RESEARCH PAPER

Sestamibi-Negative Ectopic Mediastinal Parathyroid Adenoma Presenting With Severe Hypercalcemia and Acute Kidney Injury: A Case Report

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ABSTRACT

Background: Primary hyperparathyroidism (PHPT) is most commonly caused by a solitary parathyroid adenoma. Ectopic parathyroid adenomas are uncommon and may pose significant diagnostic challenges, particularly when located in the mediastinum and when conventional localization studies are negative.

Case Presentation: A 42-year-old male presented with abdominal pain, vomiting, loss of appetite, weight loss, and generalized body ache for two months. He had previously been diagnosed with hypercalcemia-associated pancreatitis and was treated at multiple healthcare facilities. Evaluation for persistent hypercalcemia raised suspicion of primary hyperparathyroidism; however, a sestamibi scan failed to demonstrate abnormal uptake, and serum parathyroid hormone (PTH) values were variable. He was subsequently referred to our centre with severe hypercalcemia and acute kidney injury. Computed tomography Neck revealed a well-defined lesion measuring 2.7 × 2.3 × 2.8 cm in the right paratracheal region of the upper mediastinum. PTH estimation from aspirated fluid was markedly elevated at 5000 pg/mL. The patient underwent surgical excision of the lesion. His preoperative PTH was 2434 pg/mL, which decreased to 173 pg/mL intraoperatively. Histopathological examination confirmed parathyroid adenoma. At one-year follow-up, the patient remained asymptomatic with normal serum calcium and PTH levels and a serum creatinine of 1.3 mg/dL.

Conclusion: This case highlights the importance of considering ectopic mediastinal parathyroid adenoma in patients with PTH-mediated hypercalcemia despite negative sestamibi imaging. Complementary anatomical imaging and targeted biochemical confirmation can facilitate localization and successful definitive surgical treatment.

Keywords: Primary hyperparathyroidism; ectopic parathyroid adenoma; mediastinal parathyroid adenoma; hypercalcemia; sestamibi-negative; acute kidney injury.

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INTRODUCTION

Primary hyperparathyroidism (PHPT) is an endocrine disorder characterized by autonomous and excessive secretion of parathyroid hormone (PTH), resulting in hypercalcemia and disturbances in phosphate and bone metabolism. The most common cause of PHPT is a solitary parathyroid adenoma, followed by parathyroid hyperplasia.

Ectopic parathyroid glands occur because of variations in the embryological migration of the parathyroid glands. Ectopic glands may be located at various sites along their migratory pathway, including the thymus, carotid sheath, retroesophageal region, intrathyroidal location, and mediastinum. Although ectopic parathyroid tissue is relatively common, mediastinal parathyroid adenomas are uncommon and can present significant diagnostic and surgical challenges.

Parathyroidectomy is the definitive treatment for PHPT, with a high rate of cure when the hyperfunctioning gland is accurately localized and completely excised. Technetium-99m sestamibi scintigraphy is widely used for preoperative localization; however, false-negative scans may occur. Therefore, additional imaging modalities may be required in patients with strong biochemical evidence of PHPT and non-localizing conventional imaging.

We report a case of a sestamibi-negative ectopic mediastinal parathyroid adenoma presenting with severe hypercalcemia and acute kidney injury, successfully diagnosed using complementary imaging and PTH estimation from aspirated lesion fluid.

CASE PRESENTATION

A 42-year-old male, with no history of hypertension or diabetes mellitus, presented with complaints of abdominal pain, vomiting, loss of appetite, weight loss, and generalized body ache for approximately two months.

The patient had initially consulted multiple healthcare providers and was diagnosed with hypercalcemia-associated pancreatitis, for which he received treatment. As hypercalcemia persisted, further evaluation was undertaken. Primary hyperparathyroidism was suspected, and a technetium-99m sestamibi scan was performed; however, it did not demonstrate significant abnormal tracer uptake. Serum PTH values obtained during the initial evaluation were reported to be variable.

The patient was subsequently referred to our centre and admitted with severe hypercalcemia and acute kidney injury. He was managed with intravenous hydration and symptomatic treatment. With adequate hydration, there was a gradual improvement in renal function. An ultrasound examination performed previously showed normal-sized kidneys.

Further evaluation for the cause of hypercalcemia was undertaken. CT imaging of the neck and thoracic inlet revealed a well-defined iso-hyperdense lesion measuring approximately 2.7 × 2.3 × 2.8 cm in the right paratracheal region, posterior to the sternal angle at the level of the D3 vertebral body. Multiple lytic lesions were also noted.

The patient received a single dose of intravenous zoledronic acid 2 mg for management of severe hypercalcemia, and serum electrolytes were monitored regularly.

