CASE REPORT

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Hypertrophic Cardiomyopathy in Infant of Diabetic Mother

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ABSTRACT

Background: Asymmetric septal hypertrophy and hypertrophic cardiomyopathy are recognized cardiac complications reported in infants of diabetic mother. Maternal hyperglycemia especially in the third trimester of pregnancy is associated with hyperinsulinism in the fetus which results in selective organ hypertrophy among which heart is one of the organs.

Case: A large for date baby was born to mother with gestational diabetes mellitus. He had hypertrophic cardiomyopathy with low ejection fraction and had oxygen dependence without significant respiratory distress. We started him on propranolol therapy and he responded well. Now he is under regular follow up with noticeable resolution of cardiac findings in echocardiography.

Discussion: Frequently there is left ventricular outflow tract obstruction, delay in weaning of oxygen and increase in duration of hospital stay in babies of diabetic mothers. Fortunately, the condition is reversible with most cases responding well to oral medications like propranolol and recovering by six months.

Key-Words: *Asymmetric septal hypertrophy, hypertrophic cardiomyopathy, maternal hyperglycemia.*

INTRODUCTION

Hypertrophic Cardiomyopathy (HCM) accounts for 25 to 40% of all pediatric cardiomyopathy and has the highest incidence in pediatric population is reported in infants.[1] This cardiac complication in the infant of the diabetic mother has recently been named as pathological ventricular hypertrophy.[2] Maternal hyperglycemia may result in hyperinsulinemia and asymmetric septal hypertrophy, macrosomia, and hypoglycemia in infants of diabetic mothers. Hypertrophic cardiomyopathy includes thickening of one or both of the ventricular walls, hypertrophy of the interventricular septum, systolic and diastolic dysfunction, and transient hypertrophic sub-aortic stenosis is a well-recognized comorbidity in these infants.[3]

We report a neonate that was diagnosed as case of asymmetrical septal hypertrophy and hypertrophic cardiomyopathy and was born to mother with gestational diabetes mellitus who was under diet control. Baby was started on propranolol because we were unable to stop supplemental oxygen even when baby did not have other signs of respiratory distress, and the oxygen requirement decreased after 3 days of its initiation and baby was off oxygen on day 6 of propranolol therapy.

CASE

A large-for-date, 4400 grams male baby born at term gestation (38+4 weeks) to second gravida mother with history of gestational diabetes mellitus was admitted to our newborn observation unit for monitoring of hypoglycemia. Baby was born through meconium-stained amniotic fluid, was vigorous at birth and had APGAR score of 8/10 and 9/10 at 1 and 5 minutes of life. Baby had hairy pinna. Baby received Vitamin K at birth. The prenatal ultrasound was only significant for a large for gestational age fetus without any structural cardiac anomalies. He developed hypoglycemia at 1st hour of life with RBS value of 26 mg/dl. Hypoglycemia was not corrected by oral feeds so he was started on dextrose infusion.

Tachypnea soon after birth which developed to respiratory distress in the form of tachypnea, subcoastal retractions, cyanosis and nasal flaring at 2 hours of life. We started oxygen via indigenous bubble CPAP (PEEP: 5 cm of water). X-ray was done which revealed hyperinflation and no significant infiltrates in lung fields.

We shifted the baby to NICU and managed him as a case of meconium aspiration pneumonitis with hypoglycemia. Hypoglycemia was corrected after starting Glucose infusion rate at 6 mg/kg/min. we started him on first line antibiotics- Injection

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Ampicillin-Cloxacillin and Gentamycin. Laboratory reports and x-ray chest at admission are attached. Feeding was started via orogastric tube. Out of total fluid intake, 50% was given as feeds and remaining 50% as fluids. Formula feeds were given till availability of expressed breast milk. Glucose infusion was stopped next day and baby was kept under full volume feeds. Oxygen was continued via bubble CPAP. ECG showing septal hypertrophy pattern (Fig 1) and echocardiography were done on second day of life. Echo showed a hemodynamically significant PDA (2.3mm) along with asymmetric septal hypertrophy (Fig 2A). Echo findings are mentioned in the index. Electrocardiography showed T-wave inversion in V3-5 chest leads.

