RESEARCH PAPER

Multiloculated Lytic Lesions of the Jaw: A Radiologic Case Report of Brown Tumours Caused by Parathyroid Adenoma

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ABSTRACT
This case report describes the role of contrast-enhanced computed tomography (CT) in diagnosing multifocal brown tumours of the mandible secondary to primary hyperparathyroidism. A 35-year-old male presented with painless jaw swellings. CT revealed extensive multiloculated lytic lesions involving multiple mandibular sites, with cortical thinning and focal breaches. Crucially, the scan also identified an incidental, well-circumscribed lesion inferior to the right thyroid lobe, suggestive of a parathyroid adenoma. Histopathology of a jaw lesion confirmed a brown tumour, and biochemistry showed severely elevated parathyroid hormone levels. This case underscores the value of comprehensive CT imaging in characterizing these lesions and identifying the underlying endocrine aetiology, thereby guiding appropriate management and preventing misdiagnosis..

Keywords: Brown Tumour, Parathyroid Adenoma, Jaw lesion, Radiology.
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INTRODUCTION

Brown tumours, also known as osteitis fibrosa cystica, are non-neoplastic, osteolytic bone lesions that arise as a direct consequence of prolonged and excessive parathyroid hormone (PTH) activity, typically in the setting of hyperparathyroidism [1]. They represent a localized form of bone resorption, where increased osteoclastic activity leads to the replacement of normal bone with fibrovascular tissue, hemosiderin deposits, and osteoclast-type giant cells, giving them a characteristic brownish hue macroscopically [2]. While any part of the skeletal system can be involved, the jaws particularly the mandible, are among the most frequently affected sites [2, 3].

Clinically and radiologically, these lesions pose a significant diagnostic challenge. Their presentation as expansile, lytic, and sometimes multifocal bony lesions can closely mimic aggressive neoplastic conditions such as giant cell tumours, central giant cell granulomas, metastatic disease, or primary bone malignancies like osteosarcoma [1, 4, 5,]. This potential for misinterpretation can lead to unnecessary and invasive diagnostic or therapeutic procedures. Therefore, a high index of suspicion, coupled with a systematic diagnostic approach integrating imaging, histopathology, and biochemical correlation, is paramount. This report illustrates a case of multifocal mandibular brown tumours, where contrast-enhanced CT played a pivotal role not only in characterizing the jaw lesions but also in incidentally identifying the underlying parathyroid adenoma, thereby establishing the diagnosis.

Case Presentation

A 35-year-old male presented to the maxillofacial surgery department with a chief complaint of gradually progressive, painless swellings in his jaw region over several months. There was no associated history of trauma, dental pain, paraesthesia, or constitutional symptoms like fever or weight loss. The patient's medical history was unremarkable, with no prior diagnosis of renal disease or endocrine disorders. A panoramic radiograph obtained externally had raised suspicion for a giant cell reparative granuloma versus a malignant bone lesion, prompting referral for advanced imaging.

The patient subsequently underwent a dedicated contrast-enhanced computed tomography (CT) scan of the facial bones and neck for definitive characterization. Following the imaging findings, an incisional biopsy of the most accessible mandibular lesion was performed for histopathological examination. Concurrently, serum biochemical assays were requested to evaluate calcium and parathyroid hormone levels.

CT Findings

The contrast-enhanced CT examination revealed extensive and multifocal bony abnormalities. The primary involvement was within the mandible, which showed multiloculated, expansile lytic lesions. These lesions were identified in the parasymphyseal region, both mandibular bodies, bilateral rami, and extending to the left mandibular condyle (Figures 1-3).

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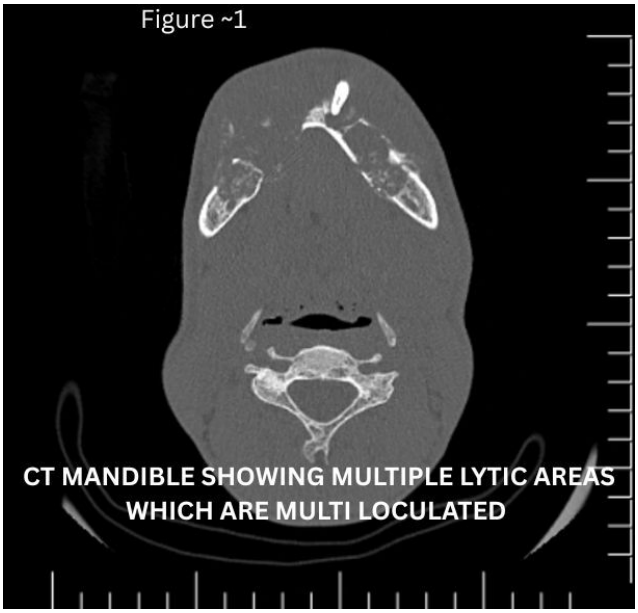

