot be excluded that prolonged aerosol corticosteroid therapy (fluticasone propionate) contributed to cardiac rupture in our patient with silent myocardial infarction.

The natural history of left ventricular pseudoaneurysm is still a question of debate. Several investigators recommend surgery as the treatment of choice due to the high risk of cardiac rupture. Improvements in surgical techniques have decreased the perioperative mortality to less than 10%[2]. However, a prolonged survival has been observed even in patients who were treated conservatively[3-5]. Natarajan et al.[1] reviewed the management of post-infarction pseudoaneurysm, described in 35 articles published between 1984 and 1993 (66 patients), suggesting that medical treatment of a chronic pseudoaneurysm (> 3 months) was not associated with an increased risk of cardiac rupture. In our case, the large dimensions of the pseudoaneurysm and of its orifice, and the supposed recent onset were both factors which strongly supported the surgical option.

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