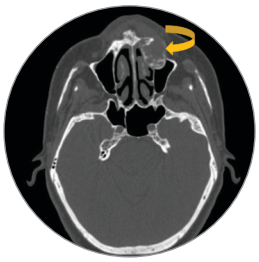


Multifocal Brown Tumor as A Manifestation Of Primary Parathyroid Carcinoma. A Case Report

Tumor pardo multifocal como manifestación inicial de carcinoma paratiroideo. Presentación de caso

María Berenice Reyes[1]
Silvia Lissett Espinoza Alvarado[2]
Didier Armando Robles López[3]
Carlos Rivera Argeñal[4]
Yaritza Turcios[5]

Key words (MeSH)

Osteitis fibrosa cystica
Parathyroid neoplasms
Primary hyperparathyroidism
Radiography

Palabras clave (DeCS)

Osteítis fibrosa quística
Neoplasias de las paratiroides
Hiperparatiroidismo primario
Radiografía

Summary

Parathyroid carcinoma is an exceptional cause of primary hyperparathyroidism. Brown tumors develop as skeletal manifestations of primary hyperparathyroidism in the context of advanced disease. They are highly vascular, lytic bone lesions with a reparative cellular process instead of a neoplastic process. They can be identified in the secondary forms of the disease, however, they are more frequent in primary hyperparathyroidism. We present a case of multiple brown tumors secondary to parathyroid carcinoma. The initial manifestation of primary hyperparathyroidism was a pathological fracture secondary to brown tumor associated with hypercalcemia and elevated levels of parathyroid hormone (PTH), which is rare due to the fact that in recent decades the incorporation of serum calcium in routine laboratory tests detects this disease in asymptomatic or minimally symptomatic stages. Ultrasound detected a vascularized solid lesion in the anatomical site of the left lower parathyroid and nephrocalcinosis. The lesion was surgically resected in the left hemicolumn and the biopsy reported parathyroid carcinoma. Brown tumors are non-neoplastic lesions that do not have malignant potential and are therefore considered repairing granulomas.

Resumen

El carcinoma de paratiroides es una causa excepcional de hiperparatiroidismo primario. Los tumores pardos se desarrollan como manifestaciones esqueléticas de hiperparatiroidismo primario en el contexto de una enfermedad avanzada. Son lesiones óseas líticas, altamente vasculares con un proceso celular reparador en lugar de un proceso neoplásico. Pueden identificarse en las formas secundarias de la enfermedad; sin embargo, son más frecuentes en el hiperparatiroidismo primario. Se presenta el caso de múltiples tumores pardos secundarios a un carcinoma de paratiroides. La manifestación inicial del hiperparatiroidismo primario fue una fractura patológica secundaria a tumor pardo asociado con hipercalcemia y niveles elevados de hormona paratiroidea (PTH), lo cual es infrecuente debido a que en las últimas décadas la incorporación del calcio sérico en las pruebas de rutina de laboratorio detecta esta enfermedad en estadios asintomáticos o mínimamente sintomáticos. Por ultrasonido se detectó lesión sólida vascularizada en sitio anatómico de paratiroides inferior izquierda y nefrocalcinosis. Se sometió a resección quirúrgica de la lesión en hemicuello izquierdo y la biopsia reportó carcinoma de paratiroides. Los tumores pardos son lesiones no neoplásicas que no tienen potencial maligno por lo que se consideran granulomas reparadores.

Introduction

In 1864, Gerhard Engel first described the brown tumor and in 1925 Mandl associated it with hyperparathyroidism. The name brown tumor originated from its macroscopic histological appearance, as the brown mass was made up of a combination of microfractures, recurrent in various stages of remodeling, with blood, hemosiderin, fibrous tissue and connective tissue. Although they are called tumors, they are not; according to this author, brown tumors are not the result of a neoplastic process. They are focal bone lesions as a result of bone remodeling by hyperparathyroidism or paraneoplastic syndrome (1).

The increase of paratohormone can be the result of primary hyperparathyroidism, which, in 80-90% is secondary to a parathyroid adenoma, 5-10% is produced by multiglandular disease and in less than 1% by parathyroid carcinoma. It can also be caused by a secondary hyperparathyroidism, such as chronic renal failure and intestinal malabsorption, or tertiary hyperparathyroidism, as an autonomous functioning of the parathyroids in cases of long-standing hyperparathyroidism (2).

[1]Radiologist, Hospital Escuela Universitario. Hospital Escuela Universitario. Tegucigalpa, Honduras.

[2]Radiologist, postgraduate in Radiology. Universidad Nacional Autónoma de Honduras (UNAH).

[3]Radiologist, postgraduate in Radiology. Universidad Nacional Autónoma de Honduras (UNAH).

[4]Interventionist radiologist. University Hospital School (HEU). Tegucigalpa, Honduras.

[5]Third year resident, postgraduate degree in Radiology. Universidad Nacional Autónoma de Honduras (UNAH).

