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Kasuistika | Case report

Unexpected echocardiographic findings in one vessel coronary artery disease

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Klíčová slova:
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Nadměrná konzumace alkoholu

SOUHRN

Úvod: Aneurysma levé komory je jednou z nejvýznamnějších komplikací infarktu myokardu; předpokládá se, že k jeho tvorbě dochází u 5–10 % všech osob s akutním infarktem myokardu.
Kasuistika: Padesátiletý muž s anamnézou chronické nadměrné konzumace alkoholu a rizikovými faktory ischemické choroby srdeční byl dvakrát vyšetřen v krajské nemocnici pro srdeční selhání. V prvním případě nebylo echokardiografické vyšetření provedeno. O tři roky později byl dopraven na oddělení urgentního příjmu s bolestí na hrudi jako hlavní zdravotní obtíží. Echokardiografické vyšetření prokázalo jizvu zadní stěny levé komory s aneurysmatem na jeho bazální části, kde byl rovněž přítomen trombus. Následné vyšetření transezofageální echokardiografií potvrdilo rupturu komisurálních šlašinek posteriorního cípu mitrální chlopně spolu s těžkou regurgitací. Vzhledem k těmto nálezům byl pacient odeslán na koronarografické vyšetření, které prokázalo ischemické postižení jedné koronární tepny. U pacienta byla následně provedena náhrada nativní mitrální chlopně mechanickou spolu s odstraněním aneurysmatu v levé komoře.
Závěr: Vzhledem k anamnéze nadměrné konzumace alkoholu je pravděpodobné, že u nemocného proběhl němý infarkt myokardu zadní stěny (bazální) komplikovaný vznikem aneurysmatu na stěně levé komory.

ABSTRACT

Introduction: Left ventricular aneurysm is one of the most significant complications of myocardial infarction and it is thought to develop in 5–10% of all patients with acute myocardial infarction.
Case report: A 50-year-old male patient with a history of chronic alcohol abuse and risk factors for coronary heart disease was treated twice in the regional hospital center because of heart failure. Initially, echocardiography was not performed. Three years later he was admitted to the Urgent Center with a chief complaint of chest pain. He was evaluated by echocardiography, which showed a scar affecting the inferior wall of the left ventricle with an aneurysm on its basal portion which contained a thrombus. The patient also underwent transesophageal echocardiography which confirmed that there was a rupture of the commissural chordae tendineae of the posterior mitral leaflet with severe regurgitation. In consideration of these findings, the patient was referred to undergo coronary angiography, which revealed single vessel coronary artery disease. The patient subsequently underwent cardiac surgery for mitral valve replacement with an artificial valve, along with repair of the left ventricular aneurysm.

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Keywords:
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Bacterial endocarditis
Left ventricular aneurysm
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Conclusion: With consideration of his history of alcohol abuse, our patient likely experienced a silent inferior-basal myocardial infarction complicated by the development of an aneurysm of the ventricular wall. Subacute bacterial endocarditis may have been a contributing factor leading to infarction and rupture of the mitral valve chordae tendoneae, and causing symptoms of heart failure and chest pain in our patient.

Introduction

Left ventricular aneurysm (LVA) is one of the most important complications of myocardial infarction (MI) and is thought to develop in five to ten percent of all patients with acute MI. LVA is defined as an area of the ventricle in which there is only thin scar tissue devoid of muscle. The area is well-delineated and characterized by its walls bulging outward during systole expansion [1].

Other than MI, possible etiologies of LVA include hypertrophic cardiomyopathy, trauma, iatrogenic injury, Chagas' disease, sarcoidosis, mucopolysaccharidosis, congenital LVA, and idiopathic causes. Medical history, clinical examination, and laboratory test results may be used to evaluate for these conditions [2].

The case we present describes a patient who developed a true ventricular wall aneurysm, but is unusual because of the posterior location of the aneurysm. Only 3% of aneurysms, based on data, are posterior or inferior [3]. These data are from an in vivo series, different from an autopsy series that shows an equal distribution of aneurysms respectively in the anterior and posterior locations [4].

