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# **Oral Oncology**

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## Brown tumour of mandible in association with tertiary hyperparathyroidism: A rare case report



Hyperparathyroidism (HPT) is a disease characterised by the parathyroid glands secreting excessive amounts of parathyroid hormone because of a defect in the secretion mechanism of parathyroid hormone. Because of this, the production of parathyroid hormone and primary, secondary and tertiary forms of HPT can be seen [1].

The tertiary form, first described in 1963, is the rarest form of HPT. In patients with long-standing secondary HPT, it enhances the autonomic functioning of the parathyroid with hypersecretion of PTH because of renal failure<sup>2</sup>.

The brown tumour is named after its brown appearance. The colour is caused by increased osteoclastic activity and penetration of hemosiderin and fibroblastic tissue into the spaces formed in the bone matrix. This increased osteoclastic activity causes bone enlargement well above the normal contours of the bone. As a result of this pathological bone growth, fibrovascular tissue is seen in the osteoclastic lesion, which should be healthy bone [2].

A 47-year-old male patient was admitted with complaints of swelling and paresthesia on the left side of his mandible. In the anamnesis taken from the patient, we learned he was on dialysis because of chronic kidney failure and had swelling in his throat.

The tumour's size was reduced by administering triamcinolone acetonide 1 ml/4 mg and lidocaine 20 mg/ml to the tumour once a week for 6 weeks. The tumour size decreased to 25–30 mm after steroid treatment.

We reached and removed the tumour by lifting the full-thickness flap under local anaesthesia, preserving the surrounding healthy tissues. Mental nerve continuity was not impaired during surgery (Fig. 1). In the follow-ups after the surgical procedure, the patient did not experience numbness in the lip, his morbidity was reduced and he could use his teeth more comfortably.

The patient came for 1-year follow-up after the surgical procedure. We found no intraoral or extraoral pathology. According to the conebeam computed tomography (CBCT) results, the tumour did not recur, and the jawbone and soft tissue appeared healthy.

The project was approved by an institutional ethics committee and this is reported in the manuscript. For human subjects, the investigation was conducted in accordance with the Declaration of Helsinki of 1975. A statement to this effect is included in the manuscript.

According to the tomography results, a unilocular lesion with a 55–40 mm diameter was detected between the apex of teeth 32–35 on the left side of the mandible. When the patient's hormone levels were examined, the serum parathormone level was 965 pmol/L (normal value is 15 pmol/L –65 pmol/L), serum calcium level was 10 mg/dL (normal value is 8 mg/dL –10 mg/dL), phosphate level was 3.2 mg/dL (normal value is 2.5 mg/dL –4.5 mg/dL) and the eGFR value was 8 ml/min (normal value 90 ml/min). According to the biopsy and hormone values, the patient was diagnosed with a brown tumour because of tertiary HPT.

This article describes the therapeutic management of a brown tumour with chronic renal failure and tertiary HPT. When the cases in the literature are examined, the brown tumours associated with tertiary HPT are extremely rare. Therefore, the case that is the subject of our article is extremely rare [3].

HPT is classified according to the cause of the underlying hypersecretion into primary, secondary and tertiary types. Primary HPT is a common endocrine disorder. The most common cause of primary HPT is a single-gland adenoma, representing approximately 90% of cases [3,4]. In most cases, primary HPT is diagnosed incidentally by hypercalcemia, and patients are often asymptomatic [1,2]. In rare cases, patients with primary HPT may present with symptoms, including muscle weakness and kidney osteoporosis [1,3]. Calcium deficiency and phosphate excess are observed in the blood values of secondary HPT patients, unlike primary HPT [1]. A long-term chronic presence of secondary HPT is a factor in developing tertiary HPT. Parathyroid glands continue to secrete PTH, even if the functions of kidney transplant patients improve, and the secreted parathyroid hormone prepares tertiary HPT. Tertiary HPT affects less than 10% of kidney transplant recipients with a history of secondary HPT. In our case, the patient had chronic renal failure and parathyroid adenoma. Although the patient had high parathormone, the calcium phosphate and magnesium values were normal.

There is a connection between the high level of parathormone in the blood and the formation of a brown tumour. In our case, the PTH level was 965 pmol/L [1,2].

Although the surgical excision of the parathyroid gland causes the regression of the brown tumour and the formation of healthy bone, the remodelling of the bone changes inversely with the increasing age of the patient [2] When no regression is observed in the brown tumour, surgical excision of the parathyroid gland is considered [1]. Some authors advocate surgical treatment for brown tumours, because the tumours appear to continue growing after parathyroid plands were not removed, and the tumour was removed after steroid injections.

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The data that support the findings of this study are available from the corresponding author upon reasonable request.

#### **Declaration of Competing Interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Fig. 1. Mental nerve continuity was not impaired during surgery.

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