Figure 1: **Axial CT image (bone window) showing multiloculated expansile lytic lesions involving the parasymphyseal region and bilateral mandibular bodies with marked cortical thinning and a focal cortical breach**

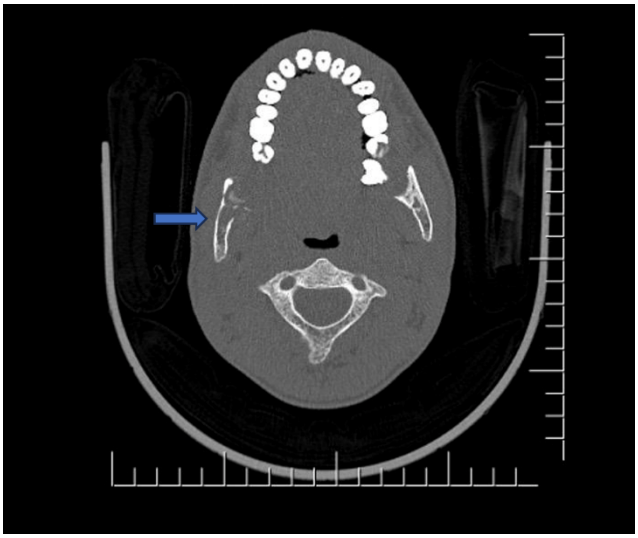

Figure 2: Axial CT image (bone window) demonstrating extensive lytic involvement of the bilateral mandibular rami with areas of focal cortical breach (arrow).

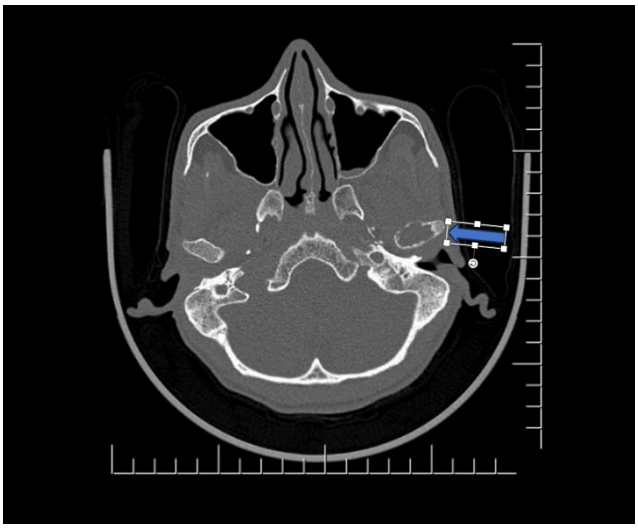

Figure 3: Axial CT image (bone window) depicting a small lytic focus in the left mandibular condyle (arrow), without significant cortical expansion.

A key observation was the presence of marked cortical thinning with several focal areas of cortical breach, most notably in the parasymphyseal and left condylar regions. Despite these cortical disruptions, the overall architecture of the mandible was maintained, and both temporomandibular joints demonstrated preserved alignment without evidence of subluxation or dislocation.

The lesions exhibited mild heterogeneous post-contrast enhancement within their fibrovascular matrix. No significant periosteal reaction or associated soft tissue mass was evident. Bilateral submandibular lymph nodes were mildly enlarged, likely representing a reactive change. The scan of the maxilla and nasopharynx was within normal limits.

A critical and incidental finding was detected in the lower neck. Inferior to the right thyroid lobe, a well-circumscribed, oval, homogeneously enhancing lesion measuring approximately 13 x 15 mm was clearly visualized (Figure 4).

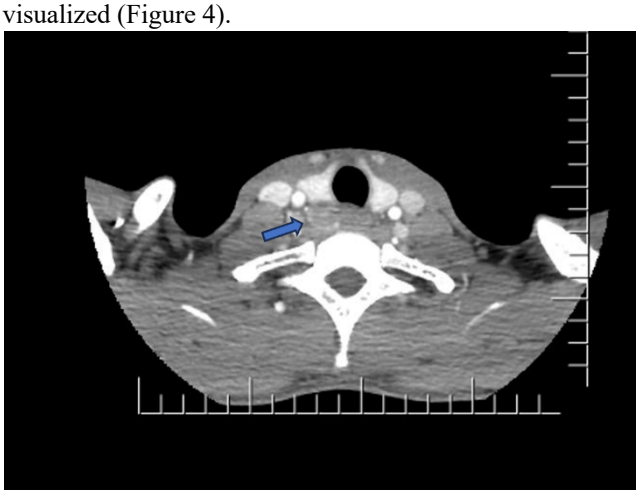

Figure 4: **Axial contrast-enhanced CT image (soft tissue window) of the neck revealing a well-defined, enhancing lesion (arrow) inferior to the right thyroid lobe, consistent with a suspected parathyroid adenoma**

This lesion was anatomically distinct from the thyroid gland and exhibited imaging features classic for a parathyroid adenoma.