Case report

We present a case of a 50-year-old man with a history of chronic alcohol abuse and other risk factors for coronary heart disease who was admitted to our Urgent Center with a complaint of chest pain. Three years previously, he was treated twice in the regional hospital center because of heart failure. Echocardiography had not been performed then. However, with the current admission, he was examined by echocardiography which revealed the presence of a scar of the inferior left ventricular wall with an aneurysm on its basal portion containing a thrombus (Fig. 1). The patient also underwent transesophageal echocardiography examination which indicated that there was a rupture of the commisural chordae tendineae with a loss of support for P3 segment of posterior mitral leaflet (Fig. 2). Severe mitral regurgitation was seen along with an enlarged left atrium (Fig. 3). On evaluation of his EKG, there was a finding of scar affecting the inferior wall (Fig. 4). As a result of these unexpected findings seen by

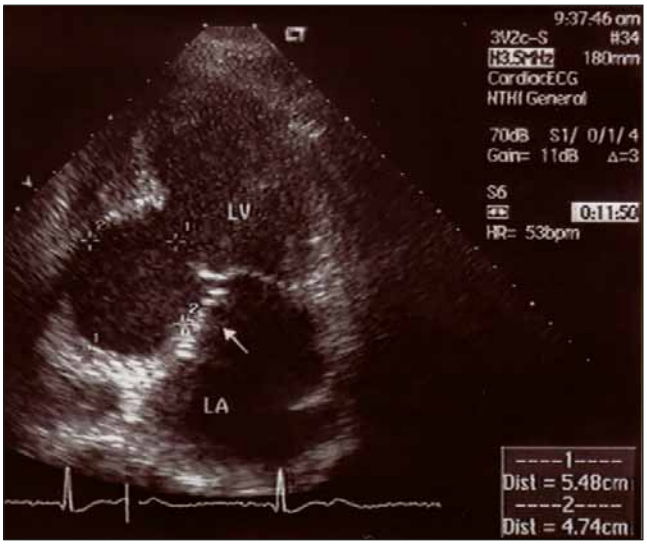


Fig. 1 – Transthoracic echocardiography showing aneurysm of basal portion of the inferior wall.

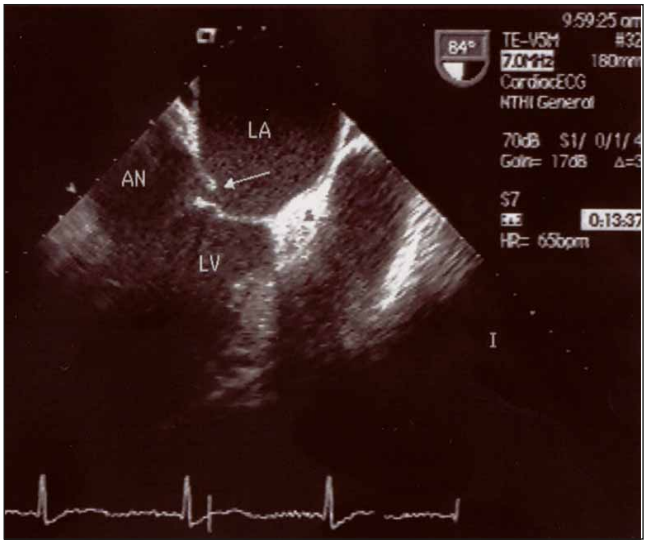


Fig. 2 – Transoesophageal echocardiography showing rupture of commissural chordae of the posterior mitral leaflet.

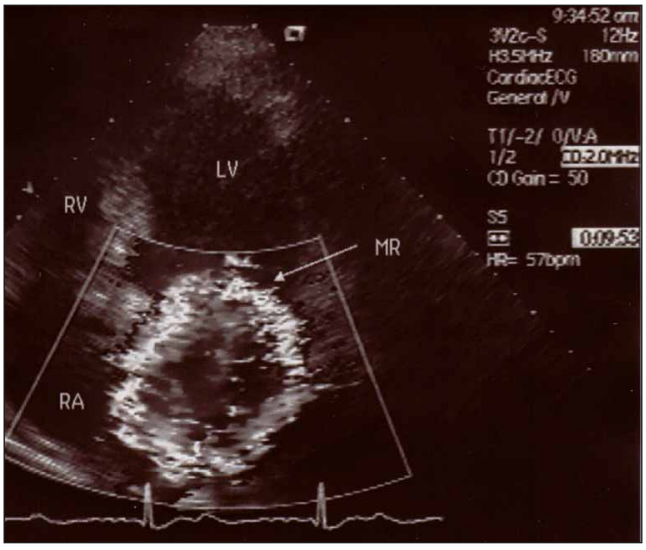


Fig. 3 – Transthoracic echocardiography – severe mitral regurgitation.

