Case report

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$P_2L_2A_0$ Emergency Lower Segment Caesarean Section (LSCS) with Atrial Septal Defect (ASD) with severe Pulmonary Arterial Hypertension (PAH) with Bidirectional flow with Pulmonary Edema with Sepsis

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Abstract:

Severe pulmonary Arterial Hypertension with Pulmonary Edema with Sepsis in a postnatal mother with Atrial Septal Defect (ASD) followed by LSCS is uncommon. Atrial Septal Defect (ASD) is the commonest adult congenital heart defect (CHD). 15 % of these patients will eventually develop pulmonary hypertension if left untreated. ASD closure is not recommended when pulmonary hypertension is irreversible. Congenital heart disease should be considered in the evaluation of dyspnoea in a young adult. The management of ASD with associated pulmonary hypertension is difficult. It is pertinent that a detailed hemodynamic assessment be undertaken. The present case report focusses on a patient with severe ASD with pulmonary hypertension with pulmonary edema and sepsis who was with 35 weeks of gestation and the control of symptoms during Caesarean section.

Key words: ASD, LSCS, Pulmonary hypertension

Introduction:

Caesarean section is an operative procedure whereby the foetus after the end of 28th week is delivered through an incision on the abdominal and uterine walls. In emergency caesarean section, operation is performed due to unforeseen or acute obstetric emergencies an arbitrary time limit of 30 minutes is thought to be reasonable from the time of decision to th start of procedure[1]. Atrial septal defect (ASD) is a form of a congenital heart defect that enables blood flow between two compartments of the heart called the left and right atria. In unaffected individuals, the chambers of the left side of the heart are under higher pressure than the chambers of the right side of the heart. This is because the left ventricle has to produce enough pressure to pump blood throughout the entire body, while the right ventricle needs only to produce enough pressure to pump blood to the lungs.[2] Hereby we are presenting a case $P_2L_2A_0$ Emergency LSCS with Atrial Septal Defect (ASD) with severe Pulmonary Arterial Hypertension (PAH) with Bidirectional flow with Pulmonary Edema with Sepsis.

Case report:

A 36-year old Indian female with an obstetrical score of $G_2P_2L_2A_0S_0D_0$ presented to the hospital (Lady Goshen Hospital, Mangalore) at 35 weeks with the complaints of breathlessness and pedal edema.

In her past history, she was a known case of ASD with pulmonary hypertension since 14 years and was on medication, Tab. Revatio 20mg half twice daily. She was married for 19 years and her first pregnancy was 2 years back and there were no specific complications in that pregnancy and the baby (Low Birth Weight -1.5 kg) was delivered by LSCS due to maternal cardiac disease . She did not have family history of any cardiac diseases or any other diseases.

In the present pregnancy, she attended regular antenatal checkups and discontinued Tab. Revatio once the pregnancy was confirmed. At 32 weeks of gestation, she presented with pedal edema and breathlessness and echocardiography was done which revealed Adult Congenital Heart Disease, large Ostium Secundum (OS) with bidirectional shunt, mildly dilated left sided chamber and severe PAH. Ultrasonography and colour doppler were done and found to be normal. Hence she was advised to take Tab. Revatio 20 mg half twice daily.

Presently, she came with severe breathlessness and pedal edema at 35 weeks of gestational age. On physical examination S1, S2, cardiac murmurs and crepitations (respiratory) were heard, pulse rate was 118beats/ min, blood pressure-170/120 mmof Hg, respiratory rate was 36 breaths/ minutes and the laboratory values were Hb-12.1gm/dl, TC-15300/cu.mm, PCV-36.3, Platelet count-2 1akhs, Urea-32mg/dl, Creatinine-0.7mg/dl, Sodium-135meq/l, Potassium-3.6meq/l, Prothrombin time-3.6 second, INR- 0.89. Hence she was posted for emergency LSCS. During the intra-operative period, tachycardia with fluctuating blood pressure was noticed and intubation done but the breathing pattern was found to be abnormal and hence re-intubation was done and put on SIMV (Synchronized Intermittent Mandatory Ventilation). The medications given during intra-operative period were Inj. Morphine 1 mg, Inj. Taxim 4 gms, Inj. Lasix 40 mg, Tab. Revatio 20 mg. Weaning from the ventilator was done on the 2nd post-operative day. She was on continuous observation; hourly vital signs were checked and intake output chart was maintained. The baby (female) weighed 2 kgs and was shifted to the NICU for observation.