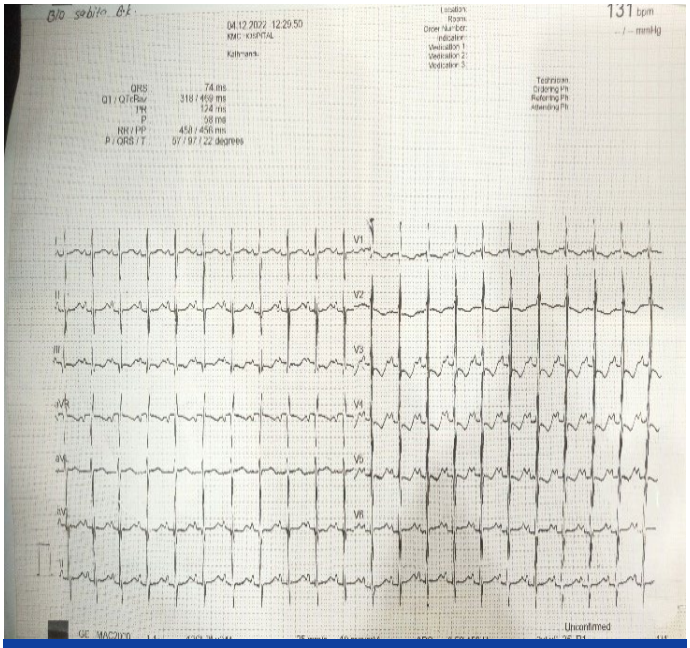


Fig 1: ECG showing septal hypertrophy pattern

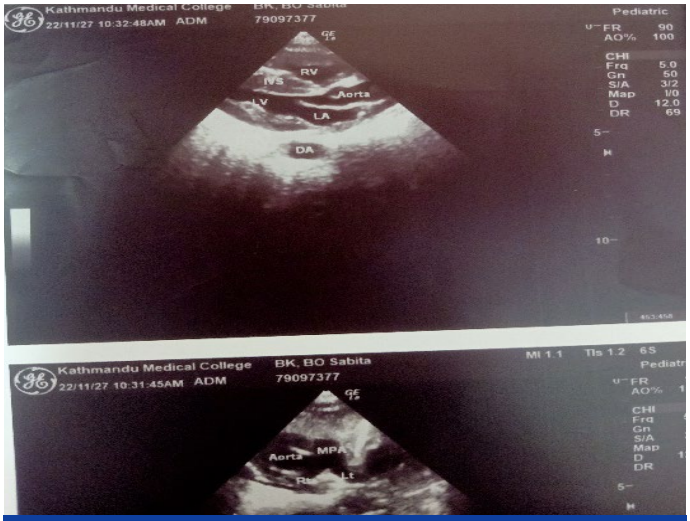


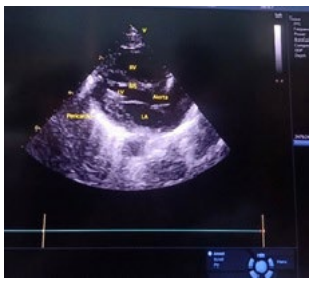
Fig 2: Echocardiography image on 2nd day of life:

Parasternal long axis view showing interventricular septal hypertrophy

Restrictive fluid approach was initiated along with use of diuretics. CK-MB level was within normal limits and Troponin I was negative. By 4th day of life breastfeeding was established and oxygen was tapered off to 1 L/min via nasal prongs. Blood culture showed no growth. There were no signs of respiratory distress and repeat chest x-ray was also within normal limits however, we were unable to stop supplemental oxygen till 6th day of life because of persistent fall in oxygen saturation below 80% in room air. Tab. Propranolol at 1mg/kg/dose, PO TDS and was started and by 12th day of life baby was able to maintain oxygen saturation in room air.

Baby was discharged on 12th day of life on same dose of propranolol and supplemental vitamin D 400IU per day. (Table 2)

Repeat echocardiography showed a slight decrease in interventricular septal diameter and left ventricular posterior wall diameter with increase in ejection fraction images of echocardiography on follow up after 2 weeks. And by day 26 of life (Fig 2B) and lastly four months (Fig2C) findings were consistent with beginning of regression of septal hypertrophy to complete disappearance, baby remaining healthy. (Table 2)



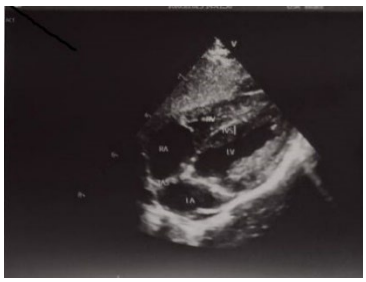


Fig 2B: Echocardiography (26 days of life) Beginning of regression of septal hypertrophy.

