CASE REPORT

Mitral valve endocarditis in hypertrophic cardiomyopathy: case report and literature review

G Morgan-Hughes, J Motwani

Heart 2002;87:e8 (<http://www.heartjnl.com/cgi/content/full/87/6/e8>)

Mitral endocarditis complicating hypertrophic cardiomyopathy occurs predominantly on the left ventricular aspect of the anterior mitral valve leaflet in the presence of outflow tract obstruction. It is a rare condition and the estimated cumulative 10 year probability of developing endocarditis in patients with obstruction is < 5%. Combined mitral valve replacement and septal myectomy has been reported in this setting. A case of community acquired *Staphylococcus aureus* mitral valve endocarditis is reported in a previously asymptomatic young man with hypertrophic obstructive cardiomyopathy. The potential treatment options are discussed.

Endocarditis complicating hypertrophic cardiomyopathy (HCM) is not commonly reported but occurs almost universally in patients showing evidence of outflow tract obstruction. The estimated cumulative 10 year probability of developing endocarditis in obstructive HCM is < 5%.¹ Studies examining mitral valves from such patients with endocarditis have found vegetations most commonly on the left ventricular aspect of the anterior mitral valve leaflet, presumably caused by mitral-septal contact during systole.² Mitral valve replacement combined with septal myectomy has been reported as a treatment for mitral valve endocarditis and HCM with severe obstruction.³ We report a case of community acquired *Staphylococcus aureus* mitral valve endocarditis in a young man with previously asymptomatic HCM and a modest basal outflow tract gradient. We review the literature and discuss the potential treatment options under such circumstances

Figure 1 Janeway lesion and vasculitis on the plantar surface of the left foot.

CASE REPORT

A 27 year old man was admitted following a three day history of general malaise, myalgia, sweats, and a high fever. Obstructive HCM had previously been diagnosed based on standard diagnostic criteria following an army medical examination a number of years earlier. There had been no related symptoms, however, and no specific treatment had been instituted. Antibiotic prophylaxis had been suggested but this advice had not been followed during dental work some days before admission. Clinical examination found a high fever, dehydration, a harsh systolic murmur, a vasculitic rash, and a Janeway lesion on the sole of the left foot (fig 1). Initial blood tests showed raised inflammatory markers (C reactive protein 220 mg/l) and thrombocytopenia ($58 \times 10^9/l$). An ECG showed major left ventricular hypertrophy and abnormal lateral repolarisation. Transthoracic echocardiography showed localised septal hypertrophy (2.4 cm) and systolic anterior motion of the anterior mitral leaflet in keeping with his underlying diagnosis. There was suspicion of a vegetation on the anterior mitral valve leaflet and mitral regurgitation was quantified as mild. A previous study under basal conditions at the time of diagnosis, using continuous wave Doppler ultrasound in the left ventricular cavity and outflow tract, had given a maximal predicted gradient of 36 mm Hg. Transoesophageal echocardiography confirmed the presence of a small (0.7 cm $\times$ 0.5 cm) vegetation on the left ventricular aspect of the anterior mitral valve leaflet and mild mitral regurgitation (fig 2). Three sets of blood cultures subsequently grew flucloxacillin sensitive *S aureus* in all bottles.

Figure 2 Transoesophageal echocardiography. Arrows mark the septal hypertrophy and vegetation attached to the anterior mitral valve leaflet.

Treatment with intravenous gentamicin and flucloxacillin was initiated and the case was discussed with a microbiologist and a cardiothoracic surgeon. Bearing in mind the virulence of the organism, early surgery was planned barring a prompt and complete clinical response to antibiotics. Following extensive discussion it was felt that the most appropriate surgical procedure, were it to prove necessary, would be septal myectomy in addition to mitral valve surgery.

Within 48 hours of treatment symptoms resolved dramatically and inflammatory markers improved. During the subsequent four week period of antibiotic treatment, transoesophageal echocardiographic surveillance confirmed no significant progression of valvar regurgitation and no new vegetations or complications. The C reactive protein concentration remained < 10 mg/l and the patient was discharged without requiring surgery. Six months following discharge he has remained well.

DISCUSSION

In the two decades preceding 1999 only 33 cases of endocarditis complicating HCM were recorded in the English language literature.¹ The information that is available suggests that the prognosis associated with endocarditis is worse if there is underlying HCM and antibiotic prophylaxis is recommended.⁴ The efficacy of antibiotic prophylaxis is, however, questionable and it is not unusual for it not to be given.⁵ Whether it is of concern in our case is dubious. It is clear from morphological studies that systolic anterior motion of the anterior mitral valve leaflet is relevant to the pathogenesis of endocarditis.² Examination of excised mitral valves has indicated that vegetations are located most commonly on the septal aspect of the anterior mitral valve leaflet, which was the case with our patient. In the most comprehensive study of prevalence and incidence (and the most comprehensive review) Spirito and colleagues¹ identified 10 patients with HCM and endocarditis. Both mitral valve involvement and outflow tract obstruction were present in all cases. These findings are typical and there are two reported cases in the literature of mitral valve surgery combined with septal myectomy under these circumstances.^{1,3} Both mitral valve surgery and the Morrow septal myectomy are accepted methods of treating symptomatic obstructive HCM refractory to medical treatment, but the largest reported series on the surgical management of HCM suggests that the two operations are rarely combined.⁶⁻⁹

Community acquired native valve endocarditis caused by *S aureus* carries a mortality of 25–47% and is itself a relative surgical indication.¹⁰ Failure to operate on patients who do not

respond rapidly to antibiotics or who have evidence of abscess formation often leads to a fatal outcome.¹¹ Both patients in the case series described by Spirito and colleagues¹ with *S aureus* and HCM required surgery.¹ The combination of obstructive HCM and endocarditis should prompt early surgical consultation, especially when the infecting organism is *S aureus*. Consideration should be given to valve surgery combined with septal myectomy. It is rare to report successful medical treatment under these circumstances.

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Authors' affiliations

G Morgan-Hughes, J Motwani, Cardiology Department, South West Cardiothoracic Unit, Plymouth NHS Trust, Plymouth PL6 8DH, UK

Correspondence to: Dr G Morgan-Hughes, Cardiology Department, South West Cardiothoracic Unit, Plymouth NHS Trust, Plymouth PL6 8DH, UK; hughesgj@talk21.com

Accepted 27 February 2002

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