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Case Report

Parathyroid Carcinoma Presenting with Severe Primary Hyperparathyroidism and Osteolytic Skeletal Lesions: A Case Report

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ABSTRACT

Parathyroid carcinoma is an exceptionally rare malignancy typically presenting with severe primary hyperparathyroidism. Diagnosis is often difficult and relies mainly on histopathological evaluation. Surgery remains the cornerstone of treatment.

We report the case of a 52-year-old man with chronic right hip pain and anxiety disorder, who presented with progressive general deterioration. Clinical examination revealed a firm, painless, anterior left-lateral basocervical mass measuring approximately 6 cm. Laboratory investigations showed marked hypercalcemia (144 mg/L) and severe hyperparathyroidism with a parathyroid hormone (PTH) level of 1,737 pg/mL (39 times normal). Imaging studies demonstrated a large heterogeneous mass adjacent to the lower pole of the left thyroid lobe, associated with multiple osteolytic skeletal lesions.

A giant parathyroid adenoma was suspected and surgical excision was performed after preoperative correction of hypercalcemia. The mass extended into the retrosternal space without invasion of adjacent structures. Intraoperative histology suggested adenoma or hyperplasia. Postoperatively, calcium and PTH levels normalized. However, definitive histopathological analysis confirmed parathyroid carcinoma, showing capsular invasion and vascular emboli.

At the six-month follow-up, the patient showed significant improvement in bone pain, with no evidence of recurrence and normal calcium levels. This case highlights the diagnostic difficulty of parathyroid carcinoma and underscores the need for long-term surveillance due to the risks of recurrence and metastasis.

Keywords: Parathyroid; Neoplasms; Carcinoma; Hyperparathyroidism; Primary; Hypercalcemia; Parathyroidectomy

Introduction

Parathyroid carcinoma (PC) is an exceptionally rare endocrine malignancy, accounting for fewer than 1% of all cases of primary hyperparathyroidism (pHPT)[1]. The condition was first documented in 1904 by the Swiss surgeon de Quervain[2] in a patient presenting with a non-functioning parathyroid lesion. Since this initial description, the global literature has progressively expanded our understanding of this uncommon neoplasm.

In this article, we describe a case of parathyroid carcinoma in a patient with persistent pHPT, reported in accordance with the SCARE criteria[3] and provide a concise review of the relevant literature.

Presentation of Case

A 52-year-old man with a history of right hip pain and anxiety disorder for the past three years presented with progressive deterioration of his general condition. Physical examination revealed a firm, painless, anterior left-lateral Baso cervical mass, mobile on swallowing, measuring approximately 6 cm in its greatest dimension, with a non-palpable inferior border **(Figure 1)**. No cervical lymphadenopathy was detected and vocal cord mobility was preserved.

Laboratory investigations demonstrated marked hypercalcemia (144 mg/L) and severe hyperparathyroidism, with an extremely elevated parathyroid hormone (PTH) level of 1,737 pg/Ml-approximately 39 times the upper limit of normal. Standard skeletal radiographs revealed diffuse bone demineralization, predominantly affecting the iliac bones, femoral necks and the right femoral diaphysis.

Cervical ultrasonography identified a heterogeneous, hypoechoic and hypervascular mass located adjacent to the lower pole of the left thyroid lobe, measuring 62 × 53 × 42 mm. Cervico-thoracic computed tomography (CT) confirmed a well-defined, solid-cystic oval lesion beneath the left thyroid lobe, with smooth margins, measuring 54 × 45 mm and extending 63 mm in height. The mass showed close anatomical relations with the left common carotid artery, situated anterior to it. Additionally, multiple costal and vertebral osteolytic lesions were noted **(Figure 2)**. Abdominopelvic CT revealed multiple osteolytic lesions involving the spine and pelvis.

