

CASE REPORT

Left Ventricle Posterior Wall Aneurysms with Calcified Thrombus in the Aneurysmal Area

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Abstract

Aneurysms can be seen as complications of myocardial infarctions. They are generally located in the apical and anterior segments but left ventricular infero-posterior wall aneurysms are rare. After a myocardial infarction, left ventricular aneurysms can cause symptomatic intractable ventricular arrhythmias. We report a rare case of a left ventricular posterior wall aneurysm containing a calcified thrombus that caused ventricular tachycardia.

Key words: Coronary artery disease, Ventricular tachycardia, Aneurysms.

Introduction

Left ventricular (LV) aneurysms are seen in about 7.6 % of cases after myocardial infarction (MI). Eighty percent of left ventricular aneurysms are in the api-

cal and anterior segments of the ventricle. Only 5-10 % of aneurysms occur in the posterior wall. Detection of posterior aneurysms are more difficult than detection of anterior aneurysms. Clinically, anterior wall aneurysms more often cause wall ruptures and fatality (1,2). Clinical presentation of aneurysms is associated with angina, left ventricular failure, emboli or arrhythmias. Approximately 15% of patients with aneurysms have symptomatic intractable ventricular arrhythmias (2-5). We report a rare case of left ventricular posterior wall aneurysm complicated by a calcified thrombus presenting with ventricular tachycardia.

Case Report

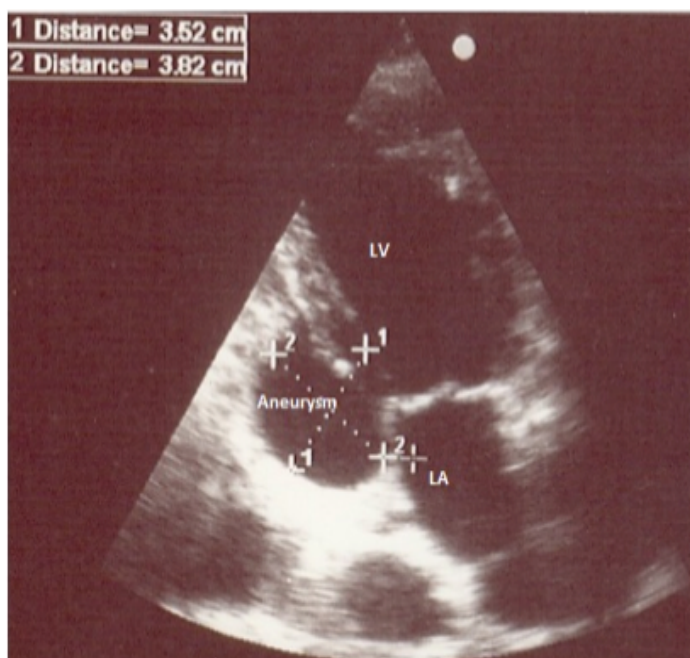
An 80-year old man was admitted to the hospital complaining of palpitations and dyspnea for one day. He had been a smoker for 70 years But He had no previous history of hypertension, diabetes mellitus or coronary

artery disease. He was hemodynamically stable and his first electrocardiogram (ECG) showed ventricular tachycardia (Figure 1). After cardioversion, his ECG showed sinus rhythm. There was an ST-depression in leads I, aVL and V4-6 and T wave inversion in leads II, III aVF. Because of persistence of the ventricular tachycardia, Continuous amiodarone infusion was started for treatment of persistent ventricular tachycardia. Despite this treatment, the ventricular tachycardia continued and required electrical