Hospital Escuela Universitario. Tegucigalpa, Francisco Morazán, Honduras

Primary hyperparathyroidism predominates in women with a 3:1 ratio and its prevalence increases with age from 0.5% in women aged 50-59 years to 1.75% in women aged 70-75 years. In Western countries, the symptomatology of primary hyperparathyroidism is very mild and its progression is very slow. The main effects are manifested in the renal and skeletal system. 75-80 % of the patients are asymptomatic; however, they can have non-specific symptoms such as fatigue, weakness, paresthesias, digestive affections and slight mental alterations. In many of these patients it is evident that fatigue and weakness were symptoms of the disease after parathyroidectomy, when the symptoms resolve (3).

The skeletal manifestations of primary hyperparathyroidism are the main cause of morbidity associated with this disease. The characteristic bone radiological findings are the subperiosteal resorption of the bone cortex in multiple locations - earlier in the radial aspect of the middle phalanges of the second and third fingers - generalized or juxtaarticular osteopenia and brown tumors (4, 5).

The brown tumor or cystic fibrous osteitis manifests itself as a lytic lesion with defined borders, characterized by focal clusters of fibrous tissue and giant cells in a highly vascularized hemorrhagic stroma (6, 7), they are usually multilocular, well-delimited and present thinning of the bone cortices as a result of a cellular repair process (8). In fact, some of the osteolytic lesions could be confused with bone metastases, with a solitary bone cyst, an aneurysmal bone cyst, a giant cell tumor, or a giant cell repair granuloma. In multiple brown tumors, the differential diagnosis includes: osteolytic metastases, multiple myeloma, multiple bone cysts, etc. It is the sclerotic margin that excludes metastasis (9).

They can occur with edema, pathological fractures and bone pain in the skeletal system. This is produced by an increase in osteoclastic resorption of the bone due to an increase in paratohormone in the blood. They mainly affect long bones, ribs, pelvis, maxilla, mandible, skull and even vertebrae (10).

The primary treatment of brown tumors is the surgical removal of the parathyroid gland, since most of them involute in a period of 6 months to 5 years. Normal serum levels of paratohormone, calcium, phosphorus, and increased bone mass in the tumor area are indicators of successful treatment. Surgical bone resection by curettage and enucleation is indicated in extensive cystic lesions with great bone destruction, which do not involute because in these the probability of mineralization is low once the cause of hypercalcemia is treated (4).

Parathyroid carcinoma is a rare endocrine malignancy, first described by De Quevain in 1909 (6). The behavior of this pathology has been described as of slow but progressive evolution, 95% are functional. It infiltrates locally, can spread to regional nodes and eventually present pulmonary metastases (11).

The incidence of parathyroid carcinoma is described as less than 1% of the cases of primary hyperparathyroidism (12).

The pathogenesis of parathyroid carcinoma is unknown. It may be sporadic or appear in the context of a genetic endocrine syndrome, such as in hyperparathyroidism/jaw tumor syndrome (HPT-JT), multiple endocrine neoplasm type 1 (MEN1), type 2A (MEN2A), and isolated familial hyperparathyroidism (IFHP) (13).

It manifests itself at an average age of 40 to 55 years, with equal distribution in the sexes. In staging it is classified as localized, metastatic and recurrent disease (14).

Parathyroid tissue is generally composed of major cells and may have oxyphilic cells, transition cells, and clear cells. Parenchymal cells generally intermingle with adipocytes in variable amounts; this adipose tissue corresponds to 10 to 30% of the glandular volume. The cellularity of a parathyroid gland varies within and between individuals. Generally, a parathyroid gland weighing more than 40 mg is abnormal (15).

With the evaluation of clinical, laboratory and radiographic characteristics, most parathyroid lesions can be easily classified. However, the diagnosis of parathyroid carcinoma can be difficult. Blood calcium and PTH levels are much higher in carcinoma than in benign causes of hyperparathyroidism. In metastatic parathyroid carcinoma, the main problems stem from the uncontrolled effects of excess PTH, and the main causes of morbidity and mortality are hypercalcemia and bone disease (16).

Parathyroid carcinomas are generally larger than adenomas, but there may be overlap in size. Carcinomas are generally associated with higher serum calcium levels than adenomas, but adenomas may overlap. Parathyroid carcinomas usually have higher mitotic rates than parathyroid adenomas, but mitotic activity also occurs in adenomas. Atypical mitoses are seen only in parathyroid carcinoma. Atypical parathyroid adenomas have some characteristics often seen in carcinomas, such as mitotic activity, cytological atypia, fibrous bands, adhesion to adjacent structures, trabecular growth, and tumor cells within the capsule, but lack of invasion or metastasis (17, 18).

To date, pathological vascular invasion, capsule rupture in structures adjacent to the neck or regional and distant metastases remain the criteria for malignancy (19).

