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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