A 59-year-old man had, over a period of 5 years, three different clinical scenarios which led to dynamic left ventricular outflow tract obstruction (LVOTO). Initially, this man who suffered from hypertrophic cardiomyopathy presented with asymmetric septal hypertrophy and systolic anterior motion (SAM) of the anterior mitral valve leaflet (MVL) along with mitral regurgitation. He was treated by septal myectomy and mitral valve repair with insertion of an artificial mitral ring. Several years later, he presented with severe LVOTO, this time related to the anteriorly displaced mitral coaptation site and the prosthetic ring. The ring and the anterior mitral leaflet were resected and a prosthetic mitral valve implanted. Several years later, the patient presented with LVOTO for a third time. It was now SAM of the remaining posterior leaflet that was responsible for the LVOTO.

In 2005, this 59-year-old man started to complain of shortness of breath on exertion (SOBOE). An echocardiogram showed asymmetric septal hypertrophy (ASH) with systolic anterior motion (SAM) of the anterior mitral valve leaflet (MVL) which caused significant left ventricular outflow tract obstruction (LVOTO) with a late peaking resting gradient of 65 mmHg and moderate mitral regurgitation (MR). It was felt that the MR was not due to SAM alone but also intrinsic MR; therefore, the patient underwent septal myectomy and mitral valve repair with insertion of a 34 mm Cosgrove Posterior Anuloplasty Band (Edwards Life-sciences, Irvine, CA, USA) after medical treatment provided no relief. While there was no significant LVOTO immediately after the operation, he later developed severe SOBOE. Echocardiogram revealed successful myectomy with septal thickness of 12 mm at the outflow tract. There was, however, severe SAM of the anterior MVL with significant LVOTO (59 mmHg) and mild MR. Medical treatment was again unsuccessful and the patient was operated on again. At that time, the annuloplasty band was removed, the anterior MVL excised, and a 27 mm ATS MV prosthesis (ATS Medical, Minneapolis, MN, USA) was set in place. The posterior MVL was left intact.

Four years later, the patient complained of fatigue and SOBOE. An echocardiogram showed normal prosthetic valve function. There was no LVOTO by the prosthetic valve. However, there was SAM of the (remaining) posterior MVL (Figure 1A and B, Supplementary data online, Videos 1 and 2) which resulted in significant LVOTO with a dynamic gradient up to 90 mmHg (Figure 1C and D).

Discussion

This patient was particularly prone to LVOTO given his small left ventricular diameter (left ventricular end-diastolic dimension of 3.5 cm) and septal hypertrophy. He has three mechanisms which resulted in LVOTO:

(i) Before surgery—the garden variety ASH and SAM resulting in LVOTO.¹

(ii) After the first operation, there was SAM associated with mitral annuloplasty. LVOTO after this procedure occurs in 5–10% of operated patients, and is associated with anterior displacement of the mitral coaptation site and change of overlapping leaflet length and position.²

(iii) After the second surgery with removal of the anterior leaflet, there was SAM of the posterior MVL. This phenomenon has rarely been observed.³ Preservation of the native subvalvular apparatus (or one of the leaflets) is thought to maintain left ventricular function.⁴ Since the anterior leaflet was removed and not opposing the posterior leaflet during systole, the posterior leaflet could move into the left ventricular outflow tract causing its obstruction.

⁴ Corresponding author. Tel: +1 212 434 2000; fax: +1 212 434 2111, Email: jchen@lenoxhill.net
Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2011. For permissions please email: journals.permissions@oup.com
Supplementary data

Supplementary data are available at European Journal of Echocardiography online.

References