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Case Report

Late resolution of brown tumor two years after parathyroidectomy: Case report with eight years follow-up

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ABSTRACT

Brown tumors are a type of non-neoplastic lesion characterized by the presence of osteolytic bone lesions in patients with hyperparathyroidism. These lesions are more commonly seen in females, older patients, and can also occur in other bones such as the femur, clavicle, ribs, and pelvis. This case report aims to highlight a rare case of a male patient who was diagnosed with a brown tumor two years after undergoing parathyroidectomy for parathyroid carcinoma. PTH, calcium, and phosphate levels were within normal ranges. Histopathological analysis revealed features indistinguishable from a giant cell–rich lesion; however, normal PTH, calcium, and phosphate levels excluded active hyperparathyroidism, supporting the diagnosis of a residual brown tumor. The lesion was considered an old brown tumor caused by hyperparathyroidism secondary to parathyroid carcinoma. Clinical and radiological follow-ups were conducted over an 8-year period, demonstrating bone neoformation with no signs of recurrence. Prosthetic rehabilitation was completed. Comprehensive evaluation of brown tumors is essential to ensure accurate diagnosis and appropriate treatment. Parathyroid carcinoma can also be a cause of hyperparathyroidism and can be related to a poor outcome if not treated adequately. These bone lesions should be a sign for a judicious systemic evaluation to be performed to exclude malignancies and also discover if there is an underlying cause. Late detection of a jaw brown tumor after parathyroidectomy with normal biochemical parameters is exceedingly rare, particularly in male patients.

Keywords: hyperparathyroidism, parathyroid neoplasms, bone resorption, bone remodeling, parathyroid hormone

Statement of Clinical Significance

Parathyroid carcinoma is a rare cause of primary hyperparathyroidism, linked to poor outcomes if inadequately treated or monitored. We report a male patient with delayed resolution after parathyroidectomy. Bone lesions necessitate systemic evaluation to exclude malignancy and identify underlying etiologies.

INTRODUCTION

Brown tumors are a type of non-neoplastic lesion that arise due to alterations in bone metabolism in patients with hyperparathyroidism. They may present as expansible masses, leading to oral and/or facial asymmetry, tooth displacement or mobility and occasionally pain. These lesions are characterized radiographically by the presence of osteolytic bone lesions with well-defined margins in various bones of the skeleton, with the mandible being one of the most affected sites. Hyperparathyroidism (HPT) is clinically classified into three distinct types: primary results from parathyroid adenomas or carcinomas, renal diseases, excessive phosphate retention, impaired degradation or resistance to parathyroid hormone (PTH), or calcium-PTH feedback dysregulation; secondary manifests as a compensatory elevation of PTH levels in response to chronic hypocalcemia or vitamin D deficiency; tertiary the condition arises in the context of chronic secondary HPT, wherein the parathyroid glands transition to a state of autonomous hypersecretion. Sustained elevation of PTH in primary, secondary, or tertiary hyperparathyroidism leads to increased osteoclastic activity and abnormal bone remodelling [1–3]

Histopathologically, brown tumors are identical to giant cell–rich lesion in the jaws, which are benign lesions that arise from increased osteoclastic activity due to hypercalcemia caused by high levels of PTH and elevated calcium absorption. These lesions are more commonly seen in females, particularly in

older patients, and can also occur in other bones such as the femur, claviculae, ribs, and pelvis. [1,2,4,5]

Management of brown tumors centers on correction of the underlying HPT, as normalization of PTH levels often leads to lesion stabilization or regression. Treatment may include pharmacological therapy or surgical excision of hyperfunctioning or malignant parathyroid tissue. Local surgical intervention of jaw lesions should be reserved for extensive, symptomatic, or persistent lesions that do not regress after adequate metabolic control.[1,2,6]

In this report, we present a rare case of a male patient who was diagnosed with a brown tumor two years after undergoing parathyroidectomy for parathyroid carcinoma.

CASE REPORT

This study was approved by the Ethics Committee of the Faculty of Medicine (protocol 5160/24), University of Sao Paulo, Sao Paulo and Brazil National Health Council (protocol number 80750424.3.0000.0068).