Given the atypical location of the lesion and negative sestamibi imaging, an interventional radiology opinion was obtained. Image-guided aspiration of fluid from the suspected lesion was performed, and the aspirated fluid was sent for PTH estimation. The PTH level in the aspirated fluid was markedly elevated at approximately 5000 pg/mL, strongly supporting the diagnosis of ectopic functioning parathyroid tissue.

The patient and family were counselled regarding the abnormal location of the suspected parathyroid adenoma, and surgical consultation was obtained. He subsequently underwent surgical excision of the mediastinal lesion. The preoperative serum PTH level was 2434 pg/mL, which decreased to 173 pg/mL intraoperatively. Histopathological examination of the excised specimen confirmed the diagnosis of parathyroid adenoma.

The postoperative recovery was uneventful. The patient was closely monitored for electrolyte disturbances and was started on calcium and vitamin D supplementation. He demonstrated significant symptomatic improvement and was discharged with improvement in renal function.

At one-and-a-half-month follow-up, the patient was asymptomatic, with improved appetite and approximately 10 kg weight gain. At one-year follow-up, he continued to remain clinically asymptomatic. Serum calcium and PTH levels were within the normal range, indicating sustained biochemical remission. His serum creatinine was 1.3 mg/dL, demonstrating stable renal function.

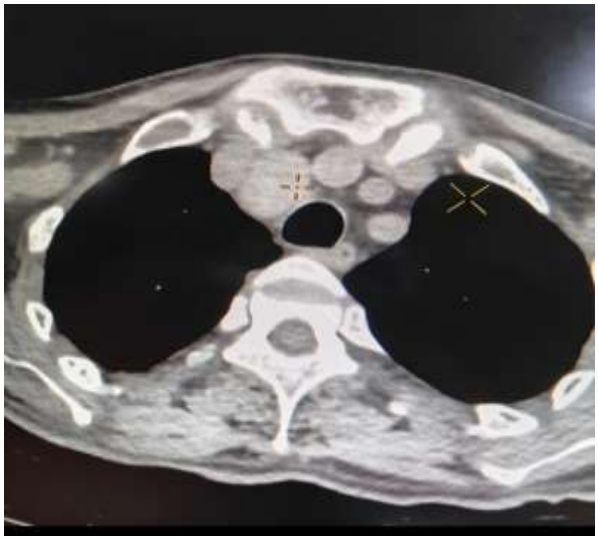

Figure 1. CT Neck Well Defined Iso Hyperdense Area, Approximately Measuring 2.7*2.3*2.8 Cm Noted in Right Paratracheal Region, Posterior to Sternal Angle at the Level of D3 Vertebral Body

