Aortic Stenosis: Pregnancy and Labour - A Case Report

Rajkumari a 30 year old gravida 2 para 1-0-0-1 with aortic stenosis, severe aortic regurgitation, mitral stenosis and mitral regurgitation, NYHA class III - IV, booked at 13 weeks, and was admitted at 32 weeks 5 days gestation on 8/7/99 with IUGR, oligohydramnios and moderate PIH. She had grade 3 proteinuria and deranged renal function tests (b. urea 136, S. Creatinine 1.4). Her heart disease was discovered incidentally during her first pregnancy 10 years ago. She had then delivered a healthy male baby without any complications. She was taking digoxin, furosemide, nifedipine and penidure. On examination, she had grade 2 hypertensive retinopathy with attenuated arterioles and arteriolo-venular changes. Her JVP was found to be normal and there were no crepitations in the chest. Bilateral pitting pedal edema was present. Echocardiography demonstrated the valve lesions and in addition a dilated left atrium and ventricle and left ventricular systolic dysfunction in the form of decreased left ventricular ejection fraction of 40%.

After admission, she had progressive deterioration of function and a falling urine output. So induction of labor was decided upon and it was carried out with the help of oxytocin infusion and artificial rupture of membranes. She was managed very carefully with strict watch on her hemodynamic parameters. She had an uneventful labor and a spontaneous vaginal delivery of a live female baby weighing 1.48 kg on 17/7/99. In the following 10 days of her confinement, her renal functions gradually improved and she was discharged subsequently.

DISCUSSION:

Valvular heart disease can be acquired or congenital. Acquired valvular disease results primarily from inflammatory or degenerative changes over time and therefore symptoms are not usually manifested until later in life.

The basic hemodynamic feature of aortic stenosis is obstruction to left ventricular ejection, which can lead ultimately to a low cardiac output. The cross-sectional areas of the valve surface must be narrowed to less than one third of normal before clinically significant obstruction is produced. In response to this narrowing, the left ventricle hypertrophies to generate stronger contractions to maintain cardiac output. The result is a larger than normal pressure gradient between the left ventricle and the aorta. A pressure gradient greater than 50 mm Hg accompanied by a disease in the aortic valve area to less than 0.8 cm² (normal = 1.5 - 2.6 cm²) indicates significant aortic stenosis. At this level of stenosis, clinical symptoms emerge that progress from dyspnea and activity intolerance to the classic triad of congestive heart failure, angina pectoris, and syncope (Schlant 1991). A grossly hypertrophied ventricle, as mentioned above may compound the effects via hypoperfusion and ischemia, which contribute further to the reduction in cardiac output.

Management of pregnancy complicated by aortic stenosis requires an accurate assessment of the severity of disease. Unlike in mitral stenosis, clinical symptoms appear very late in the course of disease. Once patients complain of angina, shortness of breath or syncope, their risk of sudden death may be out of proportion to the severity of their clinical symptoms. In the past, accurate assessment of the severity of stenosis required cardiac catheterisation and significant radiation exposure (Rapaport 1975). Now a days, intracardiac pressure gradient can be accurately measured noninvasively using the Doppler continuity equation (Otto, 1986, Whittemore, 1983).
Reduced physical activity is the mainstay of antepartum care. Unless additional stress is superimposed, patients are able to tolerate the increased cardiac demands of pregnancy. The first stage of labour is managed according to usual obstetric standards. These patients are very susceptible to supine hypotension. The second stage is managed with minimal pushing and operative assistance when adequate station of the presenting part is achieved. The third stage is managed actively to avoid potential haemorrhage. Prophylactic antibiotics are administered. Careful haemodynamic management is required during labour or caesarean section. Effective analgesia can prevent the tachycardia associated with labor pain. Peridural analgesia has been considered contraindicated in patients with aortic stenosis because of potential significant decrease in preload and afterload. Adequate time is required to carefully titrate the block and initiate compensatory actions to correct hemodynamic changes induced by the anaesthetic. Analgesia with combined local anaesthesia and narcotics is particularly useful in patients with aortic stenosis. Immediately post partum, filling pressures frequently rise, requiring a diuretic.

Neonatal outcome is usually good (Easterling, 1988). Patients with congenital aortic stenosis should be informed of a 14-26% risk of carrying a fetus with congenital heart disease (Whittemore, 1983). Fetal echocardiography may be useful.

REFERENCES: