Long-term Survival of A Patient with Asymptomatic Left Ventricular Pseudoaneurysm after Acute Myocardial Infarction

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Abstract

An 82 years old man developed a left ventricular pseudoaneurysm after acute myocardial infarction when he was 72 years old. Coronary angiography showed left main and triple-vessel coronary artery disease. On left ventriculography, a tubular-like pseudoaneurysm was demonstrated that originated from the basal inferoposterior wall of the left ventricle. He underwent coronary artery bypass surgery with no plication of the pseudoaneurysm because the surrounding tissues of pseudoaneurysm were all necrotic. The most recent follow-up transthoracic echocardiography revealed a hypokinetic basal inferior wall, impaired LV contraction with an ejection fraction of 44%, and an inferoposterior wall pseudoaneurysm. The patient was doing well more than 10 years after the myocardial infarction. The prognosis might be determined by the organized thrombi, aggressive pharmacologic treatment, and coronary artery bypass surgery. Although our patient has survived for more than 10 years with a nonsurgically treated post-infarction LV pseudoaneurysm, we could not provide an evidence to support that conservative therapy is enough for every patient with a post-infarction pseudoaneurysm. (J Intern Med Taiwan 2012; 23: 442-448)

Key Words: LV pseudoaneurysms, LV diverticulum, STEMI, CABG, Echo

Introduction

Left ventricular (LV) pseuodaneurysms develop when rupture of the free wall of the left ventricle is contained by pericardial adhesions or scar tissue[1]. Surgical resection of pseudoaneurysms is usually recommended because of the risk of spontaneous rupture[2]. However, few reports have been published on survival of patients with nonsurgically treated LV pseudoaneurysms that developed as a result of acute myocardial infarction[3-6]. Herein, we present a patient who has survived for more than 10 years with a nonsurgically treated LV pseudoaneurysm that occurred after acute myocardial infarction.

Case report

An 82-year-old man with a history of cigarette smoking, hypertension, and dyslipidemia developed an acute inferior myocardial infarction (Fig. 1 A &

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B) with no evidence of right ventricular infarction in February of 2002. The initial management was intravenous thrombolytic therapy. During the episode of acute myocardial infarction, the peak level of creatine kinase (6183 IU/L)-MB subform (12.2%) was 754 IU/L and the LV ejection fraction was 49% as measured by left ventriculography. A tubular-like pseudoaneurysm originating from the basal inferoposterior wall of the left ventricle was noted (Fig. 2 A & B). The patient underwent coronary artery bypass surgery for his left main and triple-vessel coronary artery disease in March of 2002. Plication of the pseudoaneurysm was not performed by cardiac surgeon because the surrounding tissues of pseudoaneurysm were all necrotic. No mechanical or electrical complications were noted during hospitalization. The patient made a good recovery and was discharged uneventfully. He has been managed with atorvaststin, aspirin, isosorbide-5-mononitrate, carvedilol, amlodipine, and enalapril since 2002.

The patient underwent follow-up transthoracic echocardiography in 2003, 2005, and 2011. The size of the pseudoaneurysm in 2003 (Fig. 2 C) was similar to that in 2005 (Fig. 2 D). On parasternal short-axis view at the papillary muscle level, an acute angle between the contour of the aneurysm and the left ventricle was noted, suggesting an inferoposterior pseudoaneurysm. The most recent follow-up transthoracic echocardiography in 2011

