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SEVERE OSTEOPOROSIS WITH FRAGILITY FRACTURE REVEALING PRIMARY HYPERPARATHYROIDISM

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INTRODUCTION

Primary hyperparathyroidism (PHPT) is frequently asymptomatic or detected incidentally; however, delayed diagnosis may lead to severe skeletal complications. Early recognition is essential, as timely identification and management of parathyroid disease can prevent significant morbidity, although diagnosis may be challenging when clinical and imaging findings are inconclusive.

CASE

A 46-year-old female with severe bilateral hearing impairment presented to the orthopedic clinic with 3 years' history of bilateral knee pain and was found to have a right intertrochanteric femur fracture following minimal trauma. She was referred for evaluation of suspected secondary osteoporosis. She has had intermittent constipation and long-standing oligomenorrhea since menarche. There was no history of childhood fractures or use of medications affecting bone metabolism. Examination revealed bilateral knee bowing.

Initial evaluation considered metabolic bone disease, including Paget's disease; however, skeletal survey showed no features suggestive of Paget's disease or multiple myeloma.

Biochemical investigations demonstrated persistent hypercalcemia (2.65–3.1 mmol/L) with inappropriately elevated intact parathyroid hormone (peak 7.62 pmol/L), consistent with PHPT. Serum phosphate was low-normal. Concomitant vitamin D deficiency (25-OH vitamin D 34.46 nmol/L) improved following replacement. Bone mineral density confirmed severe osteoporosis (lumbar spine T-score -5, z-score -4.1); (forearm -6.7, z-score -6.1) reflecting prolonged untreated disease.

Neck ultrasound demonstrated a mixed solid-cystic lesion posterior to the right thyroid lobe, suggestive of a parathyroid adenoma. The TC-99 m Sestamibi scan showed no definite focal uptake. However, subsequent SPECT-CT revealed focal tracer uptake at the posterior right thyroid gland, consistent with a hyperfunctioning parathyroid gland. Parathyroidectomy was done. Postoperatively, the calcium level normalized.

CONCLUSION

Severe osteoporosis and fragility fracture occur, reflecting prolonged exposure to excess parathyroid hormone and significant skeletal morbidity. Early biochemical evaluation in unexplained severe osteoporosis is essential, as timely diagnosis and definitive management of parathyroid disease are critical to halt ongoing bone loss and prevent irreversible complications.

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A CHALLENGING CASE OF PARATHYROID CARCINOMA IN AN ADOLESCENT

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INTRODUCTION

Parathyroid carcinoma (PC) is an exceedingly rare malignancy. Definitive surgical management with en bloc resection is crucial for cure, but the post-operative course can be complicated by profound metabolic derangements, most notably hungry bone syndrome (HBS). We report a case of PC in a 15-year-old female to highlight the challenges in perioperative management.

CASE

A 15-year-old female presented with a painless, palpable neck mass. There is no family history of note. Investigations revealed severe primary hyperparathyroidism (PHPT) with a corrected calcium of 3.4 mmol/L and an elevated intact parathyroid hormone level of 118.6 pg/mL. Alkaline phosphatase was markedly elevated at 1,168 U/L. Ultrasound, computed tomography neck, and a sestamibi scan identified a large, lobulated 4.7-cm mass posterior to the right thyroid lobe, suspicious of malignancy. Bone mineral density of the forearm was severely diminished, with a Z-score of -6.6. The patient underwent right hemithyroidectomy and parathyroidectomy with intraoperative neural monitoring. Intraoperative parathyroid hormone levels dropped from a pre-excision level of 120.1–16.5 pg/mL 5 minutes post-excision, confirming complete resection of the hyperfunctioning tissue. Histopathological examination confirmed the diagnosis of PC, demonstrating lymphovascular invasion and clear resection margins. The Ki-67 proliferation index was 5%. Post-operatively, the patient developed hypocalcemia, with corrected calcium dropping to a nadir of 1.95 mmol/L. This was managed with intensive calcium and activated vitamin D supplementation. At 10 months post-surgery, the patient continues to require supplementation for persistent hypocalcemia. Surveillance ultrasound at 3 months showed no evidence of recurrence, and she is planned for ongoing annual monitoring.

