Rheumatic disease mimicking an infiltrative mass of the mitral valve

Ferande Peters1, Bijoy K. Khandheria2*, Michelle L. Wong3, and Mohammed R. Essop1

1Department of Cardiology, Chris Hani Baragwanath Hospital, University of the Witwatersrand, Diepkloof 319-Iq, Johannesburg, Soweto 1862, South Africa; 2Aurora Cardiovascular Services, Aurora Sinai/Aurora St Luke’s Medical Centers, University of Wisconsin School of Medicine and Public Health, 2801 W. Kinnickinnic River Parkway, #840, Milwaukee, WI 53215, USA; and 3Department of Pulmonology, Chris Hani Baragwanath Hospital, University of the Witwatersrand, Diepkloof 319-Iq, Johannesburg, Soweto 1862, South Africa

*Corresponding author. Tel: +1 414 649 3909; fax: +1 414 649 3551. Email: publishing22@aurora.org

A 32-year-old man previously was diagnosed with sarcoidosis on the basis of bilateral lung infiltrates, hypercalcaemia, multiple renal calculi and an elevated serum angiotensin-converting enzyme level, all of which improved on corticosteroids. Nine months later he developed symptomatic, sputum-positive pulmonary tuberculosis and tuberculous lymphadenitis, which were successfully treated.

At his initial presentation, there was evidence of severe mitral regurgitation and moderate mitral stenosis complicated by severe pulmonary hypertension (Panels A and B). The medial halves of both mitral leaflets were immobilized by what appeared to be an infiltrative process with an associated pedunculated mass (Panels C–F, see Supplementary data online, Videos S1 and S2). The subvalvular apparatus and basal posterior wall were abnormal and presumed to represent a continuum of the infiltrative process. No clinical or laboratory features of infective endocarditis were present. Two years later, the morphology of the valve was unchanged except for the absence of the pedunculated mass, while the degree of pulmonary hypertension and mitral stenosis was worse. Cardiac catheterization confirmed the severe pulmonary hypertension was due almost exclusively to the severe mixed mitral valve disease. This haemodynamic abnormality was eliminated following successful mitral valve replacement.

Histology of the resected valve revealed features of chronic inflammation compatible with chronic rheumatic disease with no features suggestive of tuberculosis or sarcoid (Panel G). We postulate that the pedunculated mass might have represented nonbacterial endocarditis and that excessive scarring and chronic inflammation from untreated rheumatic disease resulted in this unusual morphological appearance of the mitral valve.

Supplementary data are available at European Heart Journal – Cardiovascular Imaging online.