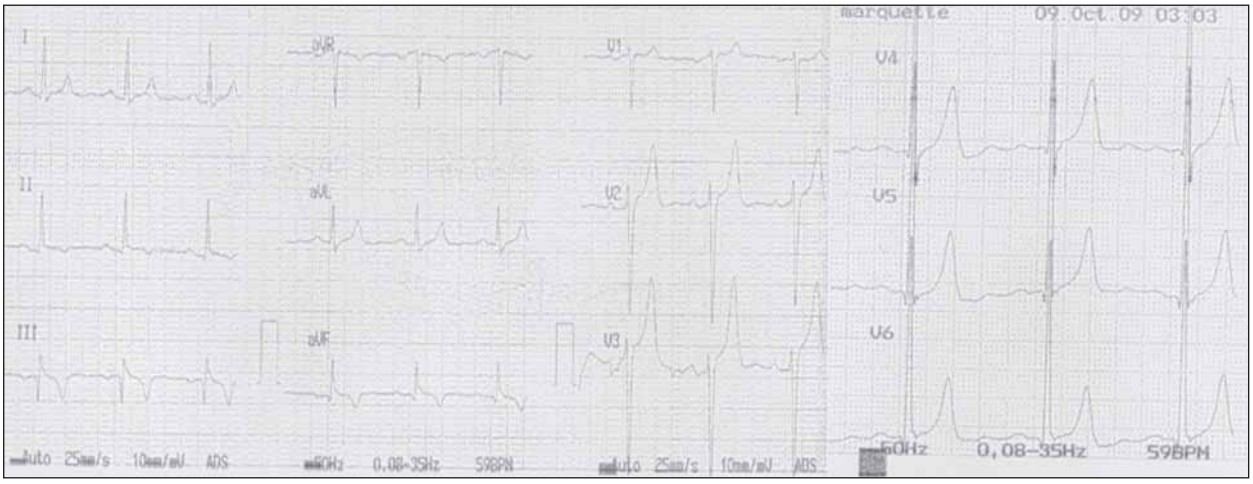
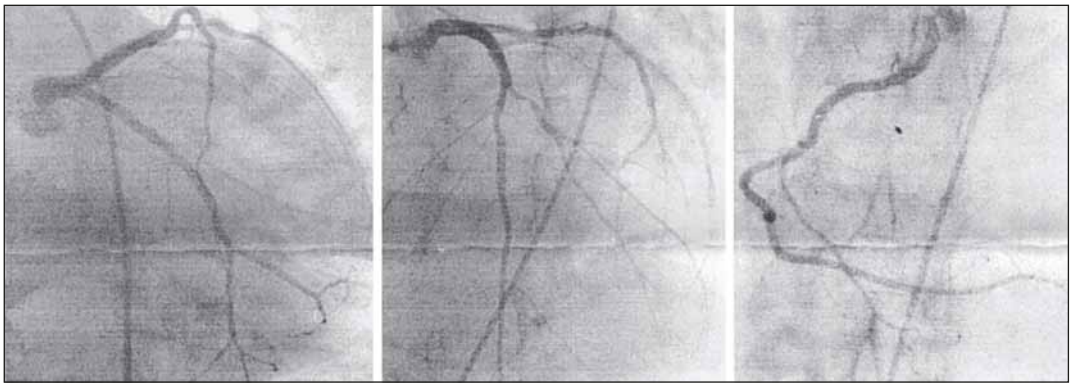


Fig. 4 – EKG – scar of the inferior wall.



LM + Cx LAD + D1 RCA

Fig. 5 – Cardiac angiography.

echocardiography, the patient was referred to undergo coronary angiography, which revealed that the first diagonal artery (D1) was obstructed up to 80% and the right coronary artery and circumflex coronary artery (Cx) were without any stenosis (Fig. 5). The patient was sent for cardiac surgery, during which the mitral valve was replaced by an artificial valve and the left ventricular aneurysm was repaired by a reconstructive technique called the Dor procedure (pioneered by Dr. Vincent Dor). Postoperative echocardiography revealed good mobility of the artificial valve with sufficient gradient across it and akinesis of basal part of the inferior wall with preserved contractility of the other segments.

Case report conclusion

Detection of one vessel coronary artery disease (significant stenosis of D1) alone does not explain the occurrence of left ventricular aneurysm detected on our patient's inferior wall. It is known that aneurysm of left ventricle with normal angiography findings could occur with sarcoidosis and some other systemic inflammatory conditions. However, these conditions were not found in our patient.

A bacterial analysis revealed findings of *Staphylococcus* in the smear taken from the surface of left ventricle and mitral valve. Therefore, we have concluded that in our patient, the possible etiology of infarction with aneurysm of the inferior wall was coronary spasm or embolic infarction that occurred as a consequence of subacute bacterial endocarditis (SBE).

Discussion

Left ventricular aneurysm (LVA) describes a discrete region of ventricular wall that is thinner than the adjacent myocardial segments, balloons outward, and exhibits either akinesis or dyskinesis [5]. LVA is strictly defined as a distinct area of abnormal left ventricular diastolic contour with systolic dyskinesia or paradoxical bulging. After myocardial infarct, fibrous scar tissue develops in about one month. Mural thrombus is present in about 50% of patients with an LVA, but rarely produces thromboembolism. True posterior aneurysms are often associated with post-infarct mitral insufficiency. Typically, LVA evolves over 6 months and is unlikely to enlarge after 1 year. The aneurysm leads to global cardiac remodeling and dilatation followed by reduction of systolic efficiency due to paradoxical movement of the aneurysm.

