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PREGNANCY AND THE BOOT SHAPED HEART:TETRALOGY OF FALLOT

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ABSTRACT We compare two pregnancies with mothers having uncorrected and corrected Tetralogy of Fallot(TOF), their obstetric management and respective foetal outcomes.

KEYWORDS :

INTRODUCTION

TETRALOGY OF FALLOT (TOF) IS THE MOST COMMON cyanotic congenital cardiac lesion, with an incidence of about 5% to 8% of all congenitally malformed hearts.[1] The four defects include, ventricular septal defect, stenosis of the pulmonary valve, overriding of aorta and right ventricular hypertrophy. During pregnancy the peripheral resistance decreases and thus the magnitude of shunt is inversely proportional to it, cyanosis may worsen causing various complications. Outcome is better in cases which are asymptomatic and in which surgical repair has been done.

Case Report

Case A:

A 23 year old primigravida 37 weeks gestation and uncorrected TOF with cardiac echo suggestive of a large subaortic VSD with bidirectional shunt with overriding of aorta and absent pulmonary valve, diagnosed during childhood, however the patient had no history of any cyanotic spells or any other chief complaints. Clinically on examination the patient had a systolic murmur with a saturation of 97. Foetal 2d echo suggestive of TOF with hypoplastic right heart syndrome with tricuspid atresia with aberrant right subclavian artery. Patient had an uneventful antenatal course with Elective LSCS done for obstetric indication under epidural analgesia. Though patient had an uncorrected TOF she was low risk due to the absence of pulmonary valve making pulmonary stenosis less troublesome. Moreover she had a left to right shunt with pulmonary regurgitation leading to volume overload, which becomes favourable for prognosis in TOF.

Foetal outcome:

Baby of 2.744 kg with apgar of 9/10, baby had TOF too and surgery has been planned for the same.

Case B:

A 29 year old primigravida, with 37 weeks of gestation with corrected TOF with cardiac echo suggestive of intra cardiac repair of TOF, residual moderate outflow tract obstruction, and severe pulmonary regurgitation. Clinically on examination patient had a murmur with saturation of 99%. Foetal 2d echo was normal. Patient had an uneventful antenatal course, progressed spontaneously, without any need for labour analgesia and delivered vaginally. Though she had a corrected TOF, she had higher risk (moderate) of periprocedural events due to the post-surgery right ventricular outflow tract obstruction and severe pulmonary regurgitation.

Foetal outcome:

Delivered by full term outlet forceps, a male child 3.204 kg with apgar of 9/10

DISCUSSION

Seen above are two patients one with uncorrected TOF who delivered by LSCS under epidural anaesthesia and the other with corrected TOF who delivered vaginally. Multidisciplinary team consisting of cardiologist, obstetrician and anaesthesiologist needs to be included to prevent any adverse events.

No maternal complication or deterioration was observed, neither in the women who had undergone repair, nor in the small subset that went through pregnancies prior to repair.[2] Pregnancy in patients with repaired TOF is possible but carries an increased risk of foetal loss, cardiovascular complications during pregnancy, and congenital heart disease in offspring. Pregnancy complications are related to important maternal hemodynamic disturbances (severe PHT, severe pulmonic regurgitation with RV dysfunction, and LV dysfunction). This reemphasizes the critical importance of a comprehensive pre-pregnancy evaluation. From the cardiovascular standpoint, vaginal delivery is preferred for most TOF patients. Women with any combination of the above hemodynamic disturbances may benefit from surgical repair, if feasible, before pregnancy. Neonatal outcomes may include miscarriages, need for operative intervention, risk of intra uterine growth retardation and premature deliveries.[3]

Congenital heart disease has been documented in 2 of 41 live infants.[4] Anaesthetic management of patients with TOF requires thorough understanding of anatomical defects and its physiological adaptations, and also the events and drugs that can alter the magnitude of R-L shunt[5]

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