

## CASE REPORTS

## Left ventricular thrombi in a patient with the antiphospholipid syndrome

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### Abstract

**A 41 year old woman with the antiphospholipid antibody syndrome presented with a cerebral embolus. This was caused by a mobile left ventricular thrombus that later resolved. There was an additional old left ventricular thrombus. Left ventricular thrombi such as these have not been previously described in this syndrome, and may have been under diagnosed.**

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**Keywords:** left ventricular thrombi; antiphospholipid syndrome.

There are major cardiovascular manifestations of the antiphospholipid antibody syndrome (APS).<sup>1,2</sup> This syndrome in which antibodies, such as anticardiolipin and lupus anticoagulant, are directed against phospholipids may occur with systemic lupus erythematosus or as a primary condition. The clinical features are protean and include valve

disease, stroke, migraine, livedo reticularis, and recurrent abortion. Intracardiac thrombosis has been very rarely seen; we describe a patient with APS who presented with a cerebrovascular accident and who had two left ventricular thrombi, one of which resolved with anticoagulation.

### CASE REPORT

A 41 year old woman was admitted with a sudden onset of dysarthria and dysphagia. She had a history of thrombocytopenic purpura diagnosed 10 years earlier and treated with steroids and subsequent splenectomy. Eight years earlier she had an episode of visual disturbance and slurred speech lasting 24 hours that was attributed to migraine. On examination she was afebrile and had no rash or abnormal cardiac signs. Her blood pressure was 140/95 mm Hg. Neurological examination showed a left upper motor neurone facial weakness, mild pseudobulbar palsy with dysphagia and dysarthria, and a slight weakness on the left arm. The electrocardiogram showed left ventricular hypertrophy. An echocardiogram was reported to be normal.

A computed tomogram (CT) of the brain confirmed an acute cerebral infarction in the right frontoposterior region. Magnetic resonance imaging identified two areas of infarction—one as seen on the CT scan and another in the left parietal lobe, which was consistent with the patient's neurological symptoms some years previously. Serological examination confirmed a diagnosis of antiphospholipid syndrome (APS) with raised titres of antidual stranded DNA antibodies, antinuclear antibodies with a homogeneous staining pattern, raised anticardiolipin antibody titres, and the presence of lupus anticoagulant. While awaiting anticoagulation she was re-admitted with a sudden worsening of dysphasia. The repeat CT scan was normal. Transthoracic echocardiography showed good overall left ventricular systolic function and mild symmetrical left ventricular hypertrophy. The mitral and aortic valves were normal and the left atrium was not enlarged. The apex was hypokinetic and there were two thrombi present—a typical well circumscribed mass in the apex of the left ventricle and a mobile irregular mass attached to the mid portion of the interventricular septum and protruding in to the left ventricular cavity (figure). Review of the previous echocardiogram

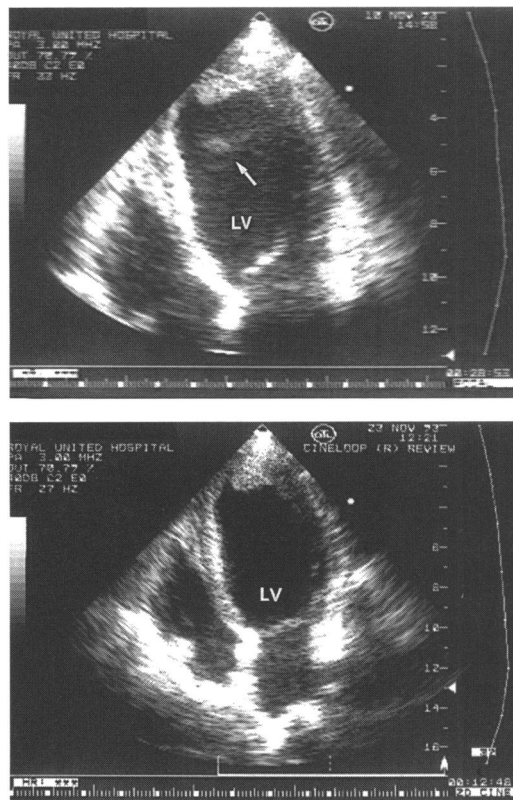
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*Echocardiographic four chamber view showing thrombi in the left ventricle (LV). The arrow in the upper frame points to the mobile irregular thrombus attached to the septum which has disappeared in the lower frame, which was taken 2 weeks later.*



gram showed poor apical views—which with hindsight may have been abnormal.

Anticoagulation with warfarin was started to maintain the international normalised ratio between 3 and 4. Heparin was avoided in view of the thrombocytopenia. Treatment with lisinopril was started for persisting mild hypertension.

Serial echocardiograms were performed each week for 4 weeks and every 3 months. At one week the septal thrombus appeared slightly larger, but at 3 weeks and 6 months it was no longer visible (fig). The apical thrombus remained unchanged.

### Discussion

The “striking clinical constellation” of the APS, described by Hughes,<sup>1</sup> is important for all specialists. There are many cardiological features, including labile and malignant hypertension, culture negative endocarditis, pulmonary hypertension, cardiomyopathy, and thrombus mimicking myxoma. Premature ischaemic heart disease may occur; however, the role of anticardiolipin antibodies in this condition is not clear.<sup>3</sup> Neurological features are also diverse and include various forms of cerebral ischaemia.<sup>4</sup> Stroke caused by cerebral embolus from cardiac chambers is thought to be rare.<sup>4</sup>

The first two reports of intracardiac thrombosis were in 30 year old women with systemic symptoms and no embolic features. One had a systolic murmur and click and was shown to have a right ventricular mass on echocardiography. She had complex calcified thrombosis attached to multiple anomalous right ventricular muscle bands.<sup>5</sup> The other patient had a systolic click and on echocardiography was shown to have a large right atrial thrombus mimicking a myxoma.<sup>6</sup> In a series of 72 patients with cerebral ischaemia three had atrial or ventricular thromboses, which were usually described as being associated with akinetic segments.<sup>7</sup> A right ventricular thrombosis was recently described in a patient presenting with fever and pulmonary infarction. At surgery the ventricular wall was partly replaced by inflammatory tissue.<sup>8</sup> The only other description of a left ventricular thrombosis was in a patient with diffuse left

ventricular dysfunction who had a mobile serpentine thrombosis.<sup>9</sup> This thrombosis embolised shortly after it was diagnosed and this caused a massive stroke.

There are several possible mechanisms for the hypercoagulability in APS.<sup>1</sup> Our patient had apical hypokinesia of unknown cause. This did not appear to be extensive enough to contribute significantly by stasis, but it may be an essential feature.<sup>7</sup> Serial echocardiograms showed the dynamic nature of one of the thrombi. The apical thrombus, which appeared fixed to the endocardium, showed no change whereas the more mobile thrombus resolved. It is uncertain whether this thrombus embolised, for which there was no clinical evidence, or lysed spontaneously. Long-term anticoagulation with high dose warfarin was given, to obtain an INR > 3.0.<sup>10</sup> Low dose aspirin may be used<sup>1</sup> although there is little evidence of additional benefit.<sup>10</sup>

The usefulness of echocardiography in young stroke patients without clinical heart disease is not clear.<sup>11</sup> We have shown a clear source of embolism in a patient with APS and we wonder whether left ventricular thrombosis has been underestimated in such patients.

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