The incidence of LVA is about 10–30% after significant MI if untreated [1]. However, the incidence of LVA has

decreased significantly due to improvements in the treatment of acute MI inducing thrombolytic therapy, HTN control, and avoidance of corticosteroids [6]. The use of thrombolytic agents has decreased the incidence of LV aneurysm from 18.8% to 7.2% [7].

Postinfarction left ventricular aneurysm is a serious disorder that can lead to premature death. Major complications arising from the development of LV aneurysm include thromboembolism, ventricular arrhythmia, congestive heart failure, refractory angina (possibly from altered hemodynamic levels), and rarely, cardiac rupture [8]

Surgical treatment is indicated for established cases that have symptoms of congestive heart failure, angina pectoris, malignant ventricular arrhythmia, or recurrent embolization from the left ventricle (LV) [9]. Indications for operation include the presence of a large aneurysm (larger aneurysm size is a risk factor for premature death). Investigators have found 5-year mortality rates of 10% to 50%, depending upon aneurysmal size [8]. Data from the Coronary Artery Surgery Study (CASS) registry showed that survival in patients with LV aneurysm was related to age, LV function, and the clinical severity of heart failure, rather than to the presence of the aneurysm itself [10]. Surgical technique involves performing a remodeling ventriculoplasty (the Dor repair, pioneered by Dr. Vincent Dor) using a patch to recreate ventricular wall architecture. The prognosis is worse if there is pre-existing dyskinesia and poor function of the LV [1].

Typically, making an evaluation of a LVA presents a diagnostic challenge, as in our case. In most cases of LVA, patients have no symptoms and an incidental detection occurs by echocardiography showing a cavity in the left ventricle wall after MI. Otherwise patients may present with chest pain, dyspnea and hypotension [11].

In our case, we postulate that there are two possible etiologies for the development of LVA. First, the LVA may have developed as a consequence of a myocardial infarct leading to weakening and dilatation of the ventricular wall. Second, the LVA may have developed as a consequence of endocarditis, leading to an embolic infarction of the ventricular wall and its subsequent weakening.

With our patient, the D1 (first diagonal) artery was obstructed, yet the aneurysm was located in the inferior-basal left ventricular wall. It is unlikely that the LVA was related to the coronary artery distribution of the myocardial infarction based on its location. One acceptable hypothesis is that coronary artery emboli originate from mural thrombi, present in some patients with nonischemic dilated cardiomyopathy (NIDCM) or LVA, which develop due to local wall infarction and fibrosis. The possibility of mural thrombus-derived emboli is unlikely [12]. It is reported that the incidence of left ventricular aneurysm in MI with absolutely normal coronary angiogram is 0.47% [12]. Myocardial ischemia/infarction with a normal coronary angiogram is commonly characterized by coronary microvascular dysfunction, coronary spasm and coronary embolism with subsequent spontaneous clot lysis [13], retraction, or recanalization [14].

We believe that it is more likely that the LVA in our case was a result of subacute bacterial endocarditis. A bacterial analysis from our patient revealed findings of *Staphylococcus* in the smear taken from the surface of left ventricle and mitral valve. Bacterial endocarditis is characterized by an inflammation of the inner lining of the heart (endocardium) and particularly heart valves due to infection. With endocarditis, clots may become attached to the damaged valves and then tend to break apart. These fragments of infected tissue are carried through the blood where they may block an artery (embolism) or spread infection to other parts of the body. We postulate that this might be the reason for an embolic myocardial infarction in our patient [15].

In addition, some reports point to a higher incidence of bacterial endocarditis in cirrhotic patients. The development of endocarditis in alcoholic patients with cirrhosis may be the cause of the hematogenous origin of spontaneous bacterial peritonitis. Several authors have described an increased incidence (up to 90%) of miliary infarctions and microembolization in patients with endocarditis [16–19]. Therefore, our patient's history of alcohol abuse may have been a contributing factor towards his development of endocarditis.

Conclusion

We conclude that due to chronic abuse of alcohol, our patient had subacute bacterial endocarditis. This could be the reason for the development of an embolic myocardial infarction and rupture of chordae that led to the onset of symptoms of heart failure and chest pain. As a consequence of that infarction, he developed a ventricular wall aneurysm. Alternately, a silent inferior-basal myocardial infarction may have been manifested by previous symptoms of heart failure in earlier hospitalizations.

Conflict of interest
None declared.

Funding body
None.

Ethical statement
Authors state that the research was conducted according to ethical standards.

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