Several findings have been described that help distinguish benign parathyroid adenomas from parathyroid carcinoma. The typical parathyroid adenoma is usually soft in consistency, round or oval in shape, and reddish-brown in color. In contrast, parathyroid carcinoma is often described as a lobed, firm to stony mass. In about 50% of cases it is surrounded by a dense, fibrous, gray-white capsule that adheres tightly to adjacent tissues and makes it difficult to separate the tumor from adjacent structures. If there is severe infiltration of the adjacent thyroid gland, nerves, muscles or the esophagus, or if there are obvious metastases in cervical nodes, the diagnosis of carcinoma is not difficult. However, any or all of these findings may be absent at the time of surgery, and pathological freezing examination is of little value in distinguishing benign from malignant disease (20).

Brown tumors are characterized by their lytic appearance on CT image and their enhancement on bone scan (21).

Currently, the Sestamibi-Tc99m scan is considered a tool with greater sensitivity (80-100%) to detect both parathyroid hyperfunction and the existence of ectopic glands (5).

Radiation therapy and chemotherapy are not effective in the management of parathyroid carcinoma. The patient is treated with calcium and vitamin D. Strict monitoring should be done in the two years following surgery, since most recurrences occur in the first 3-4 years. Without

However, relapses have been reported at the age of 10-15 years. The survival rate at 5 years is 50-80 %. Age, initial incomplete surgery and histological type (aneuploid) are factors of worse prognosis (14).

In diagnostic tests such as computerized tomography (CT) or magnetic resonance imaging (MRI), brown tumors produce images

that sometimes simulate lytic bone lesions that pose the differential diagnosis with metastasis of malignant tumors (10).

Presentation of the case

37-year-old female patient with a history of polyuria, polydipsia, asthenia and constipation, approximately two years old, with pain in her lower left limb during the last 4 months; she suffers from a fall from her own height, accompanied by weakness, edema and functional limitation of the ipsilateral hip. Upon physical examination, the left lower limb is shortened, in external rotation, with deformity in the proximal third of the thigh; no lacerations, bruises or hematomas are observed; he presents pain upon mobilization, with palpable distal pulses, without paresthesias.

Laboratory tests have the following results: paratohormone, 635 pg/mL; calcium, 14.8 mg/dL; vitamin D, 19 ng/mL; Bence Jones protein and negative tumor markers. A radiographic bone series was carried out in which multiple well-defined intramedullary lytic lesions can be seen, with thinning of the bone cortices in the right femur (Figure 1a) and a pathological fracture in the proximal third of the left femur (Figure 1b).

A radiographic bone series was made in which multiple well-defined intramedullary lytic lesions can be seen, with thinning of the bone cortices in the right femur (figure 1a) and a pathological fracture in the proximal third of the left femur (figure 1b).

An ultrasound of the abdomen identified multiple gallstones and hyperechoic, diffuse images in the medullary region of both kidneys due to nephrocalcinosis (figure 2). Also, hyperechoic images in the urinary bladder.

In the neck ultrasound, a hypo-echoic, solid, sharp-edged lesion with peripheral and central vascularity of 3×1.5×1.6 cms in its longitudinal, anteroposterior and transversal diameters with a volume of 3.9 cms^3 is identified in the anatomic site of the lower left parathyroid gland (figure 3).

In the multidetector computerized tomography (CT) of thorax and simple abdomen, oval and round lesions with defined edges were evidenced, with expansion of the bone cortices, located in the seventh right posterior costal arch, seventh and eighth left anterior costal arch, eighth and ninth posterior arches bilaterally (figure 4), in both scapulas and iliac bones, in right pubis, left ischium, left sacral wing, greater trochanter of both femurs, vertebral bodies T12, L3, and vertebral pedicle of L2 (figure 5). These lesions are characteristic of brown tumors. In addition, bilateral renal nephrocalcinosis, high-density imaging in the posterior region of the urinary bladder, and multiple lithographs in the gallbladder were identified.

In multidetector neck CT, similar lesions were observed on the inner wall of the left maxillary sinus, compressing the lower left nasal turbinate. In addition, lytic lesions with well-defined edges were observed in the right mandibular body, hard palate and in the intramedullary region of the C5 and C6 vertebral bodies (Figure 6). Subsequently, the mass was surgically resected, and freezing biopsy revealed parathyroid carcinoma (Figure 7).

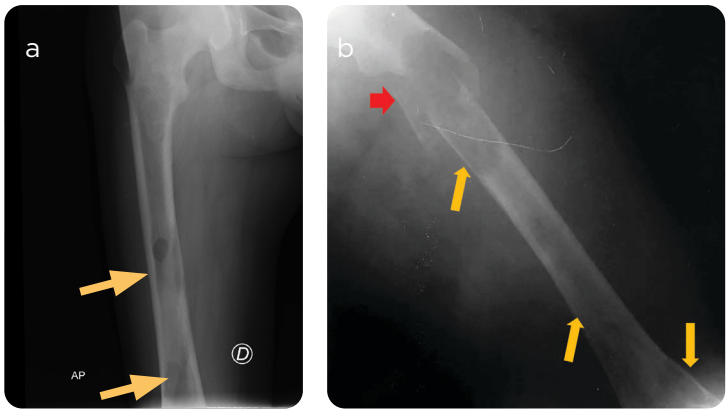


Figure 1. a) Antero-posterior (AP) projection of the right femur: In the middle and distal third of the femur there are two intramedullary oval lesions, radiolucent, with defined edges, without periosteal reaction (arrows). b) Lateral projection of the left femur: Displaced, multifragmentary fracture in the proximal third of the femur (arrowhead) associated with osteolytic lesion in the diaphyseal region and thinning of the cortex in the proximal, medial and distal third of the femur (arrows).

