

Initial biochemical results were all within normal range: calcium 2.41 mmol/L, phosphate 1.0 mmol/L, ALP 84 U/L, TSH 1.83 m IU/L, FT4 14.8 pmol/L and i-PTH 4.8 pmol/L. Overnight dexamethasone suppression test was appropriately suppressed (15 nmol/L). Her vitamin D level was insufficient at 51 nmol/L. Radiography confirmed T10 vertebral compression fracture and her DEXA scan revealed that she is osteoporotic with spine Z-score -3.2, and femur Z-score -3.5.

She was managed with oral cholecalciferol 1000 units daily and calcium carbonate 500 mg twice daily as she preferred to not use any anti-resorptive agent. Her back pain improved after cessation of lactation and with analgesics.

CONCLUSION

PLO is a rare condition and should be suspected in pregnant women who complain of back pain to ensure early diagnosis and intervention. Secondary causes of osteoporosis need to be ruled out as well.

EP_A094

SEVERE HYPERCALCEMIA UNMASKS A CAMOUFLAGED PARATHYROID CARCINOMA

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INTRODUCTION/BACKGROUND

Parathyroid carcinoma is an exceedingly rare endocrine malignancy and an uncommon cause of primary hyperparathyroidism. We report a case of severe hyperparathyroidism which required urgent surgery and unveiled a parathyroid carcinoma.

CASE

A 31-year-old Malay female, who initially presented with severe epigastric pain and vomiting, was treated for acute pancreatitis in the surgical ward. There was no history of alcohol consumption. Biochemistry results showed acute renal impairment, hypercalcemia (corrected Ca²⁺ 4.06 mmol/L), hypophosphatemia (PO₄³⁻ 0.38 mmol/L), elevated serum amylase (370 U/L) and urine diastase (663 U/L). However, no sonographic evidence of cholelithiasis was found. She was referred to the medical team for further investigations which revealed primary hyperparathyroidism with vitamin D deficiency (iPTH 705 pg/ml, 25 (OH) vitamin D 25 nmol/L, alkaline phosphatase 543 U/L). Clinically, no neck mass was observed but the neck detected a TIRADS 5 nodule in the right thyroid. Given

the discordant clinical and sonographic findings, Tc99m Sestamibi SPECT/CT was arranged but she defaulted. Subsequently, she was admitted for severe hypercalcemia manifesting as severe bone pain and requiring saline-forced diuresis and intravenous zoledronic acid. Tc99m Sestamibi SPECT/CT scan demonstrated a parathyroid adenoma measuring 2.2 x 2.6 x 2.9 cm posterior to the right thyroid gland. Further imaging work-up showed bilateral medullary nephrocalcinosis and osteopenia. She underwent urgent right hemithyroidectomy, right superior parathyroidectomy and central neck dissection successfully. Postoperatively she developed hungry bone syndrome which required intravenous calcium infusion together with high dose activated vitamin D and calcium supplements. Histopathological report confirmed the diagnosis of parathyroid carcinoma with lymphovascular and capsular invasion. Postoperatively she was scheduled for PET-CT scan but she defaulted the follow-up. She is currently pregnant.

CONCLUSION

Parathyroid carcinoma is an indolent but progressive disease. Surgery is the mainstay of treatment. Early detection with attempts to remove local recurrence and distant metastasis can provide good short- and long-term control.

EP_A095

SEVERE HUNGRY BONE SYNDROME, COULD WE HAVE PROGNOSTICATED IT BETTER? A CASE SERIES

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INTRODUCTION/BACKGROUND

Hungry bone syndrome (HBS) is characterized by serum calcium less than 2.1 mmol/L and/or prolonged hypocalcaemia more than four days following parathyroidectomy or thyroidectomy. Here, we report two cases of HBS following parathyroidectomy for primary hyperparathyroidism (PHPT).