On day 5, she was shifted to the ward and the medications were replaced by Inj. Pipzo 4.5mg, Inj. Rantac 50mg, Inj. Hydrocortisone 50 mg, Asthalin & Duolin nebulization. Her blood pressure was 120/70 mm of Hg, pulse rate was 82 beats/ min, Respiratory rate- 24 breaths/min. On day 7, she was discharged with advice of continuation of Tab. Revatio 20 mg half twice daily and Tab. Lasix 40 mg twice daily for 1 week, normal diet; restriction of heavy work load and follow-up after 1 week was advised.

Discussion:

In the case of a large ASD (>9mm), which may result in a clinically remarkable left-to-right shunt, blood will shunt from the left atrium to the right atrium. This extra blood from the left atrium may cause a volume overload of both the right atrium and the right ventricle. If untreated, this condition can result in enlargement of the right side of the heart and ultimately heart failure. Any process that increases the pressure in the left ventricle can cause worsening of the left-to-right shunt. This includes hypertension, which increases the pressure that the left ventricle has to generate in order to open the aortic valve during ventricular systole, and coronary artery disease which increases the stiffness of the left ventricle, thereby increasing the filling pressure of the left ventricle during ventricular diastole. The left-to-right shunt increases the filling pressure of the right heart (preload) and forces the right ventricle to pump out more blood than the left ventricle. This constant overloading of the right side of the heart will cause an overload of the entire pulmonary vasculature. Eventually, pulmonary hypertension may develop.

The pulmonary hypertension will cause the right ventricle to face increased afterload. The right ventricle will be forced to generate higher pressures to try to overcome the pulmonary hypertension. This may lead to right ventricular failure (dilatation and

decreased systolic function of the right ventricle).[2] Most frequently, adult patients complain of progressive shortness of breath with exertion. Studies have shown a reduction in maximum oxygen consumption in the unrepaired ASD population because of the inherent inefficiency of a continuously preload-reduced LV (Left Ventricle) in combination with a volume overload in the pulmonary circulation. A small number of adult patients also may be identified echocardiographically when a heart murmur or unrelated cardiac symptoms in the absence of exercise, rhythm, or embolic symptoms bring them to a physician's attention. Pulmonary hypertension is uncommon with ASD, even in patients with large defects, in part because of the large capacitance of the pulmonary bed. Natural history studies dating to the pre-surgical and pre-echocardiography eras suggested an incidence of ≈15% in the ASD population. The observations that pulmonary vascular disease may develop in patients with a tiny ASD and that it is absent in the vast majority of patients with large ASDs suggest that the ASD may be an associated marker of pulmonary hypertension but not necessarily causative. More recent reviews suggest a rate of 6% to 9%. Once a patient has reached adulthood with normal pulmonary arterial pressure, the natural history is established. They no longer develop significant pulmonary hypertension related to the shunt, but they may have pressure elevation, like any other patient, as a result of the development of pulmonary parenchymal disease, left-sided heart dysfunction, or obstructive sleep apnoea. It would be fair to say that the overall risk of and specific risk factors for developing pulmonary vascular disease with an ASD remain unknown.[3]

Pulmonary edema is fluid accumulation in the air spaces and parenchyma of the lungs. It leads to impaired gas exchange and may cause respiratory failure. It is due to either failure of the left ventricle of the heart to adequately remove blood from the pulmonary circulation ("cardiogenic pulmonary edema"), or an injury to the lung parenchyma or vasculature of the lung ("noncardiogenic pulmonary edema"). Treatment is focused on three aspects: firstly improving respiratory function, secondly, treating the underlying cause, and thirdly avoiding further damage to the lung. Pulmonary edema, especially in the acute setting, can lead to respiratory distress, cardiac arrest due to hypoxia, and death.[4]

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