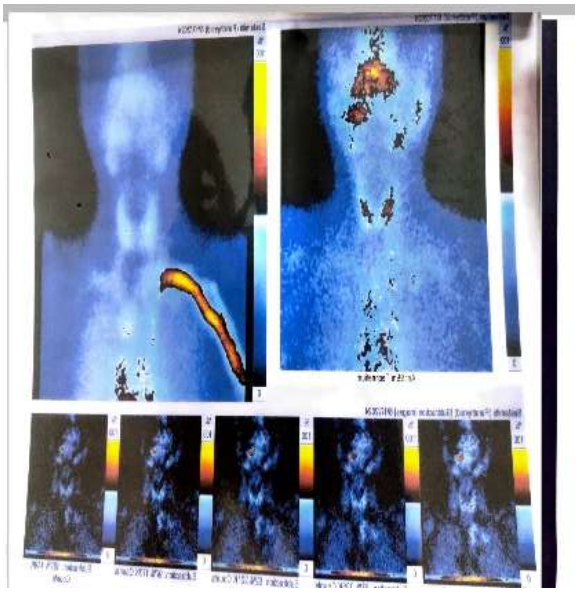

Figure 2. Technetium-99m Sestamibi-Normal

INVESTIGATIONS

Laboratory Parameters

Date	Serial laboratory findings
15 June	Intact PTH 424 pg/mL; magnesium 1.5 mg/dL; 25-OH vitamin D 34.3 ng/mL.
29 June	Serum creatinine 2.4 mg/dL; serum lipase 1000 U/L; serum amylase 245 U/L.
2 July	Creatinine 3.26 mg/dL; urea 50.6 mg/dL; sodium 139 mmol/L; potassium 4.06 mmol/L; calcium 22.6 mg/dL; phosphorus 3.7 mg/dL; total protein 6.7 g/dL; albumin 3.6 g/dL; hemoglobin 7.2 g/dL; platelet count 139 × 10⁹/L; WBC count 4200/µL.
4 July	Creatinine 2.69 mg/dL; potassium 2.74 mmol/L; bicarbonate 20.7 mmol/L; calcium 19.8 mg/dL; phosphorus 4.3 mg/dL; ALP 295 IU/L; hemoglobin 8.9 g/dL; platelet count 149 × 10⁹/L.
7 July	Creatinine improved to 1.83 mg/dL; potassium 2.93 mmol/L; bicarbonate 16.9 mmol/L; calcium decreased to 9.8 mg/dL.
11 July	Creatinine 1.19 mg/dL; potassium 3.99 mmol/L; bicarbonate 13.9 mmol/L; calcium 9.6 mg/dL; intact PTH markedly elevated at 2434 pg/mL.
12 July	Calcium 9.4 mg/dL; intact PTH 173 pg/mL.
19 July	Calcium 7.1 mg/dL; hemoglobin 10.2 g/dL; platelet count 183 × 10⁹/L; WBC count 3700/µL.
20 July	Magnesium 0.7 mg/dL; intact PTH 12.5 pg/mL.
21 July	Calcium 7.8 mg/dL; phosphorus 2.3 mg/dL; magnesium 1.1 mg/dL.
26 July	Creatinine 1.16 mg/dL; bicarbonate 15.2 mmol/L; calcium 7.6 mg/dL; ALP 979 IU/L.
12 September (follow-up)	Creatinine 1.41 mg/dL; urea 41.6 mg/dL; bicarbonate 19.5 mmol/L; calcium improved to 8.8 mg/dL; hemoglobin 11.1 g/dL; platelet count 194 × 10⁹/L; WBC count 6200/µL.

Abbreviations: ALP, alkaline phosphatase; PTH, parathyroid hormone; WBC, white blood cell; 25-OH vitamin D, 25-hydroxyvitamin D.

DISCUSSION

Primary hyperparathyroidism is characterized by excessive secretion of parathyroid hormone, resulting in hypercalcemia and associated disturbances in phosphate

and bone metabolism. Parathyroid adenoma is the most common cause of PHPT, while ectopic parathyroid tissue may occur because of variations in the embryological migration of the parathyroid glands [1,2].

Ectopic parathyroid adenomas are diagnostically challenging, particularly when located within the mediastinum. Accurate preoperative localization is essential for successful surgical management and prevention of persistent or recurrent disease. Various imaging modalities, including ultrasonography, technetium-99m sestamibi scintigraphy, and computed tomography, may be required for localization [1,3].

Technetium-99m sestamibi scintigraphy is commonly used for preoperative localization of parathyroid adenomas; however, false-negative scans can occur. Therefore, a negative sestamibi scan does not exclude the presence of a functioning parathyroid adenoma. In our patient, despite a negative sestamibi scan, CT imaging identified a well-defined lesion in the right paratracheal mediastinal region. Subsequent PTH estimation from aspirated fluid demonstrated a markedly elevated PTH level of 5000 pg/mL, supporting the diagnosis of ectopic functioning parathyroid tissue [1,2].

The patient presented with severe symptomatic hypercalcemia and acute kidney injury. Hypercalcemia can contribute to renal dysfunction, and improvement in renal function following correction of hypercalcemia and intravenous hydration supported the reversible component of kidney injury in this patient. The presence of multiple lytic lesions also suggested significant skeletal involvement in the setting of prolonged hyperparathyroidism.

The markedly elevated preoperative serum PTH level of 2434 pg/mL and its significant decline to 173 pg/mL intraoperatively further supported successful removal of the hyperfunctioning lesion. Intraoperative PTH monitoring is a useful adjunct in confirming adequate excision during parathyroid surgery [3].

Histopathological examination confirmed parathyroid adenoma. The patient demonstrated marked clinical improvement following surgical excision. At one-year follow-up, he remained asymptomatic with normal serum calcium and PTH levels. His serum creatinine was stable at 1.3 mg/dL, supporting sustained biochemical remission and long-term successful treatment.

This case highlights the importance of thorough evaluation in patients presenting with severe hypercalcemia. When biochemical findings strongly suggest primary hyperparathyroidism, negative sestamibi imaging should not preclude further investigation. The use of complementary imaging modalities and targeted biochemical confirmation can facilitate accurate localization of ectopic parathyroid adenomas and enable successful definitive surgical treatment [1–3].

CONCLUSION

Ectopic mediastinal parathyroid adenoma is an uncommon but important cause of primary hyperparathyroidism and may present with severe systemic manifestations, including hypercalcemia and acute kidney injury. This case demonstrates that a negative sestamibi scan does not exclude a functioning ectopic parathyroid adenoma. Persistent clinical suspicion and biochemical evidence of primary hyperparathyroidism should prompt further evaluation using complementary imaging modalities. In this patient, CT imaging successfully localized the mediastinal lesion, while markedly elevated PTH levels in aspirated fluid provided additional confirmation of its parathyroid origin. Definitive surgical excision resulted in significant clinical and biochemical improvement. Long-term follow-up at one year demonstrated sustained remission, with normal serum calcium and PTH levels and stable renal function. This case emphasizes the importance of a multimodal diagnostic approach for accurate localization and successful management of ectopic mediastinal parathyroid adenomas.

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