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Case Report

Occult Pulmonary Neuroendocrine Tumor Causing Severe Hyperparathyroidism: Diagnostic Challenges and Clinical Outcomes

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ABSTRACT

Background/Objective: Ectopic tumoral production of parathyroid hormone (PTH) is rare. The incidence of hyperparathyroidism and osteitis fibrosa cystica (OFC) secondary to ectopic PTH secretion has only been reported in case reports, although infrequent.
Case Report: We report a case of a well-differentiated pulmonary neuroendocrine tumor (NET) producing PTH that presented with severe hypercalcemia and OFC. Surgical removal of the pulmonary tumor resulted in resolution of hypercalcemia. Immunocytochemical analysis of the tumor tissue revealed PTH-positive staining. Recovery was complicated by severe hypocalcemia due to hungry bone syndrome.
Discussion: To the best of our knowledge, this is the first documented case of a pulmonary NET causing OFC via PTH. We further describe the successful identification and resection of a rare NET and restoration of calcium homeostasis with aggressive calcium and vitamin D repletion.
Conclusion: Although a rare cause of severe hypercalcemia and OFC, ectopic tumoral production of PTH must be considered in the differential diagnosis. Furthermore, resection of these tumors secreting PTH can lead to a protracted and severe high risk of hungry bone syndrome, which requires aggressive treatment to maintain calcium homeostasis.

Introduction

Hypercalcemia due to ectopic parathyroid hormone (PTH) production is rare with few reported cases occurring from malignancies within pulmonary and gastrointestinal systems.[1-3] The incidence of osteitis fibrosa cystica (OFC) secondary to hyperparathyroidism has been reported in a small number of cases.[4] We report a case of a 39-year-old woman with a well-differentiated PTH-secreting pulmonary neuroendocrine tumor (NET) who presented with life-threatening manifestations of hyperparathyroidism.

Case Report

A 39-year-old woman with a past medical history of chronic lumbar radiculopathy secondary to degenerative disc disease presented with 2 weeks of ambulatory dysfunction due to progressive right thigh pain. She also reported chronic nausea and vomiting, constipation, and unintentional weight loss over 9 months. Family history was notable for immediate relatives with unspecified thyroid disease and extended relatives with an unspecified bone malignancy and breast cancer.

Initial blood work revealed a severely elevated serum calcium level of 17.5 mg/dL, with a corrected value of 18.0 mg/dL (8.6-10.2 mg/dL), phosphate level of 3.4 mg/dL (2.5-4.5 mg/dL), and creatinine level of 2.17 mg/dL (0.50-1.10 mg/dL). Radiography of the right femur showed a large lytic lesion in the proximal femur measuring approximately 10.0 × 2.7 cm with cortical destruction medially and endosteal scalloping laterally. Magnetic resonance imaging of the right femur showed a lobulated eccentric expansile mass, a minimally displaced pathologic fracture of the proximal femur, and innumerable intracortical foci of signal abnormality within the

Abbreviations: CT, computed tomography; IV, intravenous; NET, neuroendocrine tumor; OFC, osteitis fibrosa cystica; PTH, parathyroid hormone; PTHrP, parathyroid hormone-related protein.

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bone with diffuse circumferential fluid (Fig. 1 *A*). The PTH, 25-dihydroxyvitamin D, and 1-25-dihydroxyvitamin D levels were >2000 pg/mL (19-88 pg/mL), 11 ng/mL (30-100 ng/mL), and <8 pg/mL (21-29 ng/mL), respectively. The PTH-related protein (PTHrP) level was 17 pg/mL (14-27 pg/mL). Hypercalcemia was treated with aggressive intravenous (IV) hydration, intramuscular calcitonin, and IV pamidronate as detailed in Table 1. Vitamin D was not supplemented prior to surgical intervention (Table 1).

