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Urosepsis with Stress Hyperglycemia – An Interesting Case Report

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Abstract

Hyperglycemia is relatively common metabolic abnormality in critically ill hospitalized children and it usually occurs as a stress response in children with previously normal glucose homeostasis. This so called stress hyperglycemia occurs primarily due to increased sympathomimetic activity, release of counter regulatory hormones and relative insulin resistance ultimately causing decreased cellular uptake of glucose promoting gluco-neogenesis and glycogenolysis. We hereby present an interesting case report of a 20 month old child presented with urinary tract infection with sepsis and stress hyperglycemia in which hyperglycemia resolved in few days without treatment with insulin.

Keywords: Blood glucose, non diabetic, stress hyperglycemia.

Introduction

Hyperglycemia is relatively common metabolic abnormality in critically ill hospitalized children and it usually occurs as a stress response in children with previously normal glucose homeostasis[1]. In diabetic children, hyperglycemia is defined as random blood glucose >200mg/dl or fasting blood glucose >126mg/dl or 2 hour post-prandial glucose >200mg/dl [2]. No clear cut-off has been defined for hyperglycemia in critically ill non diabetic children though most consider random blood glucose >150mg/dl[3,4,5] as stress hyperglycemia. This stress hyperglycemia is seen usually in critical children with diseases such as septic shock, trauma, post cardiac surgery and burns apart from other critical illnesses[1]. Uncontrolled stress hyperglycemia can cause fluid and electrolyte disturbance and also increased risk of infection[6,7]. It is shown to be associated with poor prognosis in critically ill children in some studies. We report this case of stress hyperglycemia in a child with urinary tract infection with sepsis in whom hyperglycemia resolved without insulin.

Case report

A 20 months old female child presented with fever since 15 days, which was high grade, continuous and associated with chills and rigors. Child had abdominal distension since 5 days before admission which was followed by 10-12 episodes of watery diarrhea and vomiting (1-2 episodes) a day before presentation. Child was noticed to be irritable and drowsy on the day of admission. There was no history of similar episodes in past and family history was negative for diabetes mellitus.

On examination the child was febrile, semi conscious (GCS-9/15), pale, tachypneic, HR-125/min, RR-45/min (no chest wall in drawing), SpO2-92 % at room air (99% with oxygen by nasal cannula at 2 l/min), BP-70/40mmHg right arm supine, blood sugar-246 mg % (dextrostix). P/A-child had liver palpable 3cm below right costal margin in mid clavicular line (liver span -8 cm), CNS- examination revealed a stupurous child with bilateral normal and equally reacting pupils, with no cranial nerve involvement/ meningeal signs, deep tendon reflexes were sluggish and plantars were equivocal. Investigations revealed (table-1) Hemoglobin – 6.8 gm%, Total leukocyte count-28,900 cells/mm^3, RBS (lab) - 258mg/dl, C-Reactive Protein-129.6 mg/dl. Urine ketones were negative. Serum electrolytes, serum creatinine and liver function tests (liver enzymes, bilirubin, albumin and PT/APTT) were normal. Urine analysis revealed 30-40 pus cells/mm^3. Thyroid profile was normal. Arterial Blood Gas-revealed metabolic acidosis with respiratory compensation. CSF analysis including CSF sugar was normal. After sending blood and urine for culture, child was started on empirical antibiotic therapy and Intra Venous fluids (normal saline). Child had persistently high blood sugar levels (fasting and random) in spite of appropriate non dextrose containing fluids. The blood glucose levels were monitored and it returned to normal in three days. Insulin was not given as hyperglycemia was not associated with ketonuria / ketonemia (Criteria for diabetes mellitus). Urine culture and blood culture were sterile (child was treated with antibiotics elsewhere). Blood transfusion was given (packed red cells) in view of anemia (Iron deficiency anemia).Child gained consciousness gradually with this management and was started on appropriate naso-gastric feeds followed by oral feeds. Child recovered fully without any sequel and her blood sugars were normal on follow up (table-2)

Table-1
Diagnostic findings in the case on different days

Investigation	Day 1	Day 2	Day 3	Day 6
Hemoglobin (gm %)	6.8	9.0	9.2	9.4
Total WBC count (/cubic mm)	28,900	15,800	13,400	12,900
Differential WBC Count (%)	N75 L22 M2 E1	N80 L16 M2 E2	N70 L26 M2 E2	N66 M27 M4 E3
C-Reactive Protein (iu/ml)	129.6	185.5	80.4`	3.5
S.Bilirubin (mg/dl) (Total/direct/indirect)	1.22/1.18/0.04	-	-	-
SGPT/SGOT (units/l)	45/40	-	-	-
Urine analysis (pus cell/hpf)	30-40	7-8	4-6	-
Urine dipstix ketones/sugar	nil/1+	nil/1+	nil/1+	-
Serum electrolytes (sodium/potassium)	138.3/3.39	139/2.76	134/3.77	-

*Other investigations including CPKMB, Serum amylase, Serum lipase, Glycosylated hemoglobin were normal. Blood, urine and CSF cultures were not able to isolate any organisms.

Table-2
Serum glucose levels measured every 4 hourly during first 3 days (mg %)

Time	Day 1	Day 2	Day 3	Day 4
6AM	-	264	305	98
10AM	-	226	418	163
2PM	-	212	233	124
6PM	-	182	111	107
10PM	-	144	113	102
2AM	258(admission)	219	160	74

Discussion

Stress hyperglycemia is relatively common in critically ill children. This is mediated by increased sympathomimetic activity and elevated levels of counter regulatory hormones (epinephrine, glucagon, cortisol and growth hormone) coupled with elevated pro-inflammatory cytokines [tumor necrosis factor-α, interleukin-1, and interleukin-6[8-10]. Hyperglycemia is thought to be an adaptive response where in the body adapts to stress to improve survival but severe persistent hyperglycemic responses to stress are unusual[11-13]. This stress hyperglycemia is seen usually in critical children with diseases such as septic shock, trauma, post cardiac surgery and burns apart from other critical illnesses[1]. In non diabetic critically ill patients, this resolves within few days without treatment with insulin. However as children with hyperglycemia are prone to develop insulin resistance (secondary to increased counter regulatory hormones), metabolic syndrome (obesity, hypertension, hyperlipidemia and hyperglycemia) and type 1 diabetes mellitus later in life[14,15] their follow up is essential. The hospital mortality increases with higher peak glucose levels and its duration[1]. The child admitted with us with stress hyperglycemia did respond well to timely intervention and adequate supportive care without the use of insulin.

Conclusion

Even though stress hyperglycemia is transient in most children, follow up of these children is needed to detect covert diabetes mellitus or insulin resistance**.**

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