CONCLUSION

This case illustrates the need for a high index of suspicion for PC in young patients with severe PHPT. The post-operative course highlights the challenges in managing HBS, hypoparathyroidism, and long-term surveillance.

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PACLITAXEL-INDUCED HYPOCALCEMIA IN A PATIENT WITH METASTATIC BREAST DISEASE AND UNDERLYING HYPOPARATHYROIDISM

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INTRODUCTION

Hypocalcemia in patients with advanced malignancy is usually attributed to bone metastases, vitamin D deficiency, renal impairment, or antiresorptive therapy. Paclitaxel, a taxane-based chemotherapy agent widely used for breast cancer, is not commonly associated with calcium disturbances. Proposed mechanism includes renal tubular dysfunction, renal salt wasting, and disruptions in bone metabolism. In patients with underlying disorders of calcium homeostasis such as hypoparathyroidism, taxane-based chemotherapy such as Docetaxel and Paclitaxel may exacerbate calcium imbalance. We reported a case of recurrent hypocalcemia associated with paclitaxel therapy in a patient with metastatic breast cancer.

CASE

A 42-year-old female with metastatic breast cancer, involving the liver and bones, had previously undergone neoadjuvant chemotherapy, mastectomy, and adjuvant radiotherapy. Following the disease progression, she was commenced on weekly intravenous paclitaxel at a 20% dose reduction due to prior complications and underlying metabolic risk. She had a history of post-thyroidectomy hypoparathyroidism and had previously been intolerant to docetaxel during the neoadjuvant chemotherapy, which was complicated by hypocalcemia, likely secondary to renal salt wasting.

During paclitaxel treatment, she developed recurrent symptomatic hypocalcemia, requiring multiple hospital admissions and repeated intravenous calcium gluconate infusions despite ongoing oral calcium and calcitriol supplementation, which were temporarily increased during

the chemotherapy. These episodes occurred intermittently in temporal association with paclitaxel administration, with other causes of hypocalcemia were considered less likely.

CONCLUSION

Hypocalcemia associated with paclitaxel is rarely described in literature. This case highlights the importance of monitoring calcium level in patients receiving paclitaxel, particularly in those with pre-existing hypoparathyroidism.

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BEYOND THE OBVIOUS: UNMASKING PARATHYROID CARCINOMA IN AN ATYPICAL PRESENTATION

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INTRODUCTION

Incidental hyperparathyroidism with hypercalcemia is not uncommon. However, parathyroid carcinoma is an extremely rare endocrine malignancy, accounting for less than 1% of cases of primary hyperparathyroidism.

CASE

A 57-year-old Malay female with well-controlled type 2 diabetes was incidentally found to have hypercalcemia during hospitalization for pyelonephritis. She was asymptomatic, with no history of calcium supplementation or family history of endocrine disorders. Physical examination revealed no obvious neck swelling, and other systemic examinations were unremarkable. Investigations showed elevated serum calcium (2.87–3.4 mmol/L), low phosphate (0.34–0.76 mmol/L), and elevated intact parathyroid hormone (iPTH) (6.81 pmol/L). Her 25-OH vitamin D was deficient (45.58 nmol/L). Ultrasound of the neck revealed a solid lesion (1.6 × 2.0 × 2.8 cm) posterior to the right thyroid lobe with bilateral thyroid nodules, with the highest TR4, and normal cervical lymph nodes. Sestamibi scan showed multinodular goiter with cold nodules in bilateral thyroid lobes and a soft tissue lesion posterior to the right thyroid lobe. Subsequently, she underwent right hemithyroidectomy with intraoperative nerve monitoring and right inferior parathyroidectomy with intraoperative iPTH monitoring; the intraoperative iPTH level dropped appropriately from 24 to 2.4 pmol/L. Histopathological examination of the excised right parathyroid gland revealed parathyroid carcinoma, while the right thyroid lobe showed nodular hyperplasia. Post-operatively, calcium and iPTH levels normalized, and she remained well under follow-up.