Subaortic membrane: correlation of imaging with pathology

Albert Teis1*, Mary N. Sheppard2, and Francisco Alpendurada1

1Cardiovascular MR Department, Royal Brompton Hospital, Sydney Street, London SW3 6NP, UK; and 2Histopathology Department, Royal Brompton Hospital, London, UK

* Corresponding author. Tel: +44 20 7351 8824, Fax: +44 20 7351 8816, Email: a_teis@yahoo.es

A 31-year-old lady with a 6-month history of breathlessness and fatigue and presumptive severe aortic stenosis was referred to our institution for surgical evaluation. Initial transthoracic echocardiogram was of suboptimal quality but suggested the presence of turbulent flow in the left ventricular outflow tract (LVOT). A cardiovascular magnetic resonance study noted a discrete subaortic membrane causing severe subaortic stenosis (Panel A, arrows). No aortic valve disease, supra-aortic membrane, or aortic coarctation was identified.

Intraoperative transoesophageal echocardiogram (TOE) confirmed the presence of a subaortic membrane (Panel B) causing severe subaortic stenosis with turbulent flow on colour-Doppler images (Panel C, arrows). A discrete circular subaortic membrane was successfully resected with restoration of laminar flow on colour-Doppler images (Panels D and E). Macroscopic examination of the excised membrane showed multiple fragments of dense fibrous tissue (Panels F and G). Histological examination (Verhoeff’s van Gieson staining) revealed layers of fibroelastic tissue (collagen fibres in pink and elastic fibres in blue) consistent with a subaortic membrane (Panel H).

Subaortic membrane is a rare condition but remains an important differential diagnosis in hypertrophic cardiomyopathy with LVOT obstruction and in aortic stenosis, particularly at a young age or in the presence of family history. It is generally considered to be the result of a combination of factors that include an underlying genetic predisposition and various geometric and anatomical variations of the LVOT leading to flow turbulence at this level. The resulting turbulent flow may damage the endothelium and promote fibrin deposition which could ultimately progress to fibroelastic obstruction.

Supplementary material is available at European Heart Journal online.