Fig 2C: Echocardiography at 4 months Complete resolution of asymmetric septal hypertrophy.

Table 1: Investigation findings at admission

Investigations/ Date	At admission	5th day of admission	10th day of admission
Blood group	O positive		
Hemoglobin/PCV	20.3/63.3		
Total count	26000/mm3		
Differential count	`Neutro-phils:75% Lymphocytes 22%		
platelets	150000/mm3		
CRP	3.1mg/L		
Ionized calcium	1.1 mmol/L		
Blood C/S	Sterile after 72 hours of incubation		
RBS	26mg/dl		
Urine RE/ME Urine culture		Pus cells: 18-20/HPF Epithelial cells: 3-5/HPF No growth	
Urea			50mg/dl
creatinine			0.4 mg/dl
CKMB			23U/dl
Troponin I			negative

Table 2: Echocardiography findings

	On 2nd day of life (DOL)	After 2 weeks follow up (26th DOL)	At 4 months follow up
1	Patent ductus arteriosus: 2.3 mm with left to right shunt LA/Ao ratio: 1.48 Mitral valve E/A ratio: 0.74	PDA closed (Note: Inj Paracetamol was used during NICU stay for PDA closure)	No PDA
2.	Asymmetric septal hypertrophy with biventricular hypertrophy • Interventricular septum diameter, diastolic (IVSDd): 0.36cm • IVSs: 0.28cm • LVIDd: 1.46cm • LVPWDd: 0.53cm • LVPWDs: 0.45 cm • End diastolic volume: 5.64ml • End systolic volume: 2.9 ml • Ejection fraction: 48.6% • Fractional shortening: 22.2% • Stroke volume: 2.74 ml	Asymmetric septal hypertrophy with biventricular hypertrophy • Interventricular septum diameter, diastolic (IVSDd): 0.3cm • IVSs: 0.3cm • LVIDd: 1.8 cm • LVPWDd: 0.4cm • LVPWDs: 0.4cm • End diastolic volume: 9.0ml • End systolic volume: 4.0ml • Ejection fraction: 56% • Fractional shortening: 27% • Stroke volume: 5 ml	No septal hypertrophy IVSs: 0.5 cm IVSd: 0.6cm LVIDd: 2.2cm LVIDs: 1.5cm LVEF: 63% LVSF: 32%

DISCUSSION

In healthy pregnancies, there is little fetal gluconeogenesis, and the fetus is dependent on glucose supply from the maternal circulation. Glucose is transported across the placenta by facilitated diffusion, and net transplacental transfer is dependent on the maternal–fetal concentration gradient.

Fetal hyperinsulinemia in response to maternal hyperglycemia has been implicated as the cause of HCM in infants of the diabetic mothers. In our report we haven't estimated the serum insulin level but assumed the baby to be in hyperinsulinemic state based on history of gestational diabetes mellitus in mother and examination findings in baby: large for date baby with normal head circumference, hairy pinna and hypoglycemia soon after birth.

Poor maternal glycemic control is associated with neonatal hyperinsulinemia. Hyperinsulinemia increases the total body weight and causes selective organomegaly because of hypertrophy of the insulin-sensitive tissues, including the heart and increased expression and affinity of insulin receptors. There is biventricular hypertrophy that may occasionally be associated with left ventricular outflow tract (LVOT) obstruction because of marked interventricular septal hypertrophy. Spontaneous regression of hypertrophy usually occurs as plasma insulin concentrations normalize within the first few months of life.[3]

Typical management of an infant of diabetic mother with LVOT obstruction and low cardiac output includes maintaining adequate intravascular volume and β-adrenergic blockade[9]. B-blockers reduce the heart rate and myocardial contractility thereby decreasing the myocardial oxygen demand, improving coronary perfusion and LVOT obstruction, if present.[4]

However, monitoring for hypoglycemia and bradycardia is required. In our patient we did not observe such adverse events

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