Computed tomography (CT) angiography of the thorax noted a well-circumscribed homogeneous soft tissue nodule in the right lower lung lobe measuring 3 × 3 × 3 cm, with concern for a pulmonary NET (Fig. 1 *B*). With concern for malignancy, cross-sectional imaging was performed for diagnostic and staging purposes. CT of the abdomen and pelvis without contrast demonstrated nonobstructing nephrolithiasis of the left kidney. CT with and without contrast of the neck identified a possible 6-mm left inferior pole parathyroid lesion; however, ultrasound of the neck was unremarkable. Orthopedic intervention with open reduction, internal fixation, and biopsy of the femoral lesion was performed. Pathology showed a bone with increased resorption, peritrabecular fibrosis, granulation-like tissue with hemosiderin, as well as hemorrhage and giant cells without evidence of malignancy. These findings were compatible with a brown tumor and OFC, and staining ruled out metastatic carcinoma and plasmacytoma.

A whole-body technetium-99m sestamibi scan showed no uptake in the neck. However, there was mild to moderate uptake in the pulmonary mass and fundus of the stomach (Fig. 1 *C*). Biopsy of the lung mass was consistent with a well-differentiated NET. Immunohistochemical staining was positive for chromogranin A, CD56, and low Ki-67 level (<2%) and negative for synaptophysin. PTH staining was positive (Fig. 2 *A* through *F*).

Hypercalcemia was minimally responsive to medical therapies (corrected calcium level of 15.7 mg/dL). The patient underwent right robotic-assisted thoracoscopy with mediastinal lymph node dissection and right lower lobectomy on hospital day 14. The PTH levels decreased from 687 pg/mL intraoperatively to 177 pg/mL 3 hours after surgery and normalized at 24 hours to 44 pg/mL. Surgical pathology confirmed the diagnosis of a pulmonary NET secreting PTH (Fig. 2 *H*).

After tumor resection, the patient developed symptomatic hypocalcemia, with nadir on postoperative day 4 (Tables 1 and 2 and Fig. 2 *H*). She had a prolonged course of hungry bone syndrome for 1 month. On postoperative days 0 to 15, the ranges of levels were as follows: (1) calcium, <5.0 to 8.9 mg/dL; (2) PTH, 44 to 363 pg/mL; (3) phosphorus, 1.3 to 3.4 mg/dL; (4) potassium, 3.8 to 4.5 mmol/L; and (5) magnesium, 1.4 to 1.8 mg/dL (Table 2). Calcium gluconate infusions, oral calcium, vitamin D, and calcitriol were used to maintain the calcium level within the normal range (Table 3). Furthermore, the native parathyroid began producing PTH at levels of 44, 205, and 170 pg/mL on postoperative days 1, 2, and 3, respectively. By postoperative day 15, the PTH and calcium levels normalized. She was discharged on calcitriol 2 μg twice daily, calcium carbonate 5000 mg every 6 hours, and vitamin D 10 000 units daily.

Following discharge, endosonography of the gastric fundus did not show an intramural or subepithelial lesion significant for an NET. Normalization of the PTH levels along with the absence of pathology on upper endoscopy provided objective evidence that there was no residual disease postoperatively. At 1 month postoperatively, the 25-hydroxyvitamin D level improved to 49 ng/mL (30-100 ng/mL) with supplemental vitamin D. She also completed chest CT without contrast, which showed no evidence of recurrent or residual disease status post right lower lobectomy.

Highlights

- To the best of our knowledge, this is the first reported case of a well-differentiated neuroendocrine tumor of the lung secreting ectopic parathyroid hormone (PTH), leading to a brown tumor
- A whole-body technetium-99m parathyroid sestamibi scan is important to determine the location of PTH secretion
- Hypocalcemia secondary to hungry bone syndrome can be severe and have a protracted course. It requires prompt treatment because of its severity and may require massive doses of calcium supplementation to maintain calcium homeostasis

Clinical Relevance

This is the first reported case of a well-differentiated neuroendocrine tumor of the lung secreting ectopic parathyroid hormone, causing a brown tumor. It also highlights the rarity of hungry bone syndrome, its severity and duration, and how it is successfully managed.