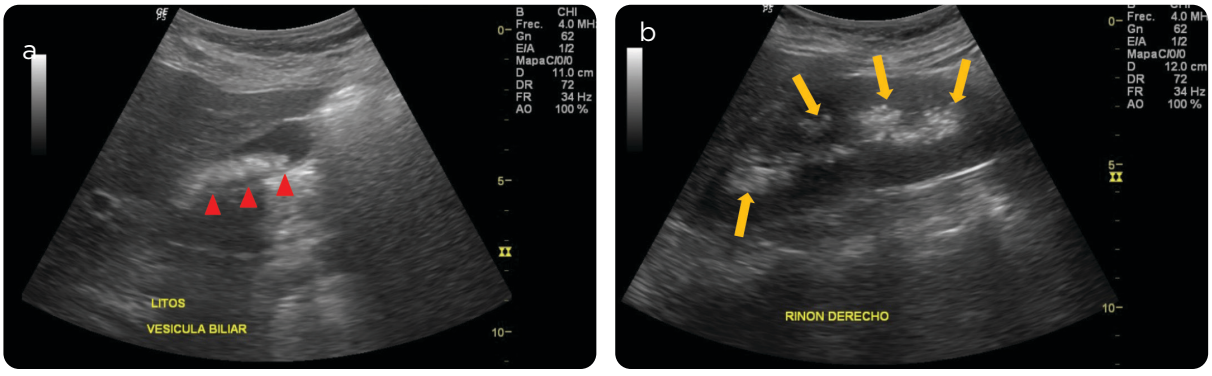


Figure 2. Ultrasound of upper abdomen. a) Longitudinal section of the gallbladder: multiple hyperechoic images with posterior acoustic shadowing in the gallbladder. b) Lateral view of the right kidney longitudinal axis: multiple hyperechoic images in the medullary region of the kidney confined to the renal pyramids.

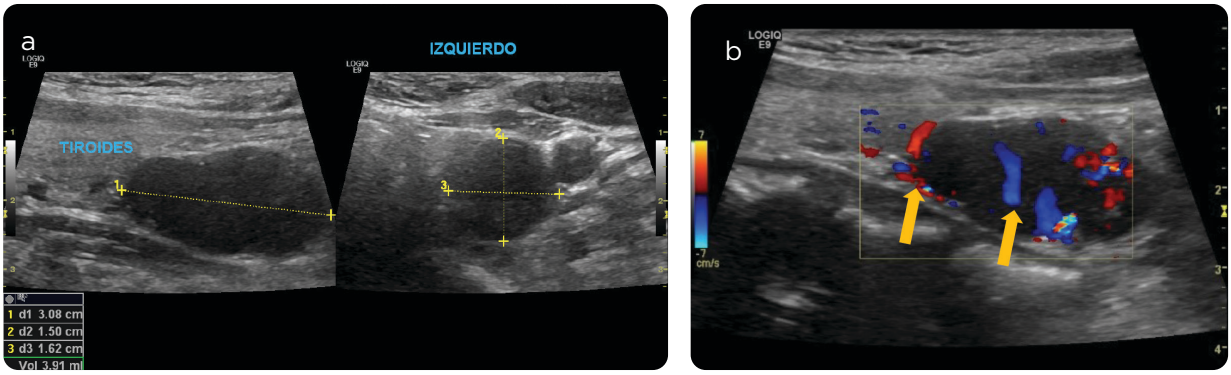


Figure 3. Neck ultrasound. a) In anatomical location of lower left parathyroid gland: oval, solid, hypoechoic, sharp-edged lesion. b) Color Doppler ultrasound: peripheral and central vascularity suggestive of malignancy (arrows).

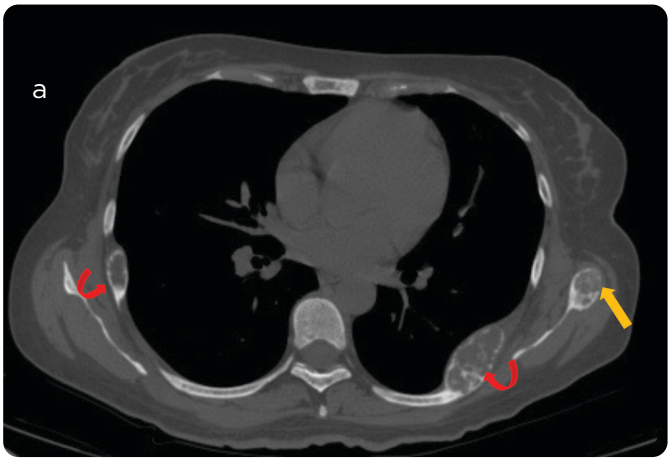


Figure 4. Bone window CT, axial section. In the seventh and eighth costal arches there are identified in the posterior segment and in the lateral edge of the left scapula lytic lesions that expand the cortex, characteristic of brown tumors.

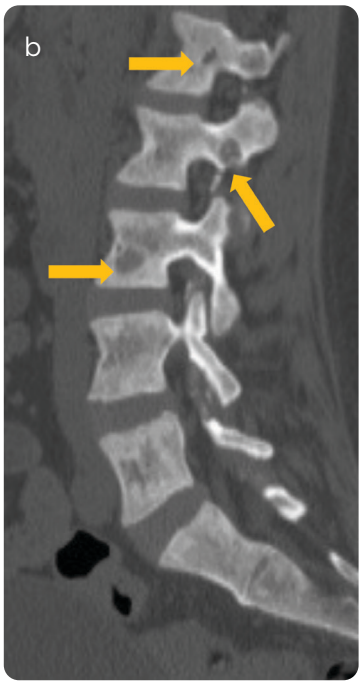


Figure 5. CT sagittal reconstruction of the spine: lithic, oval, intramedullary lesions that do not cause destruction of the cortical of the vertebral bodies of T12, L3 and in the pedicle of L2.

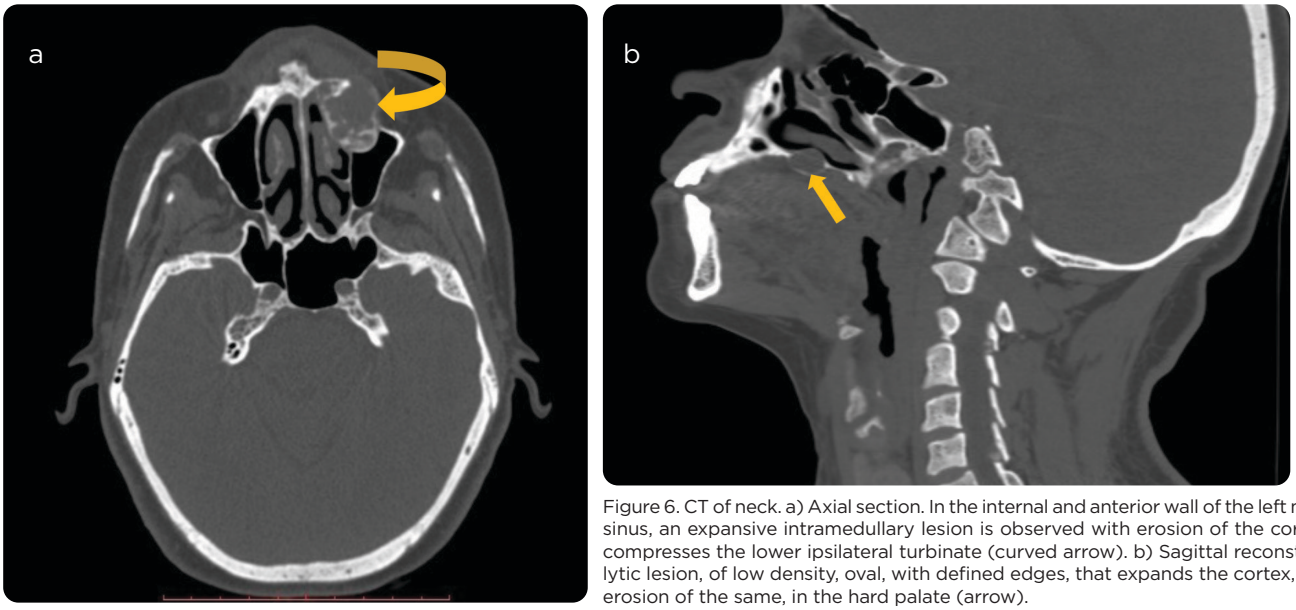
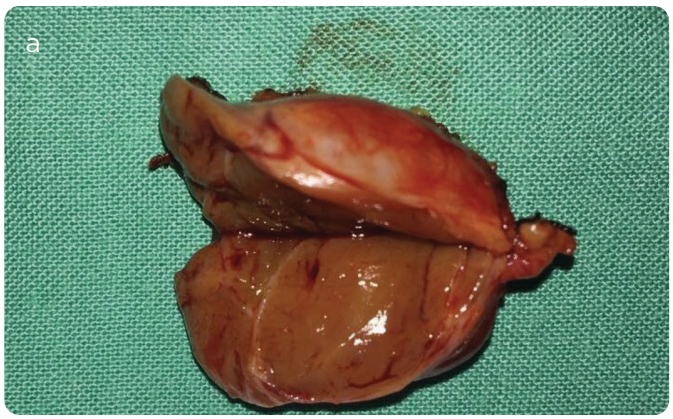


Figure 6. CT of neck. a) Axial section. In the internal and anterior wall of the left maxillary sinus, an expansive intramedullary lesion is observed with erosion of the cortex that compresses the lower ipsilateral turbinate (curved arrow). b) Sagittal reconstruction: lytic lesion, of low density, oval, with defined edges, that expands the cortex, without erosion of the same, in the hard palate (arrow).



