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Multiple Left Ventricular Aneurysms in Silent Coronary Artery Disease — A Case Report

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Abstract

Myocardial infarction leading to left ventricular aneurysm was found in less than 5% of patients. Usually, aneurysm is located at anterior wall, and is rare in other segment. Heart failure could be the major clinical manifestation in a patient with aneurysm, and surgical approach may provide clinical benefit. Here, we report a case with coexistent multiple left ventricular aneurysms without previous chest pain presenting with progressive dyspnea at rest. Reduction myoplasty was successfully performed with bypass graft of coronary arteries leading to subsequent functional improvement. (J Intern Med Taiwan 2006; 17: 244-248)

Key Words ： Left ventricular aneurysm, Heart failure, Reduction myoplasty

Case Report

A 61 year-old, male who visited our emergency depart denied any systemic diseases before. He denied chest discomfort and angina sensation during exercise. Progressive dyspnea on exertion since about 2 months ago with decreased urine output was noted with recent aggravation. Orthopnea developed one week ago, and paroxysmal nocturnal dyspnea with resting dyspnea was also noted at the same time. At emergency department, chest radiology revealed right side pleural effusion with pulmonary congestion. Electrocardiography revealed pathological Q wave over leads II, III, AVF and V5, V6. Cardiac en-

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zymes were within normal limits, and other blood chemistry data was unremarkable. Diuretics were prescribed and he was admitted for further evaluation.

Echocardiography revealed enlargement of four cardiac chambers. Left ventricular ejection fraction was 30%, and a moderate mitral regurgitation was observed. The myocardial thinning and dyskinetic motion at left ventricular apical, apical-inferior and basal-inferior segments were noted (Figure 1). A mild degree pulmonary hypertension with right ventricular systolic pressure of 42mmHg was measured using tricuspid regurgitation Doppler flow velocity calculation by Bernoulli equation. Cardiac catheterization was performed later, which showed a 70% steno-

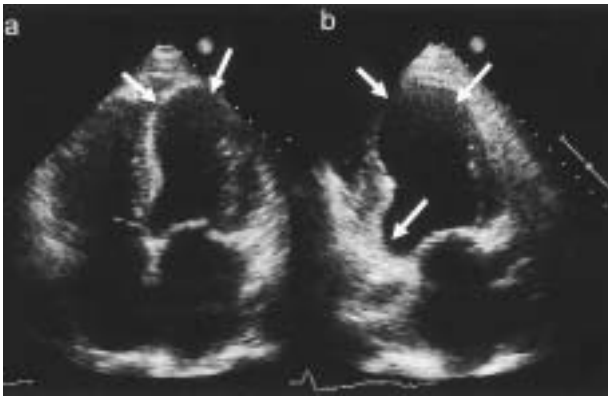

Fig.1. Echocardiography revealed multiple pouches with wall thinning character during systolic phase from apical four chambers view (panel a, white arrow), and from two chamber view (panel b, white arrow). Apical aneurysm was observed in panel a, and panel b revealed baso-inferior aneurysm (lower arrow) and another infero-apical aneurysm (upper arrow).

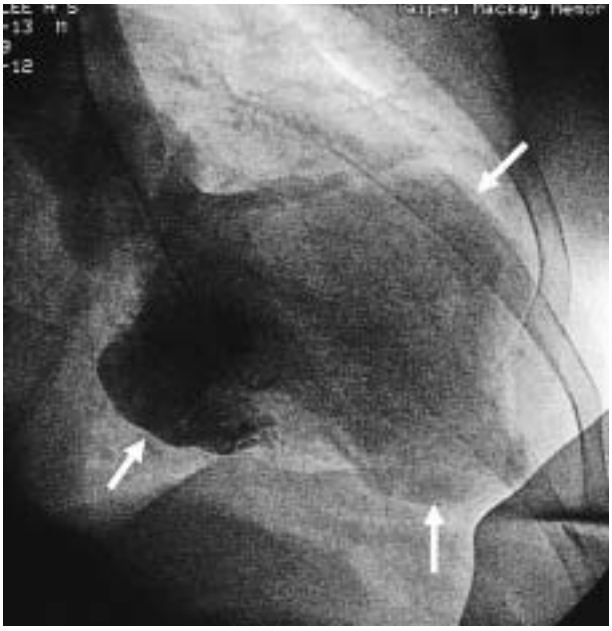

Fig.2. Left ventriculography during catheterization demonstrated multiple aneurysms formation distributed at left ventricular antero-lateral (white arrow, right-upper location), apical-inferior (white arrow, right-lower location) and baso-inferior segments (white arrow, left-lower location).

