

Left Ventricular Aneurysm with Normal Coronary Angiogram: A Case Report and Literature Review

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Most left ventricular aneurysms (LVA) develop in the atherosclerotic occlusive coronary artery disease following a transmural myocardial infarction. However, occasionally, LVA associated with normal coronary artery was found as well in dilated cardiomyopathy, hypertrophic cardiomyopathy, Chagas's disease, sarcoidosis, chest trauma, and congenital malformation. We report a 53-year-old male who had systolic heart failure owing to a posterior wall aneurysm of the left ventricle, which was found unintentionally during echocardiographic examination. Subsequent stress thallium scan revealed irreversible defects in the inferolateral wall and apex, and coronary angiography showed normal coronary arteries. The patient remained well under medical control for a follow-up period of 40 months. Previously undiagnosed inferoposterior wall myocardial infarction caused by thromboembolism with spontaneous reperfusion may be considered in this patient.

Key Words: Left ventricular aneurysm • Coronary artery disease • Normal coronary artery • Systolic heart failure

INTRODUCTION

Left ventricular aneurysm (LVA) is defined as a circumscribed, thin-walled, noncontractile outpouching of the ventricle.¹ The majority of LVAs occur after myocardial infarction and are usually located at the anterior or apical segment of the territory of the left anterior descending artery.² Occasionally, LVAs associated with normal coronary artery have been reported.³⁻⁶ Herein we present a case of LVA with normal coronary artery and review recent relevant literature.

CASE REPORT

A 53-year-old male visited our cardiology clinic on September 18, 2000, owing to intermittent chest tightness. Upon physical examination, a grade II/VI systolic murmur was heard at the cardiac apex. The resting electrocardiogram (ECG) (Figure 1) showed normal sinus rhythm with right bundle branch block. Echocardiogram (Figure 2) revealed a left posterior ventricular aneurysm and moderate mitral regurgitation. The left ventricular ejection fraction (LVEF) was 37% in M-mode. Dipyridamole stress-redistribution Thallium-201 myocardial perfusion image scan showed irreversible defects in the inferolateral wall and the apex (Figure 3). Cardiac catheterization was performed on November 6, 2000, and the coronary artery angiogram was normal (Figure 3). Left ventriculogram (Figure 4) showed a posterior wall aneurysm with systolic bulging and moderate mitral regurgitation. The LVEF was 45%. Laboratory examination including biochemical and serological tests showed no evidence of myocarditis or myocardial infarction. The

Received: July 29, 2004 Accepted: November 17, 2004

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24-hour Holter ECG showed 478 beats of ventricular premature contractions. After excluding other possible causes of LVA, undiagnosed previous myocardial infarction or coronary spasm were considered in this case. Surgical treatment was suggested but the patient refused. He then was discharged. During follow-up, radionuclide cardiac function test showed biventricular heart failure (2000/10/18 LVEF 45% RVEF 28%, 2004/07/06 LVEF 31% RVEF 30%). He remained well under medical control for a follow-up period of 40 months.

DISCUSSION

LVAs mainly occur after myocardial infarction, especially at the anterior or apical segment belonging to the territory of the left anterior descending artery.² The incidence of LVA was about 5% to 10% of all patients with acute myocardial infarction.² LVAs occurring in the absence of coronary artery disease are rare. According to Toda et al, its incidence was 0.47% (11 of 2348 cases).⁷ The etiologies include hypertrophic cardiomyopathy, myocarditis, arrhythmogenic right ventricular dysplasia, Chagas's disease, glycogen storage disease, and sarcoidosis.⁷ In addition, congenital LVA and dilated cardiomyopathy also have been reported.⁴⁻⁶

There are no pathognomonic signs in LVA, but suspicion should be raised by the occurrence of congestive

heart failure, angina pectoris, ventricular arrhythmia, embolism, or endocarditis in a patient with prior myocardial infarction, especially when accompanied by a diffuse, displaced, double, or heaving point of maximal impulse, ventricular gallop, or apical systolic murmur.⁸ The electrocardiogram is generally considered insensitive and nonspecific but may be helpful when it reveals old MI or persistent ST segment elevation. Chest radiography is helpful only when an enlarged LV is associated with a bulge or calcification, and little is gained by the addition of fluoroscopy.⁸ The history, physical examination, ECG, and x-ray are helpful in only 75% of patients

