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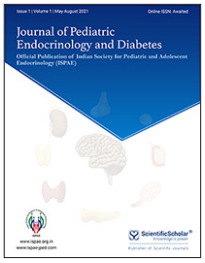

Case Report

Postprandial hypoinsulinemic hypoglycemia as a clue to hidden purging behavior in an adolescent boy with disordered eating

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ABSTRACT

Postprandial hypoglycemia, hypoglycemia occurring within four hours of intake of meals, is an extremely uncommon condition in children. We report the case of an adolescent boy who presented with excessive hunger, poor weight gain, anasarca, hypoalbuminemia, and electrolyte imbalance. Investigating the history of post-meal drowsiness showed postprandial hypoglycemia, which on subsequent evaluation revealed elevated fasting insulin and suppressed postprandial insulin levels. This eventually led to the uncovering of a deliberately and cleverly concealed purging behavior. A meticulous interview of the family and the child confirmed the diagnosis of anorexia nervosa, binge-purging type, and averted unnecessary further investigations into the cause of failure to thrive.

Keywords: Anorexia nervosa binge-purge, Hypoinsulinemic hypoglycemia, Postprandial hypoglycemia, Insulin resistance, Adolescent behavior

INTRODUCTION

Hypoglycemia occurring within four hours after meal intake is defined as postprandial hypoglycemia.[1] Postprandial hypoglycemia is an extremely uncommon condition in children and can be seen in hereditary fructose intolerance, or it could be insulin-mediated: insulinoma, non-insulinoma pancreatogenous hypoglycemia, or factitious due to exogenous insulin administration (Munchausen syndrome or Munchausen syndrome by proxy) or sulfonylurea ingestion.[2,3] Eating disorders (EDs) like anorexia nervosa (AN) can cause fasting hypoglycemia, which can be symptomatic or subclinical, predominantly due to liver glycogen depletion.[4] We report the case of an adolescent boy who presented with excessive eating, anasarca, and metabolic abnormalities and how a chance identification of postprandial hypoinsulinemic hypoglycemia led to the discovery of a carefully hidden purging behavior.

CASE REPORT

An adolescent boy (13 y and 6 mo) presented to a tertiary care hospital with an altered eating pattern, a one-week history of facial puffiness, pedal edema, and poor weight gain. Until six months ago, he had a poor appetite and lost 10 kg over 15 months. His pediatrician diagnosed him with hypothyroidism (thyroid-stimulating hormone (TSH): 8.6 uIU/mL, thyroxine T4: 8.2 µg/dL) and started treatment with 25 µg levothyroxine daily. A psychiatrist consultation

was sought at the time, but no diagnosis was made, and the boy was advised to follow-up with his pediatrician. At this time, the child was living with his mother and maternal grandparents and was attending school while his father was working in a different city. The child's appetite improved, but a month later he started eating excessively all through the day and was noted to be always hungry. Grandfather was so alarmed by the child's food consumption that he even consulted the village shaman, believing that the child must be possessed. The child's diet history included 0.5 kg chicken, 250 g sweets, and twice the usual quantity of each meal daily three times. Mother and grandfather denied any history of vomiting. However, his mother complained of frequent washroom usage, probably for bowel movements, and witnessed what looked like undigested food stuck to the toilet bowl, which was difficult to flush after the child used it. His weight remained almost the same during the past six months despite excessive food consumption. Three months ago, he was evaluated by a gastroenterologist, and tissue transglutaminase antibody and duodenal biopsy were negative, excluding celiac disease.

His weight was 24 kg (−2.93 standard deviation score [SDS]), height 142.5 cm (−1.86 SDS of mid-parental height), and body mass index (BMI) was 11.9 kg/m^2 (−2.89 SDS). He was in early puberty with Tanner stage P2G2. He had a pale and puffy face, bilateral pitting pedal edema, and a distended, non-tender abdomen. Initial workup revealed severe hypokalemia (2.3 mmoL/L), hypochloremic metabolic alkalosis; hypochromic, microcytic anemia, low white blood cell count, hypoalbuminemia (2.8 gm/L), mild (1+) proteinuria, and ascites. Random blood glucose was 84 mg/dL, and blood pressure was 96/68 mm Hg [Table 1].

Table 1: Investigations of the index case at presentation.

Biochemical parameter	Value (normal range)
Serum sodium (mmoL/L)	136 (135–145)
Serum potassium (mmoL/L)	2.3 (3.5–5)
Serum chloride (mmoL/L)	96 (95–105)
Venous blood gas	
pH	7.47 (7.35–7.45)
HCO_3	37.7 mmoL/L (18–22)
BE	+17 (−3 to+3)
Serum calcium (mg/dL)	8.4 (8.5–10.2)
SGOT/SGPT (U/L)	40/43 (5–30)
Serum albumin (g/dL)	2.8 (3.5–5)
Alkaline phosphatase (U/L)	32 (50–150)
Blood urea (mg/dL)	10.5 (6–24)
Serum creatinine (mg/dL)	1.1 (0.8–1.3)
Hb (g/dL)	7.9 (12–15)
WBC (×10^9/L)	3.2 (4–10)
Platelets (×10^9/L)	166 (150–400)
Free T3 (pg/mL)	1.27 (2.1–3.8)
Free T4 (ng/dL)	1.01 (0.9–1.5)
TSH (μIU/mL)	6.4 (0.5–5)
Serum cortisol (μg/dL)	32 (5–23)

BE: Base excess, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamate pyruvate transaminase, Hb: Hemoglobin, WBC: White blood count, T3: Triiodothyronine, T4: Thyroxine, TSH: Thyroid-stimulating hormone, pH: potential hydrogen, HCO_3: Bicarbonate.