A 63 years-old male patient was referred to the Cancer Institute of São Paulo State (Instituto do Câncer do Estado de São Paulo, ICESP) with a history of tibia and femur pathologic fractures and a histopathological diagnosis of giant-cell tumor. At that stage, no systemic biochemical correlation had been performed, which contributed to the initial diagnostic interpretation. At the first evaluation, he presented with hypercalcemia, confusion and a femur fracture untreated. In addition, the patient had high PTH levels (959 pg/ml) with no vitamin D alterations. The main hypothesis was that the tumors were due to hyperparathyroidism.

Imaging showed multiple osteolytic bone lesions affecting the left tibia and the right femur, and scintigraphy **(Figure 1)**

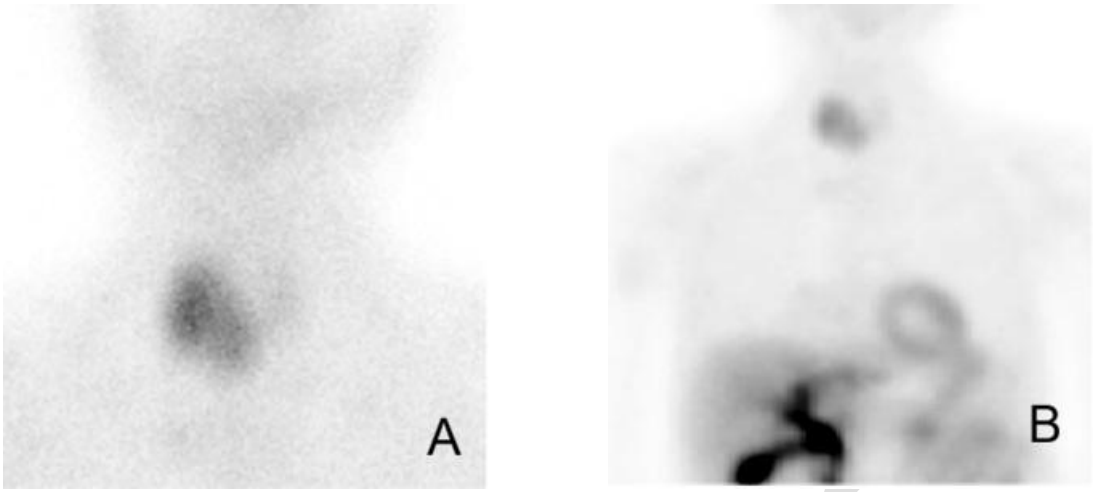


Fig 1. Parathyroid scintigraphy scans showing an increased uptake in the anterior cervical (A) and thorax (B) .

and magnetic resonance imaging (MRI) identified an expansive solid lesion in the tracheoesophageal sulcus with 4,5 cm x 3,7 cm of extension. After incisional biopsy was diagnosed as parathyroid carcinoma. Biopsy was performed in bone lesions, resulting in numerous multinucleated giant cells osteoclast-like, mononucleated cells with low mitotic index in the stroma and regions bone remodeling. The femur fracture was surgically corrected one year after our first evaluation. Hip arthroplasty was performed in the same year.

In 2015, a right total parathyroidectomy was performed with thyroid lobectomy, isthmectomy, and neck dissection. Histopathological analysis confirmed parathyroid carcinoma graded as T4apN0. At this point, no jaw lesions were observed. Between 2019 and 2020 he was treated with total thyroidectomy after local recurrence. Two PET-CTs were realized in 2022 and 2024. No other areas with abnormal increase in glycolytic metabolism was observed

In 2017, two years after carcinoma treatment and endocrinologist support, the patient was referred to the Dentistry Department to investigate a persistent bone lesion in the mandible. Clinically, discreet and consistent swelling was observed in the left mandibular body, near the inferior first premolar, with no signs of infection, and the patient had any complaints **(Figure 2).**