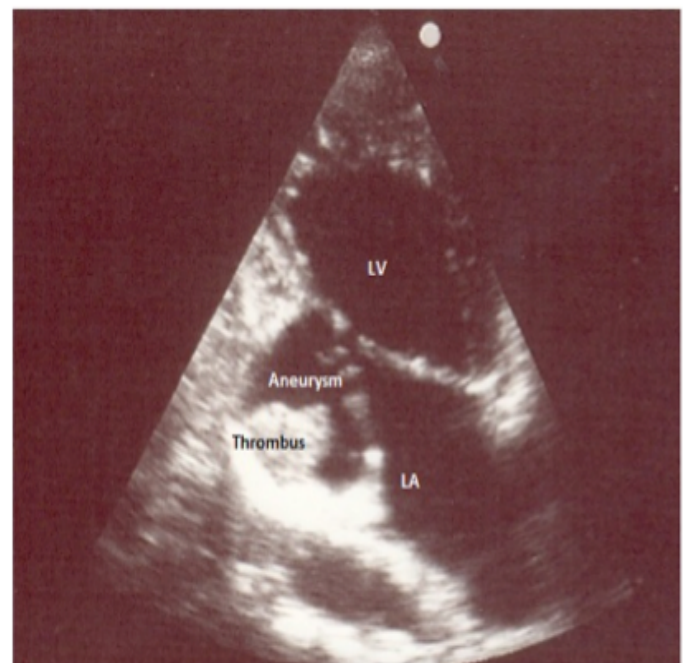
cardioversion. Twelve hours later, the ECG remained in sinus rhythm, T wave inversion continued in II,III, aVF and ST depression in V5-6. A transthoracic echocardiogram (TTE) showed a dyskinetic posterior left ventricular wall and mild mitral regurgitation, with moderate reduction of ejection fraction ($EF = 40\%$). An aneurysmal enlargement was noted in the posterior wall and a calcified thrombus was revealed within the aneurysmal area (Figure 2a and 2b). A coronary angiogram revealed left anterior descending artery



Figure 1. When patient admitted the electrocardiogram was ventricular tachycardia.



A



B

Figure 2. Transthoracic echocardiogram showed left ventricular posterior wall aneurysms (A) and detected a thrombus in the aneurysmal area (B).

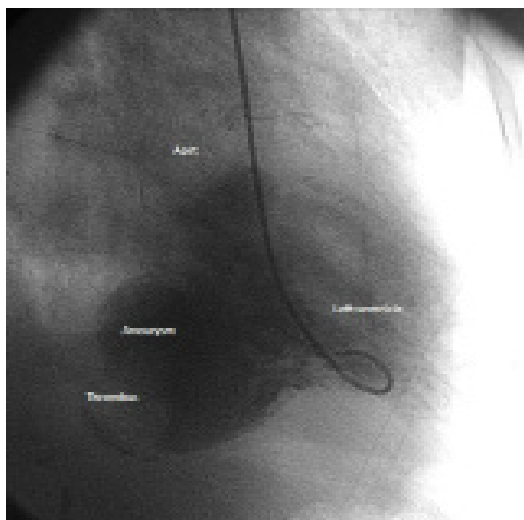


Figure 3. Contrast ventriculography showed an enlargement of the left ventricle with a large dyskinetic cavity localized in the diaphragmatic region and thrombus revealed in the aneurysm.

stenosis of 70% after the first diagonal branch, 80% before the second diagonal branch and 40% in the circumflex arteries. The right coronary artery stenosis was 30% proximally, 50-60% in the mid segment, and 50% distally. Contrast ventriculography showed an enlargement of the left ventricle with a large dyskinetic cavity localized in the diaphragmatic region and thrombus revealed in the aneurysmal area (Figure 3). Coronary bypass surgery was recommended but the patient refused. An internal cardiac defibrillator was inserted for treatment of ventricular tachycardia.

Discussion

Left ventricular aneurysms are generally seen as a complication after myocardial infarction. They are divided into two groups; true aneurysms and pseudoaneurysms. True aneurysms of the left ventricle are more common; characterized by a mouth or neck that is the largest part of the aneurysm and by the containment of myocardium and coronary arteries in their walls. Pseudoaneurysms are rare, they have a narrow neck and are restricted by adherent pericardium. These aneurysms don't contain myocardial elements in their walls and thus they are highly likely to rupture. After a myocardial infarction, a thin or disrupted myocardium moves dyskinetically,

or is non-contractile, leading to congestive heart failure. Dangerous ventricular arrhythmias can be seen with both true and pseudoaneurysms, as in our case (3,4). Complications of pseudoaneurysms are similar to those of a true aneurysm, but in contrast, they require emergency surgery. As both types of aneurysms present in a similar way, distinguishing one type from the other is often difficult (2).

A disastrous complication of the ventricular aneurysms is left ventricular free wall rupture. Rupture is generally seen early in the course of events and late rupture is extremely rare. Therefore, differentiation of true or pseudo-aneurysm is imperative and treatment of aneurysms is important (2). Many pseudoaneurysms may have pericardium as part of their wall. Posterior infarcts that cause posterior wall aneurysms may be lethal because of the involvement of the papillary muscles and consequent severe mitral valve regurgitation (2,3,6). Stagnant flow in the aneurysmal cavity may lead to thrombosis, or embolic events. Detection of aneurysms is difficult when a mural thrombus has filled the aneurysmal cavity (6). Echocardiography and contrast ventriculography, both are accepted ways for detection of aneurysm formation. (2).

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