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Case Report

Growth hormone deficiency and hypoglycemia: a case report

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ABSTRACT

Recurrent hypoglycemia in childhood may be caused by various endocrine or metabolic disorders, of which growth hormone deficiency is a rare cause. A 5-year-old girl presented to us with short stature and recurrent episodes of hypoglycemic seizures. On evaluating for the same, critical sample showed normal cortisol and low c-peptide levels. Neuroimaging ruled out pituitary anomalies. Growth hormone deficiency was diagnosed after GH stimulation test (with clonidine and insulin) revealed low basal and post-stimulation values. Following GH supplementation, no further hypoglycemia was noted. This rare case is being reported to highlight the importance of treating the underlying etiology of hypoglycemia to prevent adverse neurological outcome.

Keywords: Growth hormone, Hypoglycaemia, Short stature, Hypopituitarism

INTRODUCTION

Glucose is the core substrate for cerebral functioning. Hypoglycemia in children is defined as a whole blood glucose value of less than 55 mg/dL.[1] Recurrent hypoglycemia in childhood is associated with a dangerous possibility of cognitive and neuro-psychologic sequelae.[2] While transient, asymptomatic hypoglycemia may not lead to severe sequelae, repeated episodes during the period of rapid brain growth in early childhood can retard neurodevelopment and function.[3] The possible causes for recurrent childhood hypoglycemia include hyperinsulinism, ketotic hypoglycemia, glycogen storage disorders, disorders of gluconeogenesis, glucose transporter defects, endocrine etiology: panhypopituitarism, isolated Adreno-corticotropic Hormone deficiency or rarely Growth Hormone (GH) deficiency.[4-6] Quarter to half of the cases with recurrent symptomatic hypoglycemia in early infancy may have permanent sequelae.[1]

CASE REPORT

A 5-year-old girl, 2nd born to a non-consanguineously married couple presented with history of multiple episodes of hypoglycemia. With a birth-weight of 2.5 kg, there was no significant antenatal, birth or family history suggestive of any metabolic disorders. At 2 years 8 months, she had one episode of hypoglycemic seizures which subsided with intravenous dextrose. At 3 years of age, she developed another episode of generalized tonic-clonic seizures at a plasma glucose level of 45 mg/dL and she was found to have short stature with a height of 84 cm (<-3SD). The renal functions, thyroid function tests, electrolytes and complete hemogram reports were normal, urine ketone bodies were negative. 6 months later, child had another episode of hypoglycemic seizures with a plasma glucose level of 29 mg/dL. None of these hypoglycemic episodes had any history of fasting, associated illness or drug intake.

At 5 years of age, child presented to us for not gaining height with a significant past history of recurrent hypoglycemic seizures. Her fasting and post prandial blood sugars were 87 and 69 mg/dL respectively, urine sugars were absent and glycosylated hemoglobin was 5.94%. GH stimulation test done using Clonidine (5 µg/kg) revealed low levels: Basal and post-stimulation levels at 30 min, 60 min, 90 min (ng/mL) were 0.11, 0.24, 5.44 and 6.24 respectively. Critical sample (plasma glucose: 39 mg/dL) showed normal cortisol (7.67 µg/dL), low beta- hydroxybutyrate (0.34 mmol/L) and appropriately low c-peptide level of 0.15 ng/mL. Second GH stimulation test done using insulin showed similar results, hence confirming GH deficiency. Magnetic resonance imaging of the brain did not show any evidence of pituitary abnormalities. After starting GH replacement, child did not develop any further episodes of hypoglycemia and height improved from 99 cm at 5 years to 104 cm at 6 years.

DISCUSSION

Evaluation and treatment of hypoglycemia in children differs from that of adults. Clinical hypoglycemia as defined by the Pediatric endocrinology society is a plasma glucose value that is low enough to result in signs and symptoms of brain function impairment.[4] GH deficiency presenting with hypoglycemia, although rare, is significant as adverse sequelae can be prevented with treatment. Though recognition of hypoglycemia in pediatric age group can be challenging due to the non-specific clinical presentation, it is important to recognize it and treat at the earliest. The impact of GH deficiency on the neurodevelopmental outcome in children may be prevented with early detection and treatment, but the possibility of reversal of effect with treatment is as yet unknown.[7]

In the case report of an 18-month-old male child by Boro H et al, while the child was diagnosed to have hypoglycemia secondary to GH deficiency, there was no associated short stature. Similar to our case, midline defects were absent and there was no significant contributory history, Whipple's triad for the confirmation of hypoglycemia was fulfilled-signs and symptoms consistent with hypoglycemia, low documented plasma glucose and reversal of signs and symptoms with normalization of plasma glucose value.[5] Hypoglycemic seizures due to reduced GH can be overcome with GH replacement and the short stature due to GH deficiency can be improved with early replacement therapy.[8]

The stored glycogen reserve of the brain lasts only for a short duration of minutes after the plasma glucose levels drop, hence, prolonged severe hypoglycemia can cause permanent neurological injury.[3,4] Among the neuro-endocrine protective mechanisms against hypoglycemia, the role of growth hormone and cortisol in maintaining plasma glucose is important once the level falls below 65 mg/dL.[4,9] Repeated episodes of hypoglycemia can lead to hypoglycemia-associated autonomic failure, which in turn escalates subsequent risk of hypoglycemia and may result in hypoglycemic unawareness. Hence, targeted treatment to maintain plasma glucose values above 70 mg/dL and avoid activating the neuroendocrine defenses is necessary.

CONCLUSION

This case highlights the importance of targeted treatment of the underlying etiology of hypoglycemia to prevent adverse neurological outcome. Early detection of GH deficiency and supplementation of GH can control the hypoglycemic seizures as well as improve the final height attained.

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