

[CASE REPORT]

Left Ventricular Pseudo-aneurysm with Ventricular Septal Rupture Due to Anterior ST-segment Elevation Myocardial Infarction

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Abstract:

We report a case with the simultaneous occurrence of pseudo-aneurysm of the left ventricle and ventricular septal rupture, which was successfully surgically repaired. A 77-year-old woman with a history of aortic valve replacement and coronary bypass graft presented to our clinic due to chest pain. She was diagnosed with anterior ST-segment elevation myocardial infarction (STEMI) based on an electrocardiogram. Echocardiography revealed pseudo-aneurysm of the left ventricle and ventricular septal rupture. Coronary angiography revealed 99% stenosis with delayed contrast filling in the mid left anterior descending artery. Surgical repair with a bovine pericardium patch was performed, and the postoperative course was uneventful.

Key words: left ventricular pseudo-aneurysm, ventricular septal rupture, ST-segment elevation myocardial infarction

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Introduction

A left ventricular (LV) pseudo-aneurysm is usually formed by cardiac rupture contained by adherent epicardium or scar tissue. This condition has a variety of causes, including myocardial infarction, cardiac surgery, trauma, and infection. Surgical resection is considered to be the most appropriate treatment for LV pseudo-aneurysm because of the high risk of rupture. On the other hand, LV septal rupture is a fatal complication after myocardial infarction, and which requires urgent surgical treatment. We report a case of co-existing LV pseudo-aneurysm and LV septal rupture that was successfully treated by surgical repair.

Case Report

A 77-year-old woman with a history of aortic valve replacement and coronary bypass graft (CABG) (a left internal thoracic artery graft to the ramus intermedius, and a saphenous vein graft to the right posterolateral branch) presented to our clinic due to chest pain. An electrocardiogram revealed normal sinus rhythm with ST elevation in leads V1-V4 and II, III, and aVf, and ST depression in leads I and aVL (Fig. 1A). A diagnosis of anterior ST-segment elevation myocardial infarction (STEMI) was made. The physical findings on admission revealed a holosystolic apical murmur. A physical examination revealed the following findings: body temperature, 36.8°C; blood pressure, 92/60 mmHg; pulse rate, 86/min and regular; and oxygen saturation, 98% on room air. The findings of a laboratory analysis were as follows: white blood cell count, 8,600/ μ L (with a normal differential); red blood cell count, 462 \times 10⁴/ μ L; hemoglobin, 14.1 g/dL; platelet count, 14.4 \times 10⁴; creatinine kinase (CK), 698 IU/L (normal range: 43-151); CK-MB, 31 IU/L/37.1°C (0-25); BNP, 443.7 pg/mL (normal range: <18.4). A chest X-ray on admission showed mild pulmonary congestion (Fig. 1B). Echocardiography showed akinesis of the apical wall of the left ventricle with a left ventricular ejection fraction of 48%. Ventricular septal rupture

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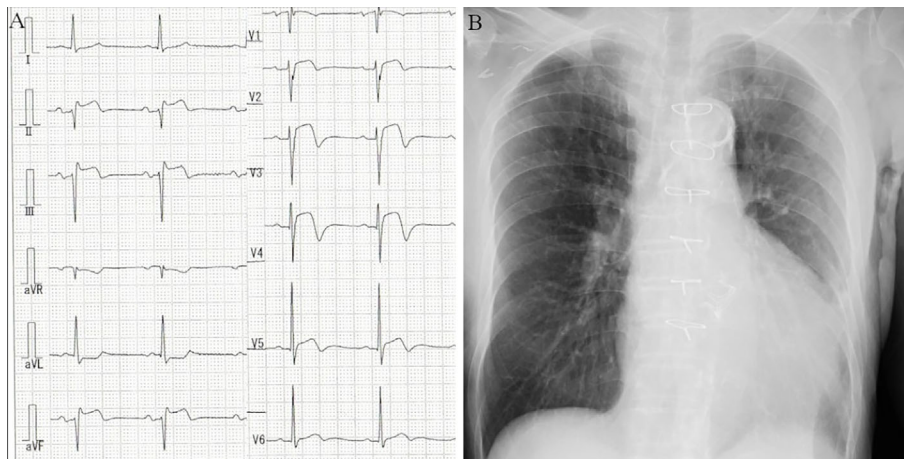


Figure 1. A: An electrocardiogram showed normal sinus rhythm with ST elevation in leads V1-4 and II, III, and aVf and ST depression in leads I and aVL. B: Chest X-ray in the supine position showed cardiomegaly and mild pulmonary congestion.

