Summary

A 38-year-old male was referred to our valve clinic for assessment. The patient had been diagnosed with untreated rheumatic mitral stenosis (MS) in 2009. The patient was diagnosed with atrial fibrillation in 2016 (on warfarin with good international normalised ratio (INR) control). Transthoracic echocardiography demonstrated severe MS (11 mmHg mean gradient across valve, 0.9 cm² valve area by planimetry), no mitral regurgitation, moderate left ventricular systolic dysfunction, volume and pressure loaded impaired right ventricle, moderate tricuspid regurgitation and severe pulmonary hypertension (estimated right ventricular systolic pressure was 76–81 mmHg with a tricuspid regurgitation maximum velocity of 4.12 m/s). A large amount of spontaneous echo contrast (SEC) was noted in the severely dilated left atrium and a transoesophageal echocardiogram (TOE) was requested for further assessment of the MS (Fig. 1, Videos 1, 2 and 3). The patient was symptomatic with shortness of breath and reduced exercise tolerance consistent with NYHA class III–IV.

The extent of SEC (representing slow flowing blood) in a patient with an acceptable INR of 2.5 is striking. The AHA guidelines (1) and ESC guidelines (2) suggest managing rheumatic MS based on stenosis severity, symptoms and the presence of left atrial clot. In this case, following multidisciplinary team (MDT) discussion, it was felt that the patient was too high risk for balloon valvuloplasty. The case was also discussed with the heart transplant service who felt that the patient was not a candidate for heart transplantation due to pulmonary hypertension. A consensus decision was then made to offer the patient a high-risk mitral valve replacement surgery with mechanical circulatory support if required. A 33 mm Sorin mechanical mitral valve was thereafter implanted in an uncomplicated operation.

The patient has been reviewed subsequently in clinic and has had a dramatic improvement in symptoms. Repeat echocardiography demonstrated a well-functioning valve and resolution of SEC. The learning point in this case is that having a therapeutic INR does not necessarily stop significant SEC from being observed on echocardiography, especially if there are important contributory factors, which in this case was severe MS. It is to be remembered that SEC is a marker of stasis and has been shown to occur due to (platelet-independent) RBC aggregation in sluggish, low-shear stress flow conditions (3).

Figure 1
Transoesophageal echocardiogram still image. Mid-oesophageal view at 0° showing spontaneous echo contrast filling a dilated left atrium with bulging of the interatrial septum to the right. Thickened mitral valve leaflets are also seen.
Video 1

Video 2

Video 3

Declaration of interest
The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of this article.

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Author contribution statement
D J prepared the manuscript; G L performed TOE and obtained images; A G edited the manuscript.

References

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