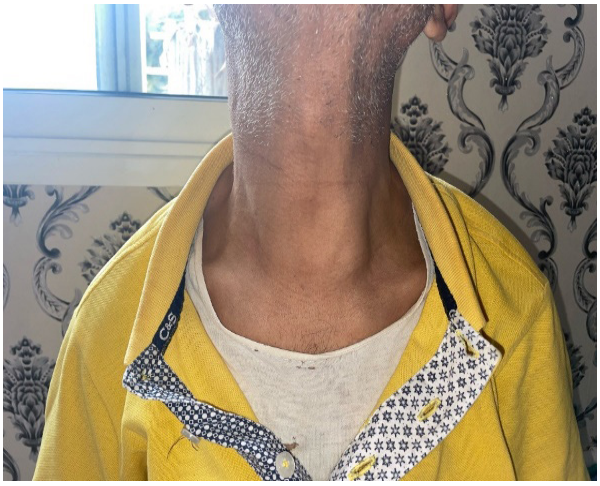

Figure 1: Physical examination revealed anterior left-lateral Baso cervical mass, mobile on swallowing, measuring approximately 6 cm in its greatest dimension, with a non-palpable inferior border.

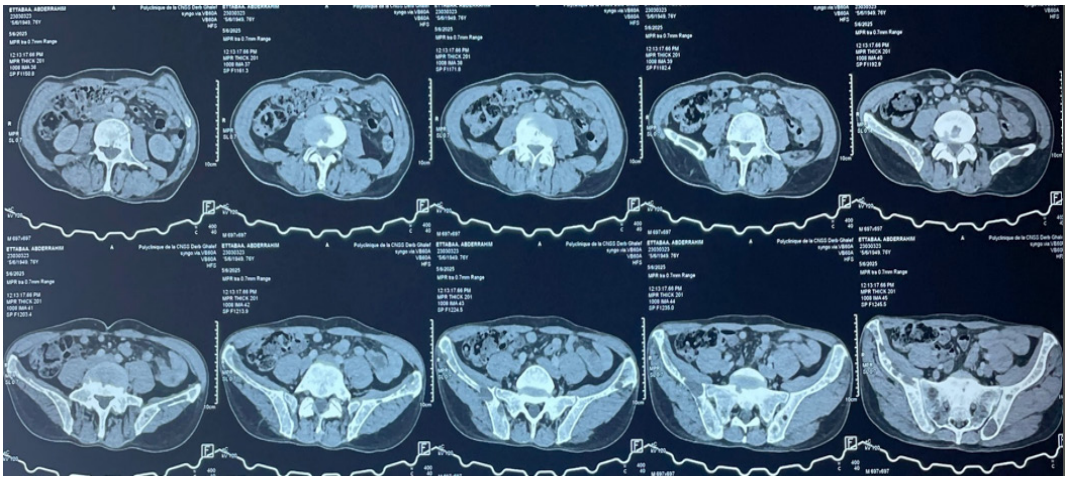

Figure 2: Axial abdominopelvic CT scan showing osteolytic bone lesions involving the spine and the pelvis

Based on these findings, a parathyroid adenoma was suspected and surgical excision was planned. Preoperative management included intravenous rehydration with isotonic saline and administration of bisphosphonates to correct the hypercalcemia.

Intraoperatively, the mass was found to be plunging into the retrosternal space, with no evidence of infiltration or continuity with the lower pole of the left thyroid lobe, which appeared macroscopically normal. A left inferior parathyroidectomy was performed and the excised specimen was sent for intraoperative histopathological examination, which suggested either an adenoma or parathyroid hyperplasia without features of malignancy.

The immediate postoperative course was uneventful. Biochemical assays on postoperative day 1 demonstrated normalization of serum calcium (103 mmol/L) and PTH levels (44 pg/mL).

Definitive histopathological analysis established the diagnosis of parathyroid carcinoma, characterized by an encapsulated malignant proliferation composed of parathyroid cells arranged in diffuse and nodular patterns, separated by fibrous septa. The tumor cells were monomorphic, with abundant cytoplasm and mildly atypical round nuclei. Mitotic activity was moderate (three mitoses per ten high-power fields). Areas of capsular invasion and vascular tumor emboli were also identified.

At the six-month follow-up, the patient showed significant

improvement in bone pain, with no clinical or ultrasonographic evidence of local recurrence. Laboratory findings confirmed normalization of serum calcium levels.

