Case report of a 26 year old primigravida with Patent Ductus Arteriosus (PDA) in heart failure

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Abstract

Congenital heart disease is an important cause of maternal morbidity and mortality during pregnancy. Pregnancy alters the circulatory and respiratory physiology with attendant deleterious effect on the mother with congenital heart disease and the foetus. Additional insult to the circulatory physiology by other factors coexisting together with congenital heart disease can further reduce the cardiac reserve in pregnancy and precipitate heart failure. These factors include anaemia, thromboembolism, hypertension, multiple pregnancy, strenuous physical activity, extremes of temperature and the normal physiological edema of pregnancy.

Patent ductus arteriosus (PDA) can present for the first time in pregnancy. Moderate to large PDA result in significant volume overload, left ventricular dilation and dysfunction. In the woman with a hemodynamically important PDA, pregnancy may precipitate or worsen heart failure. We report a successful pregnancy in a 26 year old primigravida with previously undetected patent ductus arteriosus with preeclampsia who presented in heart failure. This case highlights the importance of intensive careful examination of pregnant patients to identify such conditions.

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Introduction

Congenital heart disease is an important cause of maternal morbidity and mortality during pregnancy.[1,2] Hypertensive disorders is also associated with significant morbidity and mortality in pregnancy in comparable proportion to the general population.[3] Pregnancy alters the circulatory and respiratory physiology with attendant deleterious effect on the mother with congenital heart disease and the foetus.[4] The cardiac reserve of the pregnant woman with congenital heart disease is already reduced. Therefore additional insult to the circulatory physiology by other factors coexisting together with congenital heart disease can further reduce the cardiac reserve in pregnancy and precipitate heart failure. These factors include anaemia, thromboembolism, hypertension, multiple pregnancy, strenuous physical activity, extremes of temperature and the normal physiological edema of pregnancy. This oedema is associated with increase in total exchangeable sodium and water[4]. The ductus arteriosus derives from the left sixth primitive aortic arch and connects the proximal left pulmonary artery to the descending aorta, just distal to the left subclavian artery. Functional closure of the ductus from vasoconstriction occurs shortly after a term birth. Isolated patent ductus arteriosus (PDA) are categorized based on the degree of left to right shunting as mild, moderate and severe PDA which can be subsequently complicated with Eisemengers complex when there is reversal of shunting with progression to pulmonary hypertension. Moderate to large PDA result in significant volume overload, left ventricular dilation and dysfunction associated with atrial fibrillation and pulmonary hypertension. Pregnancy is well tolerated in women with silent and small PDA or in patients who were asymptomatic before pregnancy. In the woman with a hemodynamically important PDA, pregnancy may precipitate or worsen heart failure. Pregnancy is contraindicated in Eisenmenger syndrome because of the high maternal (≥50 percent) and fetal (≥60 percent) mortality[3,4].

We report a successful pregnancy in a 26 year old primigravida with previously undetected patent ductus arteriosus with preeclampsia who presented in heart failure.

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Case report

A 26 year old booked primigravida , who had been apparently well until few weeks earlier when she presented in the hospital with a history of progressive

dyspnoea on exertion, orthopnoea, paroxysmal nocturnal dyspnoea and bilateral leg swelling. Her estimated gestational age was 35 weeks and she was not a previously diagnosed hypertensive patient. There was no history of light flashes or epigastric pain.

There was no history to suggest significant left to right shunting in childhood. Examination revealed a young woman in respiratory distress with a respiratory rate of 32 cycles per minute, not clinically pale, afebrile, (Temperature - 36.8 ^{0}C). She was well hydrated but had bilateral pitting pedal oedema up to the knees. Examination of the chest revealed fine bibasal crepitations. In the cardiovascular system, the pulse rate was 72 beats per minute, regular and large volume. Blood pressure was 200/90mmHg. Jugular venous pressure was elevated. There was hyperactive precordium and the apex beat was displaced to the 6th intercoastal space almost at the anterior axillary line. Heart sounds were S1S2S3 with a gallop rhythm, a grade 4/6 continuous murmur heard best at the second intercostal space. Abdominal examination revealed a uniformly enlarged abdomen with gravid uterus, fundal height was 35cm and the foetus was in longitudinal lie and cephalic presentation. Urinalysis revealed significant proteinuria (+++). Echocardiography revealed dilated left atrial and ventricular chambers (LAD - 5.2cm, LVIDd-6.6cm) with systolic dysfunction (Ejection fraction- 40%, Fractional shortening- 24%).There was a patent ductus arteriosus connecting the left pulmonary artery with the descending aorta. Peak pulmonary systolic velocity was 1.0m/s (normal) while the peak aortic systolic velocity was 1.9m/s (minimally elevated) .There was no echocardiographic sign of pulmonary hypertension (peak pulmonary systolic pressure was normal). Electrocardiography revealed left atrial abnormality, left ventricular hypertrophy and non specific ST-T wave changes. She was managed conservatively with antifailure regimen including strict bed rest, antibiotics, mild diuresis (Frusemide), reduction in salt intake, antihypertensive therapy (Methyldopa and Nifedipine) and continuous fetal monitoring. She had emergency lower segment caesarean section at 38 weeks of gestation due to suspected intrauterine growth retardation with the delivery of a live male foetus, weight 2.6kg, Apgar score $7^1 10^5$. Postnatal period was not adversely eventful. She was followed up in the Cardiology Clinic and was thereafter referred to the cardiovascular surgeon for definitive surgical management.

