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Parathyroid Adenoma- The Leading Cause of Primary Hyperparathyroidism: Case Report

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ABSTRACT

Primary hyperparathyroidism is an endocrine disorder characterized by excessive secretion of parathyroid hormone (PTH) by the parathyroid glands, disproportionate to the physiological requirement for calcium concentration. This hypersecretion of PTH occurs independently of dysregulation in calcium-phosphate metabolism. The predominant etiology of primary hyperparathyroidism is the presence of a parathyroid adenoma. (7)

This case report presents a 68-year-old female patient with a history of osteoporosis, diagnosed in 2019 for osteoarticular pain and hypercalcemia. The suboptimal response to standard osteoporosis treatment was an indication to expand the diagnosis of calcium-phosphate disorders accompanying osteoporosis. Diagnostic evaluation confirmed primary hyperparathyroidism due to parathyroid adenoma and appropriate therapeutic measures were subsequently implemented.

This report highlights the nonspecific clinical presentation of the disease and the diagnostic difficulties associated with illness. A multidisciplinary approach to the patient is required due to the multifaceted nature of the disease.

Keywords: Primary Hyperparathyroidism, Parathyroid Adenoma, Osteoporosis, Parathyroid

INTRODUCTION

Primary hyperparathyroidism is the most common cause of PTH-dependent hypercalcemia and is most commonly diagnosed in older women. The clinical presentation of primary hyperparathyroidism is influenced by the duration of elevated parathyroid hormone (PTH) secretion and the severity of hypercalcemia. In the early stages, the condition is frequently identified incidentally through routine diagnostic evaluations. The disease can also be diagnosed based on symptoms such as kidney stones, skeletal disorders, hypertension, or neuromuscular dysfunction. The diagnostic challenges posed by non-specific or absent symptoms often contribute to significant delays in the recognition of primary hyperparathyroidism. Chronic hypercalcemia resulting from prolonged disease progression is associated with increased arterial stiffness, which predisposes patients to the development of atherosclerosis, hypertension, and an elevated risk of cardiovascular mortality. (1,2,3)

Incidentally diagnosed primary hyperparathyroidism is most commonly identified through blood tests. Hypercalcemia is the hallmark biochemical indicator of primary hyperparathyroidism. (3) Additional laboratory findings may include hypophosphatemia (observed in approximately 25% of patients), elevated PTH levels, decreased serum vitamin D levels, and hypercalciuria, as detected in a 24-hour urine analysis. To ascertain the etiology of primary hyperparathyroidism, imaging studies are essential.

Neck ultrasonography is typically the initial diagnostic modality due to its wide availability and cost-effectiveness. (4) Scintigraphy with technetium-99m-labeled methoxyisobutylisonitrile (99mTc-MIBI) is recognized as the most sensitive imaging technique for detecting parathyroid gland abnormalities. The combination of 99mTc-MIBI single-photon emission computed tomography (SPECT-CT) and neck ultrasound is currently recommended as the optimal approach for parathyroid imaging. (5,6)

Surgical intervention remains the most effective treatment for symptomatic primary hyperparathyroidism caused by parathyroid adenoma. The conventional approach involves bilateral neck exploration and excision of all hyperfunctioning parathyroid tissue, a technique associated with favorable outcomes and low perioperative mortality. To minimize the risk of complications, shorten hospital stays, and facilitate more rapid postoperative recovery, advanced minimally invasive surgical techniques have been increasingly adopted in clinical practice. (8) Minimally invasive parathyroidectomy is an innovative method that reduces surgery time and patient recovery. Prior to the MIP procedure, baseline serum parathyroid hormone (PTH) levels are measured. Following excision of the parathyroid lesion, intraoperative PTH levels are reassessed to confirm that the resected gland was the sole source of hypersecretion. Additionally, the use of sestamibi - single photon emission computed tomography (SPECT) allows for the identification of the pathologically altered parathyroid gland. (8,9). Numerous techniques are still under investigation to evaluate their effectiveness, for instance: minimally invasive video-assisted parathyroidectomy (MIVAP)6, video-assisted parathyroidectomy using the lateral approach (VAPLA) (10,11) In asymptomatic primary hyperparathyroidism, management may be either surgical or conservative. Surgical intervention is recommended for patients with significant deviations in parameters such as serum calcium concentration, estimated glomerular filtration rate (eGFR), age, or bone mineral density. Conservative management is reserved for patients who are either ineligible for surgery or decline surgical treatment. This approach involves regular monitoring, including annual assessments of serum calcium levels and eGFR, as well as bone mineral density evaluations every two years. Equally important is symptomatic pharmacological treatment, which includes the use of bisphosphonates such as alendronate (12), calcimimetics like cinacalcet (13), denosumab (14) and vitamin D supplementation (15).

CASE PRESENTATION

The patient, a 68-year-old female, complaints of fatigue and osteoarticular pain affecting the upper extremities, hip joints, as well as the cervical and thoracic spine, prompting the performance of a densitometry evaluation. Imaging studies revealed a T-score below -2.5, leading to the diagnosis of spinal osteoporosis and hip joint osteopenia. Considering the patient's age and elevated fracture risk, pharmacological treatment for osteoporosis was initiated with ibandronate. However, the absence of improvement in the patient's well-being and a decline in T-score values on follow-up densitometry prompted clinicians to expand the diagnostic workup. The diagnostic workups included a complete blood count (CBC), serum parathyroid hormone (PTH) levels, blood calcium and phosphate levels, daily urinary calcium excretion, and vitamin D3 [$1,25(OH)_2$] levels.

The findings of the study were as follows:

Serum Parathyroid Hormone (PTH) levels [pg/ml]	70,8 (7.10) 59,6 (8.10) 89,5 (9.10)	15.0-65.0
Blood Calcium levels [mmol/l]	2,54 (7.10) 2,56 (8.10) 2,63 (9.10)	2,15-2,60
Blood Phosphate Levels [mmol/l]	0.62 (7.10) 0,7 (8.10) 0.92 (9.10)	0,81-1,45
Daily Urinary Calcium Excretion [mmol/24h]	8,5 (8.10) 2,6 (9.10)	2,5-5.0
Vitamin D3 [$1,25(OH)_2$] levels	7,51	19,9-79,3

Elevated serum PTH levels confirmed the presence of Primary Hyperparathyroidism. To investigate its underlying cause, an ultrasound examination of the thyroid and parathyroid glands was conducted. (Additionally, a renal ultrasound was performed to assess the kidneys for nephrolithiasis, which revealed no evidence of calculi.) Parathyroid ultrasound identified a lesion near to the lower pole of the right thyroid lobe, corresponding to the anatomical location of the right upper parathyroid gland, characterized by a homogeneous, hypoechoic structure measuring 16 mm in diameter. Color Doppler imaging demonstrated rich vascularization within the lesion. To confirm the diagnosis, scintigraphy was performed, revealing increased radiotracer uptake consistent with parathyroid hyperactivity. The patient was qualified for surgical treatment.

The patient underwent a minimally invasive parathyroidectomy (MIP). Preoperative imaging effectively identified the pathologically altered parathyroid gland, eliminating the need for intraoperative exploration and preserving the remaining parathyroid glands. Prior to the procedure, the patient's serum PTH level was measured at 92.8 pg/mL. The surgery was performed under local anesthesia, with an incision made along a natural skin fold below the thyroid cartilage to ensure the postoperative scar remained inconspicuous.

The affected parathyroid gland was excised, and intraoperative PTH levels were monitored. Ten minutes after gland removal, the PTH concentration had decreased to 21.2 pg/mL, representing a reduction of over 50%. This significant decrease confirmed the success of the procedure, and the surgical site was subsequently closed. A follow-up ultrasound revealed no abnormalities.

The patient was discharged with instructions to monitor serum calcium levels and was advised about potential symptoms of hypocalcemia that could arise postoperatively. Ten days later, she returned to the hospital for suture removal; the wound was healing appropriately. The patient was further advised to monitor her PTH, calcium, and vitamin D levels every 6–12 months.

CONCLUSION

Primary hyperparathyroidism is a prevalent endocrine disorder, most commonly caused by a parathyroid adenoma. The condition may range from being asymptomatic to leading to severe, multi-organ complications. The case presented illustrates the diagnostic process and available treatment modalities, spanning advanced minimally invasive surgical techniques, traditional surgical approaches, and symptomatic pharmacological therapies.

Effective diagnosis and management of the disease are greatly enhanced by collaboration among physicians from multiple specialties, ensuring a comprehensive and multidisciplinary approach to patient care.

Continued follow-up enables the prevention of potential relapses or exacerbations and allows for prompt intervention when necessary.

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