Discussion

Parathyroid carcinoma (PC) is an extremely rare endocrine malignancy, representing less than 0.005% of all cancers and accounting for approximately 0.5-4% of primary hyperparathyroidism cases, with significant geographical variation reaching up to 5% in Japan[4,5]. In Western countries, PC usually accounts for less than 1% of pHPT cases[6]. Its incidence is estimated at 4-6 cases per 10 million inhabitants per year and a large American series reported 286 cases over 10 years[7,8]. Because of its rarity and lack of specific clinical and biological signs, PC is frequently misdiagnosed as benign primary hyperparathyroidism and is often diagnosed only postoperatively[4,7,9,1].

The etiology of PC remains poorly understood, although several environmental and genetic factors have been implicated[10,7,9]. Neck irradiation, particularly at a young age, increases the risk of parathyroid neoplasia[5,6]. Chromosomal abnormalities-including 1p, 4q, 13q losses and 1q, 9q, 16p, Xq gains-have been reported[11] and cyclin D1 overexpression is found in most tumors[12]. Parathyroid carcinoma also shows a strong association with hyperparathyroidism–jaw tumor syndrome[13]. Additional genetic abnormalities, such as RB, p53, BRCA2 and PRAD1 mutations, have been described[13].

Most PCs are functioning tumors causing severe hypercalcemia, presenting with fatigue, weakness, weight loss, anorexia, psychiatric symptoms, gastrointestinal complaints, nephrolithiasis and bone lesions including brown tumors[10,14,15]. Renal and skeletal involvement is common at presentation[16]. Dysphonia and dysphagia, resulting from recurrent laryngeal nerve invasion, are highly suggestive of malignancy[10]. Marked hypercalcemia-often above 3.5 mmol/L-is frequently observed[11,17]. Non-functioning carcinomas are extremely rare and typically present with advanced local disease[7,15,18].

Imaging plays a key role in the evaluation of PC. Cervical ultrasound may show lobulated hypoechoic lesions with irregular margins, intra-lesional calcifications or infiltration of adjacent tissues-features suggestive of malignancy[10,15]. Negative predictive features include a thick capsule, ovoid shape or absence of intratumoral vascularity[10]. Ultrasound sensitivity ranges from 50% to 90%[15]. Tc-99m sestamibi scintigraphy is useful for localization but cannot distinguish adenoma from carcinoma; however, it may detect lymph-node or distant metastases[15,19]. CT and MRI offer better visualization of soft tissue invasion and nodal involvement[14,20]. FDG-PET may show uptake in brown tumors, which can mimic metastasis[21]. Fine-needle aspiration cytology is not recommended due to false negatives and risk of capsular rupture[22,23].

Intraoperatively, PC typically appears as a firm, lobulated mass with a dense gray-white fibrous capsule adherent to surrounding tissues, making dissection difficult[17,24]. Tumors are usually large (>3 cm) and may involve adjacent structures[25]. Histopathological diagnosis is difficult. Classic criteria include trabecular architecture, fibrous bands, mitotic activity and capsular or vascular invasion[7,1,26], but these features are not specific and may also occur in benign lesions[27].

Surgery is the mainstay treatment for PC. Recommended management includes en bloc resection of the tumor with ipsilateral thyroid lobectomy and excision of involved lymph nodes[14,15,28]. Complete excision provides the best chance of cure, while incomplete resection is associated with recurrence[7,14,15,24]. Avoiding capsular rupture is essential to prevent tumor seeding[10]. Lateral lymph-node dissection is recommended only when nodal metastases are present[28,29]. Although PC is traditionally considered radioresistant, radiotherapy may improve local control in selected cases[17,20]. Chemotherapy has not shown proven benefit[7,10].

Recurrence is frequent, occurring in 25% to 60% of cases within the first 2-5 years[7,30]. Late recurrences, sometimes beyond 20 years, have been reported, requiring prolonged follow-up[7]. Recurrence often presents with rising serum calcium and PTH levels and may involve local, regional or distant metastases[31]. Follow-up includes physical examination and serial monitoring of calcium and PTH[13]. Management of hypercalcemia may require loop diuretics, dialysis or bisphosphonates[10,14].

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