Discussion

Many haemodynamic changes that occur normally in pregnancy tend to worsen and contribute significantly to the morbidity and mortality in pregnant individuals with already reduced cardiac reserve such as those with congenital heart disease.[4] Plasma volume increase by as much as 50% especially in the third trimester. This may possibly explain why our patient presented with congestive heart failure at the time she presented. The already depressed cardiac reserve is further worsened by a sudden increase in plasma volume, increased total sodium concentration, anxiety with tachycardia and other factors such as bacteriuria with associated fever or any other febrile illness.

There are several factors contributing to deterioration in cardiovascular status of pregnant subjects with congestive heart disease. Hypertensive disorders such as chronic hypertension and pre-eclampsia are important clinical variables in this patient. Normally blood pressure falls to about 105/60 mmHg by the mid trimester due to reduction in peripheral resistance. However increase in circulatory volume in the third trimester may tilt a compensated heart into a decompensated stage. Pre-eclampsia is characterized by proteinuria and elevated blood pressure in a previously normotensive pregnant woman. The elevated blood pressure is another triggering factor for decompensation into the congestive heart failure stage which this patient presented with. Anxiety especially in a primigravida can increase the heart rate and also tilt a compensated heart to a state of decompensation. This may occur towards the delivery period as she ponders on the experience of labour.

The main predictors for maternal and fetal complications in pregnant women with congenital heart diseases include pulmonary hypertension(pulmonary vascular disease), maternal cyanosis, poor maternal functional status, arrhythmias and maternal anticoagulants.[5,6,7,8] The ductus arteriosus connects the descending aorta to the main pulmonary trunk near the origin of the left subclavian artery. The physiologic consequences of a PDA are determined by its size and length as well as by the ratio of pressure and resistance of the pulmonary and aortic circulations on either end of the duct. If systolic and diastolic pressure in the aorta exceeds that in the pulmonary artery, aortic blood flows continuously down a pressure gradient into the pulmonary artery and then returns to the left atrium.[8] The left atrium and subsequently the left ventricle

dilate, whereas the right side of the heart becomes progressively affected as pulmonary hypertension develops. This will vary with the quantity of left-to-right shunting as well as with the secondary effects on the pulmonary vascular bed. Symptoms generally increase by the second and third decades and include dyspnea, palpitations, and exercise intolerance.

The incidence of PDA has been noticed to be increasing over the last two decades due to improved survival of preterm babies.[8] There is a female predilection,[8] and genetic linkage has been described.[9] Individuals with PDA have increased morbidity and mortality due to heart failure and infective endocarditis.[10] Multiple septic emboli to the lungs can also occur.

Pregnancies complicated with Eisemenger complex are at particularly high maternal and fetal risk with many of them offered termination of the pregnancy. A successful pregnancy with Eisemenger complex has however being reported recently.[11] Treatment of the patient may involve surgical ligation which is the definitive management or by percutaneous occlusion using cathether based approaches such as Amplatzer duct occlusion, coil occlusion device and Rashkind umbrella device.[12,13] Pharmacologic treatment using indomethacin or ibuprofen is useful among preterms but can be complicated by renal dysfunction.[14] The mode of delivery in pregnant patients with congenital heart disease may be by vaginal delivery if there is no contraindication to it and the functional state of the heart is adjudged adequate. However, in case of any concern about the functional and / or structural state of the heart, circulation or the birth canal, induction or caesarean section as appropriate may be relevant. The treatment of the preeclampsia involves the use of antihypertensives such as calcium channel blockers and methyldopa.

Conclusion

This case highlights some points. First, congenital heart disease can present for the first time in pregnancy. Secondly, several factors such as anaemia, hypertension, tachycardia, infection, and arrhythmias may precipitate acute decompensation in pregnant subjects with congestive heart disease such as patent ductus arteriosus. Adequate antenatal cardiac auscultation screening and appropriate relevant investigation such as cardiac ultrasound are important to identify such patients to reduce the increased morbidity and mortality risk associated with this condition. As subsequent pregnancies further increase the morbidity, Contraceptive advice should be offered to such patients immediately postpartum.

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