CASE 1

A 17-year-old female was diagnosed with PHPT when she presented with bilateral femoral neck and humeral fractures. Her serum calcium was 3.69 (2.2-2.65 mmol/L),

serum phosphate 0.92 (0.81- 1.45 mmol/L), and serum intact parathyroid hormone (iPTH) 1187.2 (14.9-56.9 pg/ml). There was deficient 25-hydroxy vitamin D at 18.9 (<25 nmol/L), ALP was elevated at 1413 (47-162 U/L) and renal function was normal. Ultrasound (US) of the parathyroid showed a lobulated hypoechoic lesion at the posterior right thyroid, measuring 1.6 x 2.3 x 3.3 cm. Preoperatively she received IV bisphosphonates and subcutaneous calcitonin. Haemodialysis was initiated prior to surgery for hypercalcaemic crises. Postoperatively, she required calcium gluconate infusion for 2 weeks.

CASE 2

A 53-year-old female had an incidental finding of hypercalcemia, which was subsequently confirmed to be PHPT. Serum calcium range was 2.92-3.02 mmol/L and phosphate 0.6-0.72 mmol/L. Serum iPTH 456.9 pg/ml, 25-Hydroxy vitamin D 58.97 (25-75 nmol/L), ALP 372 U/L, and renal profile was normal. Parathyroid ultrasound showed a left extrathyroidal lesion measuring 0.6 x 0.7 x 0.8 cm, concordant with her sestamibi scan. DEXA scan showed osteoporosis. Preoperatively, she received IV bisphosphonate and cholecalciferol. She was discharged on day 3 with serum calcium of 2.05 mmol/L. However, she was re-admitted a day later for HBS (calcium 1.67 mmol/L) requiring calcium gluconate infusion for 3 days.

RESULTS

These cases illustrated severe HBS following parathyroidectomy. HBS may ensue in patients with elevated PTH and ALP, radiological evidence of bone diseases, large volume or weight of parathyroid gland, higher preoperative calcium, and vitamin D deficiency.

CONCLUSION

Rigorous proactive measures and timely management of HBS may avert the detrimental effects of hypocalcaemia. Identifying the likelihood that patients may develop HBS before surgery is of utmost importance for successful management.

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SPONTANEOUS RESOLUTION OF PRIMARY HYPERPARATHYROIDISM WITH PARATHYROID APOPLEXY

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INTRODUCTION/BACKGROUND

Primary hyperparathyroidism (PHPT) is characterized by autonomous parathyroid hormone (PTH) secretion from one or more of the 4 parathyroid glands. Most cases are due to a single parathyroid adenoma with parathyroidectomy offering a potential cure, however, spontaneous resolution of PHPT does occur following parathyroid apoplexy but has been sparsely reported in the literature.

CASE

We present a 27-year-old Malay male with no known comorbidities, who first presented in May 2023 with symptomatic hypercalcemia [Corrected Calcium 4.26 mmol/L (N 2.2-2.6); phosphate of 1.67 mmol/L (N 0.87-1.45); PTH of 159.7 pmol/L (N 1.6-6.9)] and was diagnosed with PHPT secondary to left inferior parathyroid adenoma. He was also diagnosed with polycystic kidney disease considering a positive family history, renal impairment, and the presence of renal and liver cysts on imaging. With saline diuresis and a dose of 30 mg intravenous pamidronate, his calcium level improved to 3.19 mmol/L. Unfortunately, he was subsequently lost to follow-up.

He presented 4 months later with acute pain and increased neck swelling associated with numbness over the extremities and perioral region. Chvostek's sign was positive with a palpable anterior neck swelling measuring 2 x 2 cm. There was no palpable cervical lymphadenopathy. Biochemical analysis revealed severe hypocalcaemia [Corrected Calcium 1.69 mmol/L (N 2.2-2.6); Phosphate 0.51 mmol/L (N 0.87-1.45)] with a marked reduction in PTH level to 39 pmol/L (N 1.6-6.9). Intravenous calcium, oral calcium carbonate and calcitriol were simultaneously initiated. Repeated 99 mTc sestamibi imaging showed negative uptake, confirming the diagnosis of parathyroid apoplexy. He was maintained on calcium carbonate 1 g twice daily and calcitriol 0.5 mcg twice daily with his latest corrected calcium level at 2.24 mmol/L.