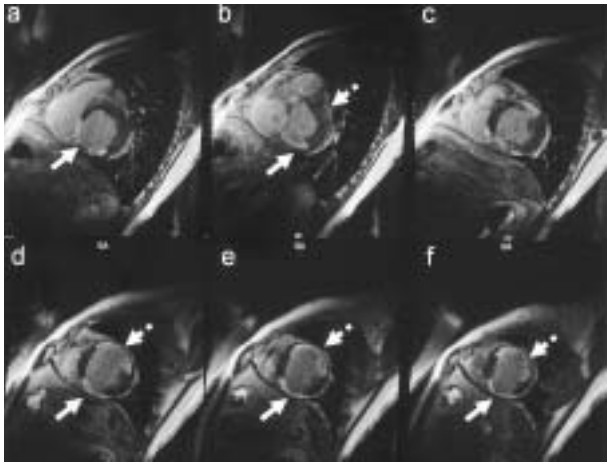

Fig.3. MRI images cutting from left ventricular basal area (upper panel) to apical area (lower panel) in a short-axis plane. Panel a to c revealed baso-inferior aneurysms formation (lower solid white arrow) with delayed enhancement, indicating infarction zone with pouch-out configure. Panel d to f revealed antero-lateral (upper dotted white arrow) and apical-inferior (lower solid white arrow) aneurysms with the same characteristics.

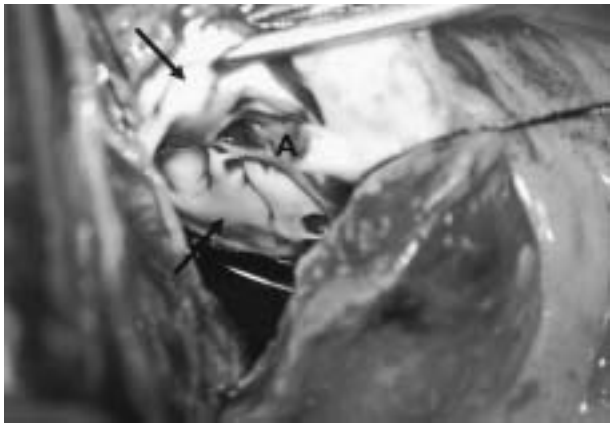

Fig.4. Scarred, fibrotic tissue indicated true aneurismal area. Upper dark arrow corresponds to antero-lateral aneurysm, and lower dark arrow corresponds to apical-inferior one. (A: viable, non-aneurysmal area located at the apex of left ventricle)

sis in middle left anterior descending artery (LAD) and a 90% stenosis at proximal circumflex artery (LcX). Right coronary artery (RCA) revealed a total occlusion at proximal to middle portion. Collateral vessels supplying RCA from left side coronary arteries were also demonstrated. Left ventriculography revealed multiple aneurysm-like dyskinetic wall motions at antero-lateral, apical and basal-inferior segments (Figure 2). Magnetic resonance imaging (MRI) was then performed for further surgical planning and assessment (1.5T MRI scanner, ECG-triggering Dark-blood sequence-Double IR, Bright blood sequence-Cine image). And a pouch-like thinning myocardial wall with delayed enhancement was noted at mid anterior, anterolateral and apical wall of the left ventricle (Figure 3). Other delayed and mild thinning myocardial wall at basal infero-septal, inferior wall, mid inferior wall of LV and antero-inferior wall of RV were also noted. Multiple true aneurysms formation with scarred and infarcted character was diagnosed for this patient.

LV aneurysmectomy (anterolateral aneurysm) and coronary artery bypass graft was successfully performed. Whitish, thinning scar tissue distributed at the multiple aneurysms area mentioned above was observed (Figure 4), which had histopathological prove of a fibrous tissue mixed with some viable myocardium after resection. This patient was discharged three weeks later and remained uneventful with a functional class two heart failure in the two months outpatient clinic follow-up.