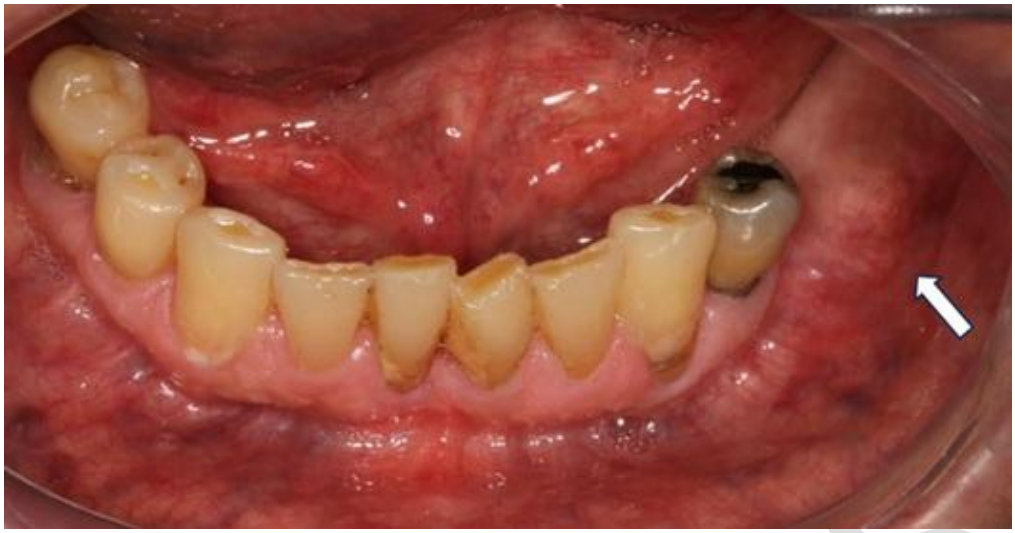


Fig 2. Clinical photograph of an occlusal view of the mandibular region. Arrow showing non-fluctuating, discrete tumefaction was observed in the left mandibular body.

No other changes were observed.

On panoramic radiography, a radiolucent lesion associated with the inferior left first premolar, well delimitated, unilocular, approximately 02 cm was the only alteration in the jaws **(Figure 3)**,

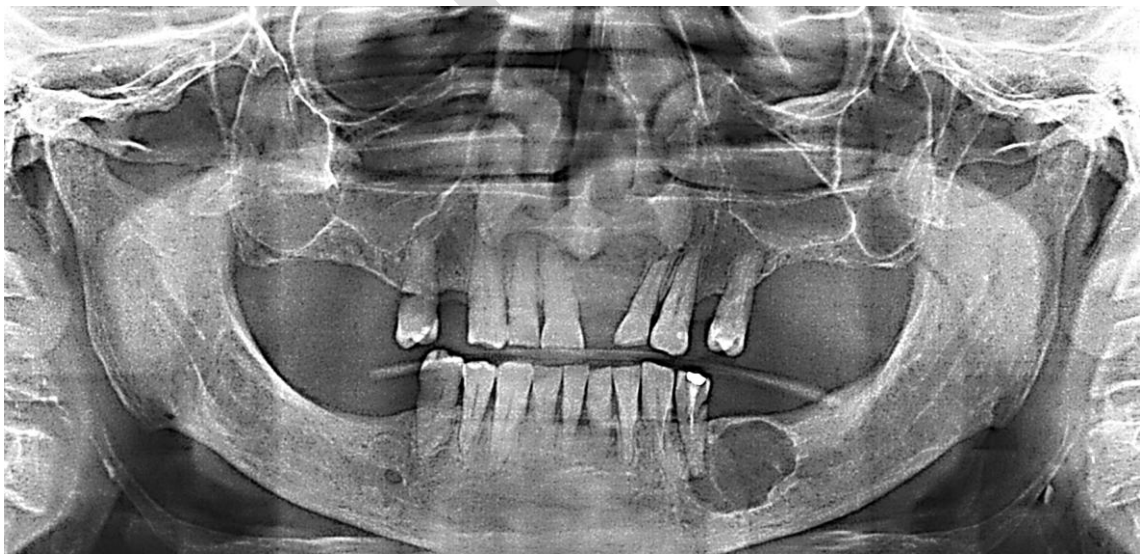


Fig 3. Panoramic radiography of a well-defined unilocular radiolucent lesion in relation to the root of the left lower premolar teeth

as seen on computerized tomography **(Figure 4)**.

