Case Report

Transient Hyperglycemia in Neonate: A Case Report

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Abstract:
Hyperglycemia has become a significant risk factor for morbidity and mortality as smaller and more fragile infants survive during the neonatal period. Historically, hyperglycemia has been attributed to excessive IV glucose infusion. More recent studies indicate that physiologic and biochemical mechanisms, leading to excess glucose production, insulin resistance, or glucose intolerance, underlie the development of hyperglycemia in preterm infants and sometimes inhibit efforts to maintain normal glycemia in the neonate. The sequelae of these disturbances in glucose metabolism are extensive. Efforts to prevent hyperglycemia should include general measures to improve the health of the infant and attempts to treat pathophysiologic conditions and diseases.1,3

Introduction:
Hyperglycemia in neonates is defined as serum glucose (capillary) level more than 125mg%. This may occur secondary to a variety of causes in the neonatal period. It is however important to distinguish transient diabetes mellitus of the neonate from other causes of hyperglycemia in the newborn period. Recently, we had under our care in BIRDEM Hospital Dhaka, a case of neonatal hyperglycemia which is being reported here.

Case report:
A 4 days old child, born of a full term normal delivery and weighing 2.2 kg, presented with loose motions and vomiting for 1 day prior to admission. The child also had oliguria for 12 hours prior to admission. The child was a first born, uncomplicated home delivery and had cried immediately after birth. Antenatal history was uneventful. On admission, the child was hypothermic, and in grade II dehydration with acidic breathing. Abdomen was distended with absent peristalsis, and palpable lumps. Investigations revealed haemoglobin 16gm%, total W.B.C count 4,400/cmm with polymorphs 75%, lymphocytes 10% and band forms 15%; serum sodium 160 meq/L, potassium 6.7 meq./L. and chlorides 104 meq./L. Blood sugar was 495 mg%, and blood gases were as follows: pH 6.92, HCO3 6.6 meq./L, PCO2 34.3 mm Hg, PO2 71.5 mm Hg and O2 saturation 88.4%. Urine showed the presence (++++) of reducing substances (confirmed as glucose). Ketones, albumin, RBcs and WBcs were absent in the urine. Stool examination revealed Gram negative bacilli on smear, 15-20 pus cells/HPF and RBCs were absent. Blood and stool culture grew Klebsiella organisms sensitive to amikacin. The child was treated with IV fluids to correct his dehydration, sodibicarb to combat acidosis, and ceftazidim and amikacin. Repeat blood sugar done after 4 hours was 390 mg% and 130 mg% after 12hours. Nasogastric tube was not inserted. Repeat blood gases and serum electrolytes were normal after 22 hours. The child died 30 hours after admission. Post-mortem showed perforation 2 cm x 2 cm in the anterior wall of stomach. Adrenals, pancreas and kidney showed hemorrhagic lesions suggestive of septicemia.

Discussion:
There are various causes of neonatal hyperglycemia. Transient neonatal diabetes mellitus: (TN DM ) This is a discreet clinical entity that simulates diabetes mellitus has been described in small for gestational age (SGA) babies during the first six weeks of life. Such states are also referred to as congenital neonatal pseudodiabetis, paradiabetes mellitus infantum or congenital temporary diabetes.1,2 The first patient identified as having this syndrome was the son of a physician who presented with ‘honeyed napkins’,3 polydypsia, polyphagia, polyuria, dry skin after birth and the child died at 6 months of age because of urinary tract infection.4 Characteristically, ketonuria is minimal or absent, but hyperglycemia, glycosuria and dehydration may be severe. The majority of these patients have temporary type of diabetes from which they recover, a few ultimately develop permanent juvenile diabetes mellitus.5 Transient neonatal diabetes mellitus is uncommon and the exact incidence is unknown.4 McDonald found 41 cases in world literature in 1974, 31 common age of presentation is 2 weeks with a range of 1 day to 6 weeks.7 The diagnosis is confirmed by documenting hyperglycemia which can range anywhere from 240-2300 mg%,4 glycosuria present in all cases and minimum to absent ketosis. Transient

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Serum insulin levels are either absolutely or relatively low for the corresponding blood sugar level. In permanent juvenile diabetes mellitus serum insulin are persistently low < 10 µU/ml.8,9,10,11 The exact sugar level. In permanent juvenile diabetes mellitus serum absolutely or relatively low for the corresponding blood glucose metabolism is concerned.

Hyper-adrenocorticism
Hyperglycemia may be present due to increased cortisol levels.10

Idiopathic:
In some cases the cause may not be evident even after a complete workup.12

In an older child salicylate poisoning, burns and acute pancreatitis should also be considered in the differential diagnosis of a child with hyperglycemia. At times, it becomes difficult to ascertain the cause of hyperglycemia as is illustrated by the present case. From the clinical and laboratory data in the present case, it was evident that the child had gastroenteritis with hypernatramic dehydration with pre-renal failure and septicemia. Also, the child had significant hyperglycemia. This was possibly due to hypernatremia and septicemia both of which are known to give rise to hyperglycemic states. We were obviously not dealing with transient neonatal diabetes mellitus because the child was sick, had hypernatremia and oliguria. TNDM babies are usually alert, active and otherwise well but emaciated. However, in doubtful cases serum insulin can be of value, in TNDM levels in view of the clearcut clinical picture. From the therapeutic angle such babies do not require insulin because of insulin resistance. Treatment of underlying etiology is sufficient to reduce the blood sugar level.

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References:


