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Case Report

Brown Tumour in a Patient with Tertiary Hyperparathyroidism: A Case Report

Malavika M*, Anjali Sethi and Suresh Pillai

Department of ENT and Head and Neck Surgery, Kasturba Medical College, Manipal Academy of Higher Education, Manipal, Karnataka 576104, India

***Corresponding Author:** Malavika M, Department of ENT and Head and Neck Surgery, Kasturba Medical College, Manipal Academy of Higher Education, Manipal, Karnataka 576104, India.

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Abstract

Introduction: Brown tumour is a rare benign bone lesion associated with poorly controlled hyperparathyroidism, most often affecting the jaw, pelvis, or long bones, and presenting as swelling, pain, or lytic bone lesions.

Case Report: A 48-year-old male with chronic renal insufficiency and tertiary hyperparathyroidism developed facial swelling and jaw thickening. Imaging and labs confirmed brown tumours and parathyroid adenomas. Subtotal parathyroidectomy decreased PTH levels, and hypocalcemia was managed post-operatively.

Conclusion: Brown tumours signal advanced hyperparathyroidism. Diagnosis requires clinical, biochemical, and radiological correlation; parathyroidectomy typically leads to regression.

Keywords: Brown Tumor; Parathyroid; Tertiary Hyperparathyroidism; Hypocalcemia

Abbreviations

PTH: Parathyroid Hormone; CKD: Chronic Kidney Disease; THPT: Tertiary Hyperparathyroidism

Introduction

Brown tumour can present as a solitary lytic or multiple lytic bone lesions and is considered a sign of poorly controlled hyperparathyroidism [1]. A brown tumour may occur in any bone but most commonly affects long bones, facial bones, mandible, pelvis, and ribs [2]. Lesions of the jaw can present as facial swelling, difficulty in chewing, talking, swallowing and breathing [8]. It has a low prevalence of 0.1% with a feminine predominance. Its incidence in patients with chronic renal disease or end-stage renal disease is of 13% as opposed to 1.5% in case of primary hyperparathyroidism [3]. On clinical examination and using only routine panoramic radiography, the lesions may resemble osteosarcoma, bone metastases of a carcinoma, multiple myeloma, Langerhans cell histiocytosis, Paget's disease and osteomyelitis. Histologically and radio graphically, it is very similar to the other giant cell lesions such as true giant cell tumour, reparative giant cell granuloma, cherubism and aneurysmal bone cyst [4]. It is differentiated from other giant-cell tumours by the presence of hyperparathyroidism. Treatment of brown tumours must be initially directed towards correcting the hyperparathyroidism. Tumour regression may occur with resolution of the hyperparathyroidism. Persistent or large destructive tumours are treated with resection or curettage [5].

Tertiary hyperparathyroidism (THPT), arising from prolonged secondary disease in chronic kidney disease (CKD), features autonomous PTH secretion from hyperplastic glands refractory to feedback. We report a case of multifocal brown tumor of the maxilla and mandible with resolution after subtotal parathyroidectomy.

Case Presentation

A 48 year old male, presented with with 2 months of progressive left-sided facial swelling, bilateral jaw thickening, and difficulty chewing. His medical history included chronic renal insufficiency, hypertension, anaemia, tertiary hyperparathyroidism.

Examination revealed diffuse bilateral maxillary and hard palate expansion with tenderness but no ulceration or discharge. Oral intake was impaired.

Contrast-enhanced computed tomography (CECT) of the head and neck demonstrated symmetrical maxillomandibular expansion with serpiginous hyperdensities, cortical resorption, and multiple calvarial lytic lesions ("salt-and-pepper" skull). Additional findings included rib, clavicular, and upper limb osteolytic/sclerotic changes consistent with severe renal osteodystrophy (Figure a).

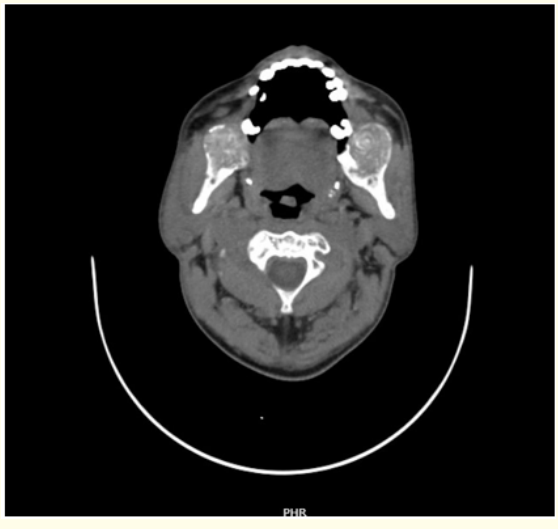

Figure a: CT image showing serpiginous hyperdensities replacing the trabeculae of B/L mandibles.

Investigations regarding the status of parathyroid glands were initiated. Serum chemistry revealed an elevated parathyroid hormone (PTH) level of 2907pg/ml (normal range, 15-65 pg/ml), serum calcium 9.0 mg/dl (normal range, 8.6-10 mg/dl), phosphorus 6.6 mg/dl (normal range, 2.5-4.5 mg/dl).

Ultrasonography of neck revealed a normal sized thyroid gland and well-defined isoechoic lesions posterior to both lobes of thyroid, 2 on the right side and 1 on the left side, suggestive of right superior and inferior parathyroid adenomas and left inferior parathyroid adenoma. A whole body Tc-99m sestamibi scan showed features suggestive of bilateral superior and left inferior parathyroid adenomas with largest being right inferior, measuring 1.7x2.2x1.7cm. Based on the medical history, clinical manifestations, and laboratory tests, the final diagnosis was brown tumour secondary to tertiary hyperparathyroidism was made.

