Inferior Wall Aneurysm of the Left Ventricle and Severe Mitral Regurgitation Following Acute Myocardial Infarction

Murat Yüksel[1], Nihat Polat[2], Ertan Vuruskan[3], İdris Ardıç[3]

ABSTRACT

Left ventricular aneurysms are seen rarely in primary percutaneous coronary interventional era. True aneurysms are usually localized to apex of the ventricle and it's very rare to develop in the inferior wall. In this paper, we presented a true left ventricle aneurysm, localized at the basal inferior wall which is developed in the course of subacute inferior myocardial infarction of a 74-years-old man. Aneurysm is detected during left ventriculography. The true ventricular aneurysm and consequent severe mitral regurgitation caused by tethering of mitral aparatus are confirmed with transthoracic echocardiography.

Key words: *True ventricular aneurysm, severe mitral regurgitation, coronary angiography, ventriculography, echocardiography*

Akut Miyokard İnfarktüsü Sonrası Gelişen Sol Ventrikül İnferior Duvar Anevrizması ve Ciddi Mitral Yetersizliği

ÖZET

Sol ventrikül anevrizmaları primer perkütan koroner girişim çağında nadir görülmektedir. Gerçek anevrizmalar genellikle ventrikülün apeksine yerleşiktir ve inferiyor duvarda yerleşimi çok nadirdir. Bu yazıda 74 yaşında bir erkek hastada akut inferior myokart enfarktüsü seyrinde gelişen bazal inferiyor yerleşimli gerçek anevrizma olgusu sunduk. Anevrizma sol ventrikülografi ile tespit edildi. Anevrizma ve yol açtığı ileri mitral yetersizliği ekokardiyografi ile doğrulandı.

Anahtar kelimeler: *Gerçek ventriküler anevrizma, ciddi mitral yetersizliği, koroner anjiyografi, ventrikülografi, ekokardiyografi*

INTRODUCTION

Left ventricular aneurysms are mechanic complications of myocardial infarction (MI). They are hemodynamically important pathologies. They were developing frequently during post-myocardial infarction period before reperfusion era; fortunately nowadays they are seen very rarely. We presented a case of elderly man who had acute inferior myocardial infarction with true aneurysm and severe mitral regurgitation (1).

CASE

A 74-years-old man was admitted to emergency department with complaint of prolonged chest pain and progressive dispnea. In his physical examination mild systolic ejection murmur was noticed at the left sternal border. Moist ralles were heard on 1/3 lower pulmonary areas. The electrocardiography revealed normal sinus rhythm with pathologic Q waves, 2-3mm ST segment elevation and T wave inversions in leads DII, III and aVF. Cardiac markers such as Troponin I, creatine kinase MB isoform and lactate dehydrogenase enzyme were elevated in the patient's serum. Prompt coronary angiography was preformed due to ongoing chest pain. It was revealed a poor distal run-off after total occlusion of proximal right coronary artery and a lesion narrowing %80 of the lumen of circumflex artery at mid level. Left anterior descending artery was normal. On left ventriculography

[1]*Department of Cardiology, Malatya State Hospital, Malatya, Turkey,* [2]*Department of Cardiology, Islahiye State Hospital, Kahramanmaraş, Turkey,* [3]*Department of Cardiology, Gaziantep State Hospital, Gaziantep, Turkey*

Correspondence: *Murat Yüksel*
Department of Cardiology, Malatya State Hospital, Malatya, Turkey
Phone: *+90 505 4450666*, **Fax:** *+90 422 3240650*
E-mail: *yukselmurat@yahoo.com*

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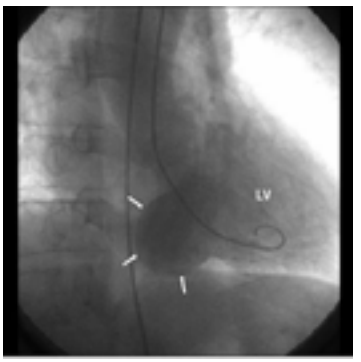

Figure 1. *Aneurysm (arrows) was demonstrated in basal region of the inferior wall on left ventriculography in right anterior oblique view in diastolic phase*

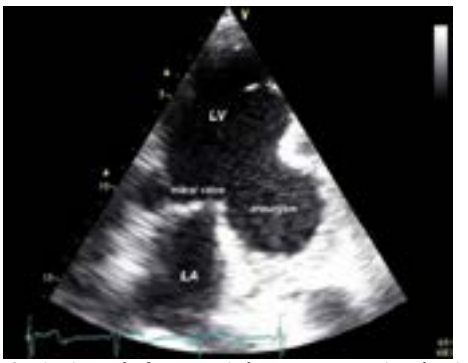

Figure 3. *A giant left ventricle aneurysm in the inferior wall showed on transthoracic echocardiography apical two-chamber view.*

at right anterior oblique view in diastole, true aneurysm was clearly demonstrated in basal region of the inferior wall (Figure 1). Transthoracic Echocardiography (TTE) performed to investigate aneurysm and mitral valve functions. On TTE, left ventricle was morphologically normal in apical four-chamber and parasternal long-axis views. However, apical two-chamber and parasternal short-axis views revealed giant (5.0x4.7 cm) aneurysm formation in the inferior wall of the left ventricle at its junction with posterior mitral leaflet. Aneurysm had thin muscular wall at similar echogenicity with the ventricular myocardium, so we defined it as a true aneurysm. Also aneurysm included a thrombus formation inside (Figure 2 and 3). Left ventricular dimensions were larger, basal and middle segments of lateral and posterior wall were hypokinetic, basal and middle segments of inferior wall were akinetic and aneurysmatic. There was a severe mitral insufficiency on TTE, because of tethering of posterior leaflet by posterior papillary muscle which originates from aneurysmatic ventricular segment (Figure 4). Urgent operation was planned due to unstable hemodynamic status. But patient rejected the operation. After coronary angiography and ventriculography were performed, patient was taken to the coronary intensive care unit and received medical treatment. His chest pain diminished after a few hours and dispnea resolved after his hemodynamic stabilization achieved with intensive medical treatment. Patient was anticoagulated with warfarin because of thrombus formation in the aneurysm. No lethal arrhythmias de-

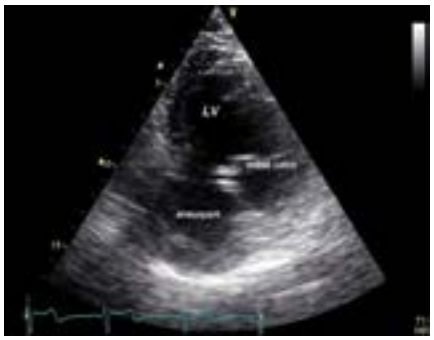

Figure 2. *On transthoracic echocardiography parasternal short-axis view revealed aneurysm in the inferior wall of the left ventricle.*

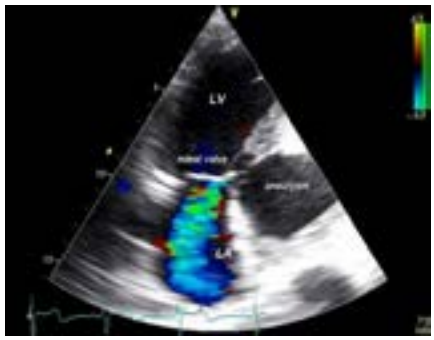

Figure 4. *Severe mitral insufficiency revealed on transthoracic echocardiography apical two-chamber view.*

veloped during in-hospital follow-up and the patient discharged from hospital at the sixteenth day of hospitalization.

DISCUSSION

Aneurysm formation is a consequence of infarct expansion (1). Clinical data suggest that infarct expansion occurs in approximately 35% to 45% of anterior MI and to a lesser extent in infarctions at other sites (1). But, true aneurysms of left ventricle are seen less than 5% of acute MI patients (2). Aneurysms are seen four times more often as a complication of anterior MI than inferior MI (2). They develop frequently after not collateralized left anterior descending artery occlusion. So, patients with three vessel disease might be protected, by their relatively richer collateral supply, from aneurysm formation during acute myocardial infarction (3). This information is about pre-reperfusion era. There are no data about in patients with promptly reperfused MI either by thrombolytic or percutaneous route. Echocardiography, magnetic resonance and left ventriculography are preferred imaging modalities. TTE is non-invasive and the most available tool. Also it gives more information about thrombus formation, pericardial effusion, myocardium and valvular functions. Magnetic resonance gives excellent three-dimensional images of aneurysm but it's unavailable in most of the emergent situations. Left heart catheterization is also a useful technique for diagnosis of aneurysm. It is also essential in the diagnosis of coronary artery disease and gives ad-hoc reperfusion treatment opportunity of culprit artery. But left ventriculography might be complicated by thromboemboli. Aneurysm formation increases the mortality of acute coronary syndrome up to six times beside the patients without aneurysms. Mortality usually occurs as sudden death (4). This is probably due to ventricular arrhythmias which is one of the most frequent complication of ventricular aneurysms (5). True aneurysms generally do not rupture to pericardium. But they steal the kinetic energy of ventricle by ballooning in systole (6). They are always a potential source of thromboemboli (7).

Mural thrombus was seen in recent case. Also they may cause moderate to severe mitral regurgitation by disrupting the anatomy of the origin of papillary muscles and consequent tethering of the mitral leaflets like in recent case. In the management of noncomplicated cases, medical treatment is consist of anticoagulation and routine heart failure medication. If the aneurysm is a focus for arrhythmias, or causes severe mitral regurgitation, or medical treatment fails in symptomatic relief, or coronary revascularization by surgery is planned at the same operation aneurysmal repair can be performed. But peri-operative mortality of aneurysmal repair is high especially if additional valvular or coronary by-pass surgery is needed (8).

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