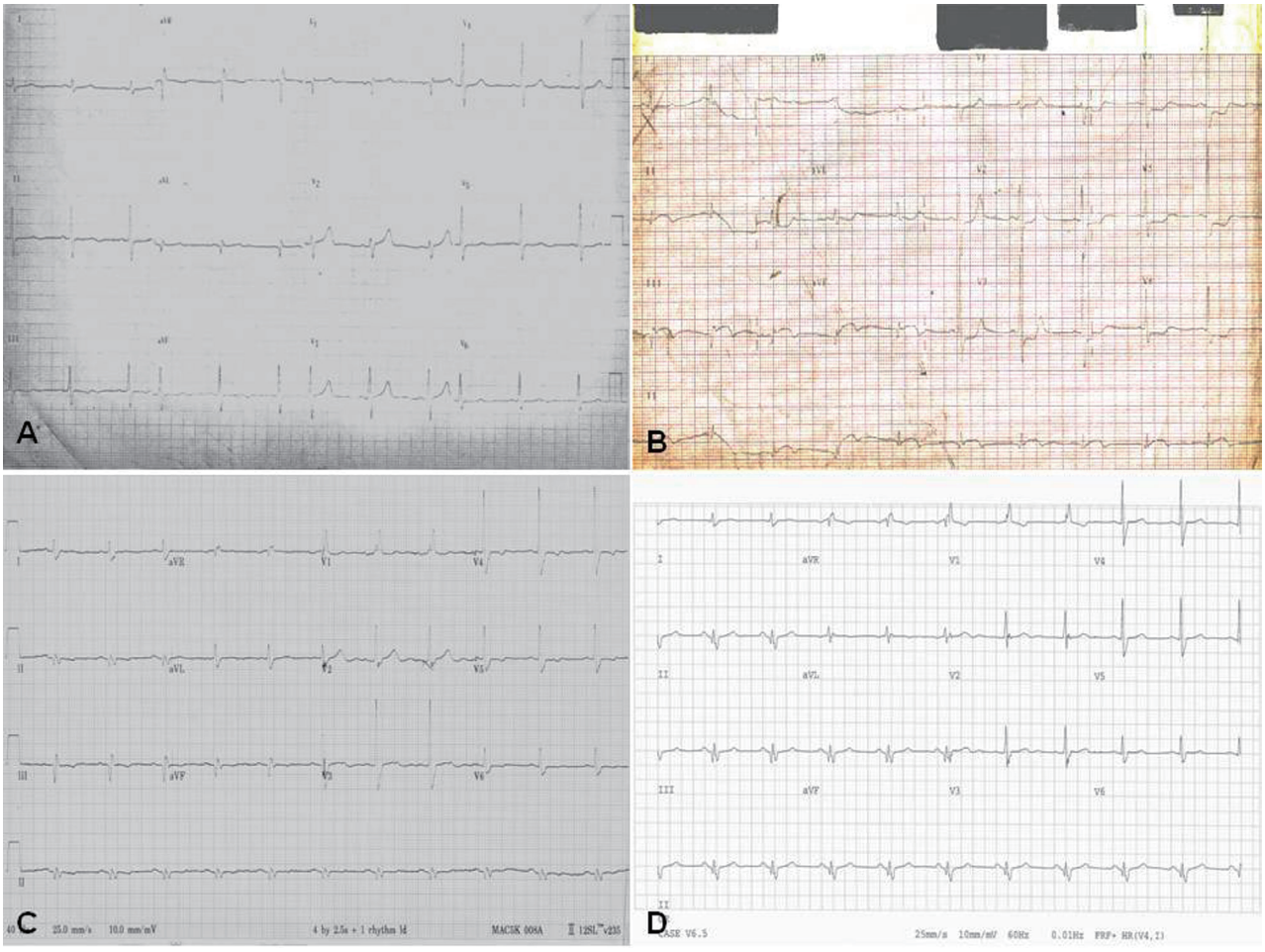

Figure 1. A series of changes of 12-lead electrocardiograms in our patient. Before (A) and after (B) acute inferior myocardial infarction electrocardiograms show a new Q-wave development (B) in inferior leads. Follow-up electrocardiograms in 2007 (C) and 2011 (D) show serial changes (Q-wave and T-wave) after inferior myocardial infarction.

revealed a hypokinetic basal inferior wall, impaired LV contraction with an ejection fraction of 44%, and an inferoposterior wall pseudoaneurysm (Fig. 3 A and B). Blood flow from the pseudoaneurysm to the LV cavity was noted (Fig. 3 C). On apical 2-chamber view, a saccular contour of the pseudoaneurysm with a narrow neck connecting it to the left ventricle was demonstrated (Fig. 3 D). There was no pericardial effusion. The follow-up 12-lead electrocardiograms in 2007 (Fig. 1 C) and 2011 (Fig. 1 D) showed serial electrocardiographic changes of the present patient. Until June of 2012, the patient was doing well more than 10 years after the myocardial infarction without any evidence of angina pectoris or heart failure.

