

# Surgical Resection of a Lupus-Related Left Ventricular Aneurysm in a Patient with Normal Coronary Arteries: Case Report



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## ABSTRACT

A 52-year-old woman with systemic lupus erythematosus (SLE) presented with shortness of breath. Echocardiography and cardiac catheterization demonstrated a discrete left ventricular aneurysm (LVA) with normal coronary arteries. Although her heart failure symptomatically improved with medical therapy, she suffered an embolic stroke from a thrombus within the LVA. She was treated with anticoagulation and rehabilitation for 6 weeks. Reevaluation with echocardiography demonstrated persistent depressed LV function and mural thrombus within the LVA. Surgical resection of the LVA was performed with evacuation of the thrombus and local repair of the LV.

## INTRODUCTION

Systemic lupus erythematosus (SLE) is associated with heart involvement in 40% to 80% of patients [Giambuzzi 1996]. Although uncommon, cardiac manifestations of SLE have included myocardial infarction [Takatsu 1985] and myocarditis [Frustaci 1996]. Although extremely rare, lupus-related left ventricular aneurysm (LVA) formation has been the subject of several case reports [Nagaoka 1993, Frustaci 1996, Giambuzzi 1996, Glower 1997]. The purpose of this brief communication is to describe another rare case of SLE-related LVA that resulted in heart failure and stroke. Surgical intervention was necessary to manage both conditions.

## CASE REPORT

A 52-year-old woman was admitted with progressive shortness of breath and bilateral pulmonary infiltrates visible on plain chest radiography. She was treated with antibiotics for presumptive pneumonia. Her past medical history was

significant for SLE for 2 years prior to admission. She was maintained on oral steroid therapy.

The patient's dyspnea progressed over the next several days. An echocardiogram showed a discrete posterior LVA with mural thrombus. No evidence of myocardial infarction was elicited by history, electrocardiogram, or myocardial enzyme analysis. Cardiac catheterization was performed and showed normal coronary arteries. The left ventricular ejection fraction was estimated at 45%.

The patient was medically treated for congestive heart failure with digoxin, angiotensin-converting enzyme inhibitors (ACE-I), and diuretic therapy. Although repeat echocardiography 1 week later showed persistent depressed LV function, her symptoms improved enough to permit increased activity. However, the following week she suffered a stroke, presumably from a thrombus within the LVA. Serial magnetic resonance imagery (MRI) of the brain showed a cerebral infarct. Carotid ultrasonography results were normal. Digital subtraction cerebral angiography did not show intracranial occlusive disease. The patient was treated with anticoagulation and rehabilitation. Six weeks after the stroke, surgical resection of the LVA was performed. Operative findings included an oval posterior LVA measuring 3 × 5 cm (Figure). Resection and repair of the LVA were performed following removal of the mural thrombus. The resected specimen demonstrated tissue fibrosis with paucity of myocardial cells consistent with classic ischemic aneurysm tissue.

The postoperative course was uneventful. Cardiac rehabilitation was initiated, and the patient was discharged on the ninth postoperative day. Coumadin therapy was resumed postoperatively for 6 weeks. The LV ejection fraction remained at 45%.

## DISCUSSION

Most reported LVAs developed as a consequence of coronary artery disease. The etiology of a LVA has been reported to be coronary disease with subsequent myocardial infarction in up to 95% of reported cases [Glower 1997]. Other causes include viral myocarditis, infective endocarditis, and aneurysm formation secondary to Chagas disease. Although uncommon, cases of LVA in association with SLE have been the subject of case reports [Nagaoka 1993, Frustaci 1996,

Received October 4, 2004; accepted November 26, 2004.

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Lupus-related left ventricular aneurysm.

Giambuzzi 1996, Glower 1997]. The etiology of the LVA in one case was reported to be a result of left anterior descending coronary artery occlusion [Giambuzzi 1996]. In another case, the LVA was caused by an occluded posterior interventricular branch of the right coronary artery [Takatsu 1985]. In a third case, lupus myocarditis with inflammatory cell aggregation caused an LVA in a patient with no evidence of arterial disease [Frustaci 1996]. The LVA in that report [Frustaci 1996] was treated with steroid therapy, which led to complete resolution of the aneurysm. A surgical approach to a case of LVA with mural thrombus associated with angiographically normal coronary arteries has been reported in only one other case [Nagaoka 1993]. In this instance, there was also histologic evidence of fibrous endocarditis or inflammatory cell aggregation.

The case presented in this brief communication documents the second instance of SLE-related LVA in a patient with normal coronary arteries. In contrast to other cases, the patient in this report was maintained on steroid therapy for years prior to her admission. In addition, she suffered an acute embolic stroke from a mural thrombus within the LVA. Although her heart failure symptoms improved with medical therapy, she continued to have a depressed ejection fraction and remained at risk for further embolic issues. Theories regarding the etiology for LVA formation in SLE include

inflammatory processes. Presumably, coronary arteritis may contribute to ischemic zones of myocardium, resulting in gross infarction. Alternatively, small vessel disease in a cardiac segment may result in transmural injury. The presence of endocarditis and inflammatory cells in 2 cases suggests a reactive process of some sort. The resolution of SLE-related LVA with steroid treatment in 1 case [Frustaci 1996] suggests that medical therapy may be effective in the acute phase. On the other hand, because of scar formation, chronic SLE-related LVA formation is most likely irreversible. In these cases surgical therapy is indicated, as it is for any standard LVA (ie, due to heart failure, arrhythmia, emboli).

## CONCLUSION

In summary, SLE-related LVA is a rare entity. Coronary angiography is necessary for determination of ischemic heart disease. Echocardiography is helpful in determining the general LV function as well as the specific presence of the LVA thrombus. Although steroid therapy may be worthwhile in newly diagnosed patients who have not previously been on chronic steroid therapy, surgical intervention should be strongly considered in symptomatic patients with SLE-related LVA, particularly if they have already been treated with steroids and the condition is considered chronic.

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