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LETTER TO EDITOR (VIEWERS CHOICE)

# NEONATAL MENINGITIS WITH HYPERGLYCEMIA MIMICKING NEONATAL DIABETES MELLITUS

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A preterm female baby born at 34 weeks 3 days with birth weight of 1600 g was referred to our pediatrics department for respiratory distress since birth. At the time of admission, respiratory rate was 76/min, heart rate was 120/min, oxygen saturation (PO2) on oxygen of 5lit/min was 97%. There were no chest retractions. Systemic examination was normal. Investigations revealed blood glucose levels of 46 mg/dl, white cell count of 16,300 cells/cumm (polymorphs 69%, lymphocytes 21%), platelet count of 2,48,000 cells/cumm, negative C-reactive protein (CRP) serum sodium 135 mEq/L, serum potassium 4 mEq/L, serum calcium 9.1 mg/dl, blood urea 24 mg/dl, serum creatinine 0.7 mg/dl. Urine sugar was 2+ with absent ketones. Chest X-ray was normal. Venous blood gas showed pH 7.4 and bicarbonate of 25 mEq/L. Aminophylline[1,2] was started to prevent apnea of prematurity along with intravenous (IV) antibiotics (amikacin and cefotaxime). Dextrose 10% was started at 60 ml/kg/day. Oxygen was continued. Random blood glucose (RBS) levels were repeated after 6 hours which was 800 mg/dl and repeat RBS after 1 hour was 760 mg/dl after which glucose infusion rate (GIR) was reduced from 4 to 3, repeat RBS after 2 hours was 560 mg/dl. At this point regular insulin was started at 0.05 u/kg/hr. Aminophylline was stopped. After overnight insulin infusion, blood glucose levels decreased to 71 mg/dl at which point insulin infusion was stopped. On the 2nd day of life, the neonate maintained blood glucose levels within the normal range. Fluids were continued according to the day of life and GIR was 3. On the 3rd day, oxygen was omitted, day-appropriate fluids were continued and nasogastric feeds were started. On the 4th day, blood was sent for insulin and C-peptide levels. Insulin was 1.30 mIU/ml (normal 5-5.5 mIU/ml) corresponding to the blood glucose of 102 mg/dl. C-peptide was 0.28 ng/ml (normal 1.2-3.6 ng/ml) indicating a decreased endogenous insulin secretion. Ultrasound of the pancreas and cranium were within normal limits. The baby developed neonatal hyperbilirubinemia (NNH) on 4th day of life requiring single surface phototherapy. Cerebrospinal fluid (CSF) analysis, done on 4th day of life showed raised proteins 220 mg/dl, sugar 38 mg/dl, lymphocytes 19 cells/cumm, corresponding plasma glucose was 89 mg/dl. CSF to plasma glucose ratio was 0.43. CSF culture did not grow any organism. In view of hypoglycorrachia and increased CSF proteins despite first line antibiotics, meningitis was diagnosed. Blood culture showed no growth. Meningitis was treated with intravenous vancomycin and meropenem for total 21 days. Repeat C-peptide levels were sent on the 7th (4.5 ng/ml) and 21st day (5.3 ng/ml) of life both which came out be within the normal range ruling out neonatal diabetes mellitus (DM). This child has been serially followed up in OPD for regular monitoring of C-peptide levels, RBS, anthropometry including head circumference. Child has been attaining milestones adequately and gaining weight appropriate for age.

Hyperglycemia occurs when the neonate is unable to adapt to glucose infusion by decreasing the hepatic glucose production or by increasing the peripheral uptake of glucose.[3] Causes of hyperglycemia include high rates of glucose infusion, prematurity, stress, drugs and neonatal DM.[4] Neonatal DM is of two types: transient or permanent. Transient neonatal DM presents within the first of week of life and usually recovers by 3 months of age (maximum age of recovery being 18 months). There may occur a relapse of DM at the time of puberty.[5] Permanent neonatal DM presents later than transient nonatal diabetes, usually within the first 3 months and requires insulin therapy for life.[6] Since the episode of hyperglycemia lasted for only a single day in our patient, it was unlikely to be transient DM.

Hyperglycemia can be due to counter-regulatory hormones released in response to clinical stressors such as sepsis, respiratory distress, and surgical procedures. These hormones lead to increased gluconeogenesis, glycogenolysis and insulin resistance as well as decreased insulin production.[7,8] It has been found that umbilical cord blood levels of glucagon are higher in preterm neonates.[9] In extremely preterm infants both defective proinsulin processing and relative insulin resistance has been found to be responsible for hyperglycemia.[10] The low levels of C-peptide and insulin in our case suggests a poor endogenous production of insulin initially. Aminophylline can also cause hyperglycemia by causing insulin resistance, but it would not decrease C-peptide and insulin levels.[11] Thus, we conclude that not all episodes of hyperglycemia in newborns are DM and underlying sepsis should be ruled out even if the child requires insulin infusion.

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**Compliance with Ethical Standards**

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