Case Report

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Primary Hyperparathyroidism Presenting with Multiple Lytic Lesions: A Case Report

Oncology Section

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ABSTRACT

Primary Hyperparathyroidism (PHPT) is a condition characterised by elevated serum calcium levels along with elevated serum Parathyroid Hormone (PTH). The main cause of PHPT is parathyroid adenoma or parathyroid gland hyperplasia. Symptoms and signs of this condition may mimic those of the first differential diagnoses, such as multiple myeloma or metastatic disease from a primary source. In this case, a lady presented with progressively increasing lower back pain and difficulty walking. Initially, the clinical diagnosis leaned towards multiple myeloma or metastatic disease of unknown origin based on Positron Emission Tomography-Computed Tomography (PET-CT) findings. However, her serum calcium levels remained persistently elevated, while her serum phosphorus levels were normal. Consequently, a serum PTH level test indicated elevated levels. A biopsy from an Fluorodeoxyglucose (FDG)-avid lytic lesion in the left iliac bone showed a giant cell-rich lesion that was positive for CD 163 and Vimentin by Immunohistochemistry (IHC), favouring a diagnosis of brown tumour of the bone. A subsequent surgical excision of the adenoma, along with the parathyroid gland, was performed. The patient received intravenous bisphosphonate therapy for the lytic lesion and was advised to follow up every three months with serum calcium and PTH levels monitored.

Keywords: Adenoma, Hypercalcaemia, Multiple myeloma, Scintigraphy

CASE REPORT

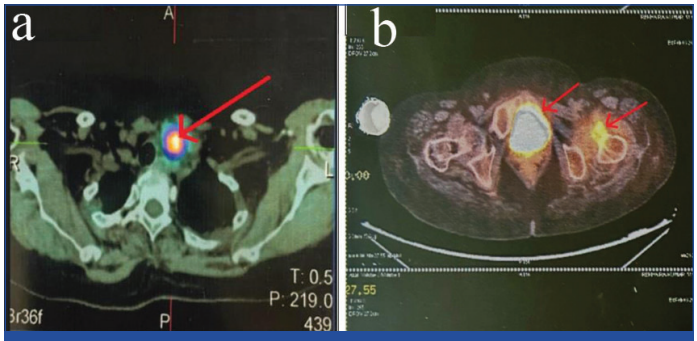
A 49-year-old female with no comorbidities presented to the day care unit with chief complaints of progressively increasing low back pain, left lower limb pain, and difficulty walking over the past three months. Clinical examination revealed no visible or clinically palpable lumps or swellings throughout the body.

Baseline blood reports, including Complete Blood Count (CBC), serum sodium, potassium, urea, creatinine, and liver function tests, were within normal range. The patient was referred for an FDG-PET scan with suspected diagnoses of multiple myeloma or metastatic lesions. The PET scan revealed multiple FDG-avid lytic lesions in the axial and appendicular skeletal systems, along with an FDG-avid hypodense lesion in the left lobe of the thyroid gland, as shown in [Table/Fig-1a,b]. These findings leaned towards multiple myeloma.

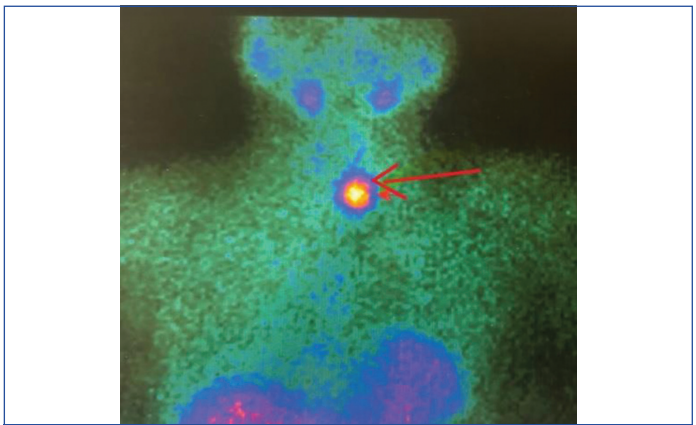
She underwent further investigations for multiple myeloma, which revealed significantly elevated calcium levels of 11.5 mg/dL and alkaline phosphatase levels of 225 IU/L, with normal phosphorus levels. A multiple myeloma panel and serum electrophoresis were negative for the 'M' band, and serum free light chain immunoassay results were within normal limits. Urine tests for Bence Jones proteins and serum carcinoembryonic antigen levels were also negative.

A series of tests, including bone scans, abdominal ultrasound, mammography, and chest radiography, were performed alongside a comprehensive physical examination, but no malignant cause was identified. Given the persistent hypercalcaemia, a PTH assay was conducted that revealed a PTH level of 1213 pg/mL (normal range: 15-68.3 pg/mL). Parathyroid scintigraphy was then advised. The scintigraphy findings demonstrated increased tracer uptake in a well-defined rounded soft tissue nodule measuring 2.3×2.2×2.3 cm in the left inferior parathyroid region, with indistinct margins adjacent to the left thyroid lobe [Table/Fig-2]. These findings were indicative of a parathyroid adenoma involving the left inferior parathyroid gland.

The gross specimen is shown in [Table/Fig-3]. A biopsy of the FDG-avid left iliac bone revealed a giant cell-rich lesion. The IHC panel supported a diagnosis of brown tumour of the bone, positive for CD 163 and Vimentin, with a proliferation Ki-67 index of 5-10%. The



[Table/Fig-1]: PET-CT Scan: a) FDG uptake in left parathyroid gland (red arrow); b) FDG uptake in left iliac crest and spine (red arrow).

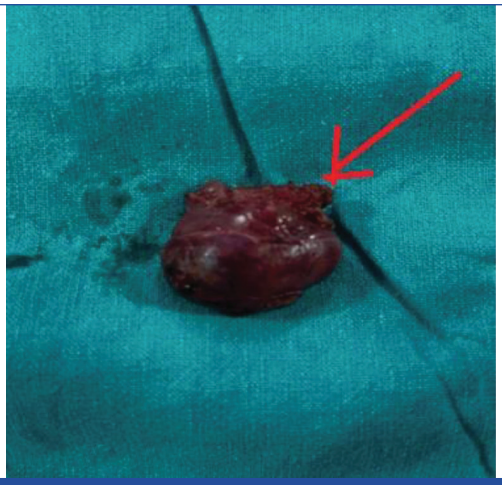


[Table/Fig-2]: Parathyroid scintigraphy report shows tracer uptake noted in well-defined rounded soft-tissue nodule in left inferior parathyroid region (red arrow).

patient underwent surgical excision of the adenoma along with the parathyroid gland. She is now receiving intravenous bisphosphonate therapy for the lytic bony lesion and is on a follow-up schedule every three months, with monitoring of serum calcium and PTH levels.

DISCUSSION

The PHPT is characterised by the abnormal production of PTH, resulting in hypercalcaemia [1]. Approximately 80% of individuals with



[Table/Fig-3]: Surgical specimen of left parathyroid gland (red arrow).

PHPT have a single, non-malignant cause, typically a parathyroid adenoma. In contrast, parathyroid gland hyperplasia is found in 15-25% of cases [2]. About 5% of cases involve multiple parathyroid adenomas, while less than 1% are attributed to parathyroid cancer [1]. Non-neoplastic lesions known as brown tumours of bone are rare and usually affect the face, clavicle, ribs, pelvis, and femur. Their origin lies in the abnormal bone metabolism associated with hyperparathyroidism [3]. Adenomas are the most common cause of brown tumours in patients with PHPT [4].

Brown tumours are histologically characterised by a vascular fibroblastic stroma and several multinucleated giant cells resembling osteoclasts, often accompanied by haemorrhagic infiltrates and haemosiderin deposits. The excessive production of PTH and the resulting hypercalcaemia are hallmarks of primary hyperparathyroidism, most commonly caused by parathyroid adenomas. Hyperparathyroidism produces bony lytic lesions known as brown tumours. Despite the term "brown tumour," these lesions are not malignant conditions. They are uncommon skeletal manifestations, affecting approximately 3% of individuals with primary and secondary hyperparathyroidism [5]. There have been numerous documented cases where brown tumours mimic skeletal metastases [6]. Very few cases have been reported in the literature where multiple skeletal lesions were later diagnosed as hyperparathyroidism [6,7].

A highly sensitive test for localised diagnosis of hyperparathyroidism is 99mTc-MIBI Single Photon Emission Computed Tomography (SPECT)/CT scintigraphy [8]. Multiple lesions are observed in about 90% of patients with bone metastases [9]. A comprehensive evaluation of the patient is essential and should include a complete medical history, a clinical physical examination, a standard complete blood profile, plain X-rays of the chest and affected bones, and CT scans of the abdomen, pelvis, and chest. In this case, no primary tumour was identified through all these tests. The patient's serum calcium level was persistently elevated, while phosphorus levels remained normal, leading to a serum PTH assay. Hypercalcaemia occurs in 5-10% of advanced-stage cancer patients.

In conclusion, PHPT should always be considered as a differential diagnosis in patients with persistently elevated corrected serum calcium levels and multiple bony lytic lesions observed in radiology. PHPT should be investigated after ruling out more common causes, such as cancer. An early diagnosis is facilitated by a strong index of suspicion. The primary treatment for PHPT is surgical removal of the affected parathyroid gland, which can lead to the regression of clinical symptoms and normalisation of biochemical values, as surgery is the most effective treatment in such cases.

CONCLUSION(S)

The PHPT is often misdiagnosed when patients present with multiple bony lytic lesions and hypercalcaemia, which are typically associated with metastatic primary tumours or multiple myeloma. It is crucial to conduct thorough clinical, radiographic, and biochemical evaluations for patients presenting with bony lytic lesions, rather than prematurely concluding a diagnosis of metastatic disease, as PHPT is the most commonly treatable pathology.

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