Discussion

Malignancy is the most common cause of severe hypercalcemia, with majority of cases occurring secondary to PTHrP.[1-3] However, our patient's severe hypercalcemia was due to ectopic PTH secreted from a pulmonary NET. To the best of our knowledge, this is the first documented case of a pulmonary NET causing OFC via ectopic PTH.[2-4] Currently, the prevalence of parathyroid bone disease in cases with ectopic PTH is unknown and is only published in case reports.[2,4] Our patient presented with pathologic fracture in the setting of OFC. This is rarely encountered in clinical practice, and the incidence of OFC from hyperparathyroidism has decreased from 80% to 15% presently.[5] On imaging, brown tumors can be seen as lytic or multilobular cystic changes, which may be misdiagnosed as bone cysts, osteosarcoma, metastases, or giant cell tumors.[4,6]

NETs are well-differentiated neoplasms with malignant potential. These tumors contain dense secretory granules and express hormones or proteins that can be detected by immunohistochemistry.[7] The most common locations for NETs include the gastrointestinal tract (48%), lung (25%), and pancreas (9%).[8] A limited number of case reports have documented hypercalcemia from NETs secreting PTH arising from the pancreas.[2] When malignancy-associated hypercalcemia was related to elevated PTH levels from a lung source, the pulmonary tumors were small cell and bronchogenic lung cancer and were not identified as NETs.[2]

On initial chest CT without contrast, the right pulmonary nodule was interpreted as highly suggestive of a pulmonary NET. A diagnosis of PTH-secreting NET was considered because of the extreme elevation of the PTH and calcium levels, with levels not typical of primary hyperparathyroidism and with no evidence of malignancy or PTHrP. Neck imaging with CT and ultrasound ruled out an atypical parathyroid lesion, particularly parathyroid carcinoma, which can present with higher calcium levels as seen in our case, generally >14 mg/dL or 3 to 4 mg/dL above the upper limit of normal as opposed to a parathyroid adenoma, which usually presents with mild hypercalcemia (within 1 mg/dL above the upper limit of normal).[9] Whole-body single-photon emission CT was obtained to investigate for ectopic PTH production. This study

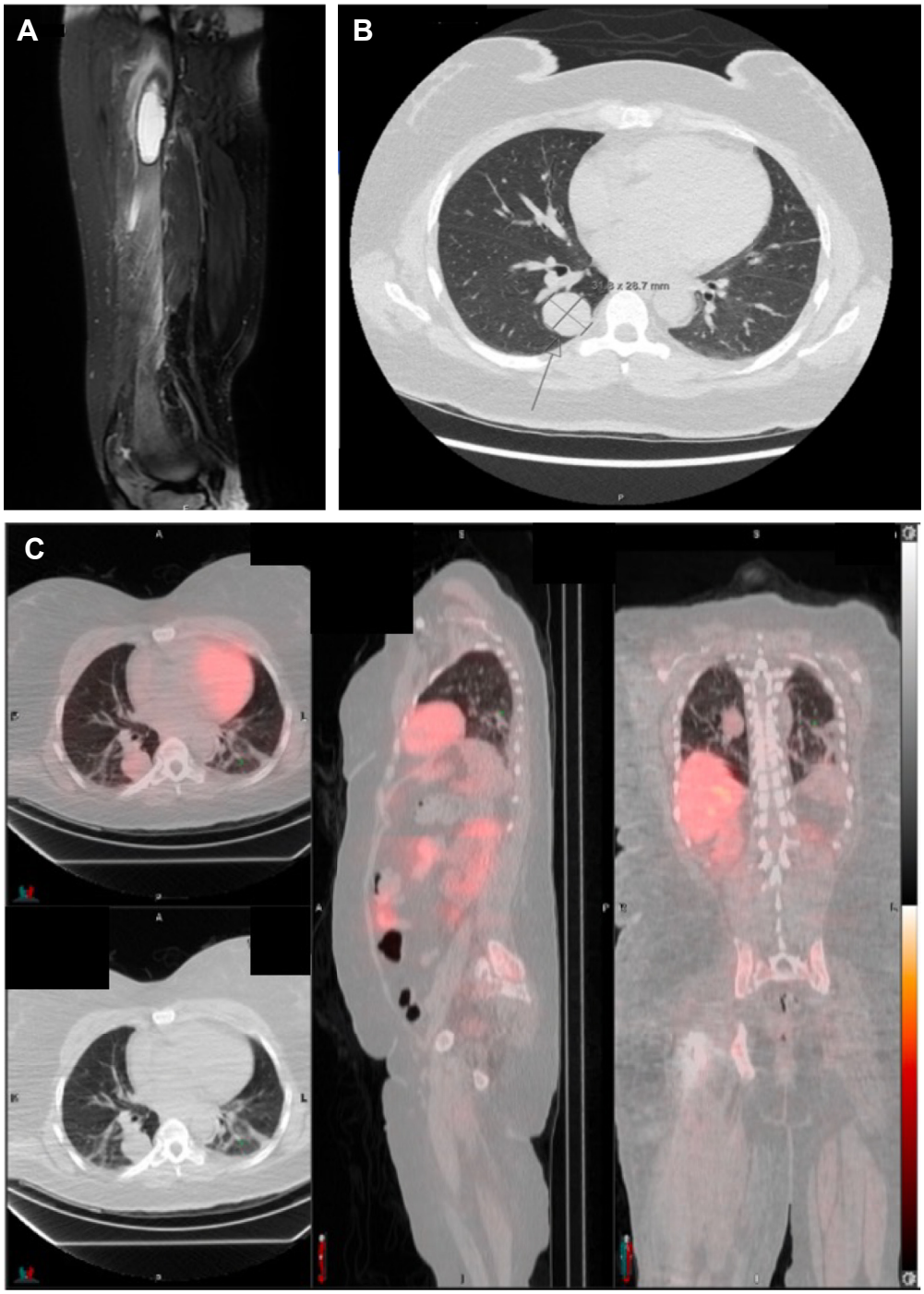


Fig. 1. Imaging of a brown tumor of the femur and neuroendocrine tumor of the lung. *A*, Magnetic resonance imaging of the femur shows a 3.4 × 2.8 × 9.4-cm expansile mass and innumerable tiny intracortical foci of signal abnormality. *B*, Chest computed tomography shows a 3.2 × 2.8 × 3.4-cm craniocaudal diameter soft tissue mass in the right lower lung. *C*, Whole-body technetium-99m sestamibi scan shows no signs of a parathyroid lesion but shows moderate uptake in a round mass in the right lower lung and intense uptake in the fundus of the stomach.

identified both pulmonary and gastric sources of tracer uptake in our patient. Biopsy and staining of the pulmonary tumor confirmed the source of PTH. However, due to concomitant uptake in the gastric fundus, there was a possibility that a gastric tumor was also producing PTH.[10] Follow-up endosonography showed no mass concerning for a gastric NET.

Postoperatively, our patient developed symptomatic hypocalcemia consistent with hungry bone syndrome. It is hypothesized that the abrupt decrease in high circulating levels of PTH arrests bone resorption while bone formation continues, depleting available calcium.[11] Thus, aggressive calcium supplementation is necessary for treatment. In cases of hungry bone syndrome, it has been suggested that IV calcium chloride or calcium gluconate should be given if the serum calcium level is <7.6 mg/dL, electrocardiogram changes are present, or the patient is symptomatic from their syndrome.[12,13] Additionally, the initial dose should be bolused (ie, calcium gluconate 10-20 mL in 50-10 mL of Dextrose 5% IV fluids and given over 5-10 minutes); then, a continuous infusion should be initiated. If the patient can tolerate by mouth and an aimed rate of 0.5 to 1.5 mg/kg/hour has been achieved, then oral

Table 1
Preoperative Laboratory Monitoring of Calcium, Albumin, and PTH and Treatment Interventions for Hypercalcemia

Hospital day	Treatment for hypercalcemia	Serum calcium level (mg/dL) pre-Tx	Albumin (mg/dL)	ALP (U/L)	Phosphate (mg/dL)	PTH (pg/dL)
Normal range	…	8.6-10.0 mg/dL	3.5-5.0 mg/dL	31-125 U/L	2.5-4.9 mg/dL	19-88 pg/mL
Admission	- IV calcitonin 350 U 1 time - Continuous IV NS	17.5	3.4	766	3.4	>2000
1-2	- IV pamidronate 90 mg 1 time - IV calcitonin 300 U twice a day for 24 h - Continuous IV NS	14.9	…	711	2.5	…
3-4	- Continuous IV NS	13.2	…	579	2.9	…
4-5	- IV calcitonin 300 U twice a day for 48 h - Continuous IV LR	12.6	…	502	2.2	>2000
6-7	- IV calcitonin 300 U twice a day for 48 h - Continuous IV LR; 1 L of D5W with bicarbonate	13.1	…	368	3.0	…
8	- IV pamidronate 60 mg - Continuous IV LR; 1 L of D5W with bicarbonate	11.8	…	361	2.9	…
9-10	- IV calcitonin 300 U twice a day for 48 h - Prednisone 50 mg orally daily (2 doses given) - IV octreotide 50 μg twice a day - Continuous IV LR	14.3	…	380	2.7	>2000
11	- IV octreotide 50 μg twice a day - Continuous IV LR	10.8	…	356	1.9	…
12	Prior to lung mass resection: - IV octreotide 50 μg twice a day - Continuous IV LR	14.3	…	372	3.2	…

Abbreviations: ALP = alkaline phosphatase; D5W = Dextrose 5% in water; IV = intravenous; LR = Lactated Ringers; NS = normal saline; PTH = parathyroid hormone. The term "pre-Tx" indicates the value obtained prior to daily treatment or intervention.

supplementation may begin.[12] In regard to oral calcium supplementation, calcium citrate, and/or calcium carbonate may be used for patients with hungry bone syndrome.[12] Varying doses for oral supplementation have been used. The doses ranged from 800 mg of elemental calcium daily to 36 g of elemental calcium daily for hungry bone syndrome status post parathyroidectomy.[14,15] In

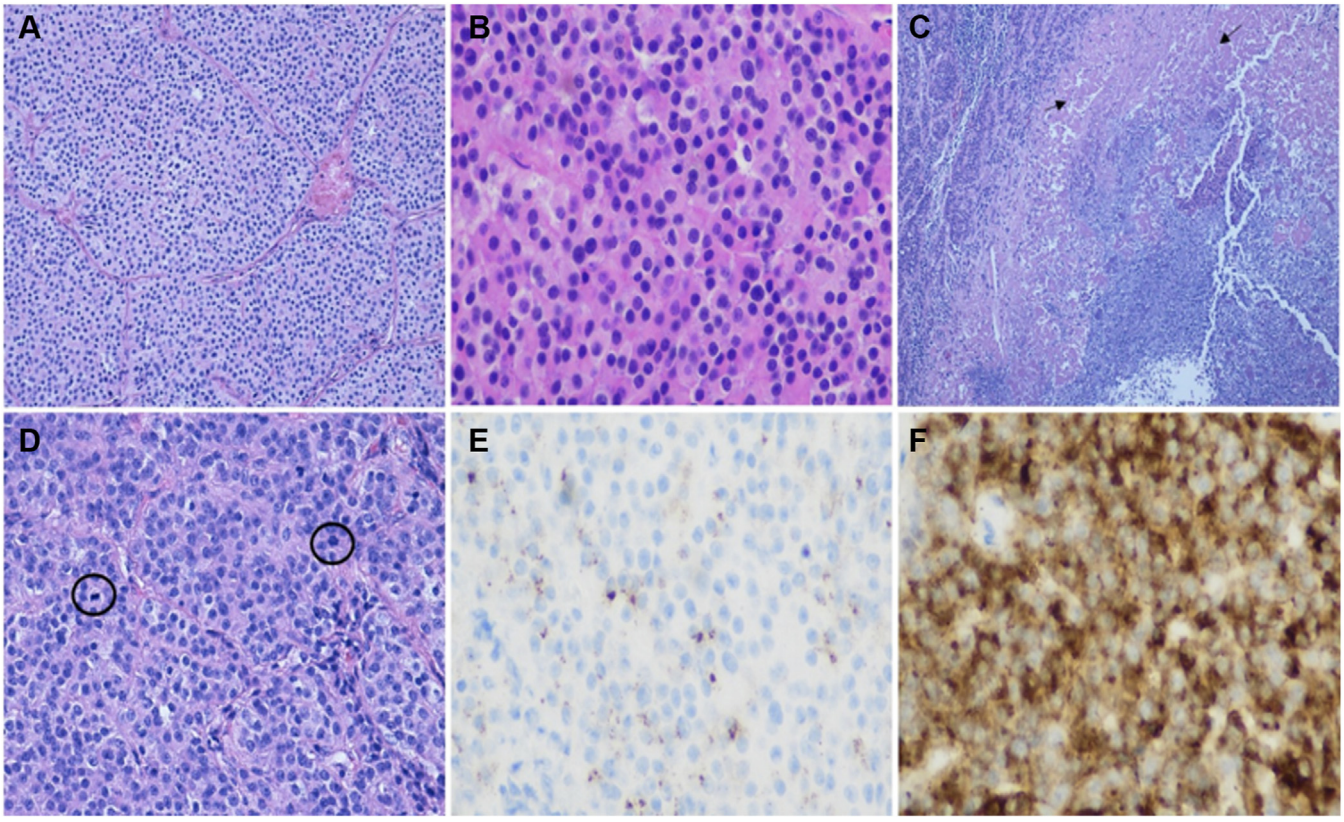
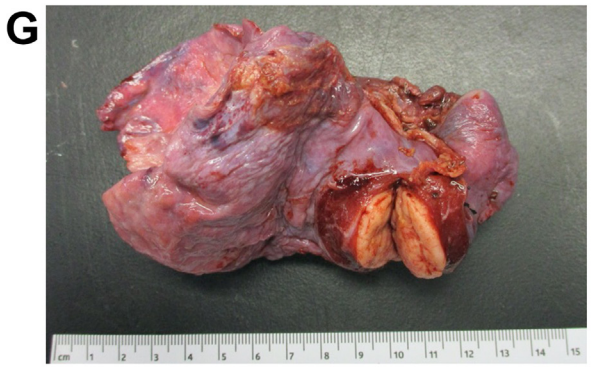


