diagnosed perioperatively. He had polydipsia and weight loss but denied other symptoms. Past medical history and family history were unremarkable. He was small in stature with a body mass index (BMI) of 12.6 kg/m^2, severely scoliotic spine and pectus excavatum and no palpable neck masses.

Skeletal survey showed multiple malunited fractures and lytic bone lesions. Biochemical investigations revealed severe hypercalcemia with a peak level of 4.38 mmol/L and a markedly elevated intact PTH (iPTH) level of 695.3 pmol/L. Initial technetium-99m-sestamibi (MIBI) parathyroid scintigraphy showed no uptake over the neck and neck ultrasound revealed no masses. A subsequent whole-body MIBI with SPECT-CT showed avid uptake at the anterosuperior mediastinum, corresponding to a contrast-enhancing mass seen on CT-scan, measuring 17 x 30 x 18 mm. Genetic screening was negative for mutations in CASR, MEN1, MEN2 and MEN4 genes.

Hypercalcemia was controlled with subcutaneous denosumab and oral cinacalcet before subsequent surgical resection. The mediastinal mass removed measured 55 mm in diameter and weighed 12.5 grams. Histopathological examination revealed tissue predominantly comprising chief cells, confirming an ectopic PTH adenoma. Post-operatively, his iPTH levels normalized and he required high doses of alfacalcidol.

CONCLUSION

Mediastinal adenomas are ectopic parathyroid adenomas that form because of aberrant embryological migration of inferior parathyroid glands. They may pose a diagnostic challenge leading to unnecessary surgical explorations. MIBI scans can improve diagnostic accuracy. Ectopic parathyroid adenomas make up 16-22% of parathyroid adenomas, of which 6-13% are mediastinal adenomas. Patients with primary hyperparathyroidism and initial negative neck imaging should prompt suspicion and work-up for mediastinal parathyroid adenoma.

EP_A038

PRIMARY HYPERPARATHYROIDISM IN PREGNANCY, INITIAL PRESENTATION WITH HYPOCALCAEMIA SECONDARY TO ACUTE PANCREATITIS AND SEPSIS

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INTRODUCTION/BACKGROUND

Diagnosis of primary hyperparathyroidism is challenging during pregnancy and requires special care to prevent complications such as nephrolithiasis, pancreatitis, preterm delivery and foetal demise. We are reporting a rare case of primary hyperparathyroidism initially presenting with severe hypocalcaemia.

CASE

A 25-year-old primigravida at 24 weeks of gestation was admitted to intensive care unit with decompensated septic shock and acute kidney injury requiring ventilatory support. Subsequently, she had chorioamnionitis and non-viable foetus resulting in premature delivery. CECT of the abdomen showed acute pancreatitis in the absence of gallstone and caecal perforation with extensive peritonitis. She then underwent laparotomy. Other laboratory results showed elevated white blood count (29.7 g/d) and serum creatinine (173 micromol/L) with reduced eGFR (35 ml/mol/1.73 m^2), hypocalcaemia (1.79 mmol/L (corrected)), hypoalbuminemia (21 g/L), with normal phosphate (0.91 mmol/L). She required multiple intravenous calcium corrections for the first 3 days. The serum calcium showed gradual increment from 2.4 mmol/l to 4.07 mmol/L within 10 days without calcium or vitamin D supplementation and adequate hydration. Intact parathyroid hormone (iPTH) was 43 pmol/L but repeat test was elevated at 148 pmol/L. She required intravenous zolendronate 4 mg with bridging calcitonin given 3 weeks apart. She had severe vitamin D deficiency of <5 ng/mL for which she was started on replacement. Patient denied familial hypercalcaemia or MEN syndrome. Ultrasound of the neck did not locate a parathyroid adenoma. Therefore, she is awaiting sestamibi scan and genetic testing to rule out familial causes of primary hyperparathyroidism is being considered.

CONCLUSION

Hypocalcaemia is a common finding in acute pancreatitis due to mesenteric calcium salt formation. It is also present in critically ill patients with sepsis which is a marker of severity with increased mortality and hospital stay.

