phosphate: 1.85-3.05 mmol/L, alkaline phosphatase (ALP): 500-800 IU/L and 25(OH)D3: 200 nmol/L. He also had hypochloraemic hypokalemic metabolic alkalosis, hyperreninemia (>550 mU/L) and hyperaldosteronemia (>3656 pmol/L). Ultrasound at 2-month-old demonstrated bilateral renal medullary nephrocalcinosis and cholelithiasis. Skeletal survey revealed no significant bony abnormalities. There were episodes of hyponatremia and hypokalemia, which improved spontaneously. Clinically, he had prominent forehead, triangular face, right hand pre-axial polydactyly and bilateral short distal phalanx of the 4th and 5th fingers with nail hypoplasia. His weight gain was poor with delayed motor development and hypotonia. At 6-month-old, his care was shared by paediatric nephrologist and endocrinologist. He was 3.57 kg with a length of 57.8 cm. His iPTH later resulted in relatively inappropriately raised level, (Ca: 2.81mmol/L, PO4: 1.87 mmol/L, ALP: 770 IU/L, iPTH: 68.3 pg/mL). Urinalysis showed profound natriuresis and hypercalciuria (24-hour urine Ca: 5.3 mg/kg/day). Ultrasound of the thyroid exhibited no abnormality. The parents' calcium profiles were normal. Pamidronic acid (1 mg/kg/dose) was given (when serum calcium >3.0 mmol/L) but the hypercalcemia only transiently improved. Eventually, he was treated with indomethacin and free water supplement. The whole exome sequencing revealed a heterozygous pathogenic variant in ROR2 gene and a homozygous variant of uncertain significance in KCNJ1 gene.

CONCLUSION

Antenatal Bartter syndrome presents insidiously during neonatal period, typically with polyhydramnios, IUGR, prematurity, polyuria and failure to thrive. It can present with nephrocalcinosis accompanied by features resembling primary hyperparathyroidism. Genetic testing enhances the diagnostic precision of various Bartter syndrome subtypes.

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BALANCING SUGAR AND STRAIN: LIVING WITH TYPE 1 DIABETES AND CHRONIC KIDNEY DISEASE

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INTRODUCTION

Effective glycaemic control is essential in the type 1 diabetes mellitus (T1DM) to prevent both acute and chronic complications of the disease. This case explores the challenges faced in daily glucose regulation and highlights the critical role of tight glycaemic control in ensuring long term health outcomes for individuals living with the disease.

CASE

A 13-year-old male arrived at the emergency department in impending diabetic ketoacidosis (DKA). He was diagnosed with T1DM at the age of 3, though his antibody work-up was negative. His medical history revealed poor adherence to medical appointments and treatment, with multiple hospitalizations for DKA between the ages of 3 and 10. At 11 years old, he was completely lost to follow-up and was managing his insulin doses independently, without proper blood sugar monitoring. After 2 years without medical supervision, he was admitted with disseminated methicillin-sensitive Staphylococcus aureus (MSSA) infection, bilateral renal abscesses and stage 3a chronic kidney disease. His HbA1c at the time of admission was 14%.

CONCLUSION

This case highlights the serious consequences of poor glycemic control in T1DM. Persistent hyperglycemia and inadequate disease management likely contributed to immune dysfunction, heightened infection risk and progressive kidney damage, ultimately leading to his critical condition. Consistent diabetes management and early medical intervention are essential to prevent such life-threatening complications.