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# Maxillary Brown Tumor Revealing Primary Hyperparathyroidism: A Diagnostic Pitfall Not to Be Missed: A Case Report

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**ABSTRACT** **Case Studies**

Primary hyperparathyroidism is a common endocrinopathy characterized by the autonomous production of parathyroid hormone (PTH), leading to hypercalcemia and hypophosphatemia. Although often diagnosed at an asymptomatic stage, its clinical manifestations can include rare but classic bone and renal complications, such as brown tumors. We report the case of a 73-year-old female patient who presented with an osteolytic swelling of the maxilla. Initial imaging and biopsy suggested a benign giant cell tumor. However, a complete phosphocalcemic panel revealed severe hypercalcemia (2.90 mmol/L), hypophosphatemia (0.65 mmol/L), and a markedly elevated intact PTH (500 pg/mL), confirming the diagnosis of primary hyperparathyroidism. Cervical computed tomography localized a right parathyroid adenoma. The patient successfully underwent targeted parathyroidectomy, which normalized biochemical parameters and led to the regression of the bone lesion. This case illustrates an atypical presentation where a maxillary brown tumor was the initial revealing sign, and underscores the crucial importance of a multidisciplinary diagnostic approach. Any giant cell tumor, particularly in the maxilla, should prompt a thorough endocrinological workup, including measurement of calcium, phosphate, and PTH. Ignoring this association could delay the diagnosis and expose the patient to serious complications. Optimal management requires close collaboration between endocrinologists, maxillofacial surgeons, radiologists, and pathologists to treat both the local tumor and the underlying metabolic disorder.

**Keywords:** Primary Hyperparathyroidism, Brown Tumor, Giant Cell Tumor, Maxilla, Differential Diagnosis, Multidisciplinary Approach, Atypical Presentation.

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## Introduction

Primary hyperparathyroidism (PHPT) is an endemic endocrine disorder with an estimated prevalence of 0.1% to 0.4% in the general population, showing a marked predominance in postmenopausal women [1]. Its pathophysiology involves clonal proliferation of parathyroid cells that acquire insensitivity to negative feedback from serum calcium, resulting in autonomous and inappropriate secretion of parathyroid hormone (PTH). The resulting chronic hyperparathyroidism disrupts phosphocalcium homeostasis: excess PTH stimulates osteoclastogenesis via the RANK/RANKL system, causing generalized bone resorption [2], and decreases renal tubular reabsorption of phosphorus, leading to characteristic hypercalcemia-hypophosphatemia. While routine biochemical screening has enabled diagnosis in most patients at an asymptomatic stage, the disease may still manifest with serious visceral complications. Bone manifestations, collectively termed fibrous cystic osteitis, and renal complications—particularly nephrolithiasis and nephrocalcinosis—remain markers of advanced disease [3]. Within this spectrum, brown tumors represent a rare but emblematic clinical and pathological entity. These are focal osteolytic lesions rich in multinucleated giant cells that may constitute the first

manifestation of the disease, mimicking a primary malignant bone tumor and misleading clinicians and pathologists [4]. Their recognition is thus a major diagnostic challenge, as their presence signifies severe and longstanding hyperparathyroidism, requiring urgent etiological management.

## Case Report

We report the case of a 73-year-old woman with no significant medical history (no medications, allergies, or prior surgeries) who presented to the maxillofacial department with a progressive, painless swelling of the right maxilla evolving over several months. Clinical examination revealed a firm, non-mobile mass measuring approximately 4 cm in its greatest diameter, associated with slight mobility of adjacent teeth. Given this presentation, contrast-enhanced maxillofacial computed tomography (CT) was performed. It revealed a large lytic lesion involving the right maxillary bone with significant soft-tissue expansion and cortical destruction, initially strongly suggestive of a malignant process such as sarcoma or metastasis.

A surgical biopsy of the lesion was therefore performed. Initial pathological analysis concluded a benign giant cell tumor without signs of malignancy. Although this result might have been considered definitive, the clinical team identified a critical point: giant cell tumors of the jaw, though benign, may represent the osseous manifestation of an underlying metabolic disorder, particularly hyperparathyroidism [5]. This clinical intuition was pivotal to the diagnosis, avoiding a diagnostic pitfall that could have delayed management of the underlying cause.

An urgent comprehensive phosphocalcemic panel was conducted. The results were striking: severe hypercalcemia (corrected calcium 2.90 mmol/L; normal <2.60 mmol/L), hypophosphatemia (0.65 mmol/L; normal >0.80 mmol/L), and an extremely elevated intact parathyroid hormone (iPTH) level (500 pg/mL; normal <70 pg/mL). These findings unequivocally confirmed the diagnosis of primary hyperparathyroidism.

