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INFANT HYPOGLYCEMIA REVEALING FACTITIOUS DISORDER IMPOSED ON ANOTHER

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INTRODUCTION

Hyperinsulinemic hypoglycemia of infancy may be congenital or acquired, and rarely due to factitious hypoglycemia imposed by another. A thorough medical and social history, critical sampling, and screening for inborn errors of metabolism are crucial.

CASE

A 5-month-old, ex-29 weeker, twin female infant, born to a non-consanguineous couple was admitted at 4 months with severe refractory hypoglycemia and seizures requiring a glucose infusion rate (GIR) of 16 mg/kg/min. Critical sampling suggested hypoglycemia due to exogenous insulin: glucose 1.1 mmol/L, ketones 0.2 mmol/L, insulin 1,208 pmol/L (17.8–173), and C-peptide 18.2 pmol/L (366–1,466). Free fatty acids were not raised; growth hormones were 2.223 µg/L (0.14–6.27) and cortisol >1,000 nmol/L (145.4–619.4). Metabolic and genetic testing excluded glycogen storage disorder. Two months prior, she was admitted with a severe human metapneumovirus and parainfluenza infection, hypoglycemia, lactic acidosis and left ventricular hypertrophy. Her twin had died of sudden infant death syndrome. Examination revealed a small puncture mark on the abdomen, but otherwise it was unremarkable. The GIR dropped dramatically over 3 days and the intravenous dextrose was discontinued. Normoglycemia was maintained on 3-hourly feeds and she tolerated an age-appropriate fast before discharge. No hypoglycemic episodes were reported. Of note, the mother suffered from bipolar disorder and had access to insulin for gestational diabetes mellitus. The infant is currently in foster care, is scheduled to have neuroimaging and continues to have growth and developmental follow-up.

CONCLUSION

Factitious hypoglycemia should be suspected when there are red flags in the clinical history and critical sampling demonstrates high insulin levels, suppressed C-peptide in the face of hypoglycemia with low ketones and low free fatty acids. A multidisciplinary approach involving paediatrics, psychiatry, and child protective services is mandatory.

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A SILENT COMPLICATION OF PROLONGED IMMOBILIZATION: FRAGILITY FRACTURE IN A CHRONICALLY ILL ADOLESCENT WITH THALASSEMIA

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INTRODUCTION

Adolescence is a crucial period for achieving peak bone mass. Risk factors such as chronic inflammation, endocrine issues, corticosteroid use, and extended immobility can lead to severe secondary osteoporosis and fragility fractures.

CASE

A 15-year-old female with transfusion-dependent thalassemia had been hospitalized for over a year following severe acute gastroenteritis complicated by septic shock, multiorgan failure, and secondary hemophagocytic lymphohistiocytosis (HLH). She received pulse methylprednisolone therapy for HLH, which was tapered over 6 months. Her clinical course was further complicated by critical illness polyneuropathy and post-infectious bronchiectasis, leading to ventilator dependence and a bedbound state.

During regular physiotherapy sessions, she developed a sudden onset of severe left shoulder pain. A radiograph of the left shoulder revealed a closed fracture of the humeral neck.

Biochemical evaluation showed normal serum calcium and phosphate levels, adequate vitamin D status, and normal alkaline phosphatase levels. However, follicle-stimulating hormone, luteinizing hormone (LH), and estradiol levels were undetectable, consistent with arrested puberty due to chronic illness. A bone mineral density scan showed a lumbar spine Z-score of -6, indicating severe osteoporosis.

She was started on intravenous zoledronate for secondary osteoporosis, and physiotherapy was resumed to improve musculoskeletal strength.

CONCLUSION

This patient was treated with bisphosphonate therapy for severe secondary osteoporosis caused by multiple risk factors, including chronic illness, prolonged immobility, corticosteroid use, and hypogonadotropic hypogonadism, with limited potential for spontaneous bone recovery. This case highlights the importance of early endocrine monitoring and bone health assessments to identify impaired bone growth and prevent fragility fractures in high-risk adolescents.