Fig. 2. *A*, Hematoxylin and eosin (H&E) staining (200×) with areas of solid sheets separated by fibrovascular septa. *B*, H&E staining (400×) showing an organoid arrangement of tumor cells with salt and pepper chromatin characteristic of a neuroendocrine tumor. *C*, H&E staining (200×), tumor cell necrosis. *D*, H&E staining (400×), highlighted mitotic activities. *E*, Immunohistochemical staining, chromogranin, focally positive. *F*, Immunohistochemical staining, parathyroid hormone (PTH) staining, strongly and diffusely positive in tumor cells. *G*, Gross pathology from right lower lung lobectomy. Pathology showed a 4-cm, benign, well-differentiated, intermediate-grade neuroendocrine tumor with focal necrosis, 3 to 4 mitoses/10 high-power fields, and 15% proliferative index with Ki-67. *H*, Trend of serum calcium, phosphorus, and PTH before and after tumor removal.



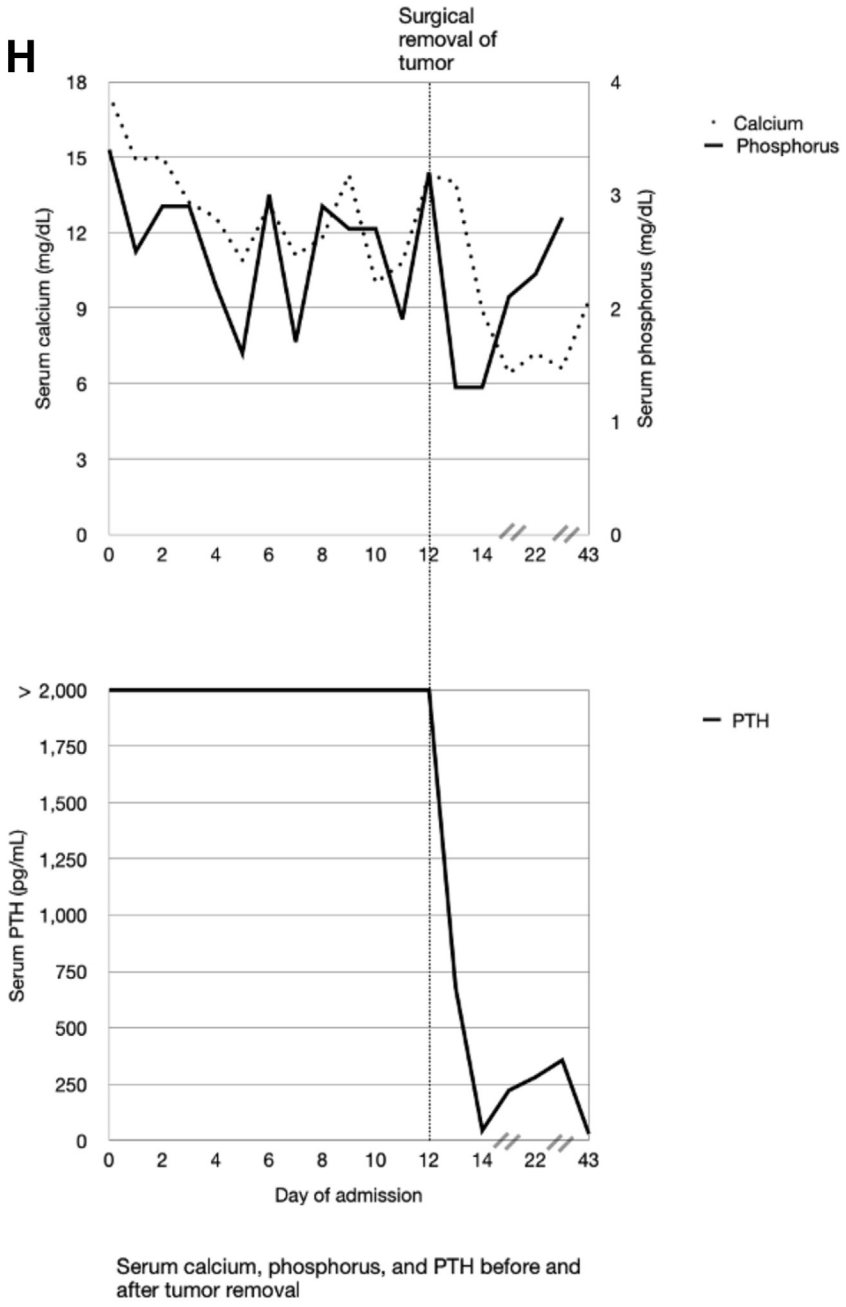


Fig. 2. (*continued*).

Table 2
Postoperative Laboratory Monitoring of the Calcium, PTH, and Alkaline Phosphatase Levels

Laboratory	Normal range	POD 0	POD 1	POD 4	POD 9	POD 14	POD 30
Calcium	8.6-10.0 mg/dL	14.0	8.9	6.4	7.2	6.6	9.3
PTH	19-88 pg/mL	678	44	222	281	356	29
ALP	31-125 U/L	372	411	722	1232	1107	521
Potassium	3.5-5.2 mmol/L	3.8	3.7	3.6	4.4	4.5	4.7
Magnesium	1.6-2.6 mg/dL	1.4	2.3	2.1	1.7	1.8	1.9

Abbreviations: ALP = alkaline phosphatase; PTH = parathyroid hormone; POD = postoperative day.

Table 3
Postoperative Treatment Course for Persistent Hypocalcemia Secondary to Hungry Bone Syndrome

