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# Brown spinal tumour secondary to primary hyperparathyroidism, a primary entity that we should not forget. Case report

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## ABSTRACT

**Introduction:** A thorough study of lytic lesions can guide us toward a more accurate aetiology in spinal column lesions. The use of imaging techniques and blood chemistry studies is useful when considering brown tumours as part of the differential diagnosis.

**Clinical Case:** A 43-year-old woman with multiple comorbidities presented with lumbar pain. A lytic lesion in the body of S2 was documented, and thoracoabdominal lesions were initially ruled out. However, during her stay, a malignant thyroid lesion was documented.

**Discussion:** Hyperparathyroidism, whether primary (adenomas, hyperplasia, or carcinoma) or secondary (vitamin D deficiency or chronic kidney disease), causes skeletal alterations in approximately 16% of cases. Among its manifestations are parathyroid tumours, which primarily affect the pelvis, rib arches, facial bones, and long bones, with less frequency in the spine. Clinically, these present progressive pain and neurological deficits. Diagnosis requires paraclinical tests and imaging studies. Treatment should address the underlying cause of hyperparathyroidism, with options ranging from medical therapies to surgical interventions.

**Conclusion:** Thyroid carcinoma as a manifestation of a brown tumour at the sacral level is a rare presentation. We consider it pertinent to perform extension studies when evaluating patients with multiple comorbidities and a single lytic lesion.

## INTRODUCTION

Brown tumor (BT) refers to the presence of one or multiple bone lesions, usually with a lytic appearance. This is generally due to metabolic conditions caused by alterations in parathyroid hormone

## Keywords

hyperparathyroidism,  
osteitis fibrosa cystica,  
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sacrum



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(PTH) levels, with primary hyperparathyroidism (PHPT) and secondary hyperparathyroidism (SHPT) being the only two causes described so far (1). It is considered that most cases of BT are primarily caused by PHPT with an incidence of up to 13%, compared to SHPT cases which account for less than 5% of the incidence (2,3).

Elevated serum PTH levels are considered to increase osteoclast activity, causing increased bone resorption and decreased mineralization, resulting in changes in trabecular bone, fibrous cystic osteitis, and hemorrhages with hemosiderin deposits, which gives it a brownish appearance; hence its name brown tumor (3, 4).

The following presents the case of a woman with a lytic bone lesion in the sacrum (S2), which was initially considered malignant in etiology. However, given the paraclinical and imaging findings, the possibility of a BT secondary to renal failure in hemodialysis was considered. Nevertheless, upon re-evaluating the case and performing extension studies, it was evident that the patient had a parathyroid lesion, later identified as a thyroid carcinoma. Therefore, the final diagnosis was BT due to PHPT.

#### CLINICAL CASE

A 43-year-old female with a history of tertiary hyperparathyroidism, stage V chronic kidney disease on hemodialysis, hypertension, and chronic heart failure presented to the emergency department with six days of severe lumbosacral axial pain, without signs of radicular compression. Physical examination revealed normal strength (5/5) in all four extremities, preserved sensation, muscular atrophy, hypotonia, and an antalgic gait.

A simple magnetic resonance imaging of the sacrococcygeal spine revealed a multilocular cystic lesion on the left lateral aspect at the level of S2. Additionally, PTH levels were found to be 2062.9 pg/ml, serum calcium was 9.5 mg/dl, and serum phosphorus was 7.2 mg/dl. Due to these findings, and considering the patient's history and symptoms, the initial possibility was a tertiary hyperparathyroidism due to mediastinal parathyroid adenomas and chronic kidney disease. Therefore, treatment was initiated with Sevelamer 800 g orally every 8 hours and vitamin D 5600 IU orally daily; parathyroidectomy was not possible due to an ongoing infectious process. Furthermore, due to the absence of spinal cord compression and pathological

fractures, surgical management of the spine was not required, and multimodal analgesic management was the alternative implemented.