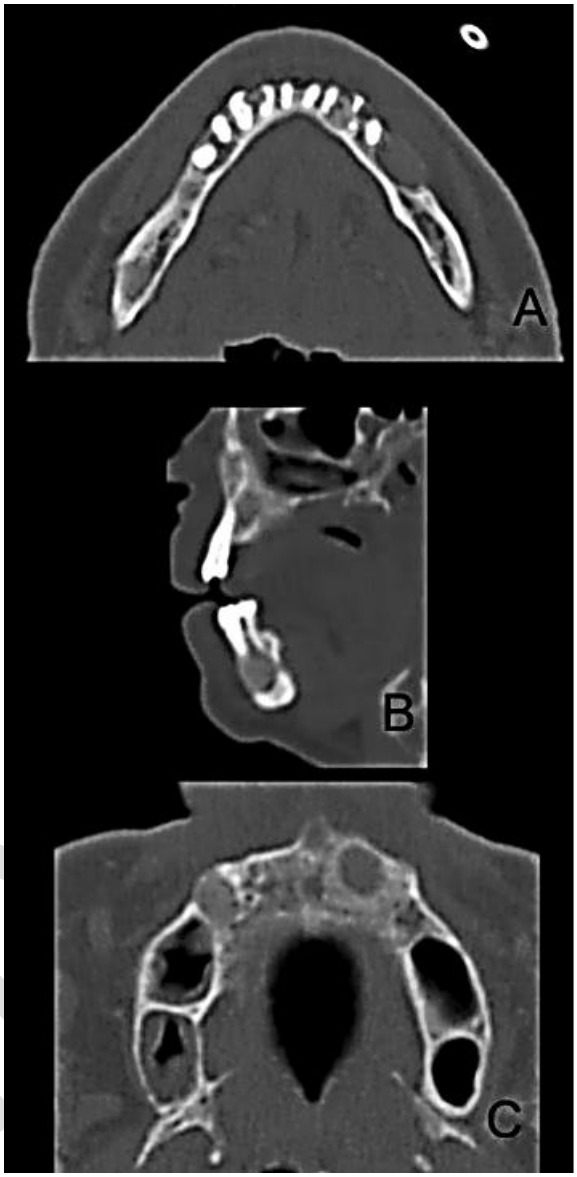


Fig 4. Computerized tomography for the evaluation of maxillary and mandibular bone lesions. A: mandibular arch (axial view) well delimitated, unilocular radiolucent area,; B: sagittal cross-section and C: maxilla (axial view) showing radiolucent area in anterior region

Additional radiolucent areas were identified in the maxilla; however, these lesions were asymptomatic, radiologically stable, and showed progressive sclerosis during follow-up PTH, calcium, and phosphate levels were within normal ranges as seen in **Table 1**

Table I.

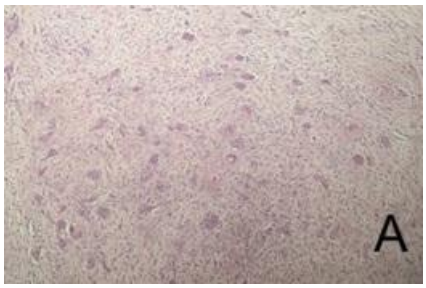
Serum levels of phosphorus, alkaline phosphatase, parathyroid hormone (PTH), ionized calcium, total ca

Year	*P (mg/dl)*	*AP (U/l)*	*PTH (pg/ml)*	*IC(mg/dl)*	*TC(mg/dl)*	*25HD(ng/ml)*
2015	1.5	267	959	6.55	13.2	44
2016	2.7	174	61	5	9.1	23
2017	2.4	UN	38	5.38	10.3	36.5
2018	2.5	105	38	5.64	10	31.7
2019	2.2	UN	75	6.11	11.9	43.3
2020	3.9	73	14	4,91	9.20	41.6
2021	2.6	73	25	5.38	10.3	45
2022	2.5	71	44	6.01	11.2	33.9
2023	2.09	51	107	6.77	11.9	35.2

UN= unfulfilled
P=Phosphorus (2.7 to 4.5mg/dl)
AP= Alkaline phosphatase (40 to 129U/l)
PTH= Parathyroid hormone (15 to 65pg/ml)
IC=Ionized calcium(4,6 to 5,3mg/dl)
TC=Total calcium (8,6 to 10,2mg/dl)
25HD= 25-Hydroxyvitamin D (30 to 60ng/ml)

that demonstrates the variation parameters.

Excisional biopsy was performed under local anesthesia by accessing the lesion and performing careful curettage and peripheral osteotomy. Histopathological analysis confirmed the hypothesis of a central giant cell lesion **(Figure 5)**,



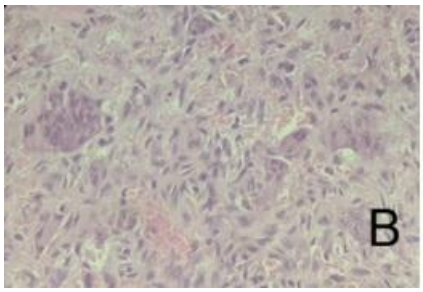
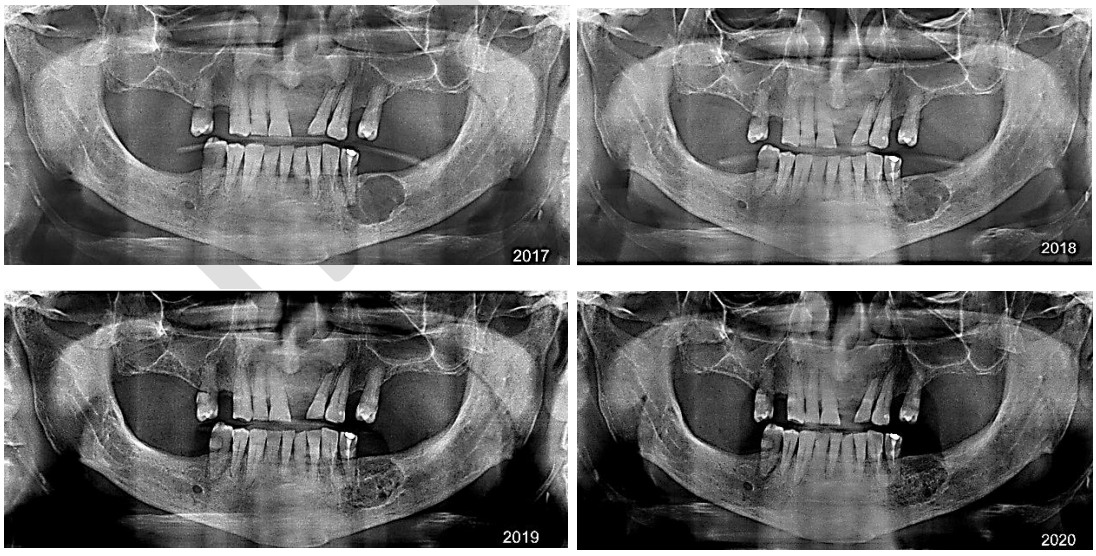


Fig 5. Photomicrographs of hematoxylin and eosin staining. A: Photomicrograph at magnification (100×) exhibiting central giant and fusiform cells; B: Photomicrograph at magnification (400×) cytomorphological aspects

but systemic evaluation showed no persistence of hyperparathyroidism.

Despite histological overlap with central giant cell lesions, the absence of biochemical hyperparathyroidism, combined with the patient's history of severe hyperparathyroidism secondary to parathyroid carcinoma and radiological stability, supported the interpretation of a residual (inactive) brown tumor rather than an active lesion

Therefore, this lesion was considered an old brown tumor caused by hyperparathyroidism secondary to parathyroid carcinoma. Clinical and radiological follow-ups were made every 06 months demonstrating bone neoformation with no signs of recurrence, including the maxillary lesions. Prosthetic rehabilitation was completed in 2022. Until now, the patient had no complaints, and the imaging results revealed no alterations **(Figure 6)**.



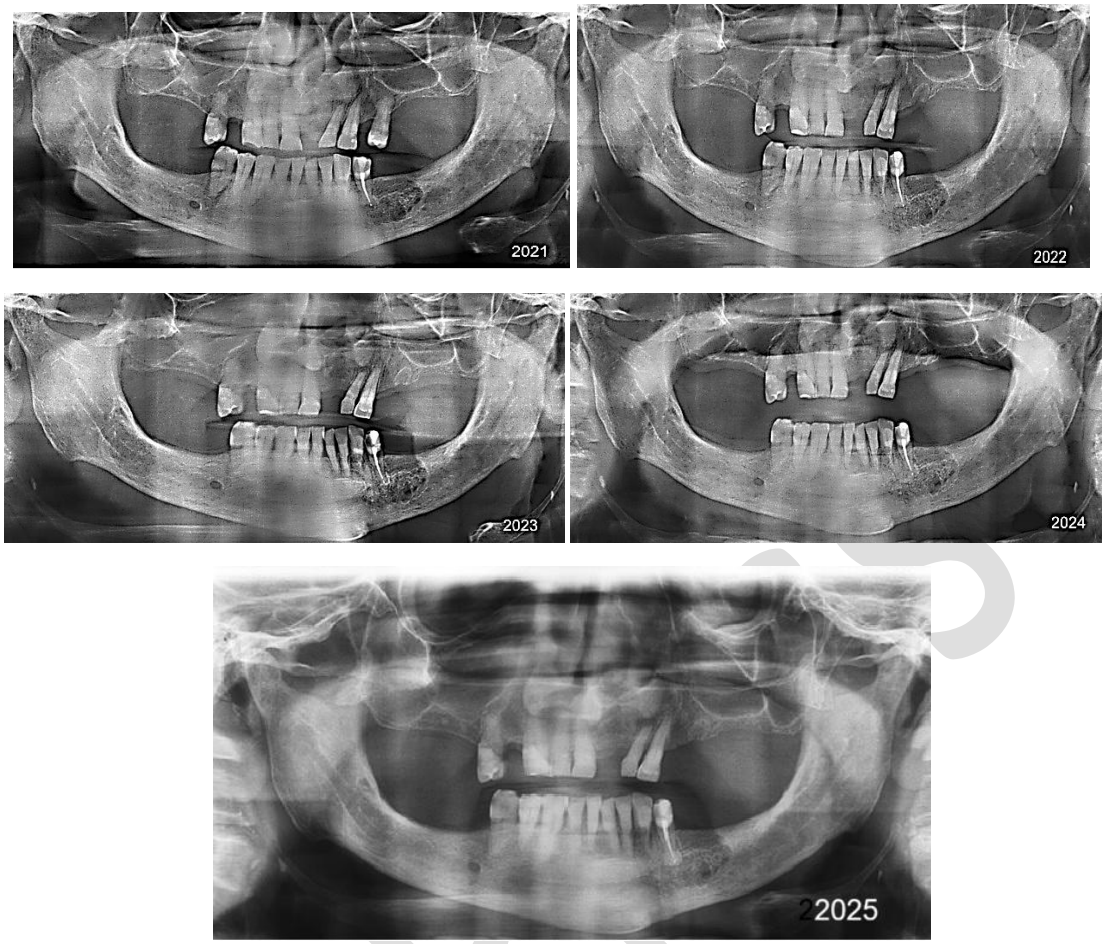


Fig 6. Panoramic radiograph demonstrating bone neoformation with 8 years follow-up, there was no progression of the lesion.

In 2024 a mass in the inferior lobe of the right lung was identified and biopsied, showing a metastatic parathyroid carcinoma, which can relate to the recurrent increase of PTH levels and also with an osteolytic lesion in the right iliac crest that suggests a new brown tumor. Attention must be given to parathyroid

carcinoma because, although rare, the prognosis may be unfavorable, with only 49% to 66% of patients surviving after 10 years, and the recurrence rate is high.[10,11,13,14] The patient is under medical assistance to determine the best treatment. Yet there is no sign of recurrence of osteolytic lesions in the mandible until 2025 (**Figure 6)**.

DISCUSSION

Hyperparathyroidism (HPT) may present as a primary disease but is also associated with other systemic conditions that lead to alterations in bone metabolism characterizing as secondary or tertiary hyperparathyroidism, and these alterations may cause intraosseous lesions called brown tumors. When it affects the head region, especially the mandible, can also show osteolytic lesions, leading to signs such as asymmetry and tooth dislocation.[7–9] Ibrahem[3] reviewed the brown tumor literature and observed that 70% of jaw lesions occurred in the mandible of females older than 50 years.

There are no histological differences between the brown tumor and a central giant cell tumor. Consequently, clinicians must conduct a comprehensive systemic evaluation when encountering osseous lesions resembling giant cell tumors. Inaccurate diagnoses can lead to significant adverse outcomes, including mortality, given that parathyroid carcinoma is one of the etiologies of HPT.[4,5] Multiple bone lesions may also lead health professionals to misdiagnose other conditions[1,10,11] as exemplified by Nguyen et al.[12] who reported a case of a female patient initially diagnosed with multiple myeloma due to osteolytic lesions. However, upon further investigation, the patient was found to have parathyroid carcinoma with subsequent HPT, and no evidence of multiple myeloma was present.

The patient reported presented with confusion, a clinical sign of hypercalcemia, and an untreated femur fracture; therefore, the systemic investigation showed a high PTH level. Imaging exams and biopsies revealed not only giant cell tumors but also parathyroid carcinoma that was causing hyperparathyroidism. Jaw lesions were managed by the dental team with

osteoplasty and endodontic treatment of the affected tooth. This lesion was not identified at first evaluation and before the first intervention of parathyroid carcinoma The patient presented the mandible lesion two years after the diagnosis but sooner than the local recurrence was identified and managed. Hence, the mandibular brown tumor can be due to this recurrence and so was investigated later.

The etiology and pathogenesis of parathyroid carcinomas are still controversial although they may occur sporadically or associated with multiple endocrine neoplasia 1 and 2 and isolated familial hyperparathyroidism-jaw syndrome. The presence of skeletal or renal lesions is not uncommon in parathyroid carcinoma.[15]

Apaydin et al.[14] demonstrated that the elevated levels of PTH and calcium are more strongly associated with parathyroid carcinoma than parathyroid adenoma; therefore, clinicians should consider this possibility in suspicious cases. Tsushima et al.[10] also observed that osteolytic lesions can mimic bone metastasis of parathyroid carcinoma; thus, biopsy is essential for accurate diagnosis. There are only a few cases in the literature published so far demonstrating the occurrence of brown tumors in the jaws with parathyroid carcinoma. In a case report, a female young patient presented with a parathyroid carcinoma associated with brown tumors in both jaws. A reduction in lesion size was observed during cancer treatment[16] as well as observed with this case report, although the mandible lesion only was diagnosed later and can be related to the local recurrence.Parathyroid carcinoma has a better prognosis if resected with free margins on the first surgery. Adjuvant radiotherapy is yet unclear although some cases report showed better local control. The metastatic form of the disease has a difficult control and the consequences of HPT must be treated.[10,13,15]

The treatment of brown tumors primarily involves addressing the underlying cause of hyperparathyroidism, followed by surgical intervention for osseous lesions that have not undergone reduction in size or regression. Surgical options include curettage, osteoplasty, or resection for larger cases. If

hyperparathyroidism is left untreated, brown tumors can relapse or new lesions may affect other sites. [1,2,6,7,9,17,18] Fedhila et al.[9] conducted a retrospective study of 16 cases of brown tumors, wherein 9 cases were resolved by treating hyperparathyroidism with parathyroidectomy. They observed complete regression of the jaw lesions. In the remaining seven cases, surgery was necessary to achieve a favorable outcome for the osseous abnormalities. In the case presented, parathyroidectomy resulted in decreased PTH levels and halted the growth of the brown tumors; however, after one year, PTH levels increased once again. A subsequent systemic investigation revealed a metastatic tumor. Consequently, the patient may experience recurrences or develop new lesions; however, the main purpose is to address the most appropriate oncologic treatment.

Even with aggressive carcinoma, PTH level control showed a favorable outcome of bone lesions, demonstrating bone remodeling and slow ossification during the years after prolonged HPT.

CONCLUSION

This case illustrates that brown tumors may persist or be detected years after biochemical normalization of hyperparathyroidism, particularly in patients with parathyroid carcinoma However with the adequate investigation the clinician

can address their origin and treat the patient in the right manner. Prognosis is favorable even in cases when osteoplasty is necessary.

Parathyroid carcinoma is not a frequent malignant lesion but can also be a cause of hyperparathyroidism and be related to a poor outcome if not treated adequately or if the follow-up is not closely observed. These bone lesions should be a sign to a judicious systemic evaluation to be performed in order to exclude malignancies and also discover if there is an underlying cause.

Long-term multidisciplinary follow-up is essential, as skeletal manifestations may not parallel endocrine control and may coexist with late metastatic disease.

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AUTHORS' CONTRIBUTIONS

Mauricio Neves Gomes: investigation, data curation , validation, visualization, writing – original draft, writing – review & editing, project administration

Ariadne Padilha Andrade: investigation, methodology, writing – original draft

Wagner Gomes-Silva: conceptualization

Priscila Abranches de Britto Pinheiro: writing – review & editing.

Rodrigo Nascimento Lopes: investigation, methodology,

Bruno Felipe Gaia: investigation, methodology, writing – original draft

Marco Aurélio Petroni Montezuma: investigation, methodology,

Luiz Guilherme Cernaglia Aureliano de Lima: investigation, methodology,

Thais Bianca Brandão: supervision, review & editing

CONFLICT OF INTEREST STATEMENT

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Ethics approval: (Autor favor confirmar)

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