Discussion

The development of LV pseudoaneurysms is a rare but potentially lethal complication of acute myocardial infarction, cardiac surgery, trauma, and infections. Most investigators consider surgery to be appropriate treatment for LV pseudoaneurysms[7] because of the risk of rupture when the condition is left untreated[8,9]. Up to now, post-infarction LV pseudoaneurysm is regarded as a surgical disease as recommended by ST-elevation myocardial infarction guideline[10]. However, some studies found that death was not attributable to cardiac rupture

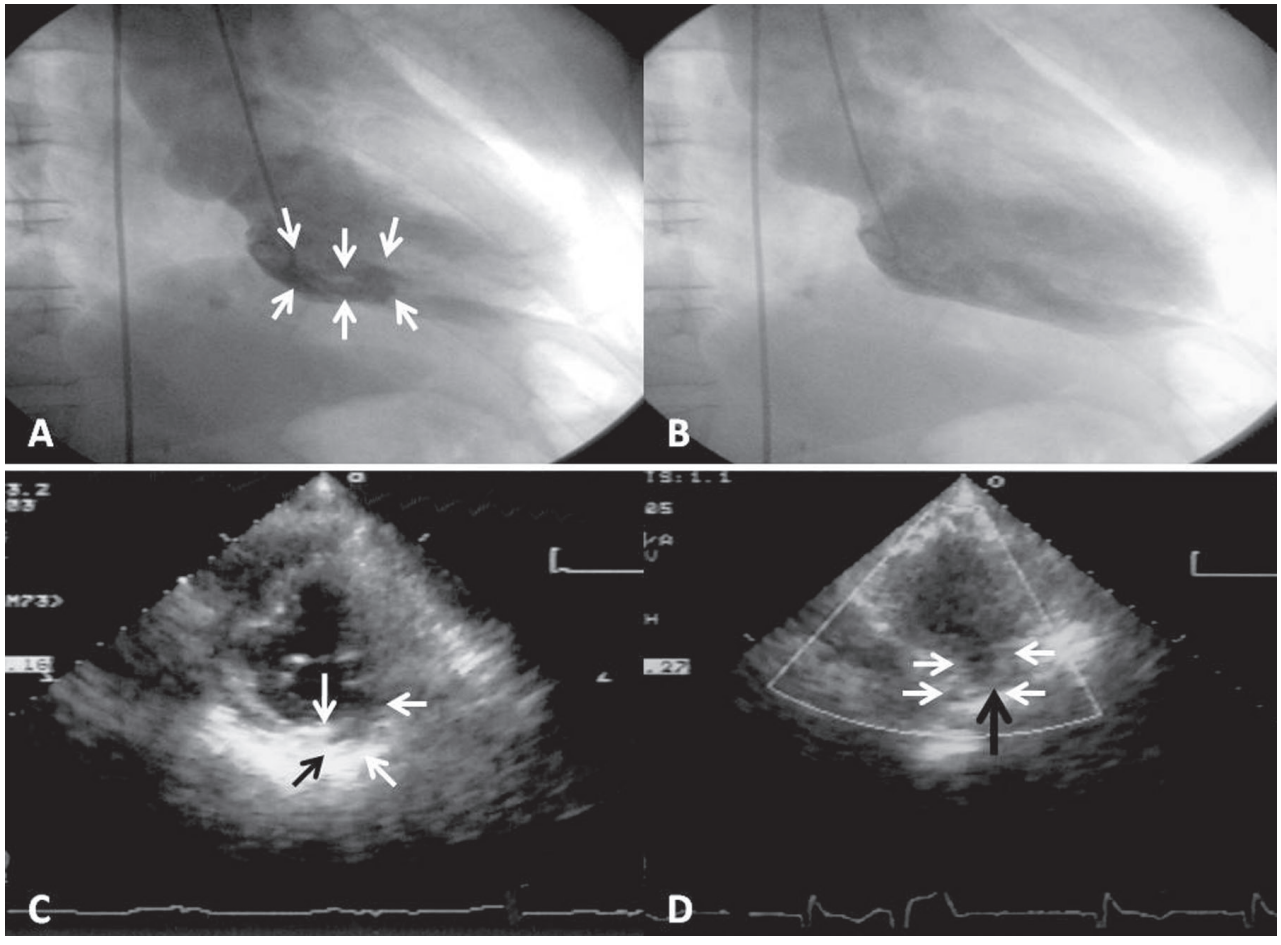

Figure 2. Left ventriculograms (upper panels) and the follow-up transthoracic echocardiograms (lower panels) of our patient, who developed a basal inferoposterior pseudoaneurysm after inferior myocardial infarction. Systolic (A) and diastolic (B) frames of the contrast left ventriculograms show a tubular pseudoaneurysm (arrows) in the basal inferoposterior wall. The follow-up echocardiograms (short-axis views at the papillary muscle level) in 2003 (C) and 2005 (D) reveal little change in the size of the inferoposterior pseudoaneurysm (arrows). Organized thrombi can be seen in panel D (black arrow).

