

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 $1.6 \times 2.3 \times 3.3$ 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 $0.6 \times 0.7 \times 0.8$ 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.

EP_A096

SPONTANEOUS RESOLUTION OF PRIMARY HYPERPARATHYROIDISM WITH PARATHYROID APOPLEXY

https://doi.org/10.15605/jafes.039.S1.107

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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.



CONCLUSION

Parathyroid apoplexy resulting in resolution of PHPT is uncommon and the best management approach (surgery or conservative) remains uncertain. As recurrence have been reported, long term monitoring is essential for patients managed conservatively.

EP_A097

A CASE OF PRIMARY HYPERPARATHYROIDISM COEXISTING WITH MONOCLONAL GAMMOPATHY OF UNDETERMINED SIGNIFICANCE (MGUS)

https://doi.org/10.15605/jafes.039.S1.108

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INTRODUCTION/BACKGROUND

Hypercalcemia is a relatively common clinical problem in hospitalized patients. Primary hyperparathyroidism and plasma cell dyscrasias such as multiple myeloma and monoclonal gammopathy of undetermined significance (MGUS) are known to be the most common causes of hypercalcemia. Although the occurrence of these disorders in one patient has been reported previously, it is still believed to be a rare phenomenon. We report a case of hypercalcemia, resulting from coexistent primary hyperparathyroidism and MGUS.

CASE

A 45-year-old female with no previous medical illness was admitted for symptomatic hypercalcemia. A review of symptoms during admission was significant for constipation, nausea, anorexia, polyuria and thirst. Initial blood investigations showed hypercalcemia (corrected calcium 4.43 mmol/L), renal impairment (creatinine 154 mmol/L) and anaemia (haemoglobin 9 g/dL). Hypercalcemia was managed with intravenous hydration, bisphosphonates and furosemide. Subsequent tests include elevated intact parathyroid hormone (820.4 pg/ml) and an elevated 24-hour urinary calcium/creatinine clearance ratio of 0.07, suggestive of primary hyperparathyroidism. This was further supported by the finding of a right parathyroid adenoma on neck ultrasound.

Due to an abnormal albumin/globulin ratio of 0.89, serum protein electrophoresis was also done which revealed IgG lambda paraproteinemia. The skeletal survey and bone scan were normal. Bone marrow and trephine biopsy showed the presence of clonal plasma cells at less than 10 percent, which confirmed the diagnosis of MGUS. The patient is currently under multidisciplinary care in endocrinology and haematology subspecialties. Sestamibi parathyroid scan has been arranged for preoperative localization. She is also being monitored closely for progression to multiple myeloma.

CONCLUSION

This case gives significant insights into potential concomitant causes of hypercalcemia. A high index of suspicion and a systematic approach to performing relevant screening tests are essential, as earlier diagnosis leads to improved clinical outcomes.

EP_A098

REVEALING THE HIDDEN MASK: A CASE ON PRIMARY HYPERPARATHYROIDISM MIMICKING PREGNANCY SYMPTOMS

https://doi.org/10.15605/jafes.039.S1.109

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INTRODUCTION/BACKGROUND

Parathyroid adenoma in pregnancy is uncommon, posing diagnostic and treatment challenges. We report a case of primary hyperparathyroidism (PHPT) due to a parathyroid adenoma successfully treated with surgery during pregnancy.

CASE

A 20-year-old female, gravida 2 para 0+1, presented with vomiting since the fourth week of pregnancy. She also reported experiencing left shoulder pain, abdominal discomfort, polyuria, nocturia, and constipation. Initially diagnosed with hyperemesis gravidarum, further investigation revealed hypercalcemia with a serum calcium level of 3.66 mmol/L, phosphate level of 0.7 mmol/L, and markedly elevated intact parathyroid hormone (iPTH) at 15.53 pmol/L (normal value: 1.6-6.9 pmol/L). Despite attempts to lower calcium levels through hydration and diuresis with furosemide, her serum calcium remained elevated at 3.14 mmol/L. She received six doses of subcutaneous calcitonin due to persistent hypercalcemia, resulting in a reduction of calcium to 2.83 mmol/L. Ultrasonography of the neck detected a left superior parathyroid adenoma. At 25 weeks of gestation, she underwent a successful left superior parathyroidectomy with intraoperative parathyroid hormone monitoring, resulting in positive outcomes for both mother and baby.