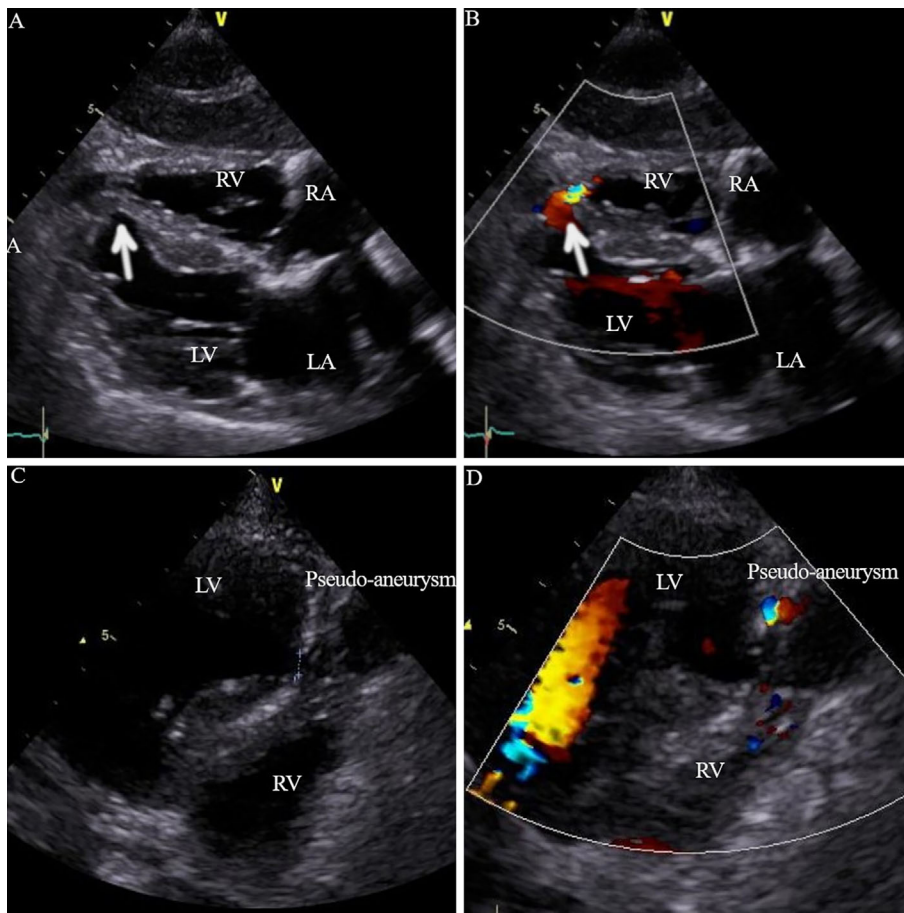


Figure 2. A and B: Echocardiography revealed ventricular septum rupture (white arrows). C and D: Echocardiography revealed pseudo-aneurysm of the LV.