in patients with pseudoaneurysms[4,6,11]. Yeo et al.[11] found that the all-cause mortality rates among patients with cardiac pseudoaneurysms were 31% (13 of 42 patients) in patients who received surgical treatment and 60% (6 of 10 patients) in patients who received medical treatment during a median follow-up period of 4 years. Sakai et al.[12] did not recommend surgery if the pseudoaneurysm was connected to the LV wall with a narrow neck, or if it occurred in the presence of the postsurgical mitral valve. In addition, thrombus is frequently found in the pseudoaneurysm. Recently, some authors found that percutaneous closure of LV pseudoaneurysms is a feasible alternative for high-risk surgical candidates[13]. However, further long-term follow-up studies are needed. The long-term survival of our patient might be related to the formation of organized thrombi within the pseudoaneurysm, which resulted in decreased LV pressure to its distal walls. Furthermore, aggressive pharmacologic treatment and coronary artery bypass surgery to decrease LV remodeling and preserve LV systolic performance might have played an important role in the long-term survival of this patient. The decline in the incidence of and mortality rate associated with cardiac rupture after acute myocardial

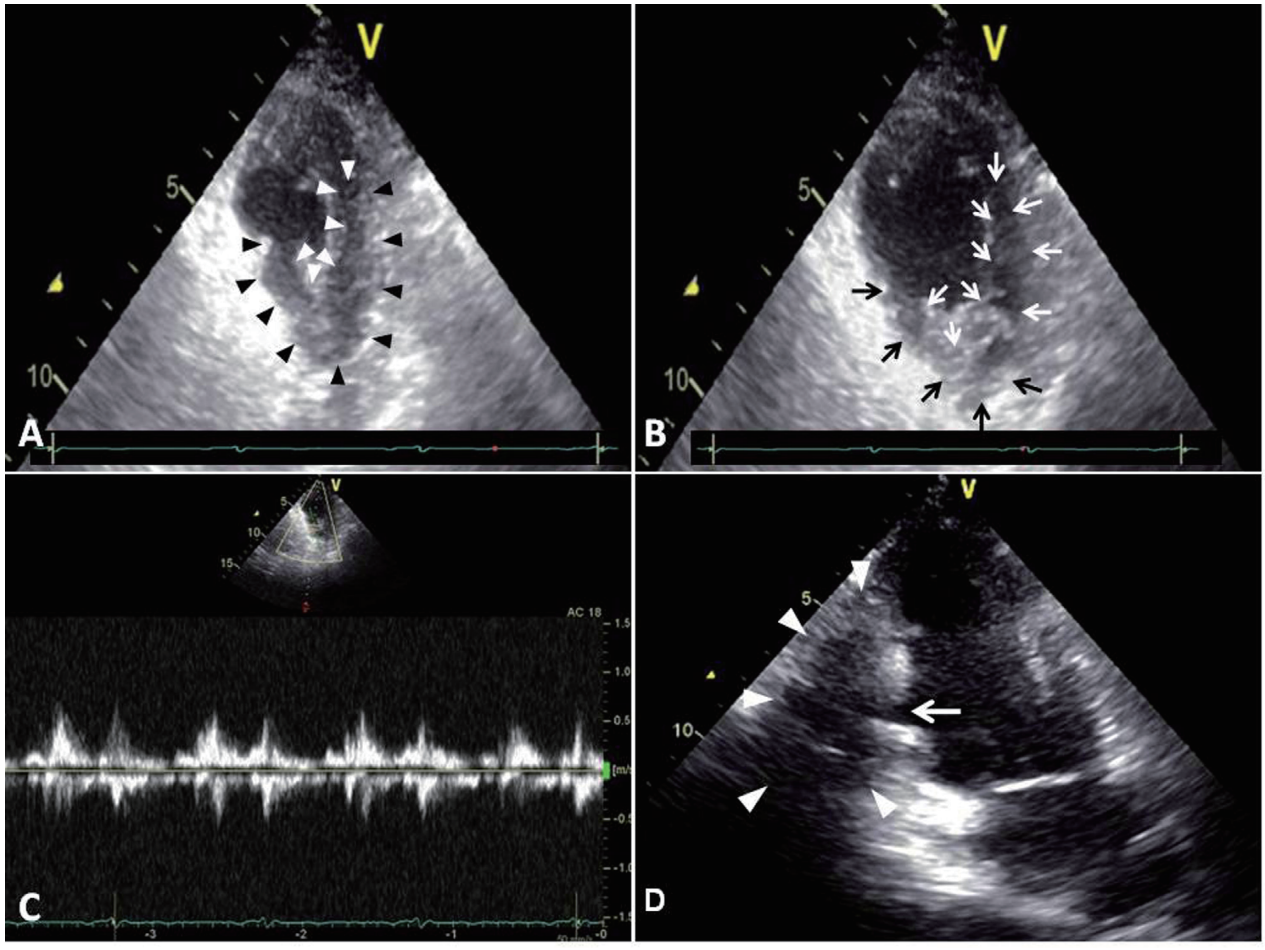

Figure 3. The most recent follow-up transthoracic echocardiograms. The parasternal short-axis views at the end-systolic phase (A) and end-diastolic phase (B) show an tubular-like echo-free space posterior to the inferoposterior wall (pseudoaneurysm, white arrows) without contractility (black and white arrowheads in A) and with organized thrombi (black arrows in B). C. Doppler flow demonstrates blood flow entering the pseudoaneurysm. D. Apical 2-chamber view shows a saccular contour (white arrowheads) of the pseudoaneurysm with a narrow neck (arrow) connecting it to the left ventricle. LA = left atrium; LV = left ventricle.

infarction over the last 30 years is associated with the increased use of reperfusion strategies and adjunct medical therapy[14]. The development of LV pseudoaneurysm is, therefore, rare[15].

Recognition of LV pseudoaneurysm after acute myocardial infarction is still important because the presentation of LV pseudoaneurysm varies. Pseudoaneurysm is most commonly detected using two-dimensional echocardiography. A narrow neck leading to the fundus is usually found. In contrast, a wide opening to the ventricular chamber is characteristic of true aneurysm. In addition, an acute angle between the contour of the aneurysm and the left ventricle, an abrupt transition from normal