

## PE-15

### UNEXPECTED ISOLATED HYPOPHOSPHATEMIC RICKETS ASSOCIATED WITH ELEMENTAL FORMULA FEEDING

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#### INTRODUCTION

Phosphate deficiency is common in rickets but is accompanied by calcium or vitamin D deficiency (also named nutritional rickets). Isolated hypophosphatemic rickets (HR) without hypovitaminosis or defective renal phosphate handling is uncommon. Phosphate is abundant in diet and its GI absorption even though primarily mediated by vitamin D, is less tightly regulated with about 30% being passive. Recently, there has been an increase in such HR cases reported associated with the use of elemental formula (EF).

#### RESULTS

The patient was born prematurely at 31 weeks and diagnosed having Tetralogy-of-Fallot (TOF) with severe pulmonary stenosis requiring Blalock-Taussig (BT) shunt at 3-month-old. Postoperatively, he had heart failure and gastroesophageal-reflux-disease (GERD), for which he was treated with heart failure drugs (furosemide, spironolactone, captopril) and antireflux (omeprazole, domperidone), respectively. At 2-months-old, he was diagnosed with Cow's Milk Protein Allergy (CMPA) and EF was commenced. Both Neocate® and Comidagen® were used interchangeably. He developed rickets at 8-month-old with initial serum phosphate 0.5mmol/L, calcium 2.46mmol/L, ALP 1432 IU/L, and 25(OH)-Vitamin D 80 nmol/L (sufficient). His urine TRP was normal and radiological findings were consistent with rickets. Notably, he developed acute severe hypocalcemia with hyperphosphatemia, immediately following oral Sandoz phosphate, despite calcium supplementation. His subsequent response, however, was partial and the hypophosphatemia persisted. He was switched to soy-based formula at 10-months-old, with ensuing improvement in serum phosphate. He achieved biochemical and radiological healing of rickets within 3 months of follow-up.

#### CONCLUSION

HR in certain infants relating to the prolonged and sole use of EF had been reported elsewhere with its etiology not fully understood but could relate to the reduced bioavailability of phosphate in EF. Replacement with an alternative phosphate form could cause transient acute severe hypocalcemia and hyperphosphatemia possibly due to sudden upregulation of Na-Pi2b cotransporter in the gut after phosphate starvation. The cessation of EF reverses the pathology.

## PE-16

### PARTIAL ECTOPIC POSTERIOR PITUITARY GLAND IN A CHILD: A VARIANT OF AN ECTOPIC NEUROPHYPHYSIS SYNDROME

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#### INTRODUCTION

Developmental abnormality of the posterior pituitary can lead to an ectopic posterior pituitary at the median eminence or along the pituitary stalk with partial or complete pituitary stalk agenesis. An ectopic posterior pituitary gland is associated with isolated growth hormone or multiple anterior pituitary deficiencies but with normal posterior pituitary function. A partial ectopic pituitary gland is a less common entity described whereby there is presence of both an orthotopic (normally located) and ectopic neurohypophysis.

#### RESULTS

The patient first presented at 2 months old with prolonged jaundice. Thyroid function screening showed central hypothyroidism and she was started on L-thyroxine. She presented again at 2 years 10 months old with a hypoglycaemic seizure. Subsequently she was referred for further paediatric endocrine evaluation. Her IGF-1 was < 20mcg/L and glucagon stimulation test confirmed severe GH deficiency (peak GH 0.54ug/L) with an optimal cortisol peak of 698 nmol/L. Pituitary/brain MRI shows a hypoplastic pituitary gland and absence of pituitary stalk. There was a bright spot at the normal expected site of the neurohypophysis in the posterior sella with an additional ectopic focus of high signal intensity on T1-weighted imaging at the infundibulum measuring 3mm x 3mm which was most likely an additional and ectopic focus of the posterior pituitary gland. The pituitary stalk was not visualized. She was started on recombinant GH therapy and remains on L-thyroxine. Regular monitoring of her 8 am cortisol remains normal and she did not have symptoms or biochemistry suggestive of diabetes insipidus on follow-up.