Case 3. An 8-year-old female with underlying mild autism was started on GnRHa injections for CPP. At every clinic visit, she will cry, shout and throw tantrums which were attributed to injection anxiety. Parents and nurses had a lot of difficulty getting her ready for injections. On her last visit, the nurse who gave her the injection reported bite marks and bruises on her arm because the patient bit her.

CONCLUSION

Although rare, one should always take extra precautions when dealing with IM injections in children. Repeated procedures carry higher risk as mentioned in this case series.

EP_P031

PITUITARY HYPERPLASIA SECONDARY TO PRIMARY HYPOTHYROIDISM – A CASE REPORT

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INTRODUCTION

Primary hypothyroidism in children can present insidiously and mimic other systemic conditions, including neurological symptoms. In rare cases, it may lead to pituitary hyperplasia due to lack of negative feedback on thyrotrophs. Timely recognition is essential to prevent complications and avoid unnecessary neurosurgical intervention.

CASE

We report a case of an 8-year-old female who presented with chronic headaches, cold intolerance, constipation and frontal scalp hair thinning. Symptoms were insidious, with persistent headaches noted since the age of six. Despite multiple outpatient visits, including private pediatric and ophthalmology consultations, no clear diagnosis was made. Ophthalmological evaluations were also normal.

Due to persistent symptoms, neuroimaging was done to exclude intracranial mass or raised intracranial pressure. MRI of the brain and pituitary revealed enlarged pituitary gland measuring 7.7 mm (AP) x 12.4 mm (width) x 9.7 mm (height), with normal posterior pituitary bright spot and pituitary stalk. Other surrounding structures were normal. Thyroid function test (TFT) performed revealed an elevated TSH of 150 mIU/L with low fT4 at 8.7 pmol/L, consistent with primary hypothyroidism. Anti-thyroid peroxidase (TPO) and thyroglobulin antibodies were positive, confirming Hashimoto's thyroiditis. Other pituitary hormones were normal. She was initiated on levothyroxine, and serial TFTs demonstrated gradual improvement. MRI features were consistent with pituitary hyperplasia secondary to long-standing hypothyroidism (PHPH), and no neurosurgical intervention was warranted. A repeat MRI scan performed 10 months after commencement of treatment showed normal study with a pituitary gland measuring 6.7 mm (AP) x 12.2 mm (width) x 5.7mm (height). Her latest TFT has normalised with TSH of 3.76 mIU/L and fT4 of 18.5pmol/L on levothyroxine 37.5 mcg *qd* Monday to Friday, and 50 mcg *qd* on weekends.

CONCLUSION

PHPH is an uncommon cause of pituitary enlargement in children. This case highlights the importance of comprehensive endocrine assessment in children with chronic headaches. Early diagnosis and thyroid hormone replacement can lead to complete resolution of symptoms and regression of pituitary enlargement, avoiding misdiagnosis and overtreatment.

EP_P032

AN UNCOMMON CAUSE OF PERSISTENT HYPERCALCAEMIA WITH NEPHROCALCINOSIS IN INFANCY

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INTRODUCTION

Hypercalcaemia with nephrocalcinosis in infants is commonly caused by excessive calcium or vitamin D supplementation, neonatal primary hyperparathyroidism, subcutaneous fat necrosis or various genetic disorders.

CASE

We present a 6-month-old Indian male infant who was born preterm at 33 weeks, via elective LSCS for poly-hydramnios with weight of 1.33 kg, length of 46 cm and head circumference of 27 cm. His mother had severe polyhydramnios, requiring amnioreduction thrice. Both parents were consanguineous. During his 3-month-stay at NICU, he had persistent hypercalcaemia with inter-mittent polyuria. Serum calcium ranged: 2.5-2.9 mmol/L,

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.

EP_P033

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.