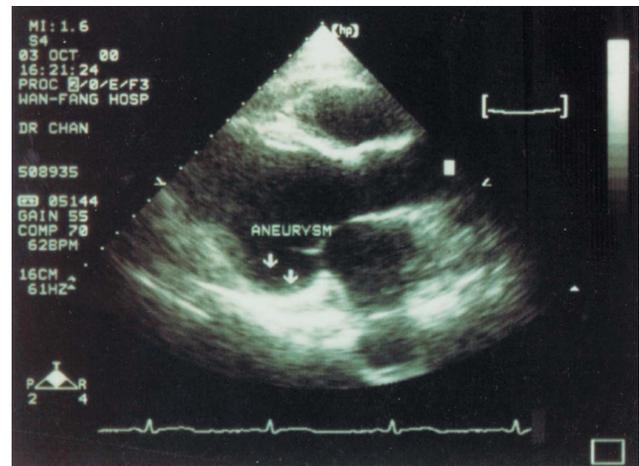


Figure 2. The echocardiography showed a posterior wall ventricular aneurysm (arrow).

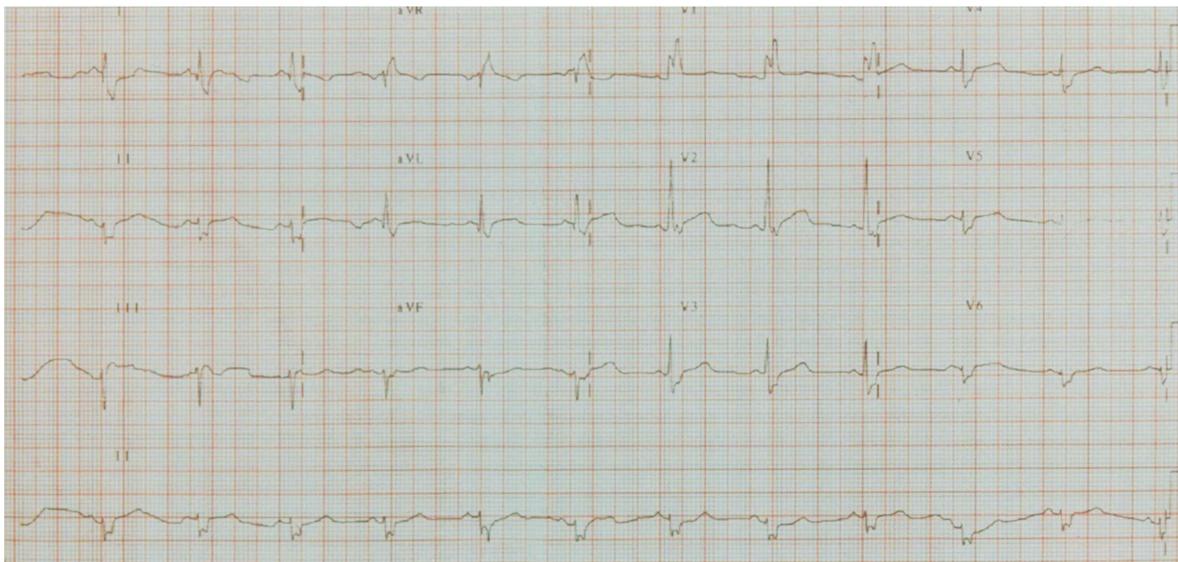


Figure 1. The resting ECG showed normal sinus rhythm and complete right bundle branch block.

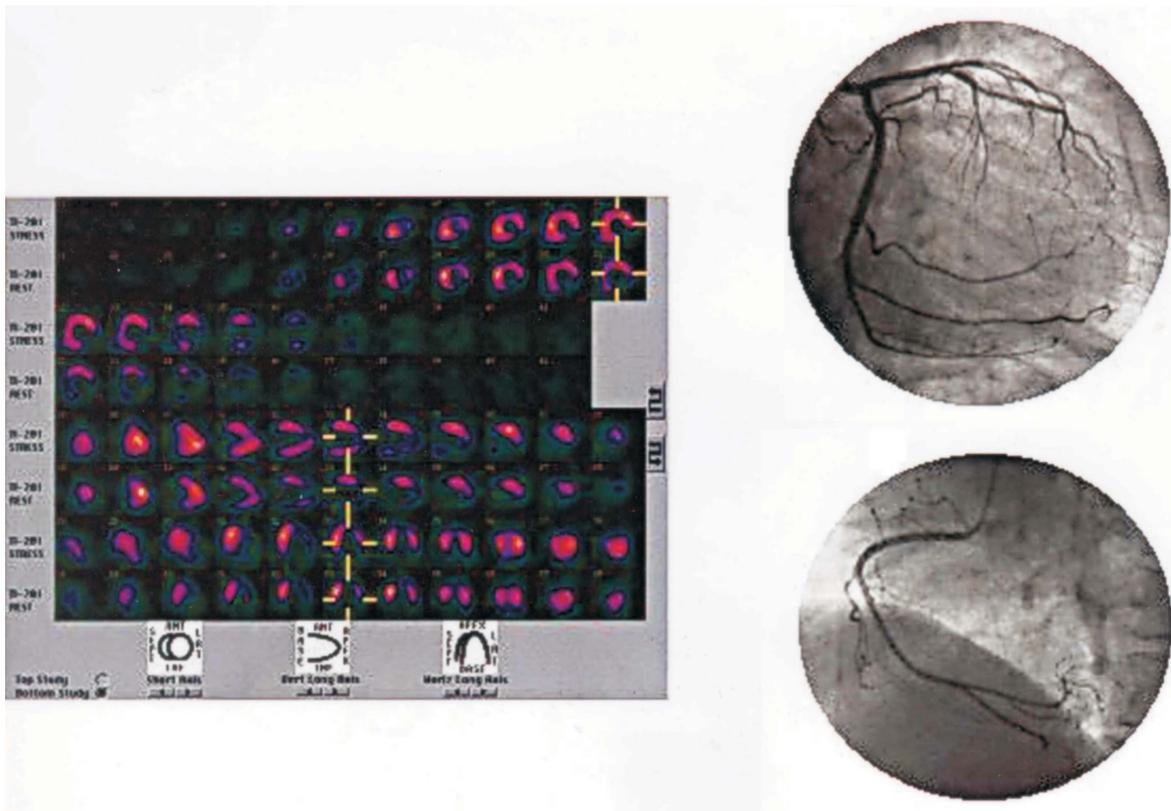


Figure 3. Dipyridamole thallium scan showed irreversible change in the inferolateral wall. The coronary angiogram showed normal coronary arteries.