(Fig. 2A and B) and pseudo-aneurysm of the left ventricle (Fig. 2C and D) were also detected. The pulmonary to systemic blood flow (Q_p/Q_s) ratio was 2.08. Computed tomography (CT) revealed LV pseudo-aneurysm (Fig. 3C and D). Coronary angiography (CAG) without left ventriculography was subsequently performed and revealed

99% stenosis with delayed contrast filling in the mid left anterior descending artery (LAD), which was considered to be the culprit lesion, as well as significant stenosis [90% of the diameter of the distal right coronary artery (RCA) and posterior descending artery (RPDA)] (Fig. 4). Based on a discussion, the cardiac team decided that revascularization of

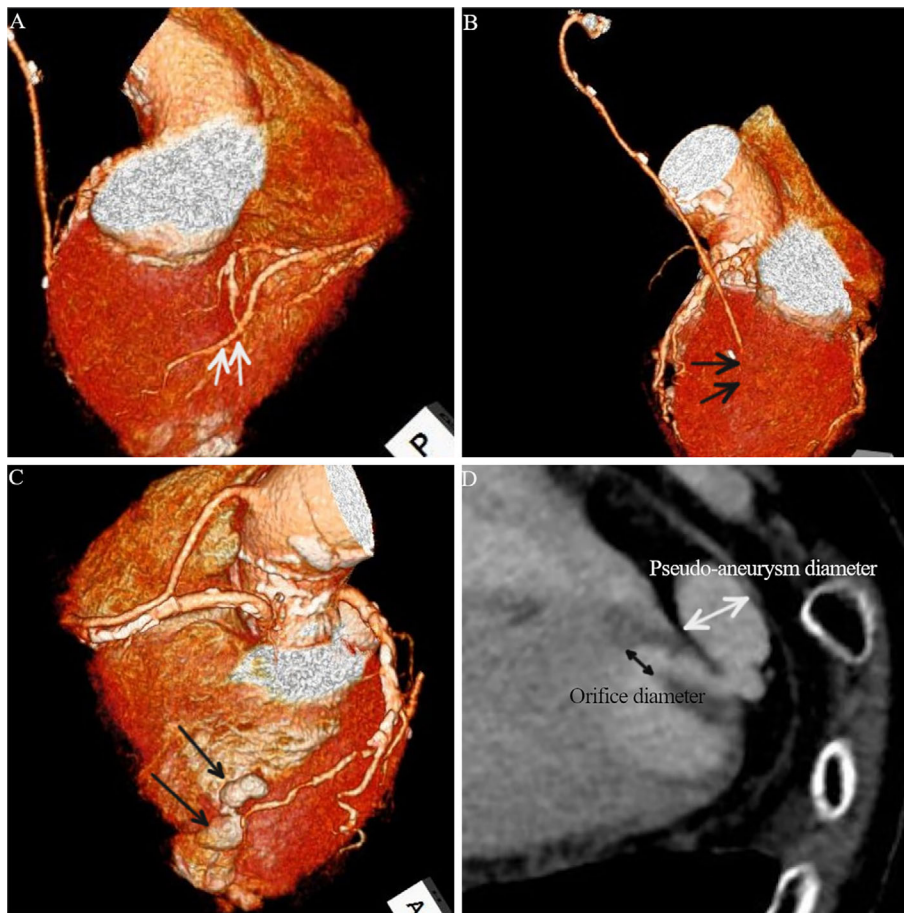


Figure 3. A and B: Coronary CT revealed the patency of a saphenous vein graft to the right posterolateral branch and a left internal thoracic artery graft to the ramus intermedius. C: Coronary CT revealed pseudo-aneurysm of the LV. D: The orifice to aneurysm diameter ratio was 0.40 (5.2 mm/13 mm).

the coronary artery was not necessary because the LV anterior wall had already infarcted and was not viable. Surgical repair was performed using a bovine pericardium patch (Fig. 5).

Discussion

LV pseudo-aneurysm is an uncommon but critical condition. It forms when a cardiac rupture is contained by adherent pericardium or scar tissue without endocardium or myocardium (1). There are various etiologies of LV pseudo-aneurysm formation, including myocardial infarction (MI), cardiac surgery, intervention, endocarditis, and chest trauma. Frances et al. reported that 55% of cases were due to MI, and of which 49% were inferior MI (1). Meng et al. reported that the most common location of LV pseudo-aneurysm formation was the posterior LV (2). Inflammatory reactions and pericardium adhesion mainly occur in the posterior wall of the LV, because patients are usually in a recumbent position after myocardial infarction (1). In the present case, cardiac oozing rupture after anterior myocardial infarction was sealed by tight adhesion of the pericardium in the apex, which resulted in pseudo-aneurysm formation. If

there was no pericardial adhesion with a history of previous cardiac surgery, cardiac tamponade and sudden cardiac death might have occurred in this case.

