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Glucose 250, MD Notified: A Case Report

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1. Abstract

Background: Hyperglycemia is common in neonates. Insulin therapy is used to treat hyperglycemia. Threshold for treating hyperglycemia varies among the neonatal practitioners. The renal threshold for glucose is 180 mg/dl, so most practitioners wait on insulin therapy till the serum glucose level reaches 180 mg/dl. We describe a case of a neonatal hyperglycemia of greater than 250 mg/dl that was successfully managed conservatively.

Clinical Findings: The neonate, who was depressed at birth, was noted to have high serum glucose levels soon after birth. Serial glucose levels within first six hours of life were persistently above 200 mg/dl. The serum cortisol was normal while serum insulin was low. Infant was provided a low rate of intravenous glucose without giving any insulin. The serum glucose declined to normal levels within 24 hours.

Primary diagnosis: Stress-induced transitional hyperglycemia.

Interventions: We performed a serial monitoring on serum glucose and provided intravenous dextrose solution at a very low rate. The glucose infusion rate used was only 2.6 mg/kg/min. We were able to bring down the serum glucose to normal ranges conservatively without using any insulin therapy.

Outcomes: Infant responded to conservative management and serum glucose was normalized within 24-hours.

Practical Recommendations: Stress-induced hyperglycemia soon after birth is transitional and could be managed conservatively without insulin therapy.

- Keywords: Hyperglycemia; Insulin; Neonate;Case report
- **3. Abbreviations:** NICU: Neonatal Intensive Care Unit; MRI: Magnetic Resonance Imaging; MD: Doctor of Medicine

4. Introduction

Neonatal hyperglycemia is defined as a plasma or serum glucose concentration greater than 150 mg/dL [1]. Early hyperglycemia has been associated with increased risk of death and brain injury in neonates [2]. Therefore, timely and adequate management of hyperglycemia is important. However, at the other end, insulin therapy should be used judiciously as neonatal hyperglycemia could be transitory [3,4]. Among the causes of hyperglycemia, stress is one of the factors [5]. The possible mechanism is the persistent glucose production and greater peripheral sensitivity to insulin [6]. Interestingly Lilien et al. [7], while studying a case series of stressed neonates, concluded that etiology of hyperglycemia cannot be attributed to hypoinsulinemia. So, the treatment with

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insulin may not be the solution to hyperglycemia. We describe a case of stress-induced hyperglycemia in a neonate that was managed conservatively.

The clinical question and medical record: Are you sure you do not want to write an order for insulin? Nurses and on-call residents were getting nervous as the infant's serum glucose levels continues to rise. The note said 'glucose 250, MD notified'.

5. Case Presentation

The infant was delivered at 38 weeks of gestation by emergent cesarean section due to fetal decelerations. At birth, no respiratory efforts were noted with heart rate of less than 60 beats per minute. Positive pressure ventilation was initiated followed by intubation. Heart rate increased to 120 bpm, however, the oxygen saturations at 10-minutes of life remained below 80. Infant looked pale on examination. There was no history of bleeding prior to delivery or observation of placental abruption. Appar score was 0, 2, 4, 4 at 1, 5, 10 and 20 minutes, respectively. Cord blood gas was 7.14/pCO₂ of 49 mm Hg (6.5 kPa/paO₂ of 31 mm Hg (4.1 kPa)/ HCO₃ of 14 mmol/L. Infant was transferred to NICU. Initial blood test showed a hemoglobin of 8 mg/dL (81 g/L). Infant was immediately transfused with packed red blood cells. No cause for bleeding was identified (infant had a normal brain MRI). Kleihauer-Betke test was not performed on the mother. Infant displayed signs of moderate encephalopathy with persistent abnormal blood gases. He was treated with therapeutic hypothermia per unit protocol.

The worries started as serial serum glucose levels continued to be way higher than normal. The team was questioning for the treatment of hyperglycemia. The resident on-call was about to order insulin when the attending physician decided to hold on. The hyperglycemia was managed conservatively. The serial values are displayed in the figure. Infant's serum insulin and cortisol level that were obtained at 8 hours of life were 1.8 mclU/mL (range 2.6-24) and $6.44~\mu g/dL$ (range 4.46-22.7), respectively. He was

extubated on day 4 and was started on oral feedings which was tolerated well. He continued to have a normal neurological examination and was discharge home in a stable condition with close planned follow up.

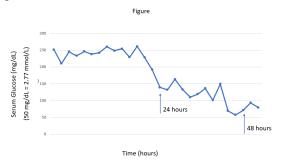


Figure 1: Graph showing the serial serum glucose levels in first 48 hours of life.

6. Discussion

The causes of neonatal hyperglycemia are iatrogenic, inability to utilize glucose, sepsis, stress and/or diabetes [8]. As insulin does not cross placenta in early gestation, insulin in neonate is all intrinsic [9]. Therefore, insulin secretion is tightly controlled by neonatal glucose levels. The most likely cause for hyperglycemia in the infant was stress at birth. Iatrogenic cause was less likely as infant was receiving intravenous glucose at a rate of only 2.6 mg/kg/min. Interestingly, we noted a normal cortisol level with concomitant high glucose level, while insulin level was low. Although we expected high cortisol level with stress, we found it within the normal range. We postulated that could be due to the blunted response secondary to transient adrenal insufficiency. Now the question is: why the infant had low serum insulin at 8-hours of life? At birth, the placental supply of glucose is cut-off resulting in active glycogenolysis and gluconeogenesis mediated by the epinephrine and glucagon [10,11]. In response to stress both these hormones are excreted producing hyperglycemia which then have a negative feedback effect on insulin secretion explaining the low insulin level observed in our case.

In conclusion, neonatal hyperglycemia in early hours of life is transitory secondary to hormonal changes at birth. It should be managed conservatively. Low serum insulin in stressed neonate is a secondary response to hyperglycemia rather than a primary deficiency. Insulin treatment should be viewed critically.

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