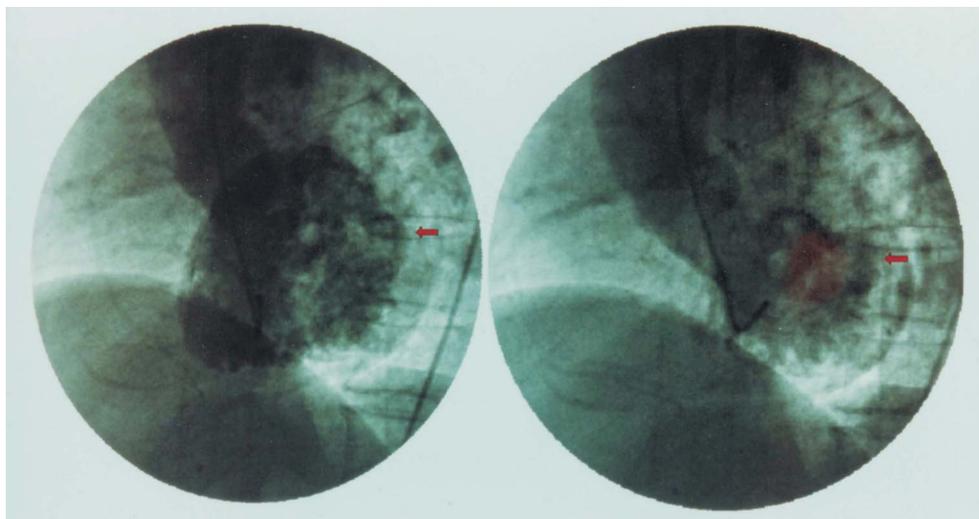


Figure 4. The left ventriculogram showed a posterior wall ventricular aneurysm (arrow).

with LVA.⁸ Two-dimensional echocardiography provides 73% to 93% sensitivity and 84% to 100% specificity in the diagnosis of LVA, and it allows assessment of LVA size and residual LV function. The criteria for diagnosis of LVA is the presence of an LV bulge

with dyskinesia.⁹ Cineangiography with catheterization remains the gold standard for diagnosis of LVA and also provides information on hemodynamics and coronary artery anatomy. The typical finding is discrete dyskinesia in an area of inadequate coronary perfusion.⁸

Complications of LVA are more common with large aneurysms, including ventricular arrhythmias, congestive heart failure, angina pectoris, and thromboembolism. Spontaneous rupture of LVA is extremely rare; such cases have been reported in patients with coronary artery disease experiencing re-infarction at the border of the LVA.¹⁰ Arrhythmias and congestive heart failure were the major causes of death.

The prognosis of LVA is worsened when the size is large, and presence of systolic dysfunction of the remaining myocardium as well as the presence of ventricular arrhythmias. However, patients with asymptomatic LVA are at low risk.¹¹

The indication for surgery for LVAs is only symptomatic LVAs. Symptoms, in order of frequency, are angina, congestive heart failure and ventricular tachycardia.¹² The first successful excision of LVA was reported in 1955.¹³ The goals of LVA resection are: (1) the eradication of the non-contractible portion of the LV as completely as possible without injury to adjacent myocardium, so as to reduce preload and afterload and maximize contractility; (2) the normalization of perfusion of all jeopardized segments; (3) the ablation of foci of ventricular arrhythmia origin and (4) the removal of mural thrombus so as to prevent embolization. These principles can result in reduction of angina pectoris, myocardial infarction, congestive heart failure, and embolism, with improvement of quality of life and longevity.⁸ LVA plication, used in 5% to 39% of patients, has been recommended in patients with angina pectoris but no congestive heart failure or ventricular arrhythmia and small LVA with minimal or no septal involvement.¹⁴

Our patient has an LVA with normal coronary artery. From the findings of stress thallium scan and echocardiography an undetected inferoposterior wall myocardial infarct may be the cause. The clinical implication for our patient is that when an LVA occurs with normal coronary artery, stress thallium scan is helpful for supporting the diagnosis of previous myocardial in-

farction caused by thromboembolism.

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正常冠狀動脈之左心室瘤—病例報告及文獻回顧

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大多數左心室瘤都是發生在冠狀動脈疾病因動脈粥狀硬化堵塞引發之心肌梗塞後的併發症。然而偶而也有左心室瘤發生在正常冠狀動脈之狀況，例如：擴張性心肌病變、肥厚性心肌病變、Chagas's disease、類肉瘤病、胸部外傷和先天性異常疾病；但一般並不常見。在此我們報告一位 53 歲男性其心臟超音波檢查無意發現有後壁左心室瘤合併左心室收縮功能衰竭且其壓力性鉍-201 核子醫學檢查呈現下側壁及心尖部位有不可逆的血液灌流缺陷，而冠狀動脈血管攝影檢查結果是正常冠狀動脈。這位病人目前已在我們內科門診追蹤了 40 個月，情況良好。由血栓造成之下後壁心肌梗塞而後自行再灌流可能是其病因。

關鍵詞：左心室瘤、冠狀動脈疾病、正常冠狀動脈、收縮性心臟衰竭。