



BROWN TUMOR OF RIGHT FEMUR IN A 55-YEAR-OLD MALE WITH BILATERAL PARATHYROID ADENOMA

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ABSTRACT Primary hyperparathyroidism (PHPT) is most commonly caused by a solitary parathyroid adenoma, while bilateral parathyroid adenomas remain a rare entity¹. Brown tumors, representing a late skeletal manifestation of severe hyperparathyroidism, are rare in the modern era due to earlier diagnosis². We report the case of a 55-year-old male who presented with progressive bone pain, multiple pathological fractures, and a painful swelling in the right thigh following minor trauma. Clinical examination revealed skeletal deformities and tenderness over the right femur. Laboratory investigations showed hypercalcemia (12.1 mg/dL), hypophosphatemia (2.1 mg/dL), elevated alkaline phosphatase (790 IU/L), and markedly increased parathyroid hormone levels (PTH: 890 pg/mL). Imaging revealed a lytic, expansile lesion in the right femur suggestive of a brown tumor². Neck Ultrasound and CE-MRI scan localized hyperfunctioning tissue in both superior parathyroid glands¹. Surgical exploration confirmed bilateral parathyroid adenomas, which were excised. Histopathological analysis corroborated the diagnosis. Postoperatively, the patient developed transient hypocalcemia managed with calcium and calcitriol (hungry bone syndrome)³. Orthopedic fixation of the right femur was subsequently performed. This case highlights the need to consider PHPT in patients with unexplained bone lesions or recurrent fractures, especially when associated with hypercalcemia². Although brown tumors are rare in developed healthcare settings, they may still occur in cases of delayed diagnosis or neglected PHPT. Bilateral adenomas require careful pre-operative localization and intraoperative PTH monitoring to ensure curative resection⁶. Early recognition and appropriate multidisciplinary management are crucial to prevent long-term skeletal morbidity in such cases.

KEYWORDS : Primary Hyperparathyroidism (PHPT) Brown Tumor Hungry Bone Syndrome Hypercalcemia.

INTRODUCTION

Primary hyperparathyroidism (PHPT) is a relatively common endocrine disorder, most often caused by a solitary parathyroid adenoma in approximately 80–85% of cases, while multiglandular involvement, including bilateral adenomas, occurs in only 10–15%¹. Although PHPT is frequently diagnosed early in developed settings due to routine biochemical screening, delayed diagnosis in resource-limited environments can lead to classical and more severe manifestations².

Among these complications, brown tumors—non-neoplastic, osteolytic lesions arising from excessive bone resorption—represent a rare skeletal consequence of longstanding PHPT². These lesions, composed of fibrous tissue, osteoclast-like giant cells, hemorrhage, and hemosiderin, derive their name from the characteristic brown color observed on gross pathology. Common sites include the pelvis, ribs, clavicle, mandible, and long bones². Clinically, brown tumors can mimic malignancies such as metastatic cancer or multiple myeloma, creating diagnostic confusion⁴.

PHPT may present subtly with non-specific symptoms such as fatigue, constipation, or bone pain, but can progress to pathological fractures, nephrolithiasis, and neuropsychiatric disturbances if unrecognized³. Advanced imaging and biochemical investigations, including parathyroid hormone (PTH) assays, calcium, phosphate, and alkaline phosphatase levels, remain essential for diagnosis⁵. Localization of the adenomas, particularly in multiglandular cases, requires high-resolution imaging like ultrasound, CT, or technetium sestamibi scan⁶.

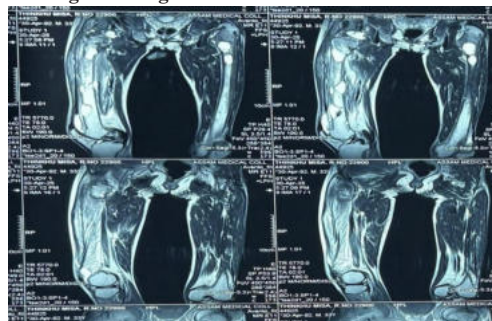
This report presents a rare case of bilateral parathyroid adenomas presenting with a brown tumor in the right femur and multiple skeletal fractures—emphasizing the importance of early recognition, multidisciplinary evaluation, and definitive surgical management to prevent debilitating complications.

CASE DESCRIPTION

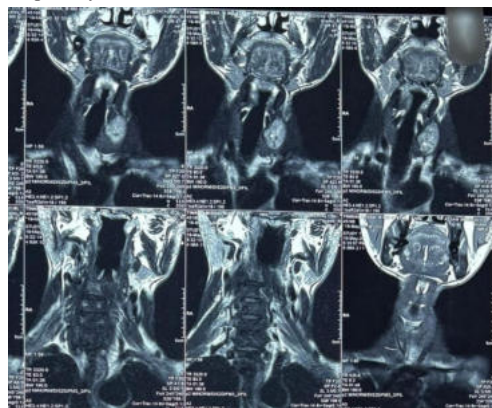
A 55-year-old male presented with chronic right thigh pain for 2 years and diffuse abdominal pain for the past few days. His past medical history was notable for low-trauma fractures of bilateral clavicles and the right superior pubic ramus. Associated symptoms included constipation, polyuria, lethargy, and decreased appetite. On clinical examination, the patient was found to be dehydrated and had mild epigastric tenderness. Neurovascular status of the limbs was intact.

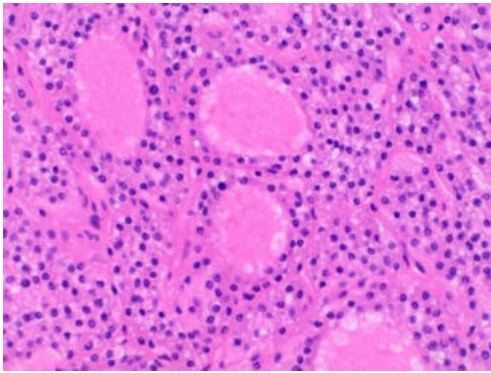
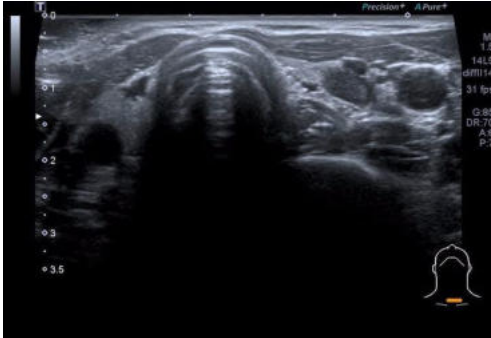
Laboratory investigations revealed elevated serum PTH and alkaline phosphatase (ALP), borderline elevated calcium levels, and low phosphate. ECG showed a short QT interval, a hallmark of hypercalcemia. CE-MRI of the neck revealed bilateral parathyroid adenomas. Skeletal imaging showed multiple osteolytic lesions in the pubic bone and a brown tumor in the right femur. Workup for malignancy and multiple myeloma was negative. A final diagnosis of primary hyperparathyroidism with bilateral parathyroid adenomas and brown tumor was made.

CE-MRI thigh showing brown tumour



xMRI of parathyroid adenoma



Histopathology image of parathyroid adenoma**Ultrasound the report Parathyroid adenoma****Investigations**

Parameter	Result	Interpretation
PTH	Elevated	PHPT
ALP	Elevated	Bone turnover
Calcium	Borderline high	Hypercalcemia
Phosphate	Low	Consistent with PHPT
ECG	Short QT interval	Hypercalcemia marker
MRI Neck	Bilateral adenomas	Multiglandular PHPT
Imaging	Brown tumor + lytic lesions	Skeletal involvement

DISCUSSION

Brown tumors are rare skeletal manifestations of advanced primary hyperparathyroidism (PHPT), resulting from persistent elevation of parathyroid hormone (PTH) levels. Chronic PTH excess stimulates osteoclastic activity and bone resorption, leading to formation of osteolytic lesions composed of fibrous tissue, hemorrhage, and hemosiderin deposits². These lesions derive their name from the brownish appearance on gross examination due to hemorrhage and hemosiderin pigmentation.

They most commonly affect the ribs, pelvis, clavicle, mandible, and long bones². Clinical presentation may include bone pain, swelling, deformity, or even pathological fractures². Radiologically, brown tumors appear as well-demarcated lytic lesions, which may be mistaken for metastasis, multiple myeloma, or giant cell tumors. In fact, up to 3% of bone lesions initially suspected as malignancies may actually be brown tumors secondary to PHPT⁴.

Multiglandular disease (bilateral or multiple adenomas) accounts for only 10–15% of PHPT cases, with solitary adenoma being far more common¹. Accurate preoperative localization via sestamibi scan or ultrasound and intraoperative PTH monitoring are crucial in such cases to confirm complete gland excision and minimize recurrence⁶.

Histological confirmation is necessary in uncertain cases, although regression of brown tumors following parathyroidectomy supports the diagnosis retrospectively. Post-surgical “hungry bone syndrome,” caused by sudden withdrawal of PTH and rebound bone remineralization, may result in hypocalcemia and requires monitoring and supplementation³. In cases with large or structurally compromised bones, orthopedic fixation is essential to prevent morbidity and improve mobility⁵.

In conclusion, brown tumors are important but often under-recognized complications of PHPT. Early diagnosis and surgical cure through parathyroidectomy can prevent irreversible skeletal damage and allow

for substantial recovery.

Treatment

Initial management included intravenous hydration and administration of bisphosphonates to correct hypercalcemia³. Definitive treatment was achieved by surgical excision of both parathyroid adenomas, guided by intraoperative PTH monitoring⁶. The postoperative phase was complicated by hungry bone syndrome, requiring calcium and calcitriol supplementation³. Given the structural compromise of the femur by the brown tumor, orthopedic stabilization was performed to facilitate recovery⁵.

CONCLUSION

Primary hyperparathyroidism (PHPT) is an under-recognized endocrine condition that can lead to significant skeletal complications if not diagnosed early. While most cases involve a single adenoma, bilateral adenomas as noted in this patient are uncommon and can complicate both diagnosis and surgical management¹. Brown tumors, though rare today, remain a significant consequence of chronic PHPT and may resemble bone malignancies radiologically².

In this case, the combination of biochemical abnormalities, including elevated calcium and PTH, alongside imaging studies, allowed for an accurate diagnosis and distinction from other causes of osteolytic lesions⁶. Surgical removal of both parathyroid adenomas led to biochemical improvement; however, the patient developed hungry bone syndrome, a known postoperative complication, which was effectively managed with calcium and active vitamin D supplementation³. Orthopedic intervention for the femoral lesion was essential to restore mechanical stability and prevent functional limitation⁸.

This case emphasizes the importance of considering PHPT in patients with recurrent fractures or lytic bone lesions. Timely identification and comprehensive management, involving surgical and orthopedic teams, are crucial to preventing permanent skeletal damage and ensuring full functional recovery⁷.

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