Radiological Impression: Multifocal lytic lesions of the mandible, highly suggestive of brown tumours in the context of hyperparathyroidism, with an incidental right parathyroid adenoma as the likely underlying cause.

Follow-Up and Correlation

The histopathological report from the mandibular biopsy described a cellular lesion composed of numerous multinucleated osteoclastic giant cells dispersed within a richly vascularized fibroblastic stroma. Focal areas of hemorrhage and hemosiderin pigment deposition were noted. There was no evidence of cytological atypia or mitotic activity. These features were diagnostic of a brown tumour [2, 5]. The biochemical serum profile provided confirmatory evidence. The patient's serum calcium level was elevated at 12.8 mg/dL (reference range: 8.5-10.2 mg/dL). Most strikingly, the intact parathyroid hormone (PTH) level was profoundly elevated at 585 pg/mL (reference range: 10-65 pg/mL), nearly tenfold the upper limit of normal. These findings conclusively established the diagnosis of primary hyperparathyroidism.

The patient was referred to the endocrine surgery team for further management of the parathyroid adenoma. Treatment with parathyroidectomy was planned, which is expected to normalize PTH levels and lead to subsequent regression or stabilization of the mandibular brown tumours. Written informed consent was obtained from the patient for the publication of this case report and any accompanying images. The patient was informed that all personal identifiers would be removed to ensure anonymity.

DISCUSSION

Brown tumours are a skeletal hallmark of severe or long-standing hyperparathyroidism. They result from unregulated osteoclastic bone resorption, triggered by excessive PTH, which creates localised areas of bone loss that are filled with fibrovascular tissue, giant cells, and degraded blood products [1, 6]. In primary hyperparathyroidism, which is most commonly caused by a solitary parathyroid adenoma as in this case, the incidence of overt brown tumours has decreased in the era of early biochemical screening but remains an important clinical manifestation [3,6].

The mandible is a predisposed site, potentially due to its high metabolic turnover and rich vascular supply [2, 3]. The multifocal presentation seen in this patient, while not uncommon, amplifies the radiological dilemma, as it can simulate multifocal metastatic disease or disseminated primary bone pathology like Langerhans cell histiocytosis [4, 5]. Key differentiating imaging features of brown tumours include their lytic, often expansile nature, a "soap-bubble" or multiloculated appearance, and the absence of a sclerotic rim. However, as demonstrated, they can cause cortical thinning and even breach, blurring the distinction from more aggressive entities [1,4].

This case powerfully illustrates the integral role of comprehensive imaging. Contrast-enhanced CT served multiple crucial functions, it precisely mapped the extensive and multifocal involvement of the mandible; it assessed the integrity of vital structures like the mandibular condyle and TMJs, which were fortunately preserved; it characterized the internal architecture of the lesions; and most significantly, it extended the field of view to successfully identify the unsuspected parathyroid adenoma in the neck. This incidental finding was the pivotal clue that directed the entire diagnostic pathway. Without it, the working diagnosis would have remained a primary jaw pathology, potentially leading to more extensive jaw surgery [7, 8].

The diagnosis hinges on a triad of evidence namely radiological appearance, histopathological confirmation, and biochemical proof of hyperparathyroidism [4,6]. Histopathology alone cannot reliably distinguish a brown tumour from other giant cell-rich jaw lesions like the central giant cell granuloma; the cellular morphology is essentially identical [5]. Therefore, the biochemical demonstration of hyperparathyroidism is the definitive discriminant. This underscores the necessity of always checking serum calcium and PTH levels when a giant cell lesion is identified in bone, especially in the jaws.

Management is directed at the underlying endocrine abnormality. Treatment of the hyperparathyroidism, typically via parathyroidectomy for an adenoma, normalizes PTH levels. This halts the pathological bone resorption, allowing for natural healing and remineralisation of the brown tumours, often obviating the need for direct surgical intervention on the skeletal lesions themselves [4, 7]. Early diagnosis is thus critical to prevent unnecessary maxillofacial surgery and to mitigate the systemic complications of hypercalcaemia, such as renal stones and cardiovascular issues.

CONCLUSION

This case of multifocal mandibular brown tumours secondary to a parathyroid adenoma highlights several key learning points. Firstly, brown tumours should be considered in the differential diagnosis of multifocal lytic jaw lesions, despite their potential to mimic malignancy. Secondly, contrast-enhanced CT is an invaluable tool that provides detailed anatomic characterization of the lesions and can serendipitously identify the underlying parathyroid pathology. Thirdly, a definitive diagnosis requires correlation across three domains: imaging findings, histopathology of the bone lesion, and biochemical confirmation of hyperparathyroidism. Finally, accurate diagnosis shifts the treatment focus from the jaw lesions to the endocrine disorder, preventing inappropriate surgery and guiding effective management. Radiologists and surgeons must maintain a high index of suspicion for this condition to ensure timely and correct diagnosis...

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