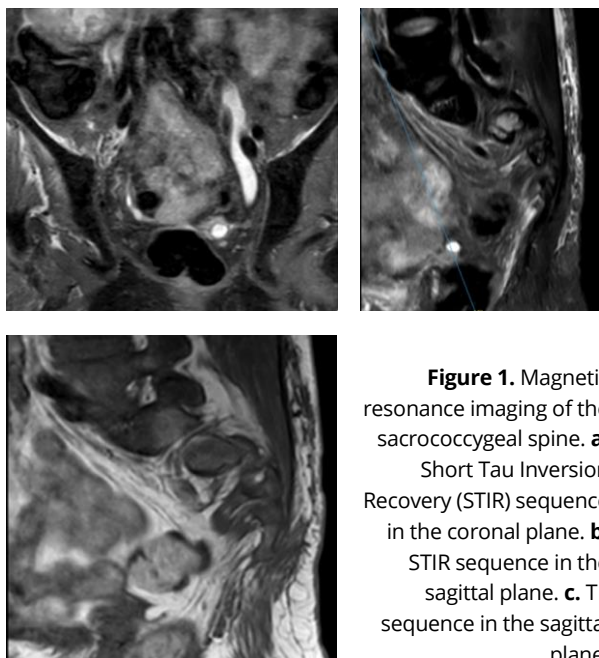
#### DISCUSSION

Hyperparathyroidism, whether of primary origin related to single or multiple adenomas, hyperplasia or carcinoma; or of secondary origin due to vitamin D deficiency or chronic kidney disease, causes skeletal system alterations in approximately 16% of cases (5). Among its manifestations, BT stands out, with an incidence of less than 5% in PHPT and approximately 15% in SHPT (5, 6), mainly affecting the pelvis, costal arches, facial bones and long bones; while a lower prevalence is observed in the spine, mainly affecting the thoracic and lumbar regions, and only four cases have been reported at the sacral level (5, 7, 8, 9).

From a clinical standpoint, vertebral BT commonly presents with progressive pain and neurological deficits, which can manifest as alterations in strength, sensation, and even impaired sphincter control; depending on the location of the lesion and the presence of spinal cord compression and pathological fractures. Likewise, it is necessary to perform paraclinical tests, such as measuring serum hormone levels, calcium and phosphorus, in addition to imaging, with the aim of providing an accurate diagnosis and distinguishing vertebral BT from its main mimics, which include giant cell tumor, giant cell reparative granuloma, multiple myeloma, and metastatic lesions (6). In our case, it correlates with the clinical presentation described in the literature when there is involvement of the S2 nerve roots.

Regarding the paraclinical results, it is common to observe elevated calcium and PTH levels, associated with low phosphorus levels (5). On the other hand, among the useful diagnostic images, there is the X-ray, where one or more well-defined and expansive lesions are usually evident; while in the computed tomography, bone erosion can be evidenced. For its part, magnetic resonance imaging allows visualizing hypointense lesions in the T1 sequence and hypo or hyperintense in the T2 sequence, with or without signs of spinal cord compression (6). Finally, bone scintigraphy shows diffuse systemic hypermetabolism. Although these examinations have advantages, their reliability is not absolute, so it is recommended to take a biopsy of the lesion, where multinucleated osteoclasts, spindle-shaped

stromal cells and a fibrous matrix can be observed (10). Correlating it with the presented case, changes in biochemical parameters were evidenced associated with extremely high serum hormonal levels, which does not agree with the expected results, but, when analyzing the case, it is determined that these findings are given by the history of chronic renal failure with hemodialysis requirement. On the other hand, the diagnostic images coincide with the descriptions found in the literature (Figure 1).



**Figure 1.** Magnetic resonance imaging of the sacrococcygeal spine. **a.** Short Tau Inversion Recovery (STIR) sequence in the coronal plane. **b.** STIR sequence in the sagittal plane. **c.** T1 sequence in the sagittal plane.

Regarding BT treatment, management should be etiological, addressing the underlying cause of hyperparathyroidism. In primary cases, phosphate binders, vitamin D, and calcimimetics are used, while in secondary cases, parathyroid surgery is employed (9, 11). This approach demonstrates positive outcomes, with normalization of calcium and PTH levels, as well as improvement in bone density and healing of pathological fractures (10). Specifically in spinal lesions, the surgical option may include tumor resection or decompression, depending on the size, location, and degree of associated neurological deficit (5).

## CONCLUSION

In conclusion, primary TP should be considered in pathologies related to PTH homeostasis, such as HPTP and HPTS. The importance of distinguishing its origin lies in the fact that treatment varies, ranging from pharmacological management to surgical intervention of the primary tumor and TP. It is

essential to conduct further studies on this entity to expand the bibliographic base and, ultimately, standardize its management.

## ABBREVIATIONS

BT	Brown tumor
PHPT	Primary hyperparathyroidism
PTH	Parathyroid hormone
SHPT	Secondary hyperparathyroidism
STIR	Short Tau Inversion Recovery

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