In the present case, concomitant CABG during surgical repair for perforation was not performed because the clinical benefit for the infarcted myocardium was not clear. Furthermore, the culprit artery was close to the left ventriculotomy and could be damaged during infarctectomy or entrapped in the closure suture line (3). Most studies have shown that revascularization has no benefit on early or late survival (3).

It is sometimes difficult to make a diagnosis of LV pseudo-aneurysm because the clinical presentation is often nonspecific. An orifice to pseudo-aneurysm diameter ratio of <0.5 on echocardiography was useful for distinguishing LV pseudo-aneurysm from LV true-aneurysm (4). In the present case, the orifice to aneurysm diameter ratio on preoperative CT was 0.40 (5.2 mm/13 mm) (Fig. 3D). Sokolskaya et al. reported the usefulness of echocardiography for the preoperative and postoperative assessment of LV pseudo-aneurysms (5). After the diagnosis of LV pseudo-aneurysm is made, treatment options should be discussed. In most of cases, surgical repair is required because of the high risk of rupture. A previous report noted that the mortality of LV

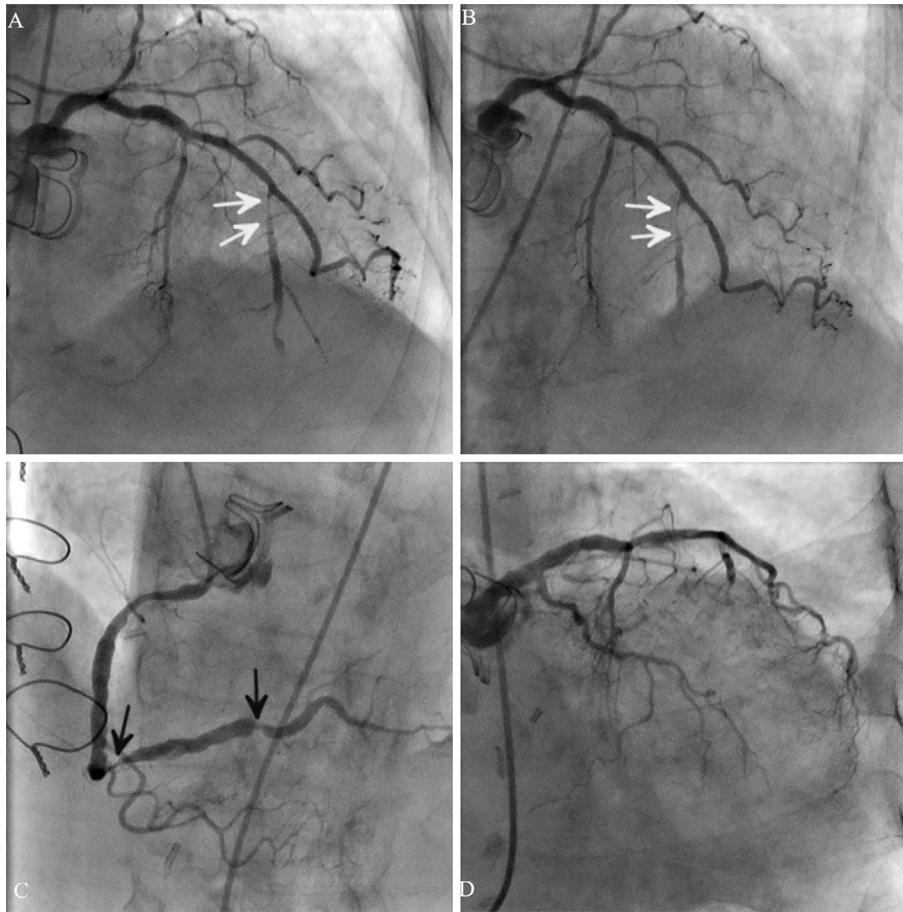


Figure 4. A and B: Ninety-nine percent stenosis was observed in the mid left anterior descending artery (white arrows). C: Significant stenosis of 90% of the diameter of the distal right coronary artery and right posterior descending artery was observed (black arrows). D: No significant stenosis was observed in the left circumflex artery.

