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Case Report

Parathyroid Adenoma Presenting with Severe Skeletal Deformities

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Abstract

Parathyroid adenoma (PA) is the most common cause of primary hyperparathyroidism. Parathyroid hyperplasia and cancer are rare, accounting for less than 1% of cases1. PA is typically identified via ultrasonography. We present a case of a 17-year-old female with severe skeletal deformities and extensive osteolytic lesions. She underwent parathyroid adenoma excision and subsequently developed hungry bone syndrome, requiring calcium and vitamin D supplementation. Isolated skeletal abnormalities as the primary manifestation of parathyroid adenoma are rare.

Keywords: Hyperparathyroidism, Hungry bone syndrome, Parathyroid adenoma

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Introduction

Parathyroid adenoma (PA) is the most common cause of primary hyperparathyroidism. Parathyroid hyperplasia and cancer are rare, accounting for less than 1% of cases[1]. Hyperparathyroidism could be Primary Hyperparathyroidism: Due to a single adenoma (most common), multiple adenomas, or hyperplasia. Rarely, it may result from parathyroid carcinoma[2]. Familial PA may be associated with MEN1, MEN2, or HPT-JT syndrome. Secondary Hyperparathyroidism: Due to chronic kidney disease or vitamin D deficiency, characterized by low calcium and high PTH. Tertiary Hyperparathyroidism: Autonomous PTH secretion following prolonged secondary hyperparathyroidism, leading to hypercalcemia e.g. renal failure.

The presentation of hyperparathyroidism is commonly summarized by the phrase: "Bones, stones, abdominal groans, and psychic moans."[3] Skeletal manifestations include selective cortical bone thinning, bone and joint pain, generalized osteoclastic bone resorption, and chondrocalcinosis. Bone resorption leads to cystic bone lesions (osteitis fibrosa cystica) and pathological fractures[3]. Neuromuscular presentations include diminished deep tendon reflexes, depression, muscular weakness, and progressive cognitive decline4.

Case Report

A 17-year-old female presented with short stature, genu valgum deformity, and muscular weakness. She had visited multiple physicians, who reassured her that her skeletal deformities were congenital and non-correctable. There was no history of neck radiation or a family history of cancer.

Investigations

- Ultrasonography of the Neck: A highly vascular soft tissue lesion (3.5 × 3.0 cm) was detected in the supraclavicular region, adjacent to major vessels, making FNAC risky.
- X-ray of the Pelvis and Long Bones: Multiple severe osteolytic lesions (2 mm–5 cm) were noted in the pubic ramus, pelvic bones, scapula, and long bones, with moderate periosteal fibrosis. No osteolytic lesions were observed in the spine, ribs, or sternum.

Laboratory Findings Serum Calcium (Total): 11.3 mg/dL (Normal: 8.1–10.5 mg/dL) Serum Calcium (Ionized): 1.79 mmol/L

(Normal: 1.15–1.32 mmol/L) Alkaline Phosphatase: 4628 IU/L

(Normal: 80–260 IU/L) Vitamin D: 14.23 ng/mL (Normal: 40–100 ng/mL) Serum Phosphate: 2.3 mg/dL (Normal: 2.5–5.0mg/dL) Serum Magnesium: 1.6 mg/dL (Normal: 1.7–2.7 mg/dL) Parathyroid Hormone (PTH): 3201 pg/mL (Normal: <56 pg/mL)

The initial diagnosis was parathyroid carcinoma due

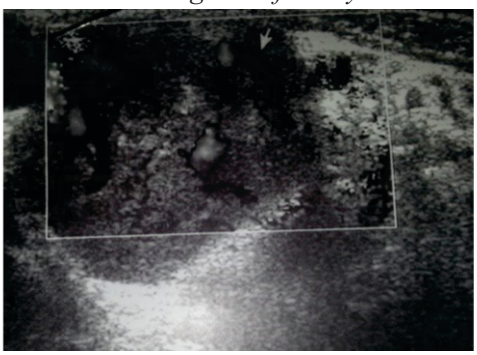
to diffuse skeletal osteolytic lesions and markedly elevated PTH.



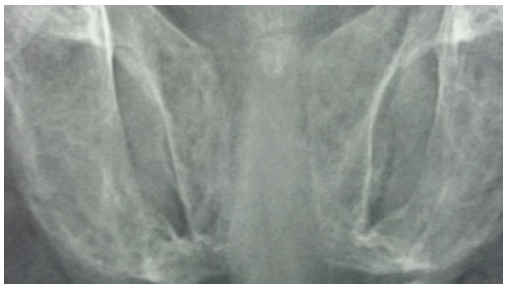
Patient is short stature and obvious kyphosis and genu valgum deformity



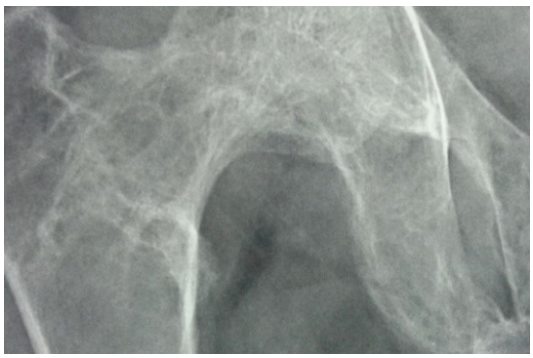
Genu Valgum deformity



Thyroid gland Ultrasonography showing vascularity of adenoma with compressed thyroid parenchyma



Severe Bone demineralization with osteolytic lesion and periosteal fibrosis



Severe Bone demineralization with osteolytic lesion and periosteal fibrosis

Management:

The patient started with intravenous hydration and furosemide. Surgical resection of the right inferior PA (3 × 4.5 cm, firm in consistency) was performed. Histopathology confirmed a benign PA without malignant cells. Postoperatively, the patient developed perioral paresthesia and carpopedal spasms. Laboratory investigations revealed: Serum Calcium (Total): 7.1 mg/dL (Normal: 8.1–10.5 mg/dL) PTH: 54 pg/mL (Normal: <56 pg/mL) She was managed with intravenous calcium gluconate and bisphosphonates. The patient was discharged on postoperative day six with a satisfactory recovery.

Discussion

This case highlights a rare presentation of isolated skeletal abnormalities in PA. PTH is regulated by serum calcium levels, primarily increasing bone resorption and renal calcium reabsorption while reducing phosphate reabsorption, leading to hypercalcemia and hypophosphatemia.

Hyperparathyroidism could be Primary Hyperparathyroidism: Due to a single adenoma (most common), multiple adenomas, or hyperplasia. Rarely, it may result from parathyroid carcinoma2. Familial PA may be associated with MEN1, MEN2, or HPT-JT syndrome. Secondary Hyperparathyroidism: Due to chronic kidney disease or vitamin D deficiency, characterized by low calcium and high PTH. Tertiary Hyperparathyroidism: Autonomous PTH secretion following prolonged secondary hyperparathyroidism, leading to hypercalcemia eg renal failure.

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and progressive cognitive decline[4]. Cardiovascular symptoms may include a prolonged P-R interval and a shortened Q-T interval. Pruritus is also a recognized manifestation. Calcium deposition in the cornea can lead to keratopathy. Approximately 85% of patients are either oligosymptomatic or asymptomatic, with symptoms such as body pain, myalgias, joint pain, and spontaneous fractures[4].

Hyperparathyroidism is more commonly observed in females, with long bones, hip bones, jaw, and ribs frequently affected. Ectopic PA is rare and may occur in locations such as near the jugular vein, carotid sheath, para-aortic region, hypoglossal nerve, and the anterior aspect of the sternocleidomastoid muscle[5]. Additional rare sites include the retropharyngeal space, paraesophageal region, aorto-pulmonary window[6], anterior mediastinum, and pericardium[7]. Parathyroid tissue originates from the third or fourth pharyngeal pouches, and ectopic lesions develop due to anomalous migration pathways[8].

Diagnostic workup primarily involves detecting hypercalcemia with an elevated parathyroid hormone (PTH) level. The differential diagnosis includes primary hyperparathyroidism, lithium-induced hypercalcemia, familial benign hypocalciuric hypercalcemia (FHH), and tertiary hyperparathyroidism. Laboratory investigations reveal elevated total and ionized calcium levels, with intact PTH being the key diagnostic marker. A combination of elevated intact PTH and raised ionized serum calcium confirms primary hyperparathyroidism. To rule out FHH, a 24-hour urinary calcium measurement is essential. Other laboratory findings include hypophosphatemia and hyperchloremic acidosis.

Ultrasonography is a widely accepted and safe tool for localizing PA, with an accuracy of 75-80%[9]. Abbound et al. reported parathyroid adenomas in 163 patients, demonstrating a positive predictive value (PPV) of 100%[10]. Nuclear medicine imaging using radiolabeled sestamibi serendipitously revealed preferential accumulation in PA, with retention beyond one hour indicating abnormal parathyroid tissue. The sensitivity of this method ranges from 60-90%. Hybrid imaging using SPECT/CT scans provides more reliable localization of PA[11]. However, ionizing radiation exposure is associated with a four-fold increase in the incidence of parathyroid tumors among individuals exposed to atomic bomb. radiation in Hiroshima[12]. Bilateral internal jugular vein sampling is rarely utilized due to advancements in radiological technology.

Management Surgical Treatment: Surgical excision is the only definitive treatment for PA. Indications for surgery, as per NIH guidelines, include:

- Serum calcium >1 mg/dL above normal
- 24-hour urinary calcium >400 mg
- 30% reduction in creatinine clearance
- BMD T-score < -2.5 at any site
- Age <50 years

For familial cases (MEN1), total parathyroidectomy with forearm autotransplantation is recommended[13].

Medical Management: Calcium (800–1000 mg/day) and Vitamin D Supplementation. Hydration and Mobility to prevent hypercalcemia, Bisphosphonates Aldronate improve BMD in primary hyperparathyroidism[14] Calcimimetics (e.g., Cinacalcet) to reduce PTH secretion[15] Estrogen Therapy in postmenopausal women is recommended without affecting PTH level. Other treatments include alcohol injections, ablation with ultrasound energy who cannot undergo surgery.

Postoperative Care and Follow-Up

Hypocalcemia post-surgery may result from hungry bone syndrome due to rapid calcium deposition in bones, as half life of PTH is 4 minutes and its level fall down rapidly after surgery. Failure to fall the level means pa-tients need further exploration. Close follow-up includes Serum calcium, vitamin D, and PTH levels at 1–2 weeks, Annual BMD monitoring and Lifelong vitamin D correction if needed[16].

Conclusion

This case demonstrates a rare presentation of PA with isolated skeletal abnormalities. Any patient presenting with skeletal deformities, hypercalcemia, and high PTH should be evaluated for parathyroid adenoma.

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Authors' Contribution

MKS: Conception
FS: Design of the work
MI: Data acquisition, analysis, or interpretation
MKS: Draft the work
FS, Mi: Review critically for important intellectual content
MKS, FS, Mi: Approve the version to be published
MKS, FS, Mi: Agree to be accountable for all aspects of the work

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