Discussion

True left ventricular (LV) aneurysm formation develops in less than 5% of all patients with ST elevation myocardial infarction and somewhat more frequently in transmural infarct, especially in anterior wall[1]. It occurs when intraventricular tension stretches the non-contracting infarcted myocardium, thin layer of necrotic muscle, and even totally fibrous tissue. The thinning myocardial wall may be occasionally mixed with viable myocardium[1]. With the passage of time, the wall of aneurysm becomes more densely fibrotic and interferes with ventricular performance through paradoxical expansion and ineffective left ventricular contraction during each systole[2]. Most aneurysms are located anterolaterally, or near the LV apex and are often associated with total occlusion of LAD with poor collateral supply. Only 5-10% of aneurysms located posteriorly, and even are more rare in inferior wall[3]. The ventricular aneurysms needs for aneurysmectomy have declinded dramatically during the past 5 years due to the new era of early reperfusion therapy in acute myocardial infarction[3]. True LV aneurysms rarely rupture early or late after its development, and late rupture almost never occur[3]. Review of previous literature[1-4], in addition to rare location, the multiple, and rarely developed location coexisted in one case, which had never been reported before.

The exact diagnosis of LV aneurysms is best made non-invasively by two-dimension echocardiography (2DE) study. 2DE is also helpful in distinguishing between a true and false aneurysm based on the demonstration of a narrow neck in relation to cavity size[4]. Left ventriculography may also provide another potential tool, however, it is an invasive method in detecting an abnormal bulge or dyskinetic wall motion in the LV contour during systole. Other clues include the presence of a persistent ST segment elevation on resting electrocardiography[5] and a characteristic bulge and calcified LV silhouette on chest roentgenogram. These findings, however, are of low specific and with limited sensitivity. We hypothesized that multiple LV aneurysms just like this case might have reciprocal effect which made no definite ST segments changes on electrocardiography, and this issue had never been discussed before. Recently, MRI has emerged as a preferred non-invasive technique for pre-operative assessment of LV shape, the degree of aneurysm thinning, and its resectability[2]. The present reported case demonstrated obviously infarcted and

scar formation with delayed enhancement in multiple aneurysmal area on MRI (Figure 3), which were compatible with surgical findings (Figure 4).

The clinical features of LV aneurysm include symptoms of heart failure (with or without angina) in 50% of patients, severe angina alone was observed in one-third of the patients, and symptomatic ventricular arrhythmias in approximately 15%[6]. The associated ventricular arrhythmia may even be refractory and life threatening[6,7]. There was no associated arrhythmia in the present case, and no angina by history taking, which made an initial correct diagnosis difficult. Mural thrombi usually can be found in almost half patients with chronic LV aneurysm, and can be detected by 2DE or by angiography[8]. Systemic embolic events in patients with thrombi and LV aneurysm were extremely uncommon at 1 month after an acute myocardial infarction[8].

Improvement of complicated true LV aneurysm can be carried out by aneurysmectomy, if there were no complicated condition, such as heart failure, angina, embolization, or life-threatening arrhythmias[2]. Coronary revascularization is frequently performed along with aneurysmectomy in patients with multivessels disease accompaning heart failure. Improvement of left ventricular function has been reported in survivors after resection of LV aneurysm[9] by removing the abnormal mechanical burden leading to a poor global LV systolic function. The nonischemic myocardium remote to the resected ischemic area also had an improvement on diastolic relaxation through a cardiovascular neuroregulatory mechanisms[9]. Standard surgical modalities in patients with multiple aneurysms have never been discussed[10]. It has been reported that symptoms of angina with heart failure would benefit more from surgical treatment as in the present reported case, the refractory heart failure also improved after surgery.

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多發性左心室瘤在無胸痛之冠心病患的臨床表現—病例報告

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摘 要

左心室瘤的產生被發現在小於5% 的心肌梗塞後的病人族群。通常左心室瘤皆位於心室的前壁，至於在其他部位則相當罕見。心衰竭可能是一個左心室瘤病患的臨床主要症狀，在此類病患族群手術能提供臨床上之改善。我們在本篇文章報告一個案例，此案例同時存在不同部位多發之左心室瘤，且無過去胸痛之病史。臨床上以漸進式的氣促及喘爲表現，心肌手術及繞道手術被成功地實施且病患在長期的追蹤裡，功能上持續的進步。