

EP_A090

INVISIBLE PARATHYROID ADENOMA WITH REFRACTORY HYPERCALCEMIA IN PREGNANCY

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INTRODUCTION/BACKGROUND

Hypercalcemia in pregnancy is an uncommon event that can have serious consequences on both the mother and foetus. We describe a patient with refractory parathyroid hormone-dependent hypercalcemia in pregnancy.

CASE

A 39-year-old female presented with asymptomatic moderate hypercalcemia (corrected calcium: 3.35 mmol/L and iPTH: 24.1 pmol/L) during the third trimester of her first pregnancy in 2021, requiring subcutaneous calcitonin before elective Caesarean section. Post-delivery, her serum calcium level remained between 2.7-2.9 mmol/L, with PTH level of 10.6 pmol/L. She underwent several imaging investigations including neck ultrasound, sestamibi parathyroid scan and computed tomography of the neck but all failed to localize a parathyroid lesion. The urine calcium creatinine ratio was inconclusive.

She conceived again in 2023 while still being investigated for primary hyperparathyroidism. During this second pregnancy, she had recurrent admissions for asymptomatic hypercalcemia, with the highest calcium level of 3.3 mmol/L, iPTH 14.8 pmol/L and PTH-related peptide <0.4 pmol/L. Neck ultrasound showed only bilateral thyroid nodules with no lesions suggestive of parathyroid adenoma. Ultrasound of the kidneys showed bilateral nephrocalcinosis. She refused to undergo exploration parathyroidectomy. Due to refractory hypercalcemia ranging from 2.9-3.3 mmol/L despite intravenous hyperhydration, she was started on cinacalcet until delivery. The delivery via Caesarean section was uneventful, and both patient and baby were well. Four days after delivery, she underwent sestamibi and CT neck that showed a small hyperfunctioning parathyroid adenoma inferior to the right thyroid lobe. Interestingly, her calcium levels decreased to 2.8 mmol/L post-delivery. She was then referred to the surgical team for parathyroidectomy.

CONCLUSION

Investigation and management of PTH-dependent hypercalcemia in pregnancy is challenging due to limited options in imaging modalities and medical interventions.

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A CHALLENGING CASE OF PERSISTENT HYPERCALCEMIA POST TOTAL PARATHYROIDECTOMY IN A DIALYSIS-DEPENDENT PATIENT

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INTRODUCTION/BACKGROUND

Tertiary hyperparathyroidism is a frequently encountered complication of advanced chronic kidney disease, characterized by an increase in parathyroid hormone (PTH) levels due to disturbances in calcium, phosphate, and vitamin D homeostasis. In most cases, total parathyroidectomy results in the resolution of hypercalcemia. However, primary hyperparathyroidism occurring in the context of tertiary hyperparathyroidism is a rare occurrence. We present a challenging case of persistent hypercalcemia in a dialysisdependent patient following total parathyroidectomy.

CASE

A 41-year-old female was diagnosed with dialysisdependent end-stage kidney disease at the age of 27 due to hypertension in the young. In 2014, she was diagnosed with tertiary hyperparathyroidism and underwent two staged operations for total parathyroidectomy in 2014 and 2020. Despite the procedure, she experienced persistent hypercalcemia, with serum calcium levels reaching 2.8-2.86 mmol/L (2.1-2.55) and phosphate levels of 1.49- 2.18 mmol/L (0.74-1.52). Her serum iPTH was 71.4 pmol/L (1.6-6.9) and 25 (OH) D was 30 nmol/L. A DEXA scan showed severe osteoporosis, with a T-score of -3.4 and Z-score of -2.6 in the lumbar spine, and a T-score of -4.6 and Z-score of -4.2 in the 1/3 radius.

Sestamibi imaging performed in December 2023 demonstrated the presence of sestamibi- avid ectopic parathyroid tissue within the inferior pole of the left thyroid lobe and superior mediastinum, measuring 1.2×1.1 cm at the level T5 vertebra. Correlating with features of post-total parathyroidectomy for tertiary hyperparathyroidism, this may represent an ectopic parathyroid adenoma. While waiting for definitive surgical management, the patient was administered subcutaneous Denosumab 60 mg every 6 months with calcium and vitamin D supplementation.

CONCLUSION

The diagnosis and management of primary hyperparathyroidism in patients with advanced chronic kidney disease presents a distinct set of challenges. In the absence of



a clearly identifiable parathyroid adenoma, it is imperative to employ a combination of imaging techniques to identify any possible ectopic focus, which yields the maximum benefit. Following localization, surgical resection continues to be the preferred mode of treatment for achieving a permanent cure.

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COMPLEX SCENARIO OF MEN 1 WITH ECTOPIC PARATHYROID GLAND: A CASE REPORT

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INTRODUCTION/BACKGROUND

In the context of Multiple Endocrine Neoplasm 1 (MEN1), primary hyperparathyroidism (PHPT) is the most prevalent form of endocrinopathy and is often the earliest endocrine manifestation among patients. It represents 2–4% of all forms of PHPT. Ectopic parathyroid adenomas (EPTA) account for a significant proportion, approximately 22% of PHPT cases. To mitigate the adverse effects of PHPT in MEN1 patients, the optimal course of treatment is parathyroidectomy. We present a complex case of MEN1 that involves an ectopic parathyroid gland.

CASE

A 54-year-old female presented with symptomatic hypercalcemia with a serum calcium of 3.63 mmol/L (2.1-2.55) along with multiple duodenal ulcers and a Hb of 8.7g/dL (12-15) in 2008. Clinical diagnosis of Multiple Endocrine Neoplasia 1 was made, as validated by primary hyperparathyroidism, microprolactinoma and non-functioning pancreatic neuroendocrine tumour grade 1. She underwent total parathyroidectomy in July 2008 with a right inferior auto-transplantation into the sternocleidomastoid muscle. Histopathological analysis confirmed parathyroid hyperplasia in all 4 glands. Ten years later, she exhibited an increasing trend of serum calcium 2.57-2.63 mmol/L and iPTH (7.05->12.89->14.4 pmol/L) (1.58-6). Neck ultrasonography revealed a welldefined elongated hypoechoic structure within the right sternocleidomastoid muscle measuring 0.2 x 0.4 x 0.9 cm (AP x W x CC). Parathyroid scintigraphy Tc99M Sestamibi with SPECT-CT demonstrated the presence of an ectopic parathyroid adenoma measuring 0.7 x 0.9 cm at the right upper paratracheal/suprasternal region. Subsequently, she underwent exploratory parathyroidectomy with the removal of the right auto-transplant parathyroid gland and right thymus. Histopathological analysis was consistent with parathyroid hyperplasia and ectopic parathyroid accordingly. Postoperatively she remained hypercalcaemic 2.7 mmol/L with non-suppressible iPTH 27.46 pmol/L. Levels of 25-OH (D) were insufficient at 40.72 nmol/L. Further localization studies were contemplated. However, 4D CT assessment was not done due to her deteriorating renal function. She was given oral cholecalciferol 1000 IU daily and cabergoline 1 mg daily.

CONCLUSION

Despite significant progress in imaging technologies and surgical techniques, the management of EPTA remains a challenging task in clinical practice. Specialized multidisciplinary input is crucial in managing such cases.

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A MORE SINISTER CAUSE OF LOWER BACK PAIN IN THE THIRD TRIMESTER: A CASE REPORT OF PREGNANCY AND LACTATION-ASSOCIATED OSTEOPOROSIS

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INTRODUCTION/BACKGROUND

Pregnancy and lactation-associated osteoporosis (PLO) is a rare but painful condition that tends to occur during the third trimester or postpartum period, with an incidence of 0.4 cases/100,000 women and 70% of those affected are primiparous. The main symptom is severe lower back pain as this condition often causes vertebral fractures which can be multiple.

CASE

We present a case of a 28-year-old female with an underlying right coronary artery fistula and endometriosis. She is para 1 and delivered her child in December 2022. During this pregnancy, she had a history of severe back pain during the third trimester. There was no history of falls or any neurological deficits. She gave a history of coccyx fracture following a fall eight years ago but recovered uneventfully. She breastfed her baby for five months post-partum and her back pain persisted during this period which prompted further investigations for her. She did not consume any steroids and there were no signs and symptoms to suggest Cushing's Syndrome.