Postoperative day	Serum calcium level before treatment (8.6-10.0 mg/dL)	Treatment for hypocalcemia
1	8.9	- Calcium carbonate 1250 mg - IV calcium gluconate 2 g
2	7.3	- Calcium carbonate 1250 mg 3 times a day - IV calcium gluconate 2 g × 3 - Cholecalciferol oral drops 800 U
3	6.8	- Calcitriol 0.25 μg twice a day - Calcium carbonate 1250 mg - Calcium carbonate 2500 mg × 2 - IV calcium gluconate 2 mg × 2 - Cholecalciferol oral drops 800 U
4	6.4	- Calcitriol 0.25 μg twice a day - Calcium carbonate 2500 mg 3 times a day - IV calcium gluconate 2 g × 4 - Cholecalciferol oral drops 800 U
5	6.5	- Calcitriol 0.25 μg - Calcitriol 0.5 μg - Calcium carbonate 2500 mg 3 times a day - IV calcium gluconate 2 g × 3 - Cholecalciferol oral drops 5000 U
6	6.2	- Calcitriol 0.5 μg - Calcitriol 1.5 μg - Calcium carbonate 2500 mg every 6 h - IV calcium gluconate 2 g × 2 - Cholecalciferol oral drops 5000 U
7	8.2	- Calcitriol 1.5 μg twice a day - Calcium carbonate 2500 mg every 6 h - Cholecalciferol 10 000 U
8	9.0	- Calcitriol 4 μg - Calcium carbonate 2500 mg every 6 h - Cholecalciferol 10 000 U
9	7.2	- Calcitriol 2 μg twice a day - Calcium carbonate 2500 mg every 6 h - Cholecalciferol 10 000 U
10	6.7	- Calcitriol 2 μg twice a day - Calcium carbonate 2500 mg every 6 h - Cholecalciferol 10 000 U
11	6.5	- Calcitriol 2 μg twice a day - Calcium carbonate 2500 mg every 6 h - Cholecalciferol 10 000 U
12	6.8	- Calcitriol 2.5 μg twice a day - Calcium carbonate 2500 mg every 6 h - Cholecalciferol 10 000 U
13	6.3	- Calcitriol 2.5 μg twice a day - Calcium carbonate 2500 mg every 6 h - Cholecalciferol 10 000 U
14	6.6	- Calcitriol 2.5 μg - Calcitriol 3 μg - Calcium carbonate 2500 mg 3 times a day - Cholecalciferol 10 000 U
15	6.4	- Calcitriol 2.5 μg - Calcitriol 3 μg - Calcium carbonate 2500 mg 3 times a day - Calcium carbonate 5000 mg - Cholecalciferol 10 000 U
16	6.7	- Calcitriol 2 μg - Calcium carbonate 5000 mg twice a day - Cholecalciferol 10 000 U

Abbreviation: IV = intravenous.

another review, the authors found that patients may require up to 12 g of oral elemental calcium per day as treatment for hungry bone syndrome status post parathyroidectomy for primary hyperparathyroidism.[16]

In our patient, vitamin D was not repleted prior to tumor removal. There is a lack of data on the safety and efficacy of preoperative vitamin D supplementation in preventing hungry bone syndrome in cases of ectopic PTH undergoing tumor resection, due to the rarity of these cases. In primary hyperparathyroidism, retrospective evidence suggests that low vitamin D levels prior to parathyroidectomy can increase the risk of postoperative hungry bone syndrome.[17] However, in a more recent prospective, randomized study, there was no statistically significant difference in the development of hungry bone syndrome in patients who were repleted with vitamin D prior to parathyroidectomy.[18] Another retrospective study found that the preoperative PTH level was the only factor that predicted the development of hungry bone syndrome.[19] An unmeasurably high PTH level in this case was the key risk factor for the development of hungry bone syndrome following resection of the ectopic PTH source. The prevalence of hungry bone syndrome in patients after parathyroidectomy with primary hyperparathyroidism has been estimated to range between 4% and 13%,[11,14] which demonstrates the rarity of this case.

Several months later, the patient ran out of her calcium for 4 days and became hypocalcemic with an increase in the PTH level from 21 to 338 pg/mL. This illustrates the importance of continuing calcium supplementation to maintain homeostasis in patients who develop prolonged and severe hungry bone syndrome following removal of the ectopic source of PTH.

Conclusion

A PTH-secreting pulmonary NET is a rare cause of severe hypercalcemia and hyperparathyroidism-associated bone disease. Sestamibi single-photon emission CT is a useful modality for localizing ectopic PTH production. Severely elevated PTH levels, like those observed in our case, can be seen with ectopic PTH production from NETs and are a risk factor for hungry bone syndrome following resection of the ectopic source of PTH production. Clinicians should be cognizant of the complications of hungry bone syndrome, including the ensuing electrolyte abnormalities, and its potential protracted course.

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