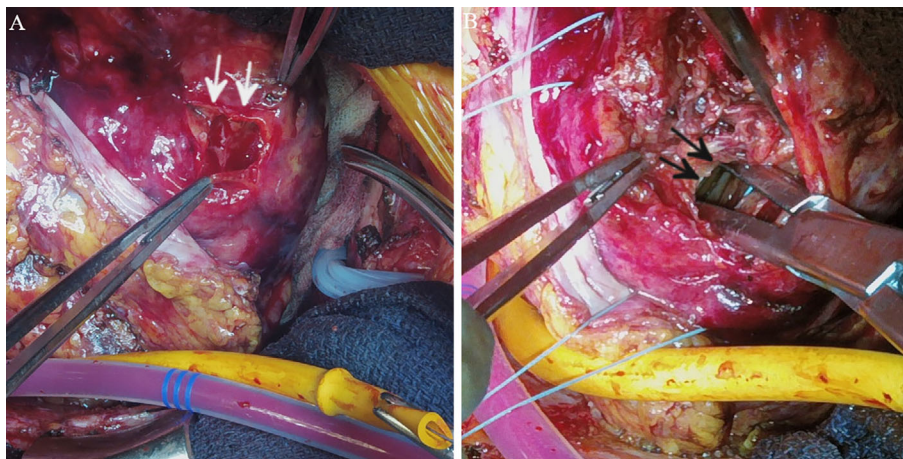


Figure 5. A: LV pseudo-aneurysm was found in the apex, which was longitudinally incised (white arrow). B: Ventricular septal perforation (10 mm) was found in the anterior infarcted septum (black arrow).

pseudo-aneurysm is considered to be higher after conservative therapy, so it should be actively treated with surgery (6). On the other hand, some retrospective studies have reported that cases involving chronic small LV pseudo-

aneurysms of <3 cm in size or patients with high surgical risk can be managed conservatively (7). However, rapidly progressive LV pseudo-aneurysm has also been reported (8), even in the case of conservative therapy, periodic follow-up

echocardiography is mandatory.

Furthermore, LV septal rupture is a fatal mechanical complication after acute myocardial infarction, which often requires urgent surgical repair because early surgical intervention offers the only realistic chance of survival (9, 10). According to a large cohort study, even in the era of increased timely primary PCI, the ventricular septal rupture-associated mortality rate remains high (11).

The simultaneous onset of LV pseudo-aneurysm and septal rupture is rarely reported. In the present case, the CAG showed very tight stenosis in the mid and distal segment of the LAD. We hypothesize that the distal LAD lesion caused pseudo-aneurysm formation and that the mid LAD lesion then caused septal perforation. Hata M et al. reported the case of reconstruction of the LV for post infarction LV aneurysm complicated by ventricular septal perforation (12). Tasaki et al. reported a case of pericardial patch repair and coronary artery bypass grafting of an LV aneurysm with ventricular septal perforation after myocardial infarction (13). Both of these conditions were fatal mechanical complications of myocardial infarction, which required urgent surgical repair in the present case. However, a case of recurrent LV pseudo-aneurysm after surgical repair has been reported (14); thus repeat follow-up echocardiography is required, even after surgical repair.

Conclusions

We herein reported a case of left ventricular pseudo-aneurysm with ventricular septal rupture due to anterior ST-segment elevation myocardial infarction. A proper diagnosis and timely surgical repair were necessary to save the patient's life.

The authors state that they have no Conflict of Interest (COI).

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