Paediatrics E-Poster

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PAEDIATRIC PRIMARY HYPERPARATHYROIDISM PRESENTING WITH BILATERAL SLIPPED UPPER FEMORAL EPIPHYSES: A CASE REPORT

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INTRODUCTION
Primary hyperparathyroidism (PHPT) is a rare endocrine disorder in children and adolescents with a prevalence of 2-5 in 100,000. PHPT in this age group is often due to single parathyroid adenoma, whereby surgery remains the definitive treatment. Postoperative transient hypocalcemia is a common complication. Nevertheless, severe hungry bone syndrome (HBS) in paediatric is uncommon and is a challenge in the post-operative management of PHPT.

CASE
A 14-year-old Malay, male presented with trivial falls and subsequently developed bilateral lower limb pain for 2 months, which led to an abnormal, painful gait for 1 week. He also had nausea, intermittent vomiting, abdominal pain, loss of weight and appetite.

Biochemical investigations were consistent with primary hyperparathyroidism. He had severe hypercalcemia, hypophosphatemia with elevated alkaline phosphatase and intact parathyroid hormone (iPTH). Pelvic x-ray revealed bilateral slipped upper femoral epiphysis (SUFE) and periosteal bone resorption at the pelvic bones. A neck ultrasound showed a hypoechoic nodule located posterior-inferior to the right lobe of the thyroid gland. A Tc-99m sestamibi parathyroid scan detected an avid lesion inferior to the right thyroid lobe.

His severe hypercalcemia was managed by hydration and loop diuretics. For preoperative optimisation, he received intravenous zoledronate and subcutaneous calcitonin. He underwent a right-focused parathyroidectomy and histopathology confirmed the diagnosis of parathyroid adenoma. Post-operatively, he developed severe HBS. He had symptomatic hypocalcemia post-parathyroidectomy for which he required continuous calcium gluconate infusion, calcitriol, calcium carbonate and cholecalciferol. Continuous intravenous calcium and intravenous alfacalcidol were given for four weeks and stopped when serum phosphorus and alkaline phosphatase levels returned to normal limits.

CONCLUSION
PHPT in children and adolescents often presents with non-specific symptoms leading to a delay in diagnosis. A diagnosis of PHPT should be considered when they present with bone pain or skeletal deformity associated with radiological imaging of osteolytic lesions.

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THE HIDDEN THREAT: DIABETES MELLITUS IN A CHILD WITH CONGENITAL RUBELLA SYNDROME

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INTRODUCTION
Congenital rubella syndrome arises from maternal infection with rubella virus, particularly during the first trimester of pregnancy. While rubella is primarily associated with ocular, cardiac and auditory defects, its effects on the endocrine system, particularly in relation to diabetes mellitus, are seldom reported. This case report underscores the necessity of close monitoring in children with a history of rubella exposure, given the potential risk for the subsequent development of diabetes mellitus.

CASE
A 1-year 9-month-old male had right corneal clouding and absent red reflex. He was born at term with a birth weight of 2.6 kg. His mother had a multinodular goitre with no history of fever and rashes during pregnancy. He was diagnosed with right eye glaucoma and left uveitic cataract. He underwent evisceration of the right eye at 4 months and left lens surgery at 6 months.

The patient presented recently with lethargy, excessive thirst, frequent urination and weight loss. He had global developmental delays and showed signs of dehydration during the examination. His growth was within percentile. Investigations revealed blood glucose of 46 mmol/L, positive serum ketone and metabolic acidosis (pH, 7.126; HCO_3, 10.1 mmol/L; base excess -22.7 mmol/L; $PaCO_2$, 20 mm Hg). He had no skin lesion and other systemic examinations were unremarkable.

He was diagnosed with diabetic ketoacidosis and was treated with intravenous fluids and insulin. Following metabolic stabilization, he was transitioned to subcutaneous insulin.