thickness of the myocardium to a thin-walled sac, a distinct discontinuity in the ventricular wall, and color Doppler studies demonstrating movement of blood from the suspected pseudoaneurysm to the LV cavity can be helpful in diagnosing pseudoaneurysms[4]. Unlike two-dimensional echocardiography, three-dimensional echocardiography provides good images of the LV posterior wall and is, therefore, an excellent tool for monitoring unruptured pseudoaneurysms. In contrast to LV pseudoaneurysm, LV diverticula are rare congenital anomalies consisting of protrusion of endocardium and myocardium from the LV free wall[16]. Histopathologically, they are classified into fibrous or muscular. The former is non-contractile as it is constituted mostly from fibrous tissue. It most commonly originates from the apical or subvalvular positions. The muscular type includes all cardiac layers and usually emerges from the apex; therefore, it has a mechanical activity. This type is often associated with other congenital defects. Few case reports showed that diverticulae are not congenital but associated with hypertrophic cardiomyopathy[17,18]. Differentiation between these 2 entities is also important because LV diverticulum is often associated with serious complications, such as systemic thromboemblism, cardiac rupture, arrhythmia, and sudden death[19,20]. Transthoracic echocardiography can show the origin, the neck (narrow or wide), the walls, and the contractility of the diverticula[21,22]. The multidetector computed tomography enables accurate evaluation of both LV myocardium and lumen[23]. Therefore, the multidetector computed tomography can establish the diagnosis of a LV pseudoaneurysm or diverticulum, which is the limitation of our case report. In our case, the outpouching echo-free space had no contractility and we believe that it was a case of pseudoaneurysm.

In conclusion, we present a patient who has survived for more than 10 years with a nonsurgically treated LV pseudoaneurysm that occurred after acute myocardial infarction. From this limited experience, however, we could not provide an evidence to support that conservative therapy is enough for every patient with a post-infarction pseudoaneurysm.

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急性心肌梗塞併發無症狀的左心室偽瘤
—長期存活之病例報告

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摘　要

一位八十二歲男性於七十二歲時急性心肌梗塞後併發左心室僞瘤。冠狀動脈攝影顯示左主冠狀動脈和三條冠狀動脈疾病；左心室攝影發現下後壁有一條狀僞瘤。他接受冠狀動脈繞道手術但因僞瘤週邊組織皆壞死，所以無法以折疊術處理左心室僞瘤。最近的經胸心臟超音波檢查顯示左心室下後壁運動低下、左心室收縮功能不良（射血分率爲44%）、左心室下後壁有一僞瘤。此病患於心肌梗塞十年後仍存活良好。這預後可是能因爲僞瘤內的組織化血栓、積極的藥物治療和冠狀動脈繞道手術。雖然此個案以非外科治療後存活十年多，但我們無法以此個案論證以後所有急性心肌梗塞後併發左心室僞瘤病患皆可只接受保守治療。