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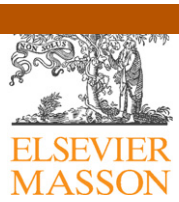



IMAGE

# Fast progression of aortic stenosis in rheumatoid arthritis

## Sténose aortique de progression rapide et polyarthrite rhumatoïde

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Valvular heart disease associated with rheumatoid arthritis is rare. In some cases, pathological studies have shown valve nodules and leaflet fibrosis that may extend to both the valvular annulus and, in the case of atrioventricular valves, the subvalvular apparatus. Valve regurgitation is common, but there are only few reports on valve stenosis. We report a case of rapid progression of aortic stenosis in a 67-year-old woman with long-standing active, uncontrolled and erosive rheumatoid arthritis for which an antitumour necrosis factor (TNF) $\alpha$ therapy had been initiated. She had a 3-year history of stable asymptomatic moderate aortic stenosis (Fig. 1, Panel A) (maximal velocity: 2.3 m/sec and 2.7 m/sec, mean gradient: 17 and 22 mmHg, aortic surface area: 0.8 $cm^2/m^2$ and 0.7 $cm^2/m^2$, respectively, 3 years and 3 months before the acute event). An episode of acute heart failure occurred 5 days after the first infusion of etanercept. Transthoracic echocardiography revealed marked deterioration of the left ventricular ejection fraction (25—30%) and now severe low gradient aortic stenosis (mean gradient: 20 mmHg, calculated aortic surface area 0.5 $cm^2/m^2$). Ejection fraction had been normal 3 months before the acute decompensation. The anti-TNF $\alpha$ therapy was interrupted. Low-dose dobutamine-stress echocardiography revealed residual contractile reserve and confirmed ''true'' severe aortic stenosis (Fig. 1, Panel B). Aortic-valve replacement was performed after ruling out coronary artery disease by coronary angiography. The operative settings showed three thickened leaflets and valve nodules. Histological examination confirmed the presence of rheumatoid nodules (Fig. 1, Panel C-a) showing large epitheloid cells

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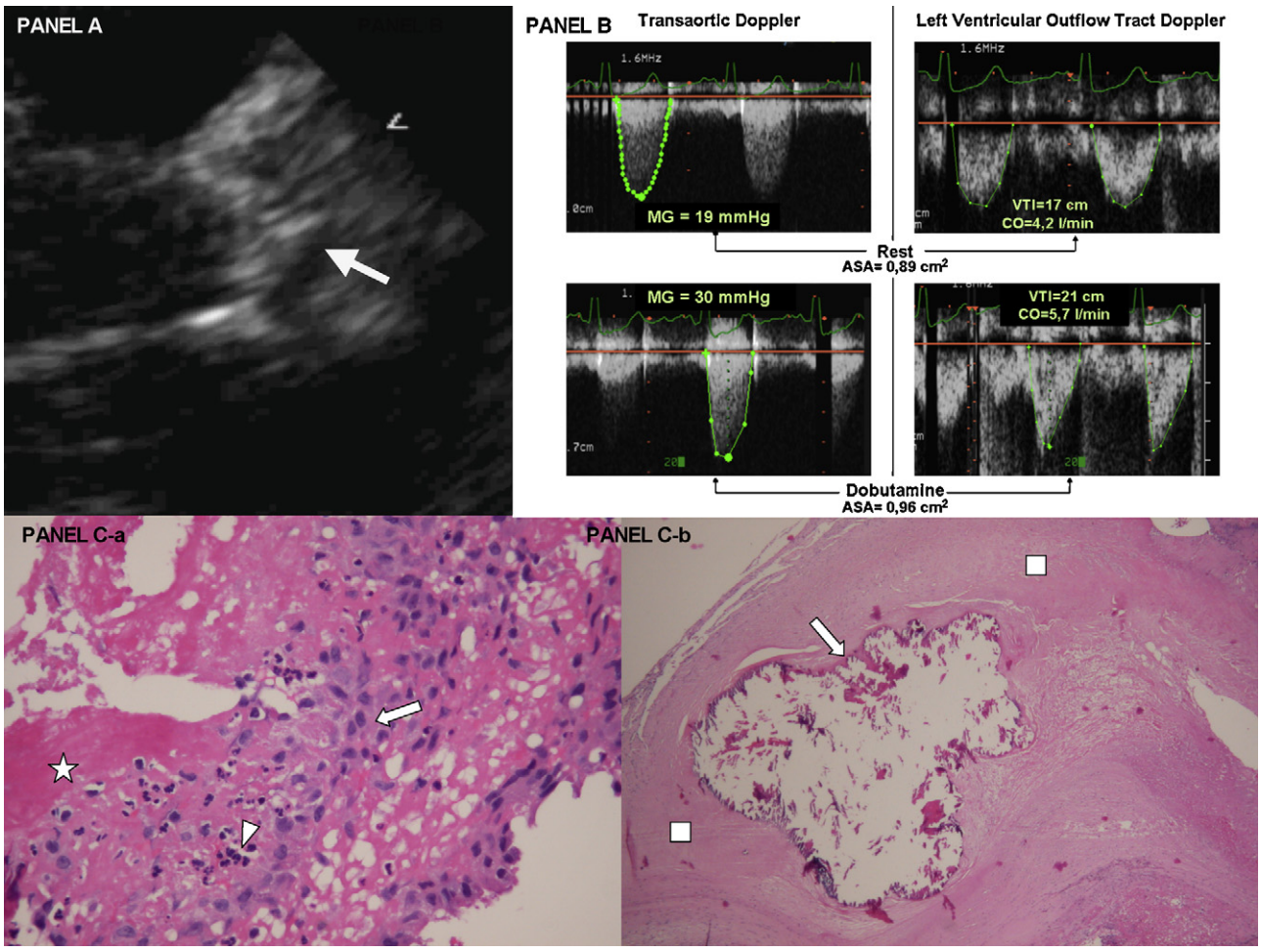


**Figure 1.** Panel A. Parasternal long-axis view of the aortic-valve (arrow) showing thickened and calcified leaflets. Panel B. Low-dose dobutamine-stress echocardiography showing residual contractile reserve and ''true'' severe aortic stenosis. Panel C. Histology (haematoxylin and eosine) showing (a) large epitheloid cells (arrow) and neutrophilic granulocytes (arrowhead) in a background of fibrin (asterisk) associated with (b) leaflet fibrosis and calcifications (squares and arrow, respectively).

(arrow) and neutrophilic granulocytes (arrowhead) in a background of fibrin (asterisk) associated with leaflet fibrosis and calcifications (squares and arrow, respectively, in Fig. 1, Panel C-b). Because of poor control of the rheumatoid arthritis, the etanercept therapy was reintroduced, without recurrence of cardiac symptoms. At follow-up 3 months after surgery the ejection fraction had recovered completely.

To our knowledge there are no data available linking active rheumatoid arthritis with fast progression of aortic stenosis. Previously reported risk factors for rapid progression are older age, high maximal velocity (>4—4.5 m/sec), aortic leaflet calcification, pathological exercise stress test, cardiovascular risk factors such as hypercholesterolaemia and smoking, renal failure or dialysis, hypercalcaemia and elevated C-reactive protein.

## Conflicts of Interest

None