The patient underwent subtotal parathyroidectomy under general anesthesia. Intraoperative PTH monitoring dropped from 2480 pg/mL (pre-excision) to 724 pg/mL post-removal of right superior/inferior and left inferior glands (right superior spared). Histopathology confirmed chief cell hyperplasia without atypia.

Postoperatively, serial monitoring of PTH, serum calcium, serum magnesium and serum phosphorous were done. PTH was stable with an average value of 700 pg/ml. There was a significant drop in serum calcium level, with the serial values being 5.8, 6.4, 7.1, 5.9 on POD 1, 2, 3 and 4 and he developed symptoms of hypocalcemia, pointing towards the development of hungry bone syndrome. Treatment was started with iv calcium gluconate along with oral calcium supplementation. Serum calcium showed a stable trend after POD5 on oral calcium supplementation. The values of serum magnesium and phosphorous were within normal limits (Figure b).

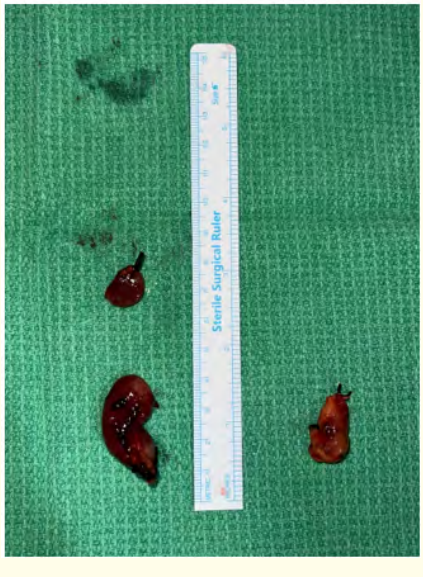

Figure b: Resected specimen of parathyroid glands.

Discharged on POD10 with oral calcium/vitamin D; 1-month follow-up showed PTH 650 pg/mL, normalised jaw function, and partial lesion regression on follow-up imaging.

Discussion

THPT is characterised by increased secretion of parathyroid hormone, which develops following prolonged secondary hyperparathyroidism due to CKD, renal transplantation, and gastrointestinal malabsorption. The parathyroid glands undergo hyperplasia and autonomously secrete parathyroid hormone, which is not suppressed by feedback control. THPT manifests as bone pain, decreased bone mineral density, osteitis fibrosa cystica with pathological fractures, pruritus, muscular weakness, nephrolithiasis, anorexia, nausea, and weight loss, which result from increased PTH levels and/or hypercalcemia [6].

Brown tumours are non-neoplastic lesions resulting from abnormal bone metabolism, arising from a group of pathological disorders (primary, secondary, or tertiary HPT) that result in the increased secretion of PTH, which causes increased osteoclastic bone resorption, primarily in cortical bone. Their histopathology is characterised by masses of soft tissue composed of giant cells in a fibrovascular stroma, cyst-like spaces lined by connective tissue, and foci of haemorrhage. Their association with hyperparathyroidism differentiates brown tumours from other giant cell lesions [7]. Brown tumours most commonly involve the ribs, clavicles, pelvic girdle, and mandible. Lesions of the jaw can present as facial swelling, difficulty in chewing, talking, swallowing and breathing [8].

Surgical removal of parathyroid glands in patients with tertiary hyperparathyroidism has been shown to normalise the levels of calcium, parathyroid hormone and increase the bone mineral density [9].

Normalisation of PTH levels will often cause the brown tumours to regress or sometimes even resolve spontaneously [10]. De Oliveira *et al*. described a case of a large brown tumour that regressed after surgical removal of a parathyroid adenoma, which measured 8.1 cm × 6 cm × 5.1 cm, which invaded the orbital cavity on the left side and a large part of the nasal cavity [11]. Nabi *et al*. also reported spontaneous regression of brown tumour after a parathyroidectomy [12]. Surgical parathyroidectomy remains the definitive therapy for THPT, restoring calcium and PTH balance and preventing further skeletal complications. Following surgery, normalisation of PTH levels frequently leads to regression or spontaneous resolution of brown tumors within 6-10 months in nearly 90% of cases. Conservative management includes calcimimetics (cinacalcet), phosphate binders, and vitamin D analogues for those unfit for surgery. Persistent or functionally compromising lesions may require local curettage or reconstructive surgery [13]. In our case, the patient underwent subtotal removal of the parathyroid gland and the PTH values normalised in 2 months following surgery.

Conclusion

Tertiary hyperparathyroidism arises when prolonged secondary hyperparathyroidism leads to autonomous PTH secretion, resulting in hypercalcemia and complications like brown tumors from excessive bone resorption. Parathyroidectomy offers definitive relief, normalizing PTH and calcium levels, improving bone density, and prompting brown tumor regression in nearly 90% of cases within 6-10 months. In our patient, subtotal parathyroidectomy achieved PTH normalization in just 2 months, effectively halting disease progression. While medical therapies such as cinacalcet provide options for those unfit for surgery, timely parathyroidectomy remains essential to prevent lasting skeletal damage and enhance patient well-being.

Statements

Informed consent of the patient obtained.

Conflict of Interest

The authors have no conflicts of interest to declare.

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Author Contributions

- Malavika M: Drafting, revision of manuscript, agreement to be accountable for all aspects of the work, corresponding author.
- Anjali Sethi: Initial drafting; agreement to be accountable for all aspects of the work.

- Suresh Pillai: Critical revision of the manuscript for important intellectual content, final approval of the version to be published, agreement to be accountable for all aspects of the work.
- All authors read and approved the final version of the manuscript.

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