Hypercalcaemia was unmasked later in this case upon the resolution of the above-mentioned conditions. During pregnancy, surgery is the treatment of choice during the second trimester in cases of severe hypercalcemia (calcium >3.0 mmol/L) because medical therapy options are unsafe.

EP_A039

A CHALLENGING CASE OF FAMILIAL HYPOCALCIURIC HYPERCALCEMIA CONCEALED BY CONCOMITANT VITAMIN D DEFICIENCY AND PAPILLARY THYROID CARCINOMA

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INTRODUCTION/BACKGROUND
Familial hypocalciuric hypercalcemia (FHH) is a rare autosomal-dominant disorder resulting from an inactivating mutation in the calcium-sensing receptor (CASR) gene. It is generally benign and characterized by longstanding parathyroid hormone (PTH) - dependent hypercalcemia. Resection of the parathyroid tissue does not normalize serum calcium.

CASE
We report a case of FHH with evaluation confounded by vitamin D deficiency and newly diagnosed papillary thyroid carcinoma.

A 63-year-old female was incidentally noted to have hypercalcemia with elevated PTH during admission for pneumonia. She had completed antituberculosis therapy for gastrointestinal tuberculosis 2 years ago. The baseline vitamin D level was 24nmol/L (NV: 75-100), and the 24-hour urine calcium creatinine ratio was 0.01. Bone mineral density revealed an osteopenic spine. Focused parathyroidectomy was performed after technetium (99mTc) sestamibi SPECT scan which revealed a left superior parathyroid adenoma. However, the calcium levels remained elevated. Histopathological examination of the postoperative sample revealed a metastatic papillary thyroid carcinoma in the lymph node. Hence, she underwent total thyroidectomy, left modified neck dissection, and total parathyroidectomy with auto-transplantation of parathyroid tissue into the presternal space. Albeit that, the serum calcium and PTH remained elevated. PTH-related protein (PTHrP) and 1,25 hydroxy vitamin D levels were within normal range. Opportunistic screening of her daughter revealed that she had raised calcium levels with inappropriately normal PTH and 24-hour urinary calcium creatinine ratio of less than 0.01. The patient's repeated 24-hour urinary calcium creatinine ratio after repletion of vitamin D was also less than 0.01. We diagnosed her with FHH based on positive family screening and longstanding asymptomatic PTH-dependent hypercalcemia refractory to parathyroidectomy.

CONCLUSION
Our case highlights the diagnostic dilemmas in hypercalcemia, the importance of screening family members and the repetition of 24-hour urine calcium creatinine ratio after correction of vitamin D deficiency.

EP_A040

TERIPARATIDE [HUMAN PARATHYROID HORMONE (PTH) 1-34] FOR THE MANAGEMENT OF POST-THYROIDECTOMY HYPOCALCEMIA EXACERBATED BY CHYLE LEAK

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INTRODUCTION/BACKGROUND
Hypocalcaemia secondary to hypoparathyroidism is a common complication of thyroidectomy. Another less common but serious complication is chyle leak which may also lead to electrolyte abnormalities, including hypocalcaemia. We report a case of refractory hypocalcaemia following thyroidectomy complicated by chyle leak which was successfully managed with teriparatide.

CASE
A 37-year-old male had undergone total thyroidectomy with central and left lateral neck dissection for papillary thyroid carcinoma. After the surgery, up to 200 cc/day of milky fluid were noted in his neck drain. Biochemical analysis showed high triglyceride content (2.3 mmol/L), consistent with chyle. He was initially treated conservatively with total parenteral nutrition, pressure dressing and subcutaneous octreotide.

On postoperative day (POD) 3, he had symptomatic hypocalcaemia (corrected calcium 1.95 mmol/L). PTH was undetectable (<0.5 pmol/L). However, despite intravenous calcium gluconate infusion, high doses of activated vitamin D and calcium supplements (calcitriol 4.5 mcg/day, alphacalcidol 2 mcg/day and calcium carbonate 6 g/day), his calcium level remained as low as 1.9 mmol/L by POD