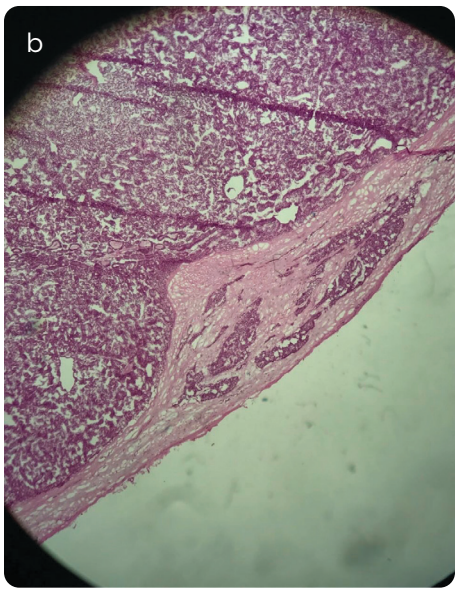


Figure 7. a) Lower left parathyroid mass of pinkish-gray surface, smooth, shiny, 2.9×1.5×1 cm and weighs 8 g. b) Histological section in which there is evidence of infiltration of the capsule, surpassing it and extending into the perithyroid tissue, with infiltration of the lower border and vascular/lymphatic permeation of the capsule vessels.

Discussion

Primary hyperparathyroidism is a relatively frequent pathology, it is the third most common endocrine pathology after diabetes and thyroid pathology. Approximately 80% of patients with primary hyperparathyroidism have a single parathyroid adenoma, 10-11% have more than one adenoma and less than 10% have hyperplasia of all four glands. Parathyroid carcinoma causes less than 1% of the cases of hyperparathyroidism (22).

Due to the ultrasound findings in the left hemicolumn, at the site of the lower parathyroid gland, multiple lytic lesions in the different bone structures, pathological femoral fracture, hypercalcemia and elevated PTH, brown tumor associated with primary hyperparathyroidism, caused by parathyroid carcinoma, was suspected. Ultrasound and its color Doppler modality can be useful for the precise diagnosis of parathyroid lesion and the differentiation of parathyroid adenoma from parathyroid carcinoma and other cervical pathologies. Their overall sensitivity, specificity and accuracy in the correct diagnosis of parathyroid adenoma have been 97%, 100% and 98.6%, respectively.

The color Doppler power image commonly shows a characteristic extrathyroid feeding vessel (typically, a branch of the lower thyroid artery), entering the parathyroid gland at one of the poles known as the single polar nourishing vessel, in parathyroid adenoma, and in parathyroid carcinoma it appears as a large mass that is densely attached to the surrounding soft tissues or to the thyroid gland, intense central and peripheral vascularization (23).

The radiological evaluation of the lytic lesions showed important characteristics: ovoid morphology, smooth edges with thinning of the cortex, regular edges without periosteal erosion or soft tissue alteration. The brown tumor is a benign tumor that, in its radiological appearance, is similar to an osteolytic metastatic tumor. The difference is that it does not invade adjacent tissues and does not induce changes around the periosteum (24).

Most brown tumors present as solitary lesions and in some rare cases as multiple lesions. The bones commonly affected are the pelvis, ribs, femurs, humerus and other long bones (25).

The location in the mandible and maxilla is unusual, 4.5%, although it is more frequent in the mandible than in the maxilla; it is even less common in both maxillary bones simultaneously (7).

Our patient had multiple bone lesions, as well as in the left maxilla, mandible, C5, C6, L3 and pedicle of L2, for which it was necessary to make differential diagnoses; even, a single lesion in the hard palate, of which, no described cases are found (Figure 6b). The clinical criteria for the suspicion of malignant parathyroid neoplasia according to Obara et al, are: age under 55 years; marked hypercalcemia and increase of paratohormone —more than 10 times the limit—; severe bone symptoms —fibrocystic osteotitis in 40% to 70% of cases— and renal symptoms —nephrocalcinosis, nephrolithiasis in 30% to 60% of cases—; recurrent laryngeal paralysis due to tumor invasion; palpable cervical inflammation —rare in benign disease (14).

Primary hyperparathyroidism is the most common cause of hypercalcemia and should be considered in anyone with an elevated serum calcium level. One of the relevant aspects of the brown tumor in this case is that it represented the primary expression of primary hyperparathyroidism, secondary to parathyroid carcinoma, which is infrequent, because in the last decades the incorporation of serum calcium in routine laboratory tests detects this disease in asymptomatic or minimally symptomatic stages (26).

