Images in cardiovascular medicine
Non-bacterial endocarditis as first evidence of systemic lupus erythematosus

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A 32-year-old woman was referred to our Department for palpitations and new-onset systolic murmur. There was no history of systemic symptoms.

At transthoracic echocardiography, the mitral valve leaflets and subvalvular apparatus appeared thickened with vegetations on the leaflet ventricular surface, provoking severe mitral insufficiency due to impaired leaflet motion (Fig. 1). The left ventricular function was normal. A mild increase in the dimensions of the left atrium (44 mm) and of the left ventricular end-diastolic diameter (58 mm) was found. Blood tests showed increased levels of antistreptolysin and conventional inflammatory markers (erythrosedimentation rate and gammaglobulin), while C-reactive protein and white cell count were in the normal range. These findings did not fulfill the criteria for the diagnosis of rheumatic carditis. Systemic lupus erythematosus (SLE) tests, rheumatoid test and Waaler Rose were negative. No bacteria were recovered from repeated blood cultures.

Five months later the patient was hospitalized for an acute thrombosis in the right arm.

The hypothesis of antiphospholipid syndrome was made because of the presence of coagulation disorders (thrombocytopenia, prothrombin time elongation) although antiphospholipid and anticardiolipin testing was negative. For this reason, oral anticoagulant and corticosteroid therapies were prescribed. Laboratory analysis revealed that the patient was negative for lupus anticoagulant antibodies but positive for antinuclear antibodies. Decreased levels of the C3 and C4 complement components were also found.

A transthoracic echocardiogram was performed 2 months later revealing a mild

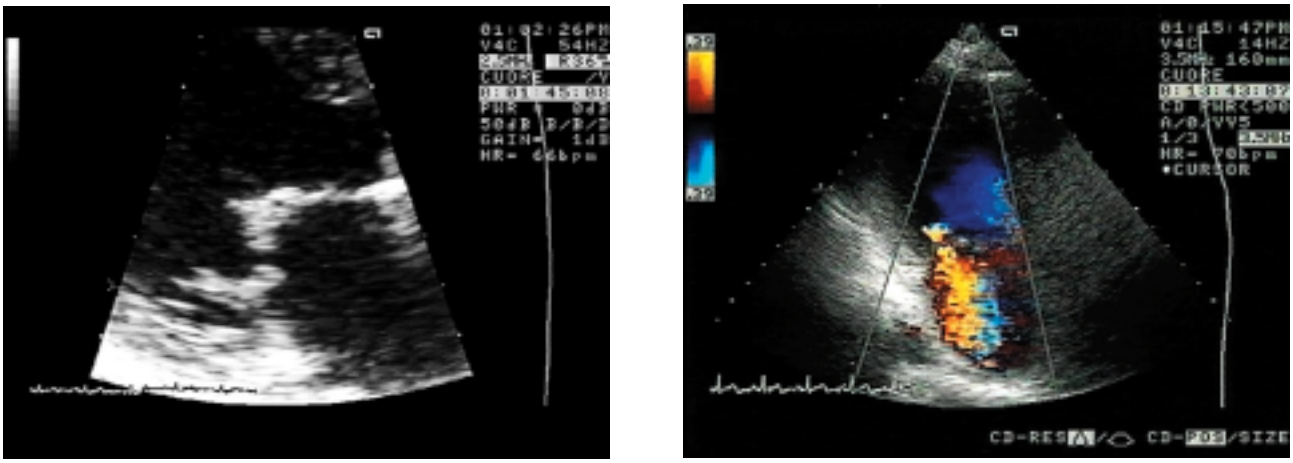

Figure 1. A: *transthoracic echocardiogram. The mitral valve leaflets are thickened and vegetations are present in the middle segment of both leaflets on the ventricular side.* B: *color Doppler flow on the mitral valve. Note the moderate to severe mitral insufficiency detected on the color map and by a large proximal isovelocity surface area surrounding the mitral orifice.*

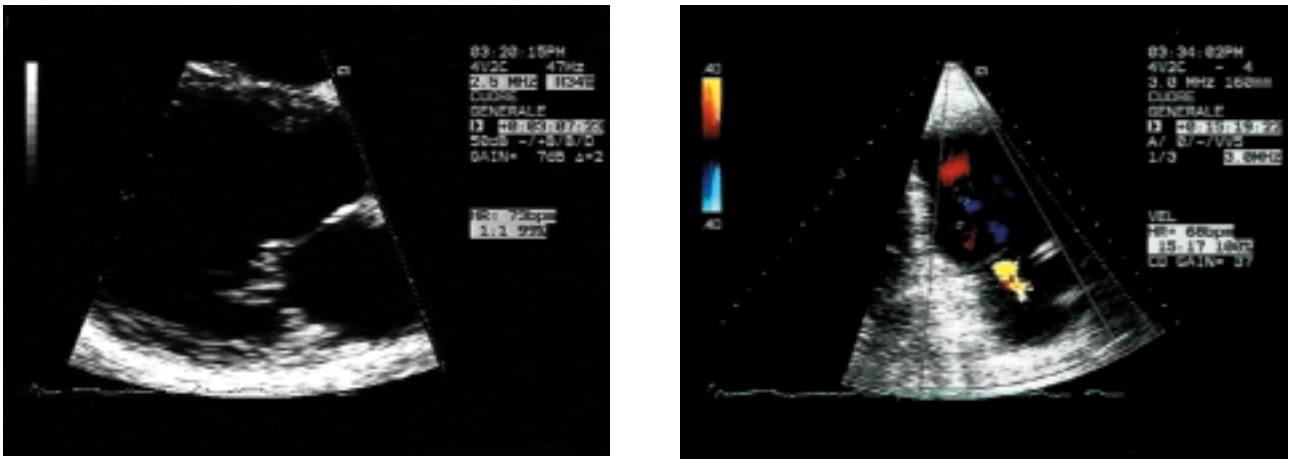

Figure 2. A: *transthoracic echocardiogram. Note the reduction in leaflet thickening and the absence of vegetations.* B: *color Doppler flow on the mitral valve. Mild mitral insufficiency on the color map, no proximal isovelocity surface area is detected.*

mitral leaflet thickening without significant regurgitation (Fig. 2). The definite diagnosis of SLE was made only 1 year after the first clinical evaluation and was based on the appearance of systemic symptoms (fever, polyarthralgia) and on the new laboratory results (lupus anticoagulant and anticardiolipin antibodies were both positive, presence of cryoglobulinemia, antinuclear antibodies with decreased C3 and C4 complement components and increased inflammatory markers).

This case shows that acute mitral valve disease may be the first clinical manifestation of SLE before laboratory marker evidence and that medical treatment may suffice to reverse it. Reduced mitral regurgitation coincided with the decrease in leaflet thickness and the regression of vegetations. It is difficult to explain the exact mechanism and specific merit of drug therapy. The effects of steroids on valvular abnormalities in SLE are not clear. It has also been reported that this therapy may facilitate valve damage.

Therefore, in the presence of apparently idiopathic acute mitral valve disease it is crucial to carry out a comprehensive evaluation in order to identify potentially reversible causes.

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