Management and outcome

The child was treated with intravenous (IV) potassium correction and IV fluids at a maintenance rate. An evaluation for possible multisystem disorder with malabsorption was considered by the treating pediatrics team. A plan was made for gastroenterology and immunology consults. A bone marrow examination was organized. Pediatric endocrinology services were sought for hypothyroidism.

A careful review of the dietary history with the mother revealed a peculiar eating pattern. The child eats quite a lot of food starting from mid-morning, demanding meals every hour till afternoon around 16:00 h, when he feels extremely sleepy and dozes off, waking up after a few hours. This prompted us to check postprandial blood glucose 90 min after a mixed meal, which was 59 mg/dL. Paired samples of glucose and insulin were obtained after overnight fasting and after one hour and two hours of mixed meal intake. This showed fasting (65 mg/dL) as well as post-meal (1 h: 55, 2 h: 78 mg/dL) hypoglycemia. Fasting insulin (36.47 mIU/mL) was elevated, while post-meal insulin levels (1 h: 1.31, 2 h: 1.88 mIU/mL) were suppressed appropriately for hypoglycemia. Urine ketones were negative, and serum cortisol was appropriately elevated. Thyroid functions were typical of non-thyroidal illness syndrome [Table 1].

The combination of postprandial hypoglycemia with suppressed insulin, electrolyte abnormality resembling pseudo-Bartter syndrome, and a history of weight loss with excessive oral intake led us to consider the possibility of binge-purging strongly. Examination of the oral cavity revealed significant dental erosion and caries. Mother and grandfather strongly denied any vomiting or purging. However, on meticulous questioning, they revealed that the child drinks almost one liter of water before starting his daily meal ritual and then keeps visiting the washroom between meals till noon. They firmly believed that these visits were for urination and bowel movements, and they never heard any retching noises from the washroom, although the child always kept the water tap on. After some explanation, the mother understood that purging was a definite possibility and realized that what she was noticing in the toilet bowl all along was not stools, but vomitus. The boy denied any vomiting and got upset when this discussion came up. Social history revealed a middle-class family with severe family discord between the parents, resulting in parental separation 18 months ago when the mother took the child to live with her parents. The child

had no siblings, used to be a witness to routine verbal abuse between parents for many months before their separation, and also sustained bullying at school due to his withdrawn and quiet nature. After the separation, he missed spending time with his father.

We discussed with and convinced the pediatrics team to defer further investigations and sought a pediatric psychiatry consultation. A diagnosis of AN – binge purging variety (AN–B/P) was considered based on the history of restrictive eating behavior with weight loss, recent binge purging, and significantly low current BMI. The child was started on fluoxetine 10 mg once a day and referred to a multidisciplinary in-patient care for complex EDs at a higher center, where he slowly improved. The child confessed to his mother that he repeatedly purges after bingeing and that he was unable to control the urge to binge purge. He denied having a distorted body image or fear of getting fat.

DISCUSSION

Although EDs such as AN and bulimia nervosa (BN) in adolescents are relatively common in Western societies, limited data exists from India. AN is the most commonly reported behavior, while BN and AN–B/P are extremely uncommon, with only a handful of case reports.[5] The metabolic complications of binge-purging are similar in BN and AN–B/P.[6]

Fasting hypoglycemia is a well-recognized complication of AN, with chronic liver injury and marked glycogen depletion being the main contributors. Although studies on insulin sensitivity in AN resulted in contradictory findings, the refeeding phase was shown to be associated with the onset of insulin resistance, partly due to increased visceral fat.[6,7] Moreover, binging episodes seem to worsen this situation, as even a single day of binge eating was seen to decrease whole-body insulin sensitivity by 28%, indicating the rapidity with which binge eating can alter glucose metabolism.[8] Our child had elevated fasting insulin levels, indicating a state of insulin resistance brought about by the binging episodes that succeeded in the previous anorexic phase. These high fasting insulin levels can dramatically drop along with plasma glucose after the purging, as was seen in a study in 8 bulimic women.[9] Moreover, patients with AN have defective glucose-dependent insulinotropic polypeptide-mediated glucagon secretion and decreased sensitivity to glucagon-induced hepatic glycogenolysis.[10] The resulting profound post-purge hypoglycemia can trigger extreme hunger and play a predominant role in the continuation of binge/purge cycles.

Our patient also presented with the classic pseudo-Bartter syndrome. Repeated vomiting results in chronic volume depletion, hypotension, hypokalemic, and hypochloremic alkalosis that stimulates the renin-angiotensin-aldosterone system, resulting in sodium and water retention, normalization of blood pressure, and edema, which might have been worsened by hypoalbuminemia.[5,11]

Research into ED is severely limited, and obscure presentations such as the index case make diagnosis challenging and delayed. Shame and fear of judgment associated with binge/purging lead to secrecy, which teenagers could go to any lengths to maintain. Atypical cases of AN and BN with no body image disturbance or fear of fatness were described mainly from Asian countries because, in these populations, stressful social and cultural influences could be the main drives for ED rather than a pursuit of thinness.[12] A high index of suspicion in adolescents with any disordered dietary habits can avoid unnecessary investigations and facilitate early therapeutic intervention.

CONCLUSION

Binge-purging should be considered as one of the differential diagnoses for postprandial hypoglycemia in adolescents. This is typically associated with postprandial hypoinsulinemia, and fasting insulin can be elevated. A high index of suspicion is needed to identify purging behavior in an adolescent with metabolic derangements, bodyweight fluctuations, and disordered eating.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

Patient's consent is not required as the patient's identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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