However, due to the lack of accessibility to hospital centers and serial studies, the disease progresses to multiple tumors of diffuse location, including infrequent locations. Manifestations such as weakness, asthenia, and constipation were also evident and have been described in the literature in patients with the disease (4).

Due to the lesion in the left jaw and the common histological characteristics, a differential diagnosis was made with other entities that affect the facial mass, such as giant cell tumor, aneurysmal bone cyst and kerubism. Radiographically it must be differentiated from

odontogenic lesions, such as ameloblastoma, odontogenic keratocysts and complex odontomas (27).

Block resection of a primary parathyroid carcinoma is the initial treatment of choice. Metabolic complications related to hypercalcemia are associated with increased mortality. Even if metastasis or recurrence is detected, aggressive resection of metastatic parathyroid carcinoma is the most effective treatment to control hypercalcemia and improve survival (28).

The disease recurs in more than 50% of the patients. Surgical resection is the primary mode of therapy for recurrence, as well as palliation for the metabolic disorder caused by hyperparathyroidism. Reoperations are rarely curative, and chemotherapy and radiation treatments are ineffective (29).

Conclusions

The brown tumor represents the most advanced stage of the bone disease associated with hyperparathyroidism. It should be noted that brown tumors are non-neoplastic lesions, which do not have malignant potential, so they are considered restorative granulomas and their main differential diagnoses are metastasis, lymphoma, the giant cell tumor, the aneurysmal bone cyst, kerubism, ameloblastoma, odontogenic keratocysts and complex dentistry.

Among the main radiological features in hyperparathyroidism, subperiosteal resorption of the bone cortex in multiple locations, generalized or juxtaarticular osteopenia and brown tumors are observed. The most frequent sites of appearance of the latter are long bones, ribs, pelvis, maxilla, jaw, skull and even vertebrae. In this case they were identified in both the axial skeleton and the appendix.

The initial treatment of the brown tumor consists in the elimination of the cause and in the control of the metabolic disease, which in this case consisted in the resection of the parathyroid carcinoma; this favors the normalization of the hormonal levels and may be enough to stimulate the mineralization and the progressive disappearance of the lesion.

