Rheumatic mitral valve disease in pregnancy

Introduction

Pregnant patients with rheumatic valve disease need careful evaluation. Those with even moderate mitral stenosis should not be considered at low risk in pregnancy.

Discussion

Rheumatic fever remains endemic in some regions of the developing world with an overall prevalence of rheumatic mitral stenosis of between 40 and 50% of cardiac disease in pregnancy (Tsiaras and Poppas, 2009).

Rheumatic mitral stenosis has the highest morbidity and mortality in pregnancy, recently re-emerging in the UK as a cause of death in pregnancy (Nelson-Piercy, 2007). Patients at high risk of complications are those with severe pulmonary hypertension, left ventricular dysfunction (ejection fraction of <40%) and tighter stenosis.

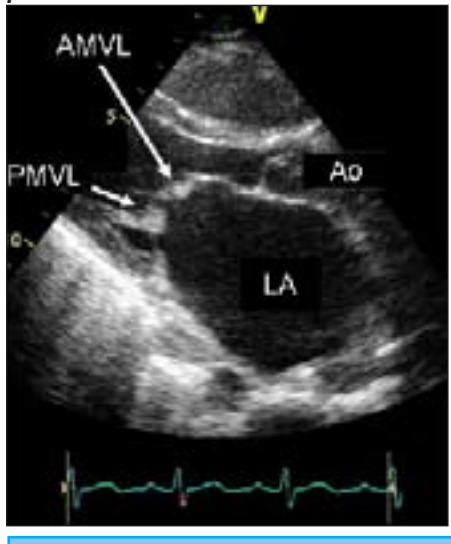
In pregnancy there is a 30–50% increase in cardiac output and intravascular volume which usually reaches a peak by the 24th week of gestation (Horstkotte et al, 2003). Patients with significant rheumatic mitral stenosis will usually develop pulmonary oedema in the third trimester, during delivery or in the early puerperium. Further cardiovascular compromise will occur if atrial fibrillation ensues, particularly with uncontrolled ventricular rates.

Mitral stenosis is the lesion that most often requires intervention in pregnancy. Increased transmitral flow and reduced diastolic filling time as a result of tachycardia can increase pulmonary artery pressure up to 50%. Pulmonary hypertension is associated with a maternal peripartum mortality of 30–56% and neonatal risk of 10–13% (Weiss et al, 1998). Patients with a mitral valve area >1.5 cm^2 can usually be managed medically with diuretics and beta blockers.

More severe stenosis may need mitral balloon valvotomy during pregnancy. In this case, this was not an option because of coexisting mitral regurgitation. Continued medical therapy was thought likely to fail and necessitate emergency valve replacement, possibly when the fetus would be viable and therefore need an emergency caesarean section before cardiac surgery. Neonatal survival to discharge with delivery at 24 weeks' gestation is only 26%, and a quarter of surviving babies are severely disabled.

Surgical options range from closed mitral valvotomy through to open mitral valve replacement with cardiopulmonary bypass, but the best interests of the mother and fetus may not coincide. In the UK, fetal mortality remains significant, at between 19 and 33%, for cardiac surgery with cardiopulmonary bypass in pregnancy (Parry and Westaby, 1996).

Figure 1. Parasternal long axis echocardiographic view. The left atrium (LA) is significantly dilated (48 mm). The mitral valve is stenosed with a typical 'hockey stick' deformity of the anterior mitral valve leaflet (AMVL). Ao = aorta; PMVL = posterior mitral valve leaflet.



Conclusions

Functionally occult rheumatic mitral valve disease can cause life-threatening cardiac compromise even early in pregnancy. Increased vigilance of pregnant women from regions where rheumatic fever might be endemic is recommended. BJHM

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Case Report

A 32-year-old Nepalese woman with known rheumatic mitral valve disease presented 17 weeks into her first pregnancy with exertional breathlessness and wheezing, leading to expectoration of foamy pink liquid. Her history included percutaneous balloon mitral valvotomy at the age of 25 years, after which she had remained asymptomatic and fully active. She had three episodes of acute pulmonary oedema and was transferred to a tertiary centre for further management. An echocardiogram suggested severe mitral stenosis (mitral valve area 1.0 cm^2) and pulmonary hypertension (mean pulmonary artery pressure 87 mmHg). She had further episodes of pulmonary oedema despite maximal medical therapy with diuretics, cardioselective beta blockers (dose limited because of systolic hypotension), digoxin, nitrates and anticoagulation. At 21 weeks' gestation, an emergency St Jude metallic valve was implanted. Unfortunately, intrauterine death occurred 2 days postoperatively, likely caused by pre-eclampsia or haemolysis, elevated liver enzymes and low platelets (HELLP) syndrome.

The patient recovered well and continues on warfarin and a small dose of beta blocker.