Hypoglycemia And Supraventricular Tachycardia In An Infant Of Diabetic Mother: A Possible Association.

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Citation

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Abstract

3 to 10 % of all pregnancies are associated with some degree of glucose dysregulation. 90% of those are related to gestational diabetes.[1] Infant born to diabetic mothers with poor glycemic control, have greater associated morbidity and mortality. Hypoglycemia is the common manifestation in such infants, with an incidence of up to 50 % in the immediate postnatal period.[2,3] Atrioventricular block has been documented in infants of diabetic mother (IDM).[4] Reactive hypoglycemia has been reported to trigger atrioventricular nodal reentry tachycardia in an adult.[5] We describe a macrosomic IDM, who developed severe hypoglycemia and simultaneous supraventricular tachycardia (SVT) in the immediate postnatal period, suggesting a possible association and biological plausibility. Sinus rhythm was established with adenosine and after institution of stable euglycemia.

INTRODUCTION

3 to 10 % of all pregnancies are associated with some degree of glucose dysregulation. 90% of those are related to gestational diabetes.[1] Infant born to diabetic mothers with poor glycemic control, have greater associated morbidity and mortality. Hypoglycemia is the common manifestation in such infants, with an incidence of up to 50 % in the immediate postnatal period.[2,3]

Atrioventricular block has been documented in infants of diabetic mother (IDM).[4] Reactive hypoglycemia has been reported to trigger atrioventricular nodal reentry tachycardia in an adult.[5] We describe a macrosomic IDM, who developed severe hypoglycemia and simultaneous supraventricular tachycardia (SVT) in the immediate postnatal period, suggesting a possible association and biological plausibility. Sinus rhythm was established with adenosine and after institution of stable euglycemia.

CASE REPORT

25-year-old G2P1 poorly controlled gestational diabetic mother (Class A2) delivered a macrosomic (Birth weight: 5050g) female at 37 and 4/7 weeks by emergency Caesarean section in view of a non-reassuring fetal tracing and Biophysical profile of 2. Apgar scores were 8 and 9 at 1 and 5 min respectively. Baby received blow-by oxygen until 2 minutes of age. The mother's Group B Streptococcal vaginal culture was positive (AROM at delivery), she was B positive, rubella immune, HbsAg negative, RPR Nonreactive and HIV negative. Antenatal medications included Glyburide 2.5 mg twice daily and prenatal vitamins. Prenatal ultrasound showed no evidence of hydrops with normal Amniotic Fluid Index. Past obstetric and medical histories were unremarkable. She denied tobacco, caffeine, or recreational drug use. Fetal heart rate had been normal on prenatal visits.

At 21 min of age, the newborn had a normal exam with Heart rate (HR) of 150/min, Respiratory rate (RR) of 53/min, SaO2 97% in room air and blood glucose of 19 mg/dl (Accucheck Glucometer, Rosche, Germany). Following a 50ml formula feed, the blood glucose at 31 min of age was 18 mg/dl.

At 1 hr of age, during a peripheral intravenous line placement, the HR suddenly increased to 258/min with BP=66/36mmHg (Mean=45mmHg) and a simultaneous blood glucose level of 27 mg/dl. On examination, the baby was diaphoretic but hemodynamically stable. Rest of the clinical exam was normal. 12 lead EKG was suggestive of SVT (Fig1).

Umbilical lines were placed for central venous access and vagal maneuvers viz, application of ice to the face and rectal stimulation, did not convert the rhythm to normal sinus. The umbilical venous/arterial catheters (UVC) and UAC were secured at 13cm and 22 cm at the umbilicus respectively.

Adenosine (0.1 mg/kg) was administered using the 'double push' method followed by D10W bolus of 2ml/kg through the UVC. D10 W at rate of 100ml/kg/day (glucose delivery rate = 6.8mg/kg/min) was started. Blood glucose level following the initial D10 W bolus was 69mg/dl. The first adenosine push resulted in the HR decreasing transiently to the 120s for about 30 min and rising back to 270/min. Chest X-ray showed no cardiomegaly or signs of RDS/pulmonary edema. The UAC was seen to be curling into the left common iliac artery and was removed. The initial arterial blood gas showed ph =7.31, PCO2=31.5, PaO2=108, BE=-9.3. The electrolytes were Na=141, K=3.3.

The second dose of Adenosine, at 0.2 mg/kg was administered at 2 hr 40 min of age. The HR decreased to the 80s transiently and increased to the 190s for about 15 min before reaching the 260s again. The blood glucose level had decreased again to 25mg/dl necessitating D10W bolus with subsequent escalation of glucose delivery rate to 8.4 mg/kg/min. Sinus rhythm was established at 3 hrs of age, around the same time when stable euglycemia was reached and established (Fig2). Ampicillin (100mg/kg/dose) IV Q12 hourly and Gentamicin 4 mg/kg/dose IV Q24hourly were started. Initial CBC showed a WBC of 22.6, hematocrit of 54, platelets of 118 with no shift to the left. The neonate was transported to the Level III NICU (Las Palmas Medical Center, El Paso, TX) for further management.

Echocardiogram revealed a structurally normal heart and EKG showed no evidence of WPW syndrome. The neonate tolerated feed advancements and maintained euglycemia during the rest of the stay in the NICU with no further recurrence of SVT. Loading dose of Digoxin was administered on day 2 and baby was discharged on maintenance digoxin on Day 5. Digoxin was weaned by 6 months of age in view of no subsequent recurrence of SVT.

Figure 1

Fig1: EKG showing SVT

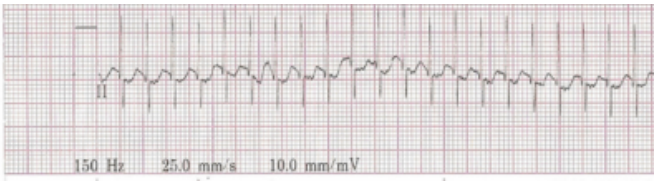
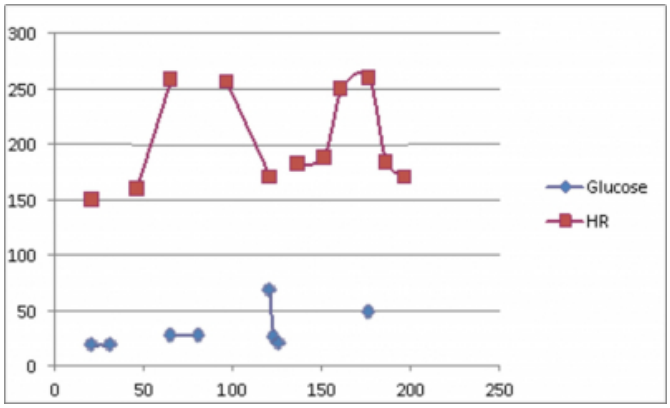


Figure 2

Fig 2: Graph showing blood glucose (mg/dl) and Heart rate (HR) over time (min)



DISCUSSION

IDMs are at increased risk for periconceptional, fetal, neonatal and long term complications.[1, 2] Their etiologies are multifactorial; however, many of them could be traced to the effect of maternal glucose control. Up to 50% of IDMs experience hypoglycemia in the immediate neonatal period [1, 3]

Arrhythmias like congenital AV heart block have been reported in IDM.[4] White SE et al, reported a hemodynamically unstable SVT associated with perioperative hypoglycemia in an infant with cyanotic heart disease, with pre-existing first degree heart block. The simultaneous blood glucose level during SVT was 28 mg/dl. Within an hour of administration of Dextrose 50% bolus and digoxin (2 doses of 2 mg/kg over 20 min), sinus rhythm was established.[6]

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.[7]

Pro-arrhythmic effects of reactive hypoglycemia have been described in adults.[5] Chelliah YR described the possible effect of hypoglycemia on the heart leading to ventricular tachycardia, which, promptly resolved with correction of hypoglycemia.[8] An association between hypoglycemia and atrial flutter has been described in diabetic patients.[9] Intensive insulin therapy in diabetics has an inherent risk of hypoglycemia that can lead to loss of consciousness, cardiac arrhythmia, seizure, and death (dead in bed syndrome). [10]

Data from intact mouse heart studies suggest a link between

impaired myocardial insulin signaling and the increased risk of arrhythmia and sudden death in patients with diabetes.[11] Role of hyperinsulinism in precipitating arrhythmias in IDM is unknown.

Supraventricular tachycardia (SVT) is the most common sustained arrhythmia to present in the neonatal period with a prevalence of 1 in 250.[12] Predisposing factors (congenital heart disease, drug administration, illness and fever) occur only in 15% of cases.[13] In our report, we would like to highlight the occurrence of SVT in an IDM within the first hour of birth with simultaneous onset of severe hypoglycemia. There was no history of fetal arrhythmia, structural congenital heart disease, neonatal sepsis, electrolyte disturbances or WPW Syndrome. SVT occurred at 1 hr of age requiring two boluses each of adenosine and D10. Stable sinus rhythm was restored only after stable euglycemia was established by 3 hrs of age. We would like to highlight the plausible pro-arrhythmic nature of hypoglycemia in view of its temporal association with the onset of SVT in IDM and the reversal to sinus rhythm with establishment of stable euglycemia.

Hypoglycemia could induce arrhythmias by the following mechanisms:

I) 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).

Role of interventricular septum glycogen loading in IDMs and arrhythmia is unknown. Intact KATP channel is mandatory for adequate myocardial repolarization under sympathetic stress, providing electrical tolerance against triggered arrhythmia. [14]Mice studies reveal that KATP channels are critical metabolic sensors in acute metabolic changes, including hypoglycemia, ischemia, and hypoxia. [15, 16]The role of hypoglycemia affecting KATP channels needs to be further studied in IDMs.

CONCLUSION

The public health problem of childhood obesity will likely result in an increased incidence of insulin resistance and type II diabetes. An early manifestation of this may be glucose intolerance during pregnancy in overweight young women. 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.

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