References

1. Xie C, Tsakok M, Taylor N, Partington K. Imaging of brown tumours: a pictorial review. Insights Imag. 2019;10(1). doi: 10.1186/s13244-019-0757-z.
2. Arias W, Ayala A, Pacheco F, Barzallo D. Tumor pardo multifocal como manifestación del hiperparatiroidismo primario por adenoma paratiroideo asociado a carcinoma papilar de tiroides. Rev Chil Cir. 2014;66(6):592-8. doi: 10.4067/s0718-40262014000600014
3. Madkhali T, Alhefdhi A, Chen H, Elfenbein D. Primary hyperparathyroidism. Turkish J Surg. 2016;32(1):58-66. doi: 10.5152/ucd.2015.3032.
4. Alfawareh M, Halawani M, Attia W, Almusrea K. Brown tumor of the cervical spines: A case report with literature review. Asian Spine J. 2015;9(1):110. doi: 10.4184/asj.2015.9.1.110.
5. Kwon J, Kim E, Lee H, Moon H, Kwak J. Neck ultrasonography as preoperative localization of primary hyperparathyroidism with an additional role of detecting thyroid malignancy. European J Radiol. 2013;82(1):e17-e21. 10.1016/j.ejrad.2012.08.003.
6. McDonald D, Parman L, Speights V. Primary hyperparathyroidism due to parathyroid adenoma. RadioGraphics. 2005;25(3):829-34. doi: 10.1148/rg.253045042.
7. Mora-Escudero I, Gato-Díez A, Blázquez-Cabrera J, Lozano-Setien E, García-de-la-Torre J. Tumores pardos pretibiales como manifestación inicial de un hiperparatiroidismo primario. Rev Clín Española. 2012;212(3):e15-e18. doi:10.1016/j.rce.2011.07.014.
8. Herrera A, Aranda P, Díaz J. Cáncer de paratiroides: revisión de literatura. Rev Española Enfermed Metaból Óseas. 2007;16(6):124-9. doi:10.1016/s1132-8460(07)73511-0.
9. Morán L, Moeinvaziri M, Fernández A, Sánchez R. Multiple brown tumors mistaken for bone metastases. Computed tomography imaging findings. Egyptian J Radiol Nucl Med. 2016;47(2):537-41. doi: 10.1016/j.ejrnm.2016.03.001
10. Rocha AL, Suazo LC, González PM, Lee ChK, Rossel DG. Hiperparatiroidismo primario y cáncer de paratiroides: Caso clínico. Rev Chil Cirugía. 2010;62(5). doi: 10.4067/s0718-40262010000500016.
11. Jakubowski J, Vélez I, McClure S. Brown tumor as a result of hyperparathyroidism in an end-stage renal disease patient. Case Reports Radiol. 2011;2011:1-3. doi: 10.1155/2011/415476.
12. Parra Ramírez P, Lecumberri Santamaría B, Álvarez Escolá C, Pallardo Sánchez L. Hiperparatiroidismo primario con osteítis fibrosa quística simulando una neoplasia ósea maligna. Endocrinol Nutric. 2013;60(2):96-8. doi: 10.1016/j.endonu.2012.02.012.
13. Ferraro V, Sgaramella LI, Di Meo G, et al. Current concepts in parathyroid carcinoma: a single Centre experience. BMC Endocr Disord. 2019;19(46). https://doi.org/10.1186/s12902-019-0368-1.
14. Fernandes J, Paiva C, Correia R, Polónia J, Moreira da Costa A. Parathyroid carcinoma: From a case report to a review of the literature. Inter J Surg Case Reports. 2018;42:214-7. doi: 10.1016/j.ijscr.2017.11.030.
15. Rosai JDR, Carcangiu ML, Frable WJ, Tallini G. Tumors of the thyroid and parathyroid glands. AFIP Atlas of Tumor Pathology. Silver Spring, Maryland: ARP Press; 2014.
16. Betea D, Bradwell A, Harvey T, Mead G, Schmidt-Gayk H, Ghaye B et al. Hormonal and biochemical normalization and tumor shrinkage induced by anti-parathyroid hormone immunotherapy in a patient with metastatic parathyroid carcinoma. J Clin Endocrinol Metabolism. 2004;89(7):3413-20. doi: 10.1210/jc.2003-031911
17. Erickson L, Mete O. Immunohistochemistry in diagnostic parathyroid pathology. Endocrine Pathol. 2018;29(2):113-29. doi: 10.1007/s12022-018-9527-6
18. Shi C, Guan H, Qi W, Ji J, Wu J, Yan F et al. Intrathyroidal parathyroid adenoma: Diagnostic pitfalls on fine-needle aspiration: Two case reports and literature review. Diagnostic Cytopathol. 2016;44(11):921-5. doi: 10.1002/dc.23528
19. Wong Y, Sharifah N, Tan G, Gill A, Ali S. Intrathyroidal oxyphilic parathyroid carcinoma: A potential diagnostic caveat in cytology? Diagnostic Cytopathol. 2016;44(8):688-92. doi: 10.1002/dc.23493.
20. Shane E. Parathyroid carcinoma. J Clin Endocrinol Metabolism. 2001;86(2):485-93. doi: 10.1210/jcem.86.2.7207
21. García J, Álvarez Moro F, Bassa P, Soler M, Llinares E, Riera E. Detección de tumores pardos por hiperparatiroidismo secundario mediante PET/TC con 11C-colina. Rev Española Med Nuclear Imagen Molecular. 2016;35(3):209-10. doi: 10.1016/j.remn.2015.07.009
22. Insogna K. Primary hyperparathyroidism. New Engl J Med. 2018;379(11):1050-9. doi: 10.1056/nejmcp1714213.
23. Piciucchi S, Barone D, Gavelli G, Dubini A, Oboldi D, Matteuci F. Primary hyperparathyroidism: imaging to pathology. J Clin Imag Sci. 2012;2:59. doi: 10.4103/2156-7514.102053.
24. Park S, Kong G, Kwon Y, Park J. Pathologic fracture of the femur in brown tumor induced in parathyroid carcinoma: A Case Report. Hip Pelvis. 2016;28(3):173. doi: 10.5371/hp.2016.28.3.173.
25. Park Y, Yoon T, Park K, Ko J. Subchondral bone restoration of supra-acetabular brown tumor secondary to parathyroid carcinoma: A Case Report. Hip Pelvis. 2018;30(2):120. doi: 10.5371/hp.2018.30.2.120.
26. Marcocci C, Cetani F. Primary hyperparathyroidism. New Eng J Med. 2011;365(25):2389-97. doi: 10.1056/nejmcp1106636.
27. Radulescu D, Chis B, Donca V, Münteanu V. Brown tumors of the femur and pelvis secondary to a parathyroid carcinoma: Report of one case. Rev Méd Chile. 2014;142(7):919-23. doi: 10.4067/s0034-98872014000700014.
28. Kim Y. Parathyroid carcinoma with lung metastasis in a thirteen-year-old girl. J Korean Surg Society. 2012;82(6):385. doi: 10.4174/jkss.2012.82.6.385.
29. Fortson J, Su R, Patel V, Lawrence G. Parathyroid carcinoma presenting with pathologic fracture: Case report and review of the literature. Head Neck. 2015;37(11):E139-E141. doi: 10.1002/hed.23965

Correspondence

Silvia Lissett Espinoza
Res. Loma Linda Norte, Bloque 180, Apart 2.
11101
Tegucigalpa, Honduras
s.lissespinoza@gmail.com

Received for evaluation: February 5, 2020
Accepted for publication: May 18, 2020