Etiological investigation was guided by cervical CT, which identified a rounded, hyperdense 1.5 cm lesion in the right paraesophageal position, highly suggestive of a right parathyroid adenoma [6]. The patient subsequently underwent successful targeted parathyroidectomy with uncomplicated postoperative recovery. Postoperative normalization of biochemical parameters was achieved within 48 hours. More significantly, clinical and CT follow-up at six months showed significant partial regression of the maxillary lesion, confirming its nature as a "brown tumor" and illustrating the remarkable capacity of bone to regenerate once the metabolic disorder is corrected [7].

| Date | Event |
|---|---|
| 3 months prior | Progressive painless right maxillary swelling noted. |
| Day 0 | Clinical examination: 4-cm firm mass with mobile adjacent teeth. |
| Day 1 | Contrast-enhanced maxillofacial CT: Osteolytic lesion with cortical destruction. |
| Day 3 | Surgical biopsy performed. |
| Day 7 | Pathology report: Benign giant cell tumor. |
| Day 10 | Phosphocalcemic panel: $Ca^{2+}$ 2.90 mmol/L, $PO_4^{3-}$ 0.65 mmol/L, iPTH 500 pg/mL. |
| Day 15 | Cervical CT: Right parathyroid adenoma localized. |
| Day 20 | Targeted parathyroidectomy performed. |
| Day 22 | Biochemical normalization ($Ca^{2+}$ 2.25 mmol/L, iPTH 45 pg/mL). |
| 6 months post | Follow-up CT: Partial regression of maxillary lesion. |

## Discussion

Our case exemplifies a classic yet complex diagnostic scenario: a maxillary brown tumor as the inaugural manifestation of primary hyperparathyroidism. This case highlights the diagnostic pitfall posed by such a lesion, whose clinical and radiological features may mimic a primary malignant tumor, and whose histological analysis may initially be interpreted as a benign giant cell tumor, leading to misdiagnosis.

### The Diagnostic Challenge: The Giant Cell Tumor Trap

The primary lesson from this case lies in the critical importance of clinico-pathological correlation [4]. The patient's clinical presentation—with an expansile lytic lesion involving soft tissues—strongly suggested malignancy. The biopsy, concluding a benign giant cell tumor (Figure 1), might have closed the case and directed toward local surgical monitoring. However, this approach would have ignored the underlying cause, leaving severe PHPT untreated, with high risks of life-threatening complications (hypercalcemic crisis, renal impairment) and progression of fibrous cystic osteitis—a spectrum of bone manifestations in PHPT resulting from excessive osteoclast activity and characterized by focal resorption, cystic changes, and brown tumors [3]. The decision to perform a phosphocalcemic panel despite "benign" histology was the decisive diagnostic step. This underscores that when faced with a giant cell tumor of the jaw, clinicians must maintain a high index of suspicion and not rely solely on the pathological diagnosis.

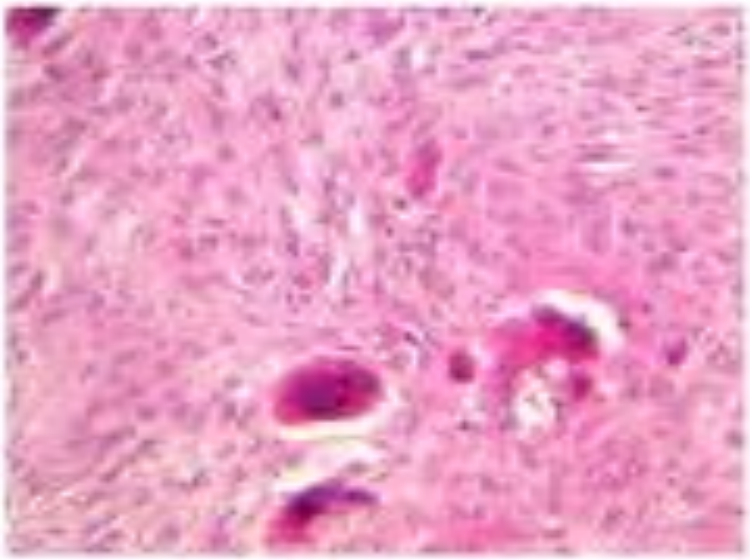

**Figure 1:** Histological slide (H & E staining) showing the giant cell tumor.

## Pathophysiology and Therapeutic Implications

Brown tumors are the local osseous manifestation of chronic hyperparathyroidism. Excess parathyroid hormone intensely stimulates osteoclastic activity via the RANK/RANKL system, causing focal bone resorption [2]. The body's reparative response attempts to counteract this destruction, explaining the proliferation of multinucleated giant cells and fibrosis that create the tumor-like lesion. The management of our patient perfectly illustrates the dual efficacy of etiological treatment. Targeted parathyroidectomy not only corrected the potentially lethal metabolic disorder but also enabled partial regression of the bone lesion. The resolution of the brown tumor after PTH normalization is a well-documented phenomenon, confirming the reactive nature of the lesion and justifying that extensive bone surgery can be avoided or minimized once hyperparathyroidism is controlled [7].

## The Indispensable Multidisciplinary Approach

Resolution of this complex case was only possible through close collaboration among multiple specialties. The maxillofacial surgeon initiated the diagnosis and performed the biopsy. The pathologist provided the initial diagnosis but also recognized its limitations in the context of the clinical picture. The endocrinologist served as the linchpin, connecting the bone tumor to the metabolic disorder and orchestrating the etiological workup and medical treatment. Finally, the radiologist played a key role in localizing the adenoma. This multidisciplinary synergy is the model for managing such atypical presentations, ensuring a holistic approach to the patient [8].

## Novelty and Clinical Take-Home Messages

While maxillary brown tumors in PHPT have been reported (e.g., Ebrahimi et al., 2012; Wang et al., 2020), this case underscores a critical diagnostic pitfall**:** the risk of delayed diagnosis when histology is interpreted in isolation. Our contribution lies in emphasizing:

1. The urgency of phosphocalcemic screeningin all jaw giant cell tumors, regardless of histological benignity.
2. The regression of brown tumors post-parathyroidectomy as a diagnostic confirmation, avoiding unnecessary aggressive surgery.
3. The life-threatening consequences of missed PHPT**,** including renal failure, cardiovascular events, and progressive bone disease.

This clinical case teaches several fundamental lessons for clinical practice:

1. Any giant cell tumor, particularly in the jaw or mandible, must undergo systematic phosphocalcemic evaluation. Neglecting this step may have serious consequences.
2. The pathological diagnosis is an essential piece of the puzzle, but it must always be interpreted in light of the complete clinical picture. Discordance between the two should systematically trigger further investigations.
3. Treatment of the underlying hyperparathyroidism is the therapeutic priority. Surgery for the brown tumor is indicated only for persistent compressive symptoms after PTH normalization.
4. Communication and collaboration among specialists are indispensable for navigating complex diagnoses and ensuring optimal patient outcomes.

The maxillary brown tumor is a formidable "diagnostic trap" that, when recognized, opens the path to the diagnosis and curative treatment of primary hyperparathyroidism. Our case reminds us that medicine is an art of synthesis, where clinical, biological, imaging, and pathological data must converge to uncover the truth.

# Conclusion

This case strikingly illustrates how a maxillary brown tumor can be the first and sole revealing sign of primary

hyperparathyroidism, constituting a true diagnostic pitfall. It emphasizes that resolving this pitfall hinges on an imperative: a multidisciplinary diagnostic approach. Thus, any giant cell tumor in the cervicofacial region must necessarily prompt an endocrine workup including measurement of calcium, phosphorus, and PTH. Neglecting this association exposes the patient to potentially fatal complications, such as advanced fibrous cystic osteitis, renal failure, or cardiovascular events. Optimal management of such complex cases therefore requires close synergy among specialists in endocrinology, maxillofacial surgery, radiology, and pathology to address both the local tumor and correct the underlying metabolic disorder.

## Declaration of Interest

The authors declare no conflicts of interest related to this article.

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## Declarations

This study had an anonymized patient data. As the research involved no direct intervention or modification of standard patient care, formal approval from an ethics committee was not required in accordance with institutional and national guidelines for observational studies. The patient data was anonymized prior to analysis to protect confidentiality and treated according to the Algerian national guidelines

**Ethical approval** was waived by the Ethics Committee of the Regional University Military Hospital Commander Abdellali BENBAATOUCHE (HMRUC), Constantine, Algeria (Reference: HMRUC-EC-2023-05), in accordance with national guidelines for observational studies involving anonymized patient data.

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## Authors' contributions

**Conceptualization:** Sihem BENSALEM; **Methodology:** Sihem BENSALEM; **Software:** Amina KHODJA; **Validation:** Sihem BENSALEM, Amina Khodja; **Formal Analysis:**Sihem BENSALEM; **Investigation:** Sihem BENSALEM, Amina KHODJA; **Resources:** Sihem BENSALEM; **Data Curation:** Sihem BENSALEM, Amina KHODJA; **Writing - Original Draft Preparation:** Sihem BENSALEM; **Writing - Review & Editing:** Sihem BENSALEM, Amina KHODJA ; **Visualization:** Sihem BENSALEM; **Supervision:** Sihem BENSALEM; **Project Administration:** Sihem BENSALEM; **Funding Acquisition:** Not applicable.

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