Hypoglycemia and supraventricular tachycardia in infant of diabetic mother- A possible association.

Sarah Paul   Senthil S
PSG INSTITUTE OF MEDICAL SCIENCE & RESEARCH, COIMBATORE

Abstract:
3 to 10 % of all pregnancies are complicated by diabetes, 90% of which is contributed by gestational diabetes. Infants born to diabetic mothers with poor glycemic control are associated with high morbidity and mortality. Hypoglycemia is the common manifestation in such infants, with an incidence of 30-40 % in the immediate postnatal period 1,2.

We describe a macrosomic IDM with severe hypoglycemia presenting with supraventricular tachycardia (SVT) in the immediate postnatal period, suggesting a possible association.

Case report
This late preterm (35wks+5days)/4360g (macrosomic) neonate was delivered by vacuum delivery to a 24 year old multiparous woman with gestational diabetes and a known case of chronic hypertension. Her blood sugar was well controlled with insulin throughout the pregnancy. Her blood pressure was well controlled throughout the pregnancy.

About 10 % of infants of diabetic mothers show evidence of cardiac dysfunction, Atrioventricular block has been documented in infants of diabetic mothers (IDM) 3. Reactive hypoglycemia has been reported to trigger atrioventricular nodal reentry tachycardia in an adult 4.
Baby had a apgar score of 8/10, 9/10 at 1 and 5 min respectively. Prenatal ultrasound showed adequate liquor with no evidence of hydrops. Fetal heart rate had been normal on prenatal visits and during active labour.

Baby was admitted in NICU with minimal oxygen support in view of respiratory distress soon after birth.

At thirty minutes of age except for the respiratory distress, examination of baby is normal.

At one hour of life, neonate was maintaining SpO2 97% in room air with stable vitals. Heart rate (HR) was 150/min, Respiratory rate (RR) was 53/min, and capillary blood glucose (CBG) was 30 mg/dl for which iv dextrose bolus was given followed by a continuous infusion of 10% dextrose.

At 2 hours of life, baby heart rate increased markedly to 284/min with a BP of 86/38mmHg in right upper limb and Capillary blood glucose revealed hypoglycemia (cbg-30 mg/dl). The baby was hemodynamically stable and the clinical examination was normal. Monitor ECG was suggestive of SVT—narrow QRS complex, absent p wave & regular RR interval (Fig1). A rhythm strip was taken and this confirmed SVT.

Two doses of adenosine were required to revert SVT to normal sinus rhythm. At 3 hours of life only stable euglycemia was established in the baby. Then after there was no recurrence of SVT. Sinus rhythm was established with adenosine and after institution of stable euglycemia. Chest X-ray ruled out cardiomegaly and signs of pulmonary edema. Serum electrolytes were within normal limit. Septic screen showed a raised CRP level and intravenous Cefotaxime was started. Initial CBC showed a WBC of 28,800, hematocrit of 54 and platelets of 77,000. Repeat CRP was negative and blood culture was sterile. Repeat platelet count was normal.

Echocardiogram done at 12 hours of life revealed 2.9mm PDA, 4.6mm ASD. This structural cardiac defects were probably not significant because it was done in the immediate newborn period. ECG showed no evidence of WPW syndrome. Baby was started on propanolol prophylaxis. The neonate tolerated feed advancements. During the NICU stay there was no recurrence of SVT. As the baby was icteric at 72 hours of life, phototherapy was started. There were no rhythm disturbances during follow up and hence Propranolol was withdrawn by 6 months of age. Follow up ECHO showed structurally normal heart.
Discussion:

Infants of diabetic mother are more prone for periconceptional, fetal, neonatal and long term complications. 30-40% of IDMs experience hypoglycemia in the immediate neonatal period. Arrhythmias like congenital AV heart block have been reported in IDM. Pro-arrhythmic effects of reactive hypoglycemia have been described in adults. SVT’s are produced mostly by electrical instability and dysfunction of cardiac conduction system in the early phase of ontogenesis.

Teuscher A et al, described resolution of fetal tachycardia (HR of 200/min) with maternal propranolol during the last 20 days of the diabetic pregnancy. In the postpartum period, the newborn developed SVT, which was controlled with propranolol. Chelliah YR described the probable effect of hypoglycemia on the heart leading to ventricular tachycardia, which, promptly resolved with correction of hypoglycemia.

The incidence of SVT in newborns ranges from 1 in 250 to 1 in 1000 neonates. The most common symptomatic arrhythmia in neonate is supraventricular tachycardia (SVT). The main mechanism of SVT in neonates is re-entry phenomenon based on accessory pathways atrioventricular re-entry tachycardia (AVRT). These tachyarrhythmias appear in preexcitation of ventricles like in Wolf-Parkinson-White syndrome (WPW), Lown-Ganong-Levin syndrome (LGL), Mahaim syndrome or other. WPW is noticed in majority cases of AVRT in newborns.

Symptoms of the arrhythmia are nonspecific like paleness, cyanosis, anxiety, breath disturbances, lack of appetite, vomiting. Atrioventricular re-entry tachycardia (AVRT) and Atrioventricular nodal re-entry tachycardia (AVNTR) are the most common type of SVT. Predisposing factors like congenital heart disease, post cardiac surgeries, drugs, cardiac tumors account for 15% of cases. SVT can also occur in children with normal cardiac morphology.

In this case report, we highlight the occurrence of SVT in an infant of diabetic mother with simultaneous hypoglycemia. Sinus rhythm was established with adenosine and after institution of stable euglycemia. Other causes like WPW syndrome, electrolyte disturbances, neonatal sepsis and fetal arrhythmias are ruled out. The structural cardiac defects were probably not significant in our case because it was done in the immediate newborn period. In this case scenario we would like to correlate the association of hypoglycemia and SVT in an infant of diabetic mother.

Hypoglycemia could induce arrhythmias by the following mechanisms:

1) Hypoglycemia induced sympathetic activation may increase ectopic activity.

2) Hypoglycemia related fall in Potassium may precipitate cardiac arrhythmias.

3) Myocardial tissue glucopenia may lead to arrhythmias.
4) Hypoglycemia related prolonged QTc and ventricular arrhythmias in adults (Dead in bed syndrome).

Conclusion

1. It is important to recognize the possibility of SVT being precipitated by severe hypoglycemia in the immediate newborn period of IDM and prevent it by early screening and establishing early prompt euglycemia.

2. SVT is frequently complicated by congestive heart failure in newborns.

3. Standard ECG and Holter monitoring are useful in the precise diagnosis of SVT type in the majority of newborns.

4. Propranolol is useful and effective in termination of SVT in selected patients.

References:


