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Dr. Lefqih Imane
Department of Thoracic Surgery IBN SINA Hospital, Mohamed V University, Rabat, Morocco

Dr. Hachem Taleb Elemine
Department of Thoracic Surgery IBN SINA Hospital, Mohamed V University, Rabat, Morocco

Dr. Labiba Sbiki
Department of Thoracic Surgery IBN SINA Hospital, Mohamed V University, Rabat, Morocco

Pr. Mohamed Bouchikh
Department of Thoracic Surgery IBN SINA Hospital, Mohamed V University, Rabat, Morocco

Pr. Abdellah Achir
Department of Thoracic Surgery IBN SINA Hospital, Mohamed V University, Rabat, Morocco

Corresponding Author:
Dr. Lefqih Imane
Department of Thoracic Surgery IBN SINA Hospital, Mohamed V University, Rabat, Morocco

Mandibular brown tumor revealing an ectopic parathyroid adenoma

Dr. Lefqih Imane, Dr. Hachem Taleb Elemine, Dr. Labiba Sbiki, Pr. Mohamed Bouchikh and Pr. Abdellah Achir

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Abstract
The case report presents a 47-year-old female patient. She was suffering a painful clinical mandibular lesion. The histological diagnosis returned in favor of a brown tumor (osteitis fibrosa cystica). The patient did not present any signs of hypercalcemia. The biology report shows an hypercalcemia and an elevated parathyroid hormone (PTH) levels.
The Sestamibi (methoxyisobutylisonitrile) scintigraphy returned in favor of an ectopic pathological parathyroid tissue in the upper mediastinal zone. The excision of this tissue revealed a parathyroid adenoma, which normalized the metabolic state and regressed the mandibular lesion.
This case study highlights the importance of the dosage of parathyroid hormones and the calcemia in identifying the origin of osteitis fibrosa cystica.

Keywords: Brown tumor, mandibular tumor, hypercalcemia, parathyroid adenoma, cervicotomy

Introduction
Brown tumor, also known as osteitis fibrosa cystica, is the result of abnormal bone metabolism in patients with hyperparathyroidism (HPT) [1].
Primary hyperparathyroidism is characterized by the hypersecretion of parathyroid hormone, which is caused by adenomas in 80-85% of cases [2].
The case report a patient who presented a brown mandibular tumor caused by a parathyroid adenoma. The purpose of this case is to take into account the dosage of the calcemia and the parathyroid hormone as soon as this tumor is suspected.

Case Report
The patient is a 47 years old female with no previous history. She reported a painful sublingual lesion that she recounts dating back to three years ago (figure 1).
Her tumor biopsy came back in favor of a giant cell lesion evoking a brown tumor

Fig 1: The sublingual lesion

Moreover, the patient did not present any bone pain or episodes of renal colic. The clinical examination found a reddish-purple maxillo-gingival swelling of 2 cm in diameter in the mouth. The cervical examination did not show any palpable swelling or lymphadenopathy.
The calcemia was 113 mg/l (normal range: 85 to 102 mg/l), the parathyroid hormone (PTH) was 89.40 pg/ml (normal range: 10 to 55 pg/ml), the calciuria was 731.40 mg/24 h (normal range: 20-275 mg/24 h) and the renal function was normal.

On cervical ultrasound showed normal.
The sestamibi (methoxyisobutylisonitrile) scintigraphy showed an aspect in favor of pathological ectopic parathyroid tissue in the upper mediastinal zone. The cervico-thoracic computed tomography showed an hypodense nodule in the left infra-thyroid situating in favor of a parathyroid adenoma (figure 2). Bone densitometry showed a T score equal to -2.

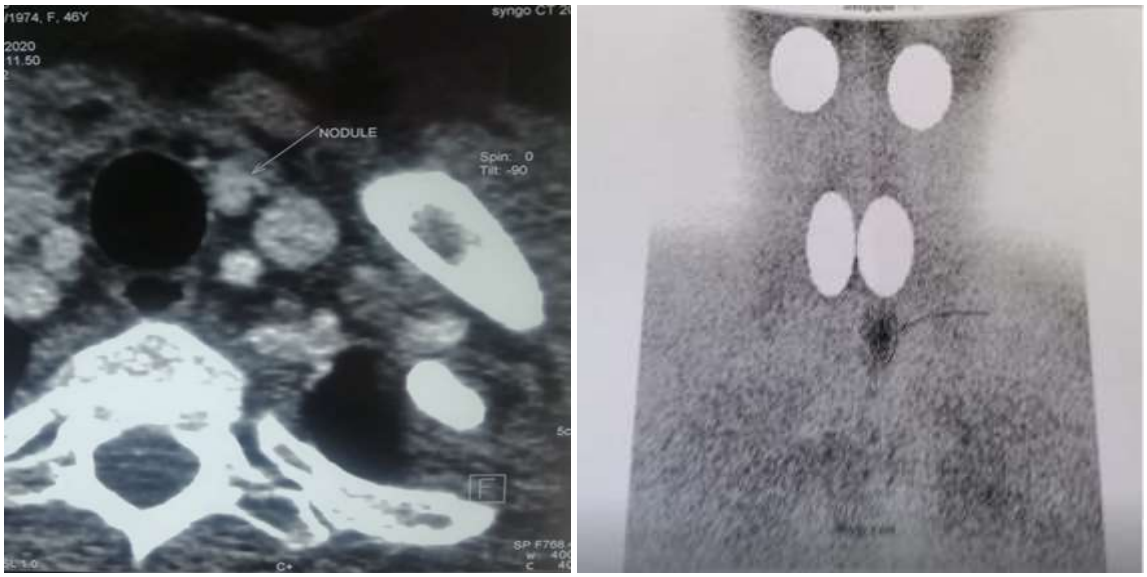

Fig 2: On the right : para-tracheal parathyroid adenoma ; ;on the left :The sestamibi scintigraphy showed an aspect in favor of pathological ectopic parathyroid in the upper mediastinal zone

This is shows a patient presenting with a brown tumor, located in the mandibule.
The brown tumor resulted by a primary hyperparathyroidism which is due to the left of laterotracheal parathyroid adenoma (figure 3).

The patient underwent an adenomectomy by cervicotomy The success of the surgery was confirmed by a falling of PTH level immediately up to 15.30 pg/ml and serum calcium level of 83.99 mg/l post operation.
The pathological examination of the resected specimen confirmed the presence of a parathyroid adenoma.

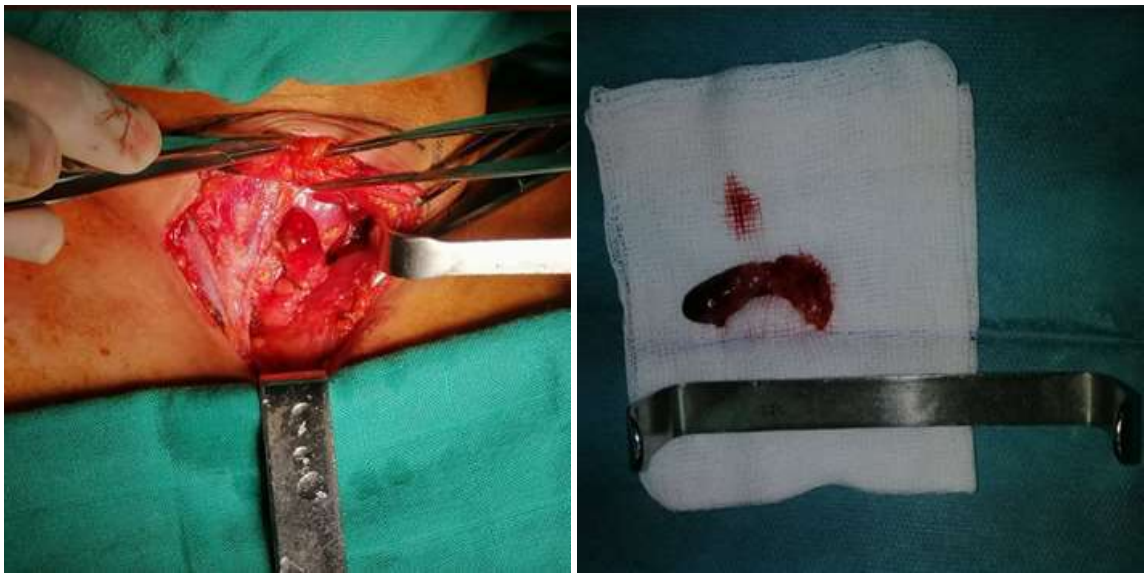

Fig 3: On the right: intraoperative view showing the location of the adenoma (by cervicotomy) on the left: parathyroid adenoma

Discussion
Brown tumor is an osteoclast hyperactivity which osteolytic process, rather than a neoplastic process. It is a lytic bone lesion. The origin of this term comes from the color of the lesion, which is caused by blood pigments and the existence of hemosiderin sedimentation in the macrophages [3].
Albright first described these tumors in 1934 at the level of the facial skeleton [4].

Brown tumors are osteolytic lesions, which in rare cases point to a patient that exhibits hyperparathyroidism. They normally occur in the terminal stage of primary or secondary hyperparathyroidism [5].
While the numbers show that brown tumors observed in the maxillar bone occurance rate is relatively low (4.5% of cases), it is important to note that hyperparathyroidism detection is majorly incidental (75 to 80% of cases)

following a blood test that shows an asymptomatic hypercalcemia [6, 4].

When diagnosing a brown tumor histologicaly, the classification of hyperparathyroidism is essential to offer the adequate treatment.

Primary hyperparathyroidism is characterized by severe hypercalcemia, an overproduction of parathyroid hormone, renal failure, hypercalciuria, recurrent nephrolithiasis, pathologic fractures as well as vertebral fractures. Fortunately, biochemical screening has become a standard practice, which leads to more people being diagnosed asymptomatically [7].

The brown tumor primary cause is parathyroid adenoma (80% of cases), then hyperplasia and exceptionally parathyroid carcinoma [6].

The diagnosis of brown tumor can be challenging when faced with a single maxillary location. This is due to the similarities shared with giant cell tumor and giant cell reparative granuloma in their histological appearance [8, 9].

The treatment depends on the bone lesion. It can range from a conservative approach with parathyroidectomy projecting the regression of the lesion - such as our case, or parathyroidectomy associated with bone surgical excision with reconstructive, for the case of a large symptomatic lesion [10]. If the lesion does not regress after one or two years, the excision of the tumor becomes mandatory [11].

Conclusion

Brown tumors resulting from primary hyperparathyroidism are very rare. The analysis of serum calcium and parathyroid hormone should be carried out systematically in the event of multiple osteolytic lesions. Then immediately intervene for conservative with parathyroidectomy.

Conflict of Interest

Not available

Financial Support

Not available

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