Brown Tumour Of Hyperparathyroidism, A Benign Latency In The Jaws (A Review of Literature)

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Abstract: Background: To inculcate awareness about the importance of thorough screening of the patients presenting with giant cell lesions in the jaw bones for clinical, biochemical and radiological features of hyperparathyroidism. Material and Methods: The history, physical examination, laboratory values, imaging and pathologic findings are described in a 32-year-old woman, presenting with brown tumour lesion in mandible, due to primary hyperparathyroidism. A systematic review of published literature from PubMed is added, which highlights the importance of a thorough diagnostic workup and selection of appropriate treatment modality. Results: In the case presented, after Parathyroid adenoma excision, within thirty minutes, the serum values of Parathormone and Calcium returned to normalcy and spontaneous regression of the brown tumour was noted. Also, the review of literature emphasized the need for systemic investigations of suspected giant cell jaw lesions and established that parathyroidectomy can be considered the primary treatment modality for brown tumours of the jaw due to hyperparathyroidism. Conclusion: Radiolucent lesions of the jaws showing giant cells on histopathology should raise suspicion of hyperparathyroidism. This case emphasizes the importance of a detailed systemic investigation for all lesions in the maxillofacial region.

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Key Words: Brown tumour, giant cell lesion, mandible, primary hyperparathyroidism

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Introduction Hyperparathyroidism, the third commonest endocrine disorder after diabetes mellitus and thyroid disease, can appear in primary, secondary, or tertiary forms.[1] Chronically raised parathormone (PTH) increases bony remodelling (with greater osteoclastic than osteoblastic effects) leading to osteopenia and osteoporosis. These bony effects can lead to brown tumours, which present as lytic, focally well-defined non-neoplastic lesions due to abnormal metabolism of the bone. In the jaws, they are a rare cause of radiolucency (roughly 1%).[2] They are characterized by a proliferation of highly vascular granulation tissue and a matrix of multinucleated osteoclast-type giant cells.[3,4]

Primary hyperparathyroidism is caused by a hypersecretion of parathyroid hormone. This usually occurs mostly due to idiopathic hyperplasia of parathyroid tissue, less commonly because of parathyroid adenoma, and very rarely owing to carcinoma of the parathyroid gland.[5,6] Most cases are diagnosed incidentally on routine blood chemistry analysis. Some are discovered from the classic constellation of symptoms described by the mnemonic ‘‘stones, bones, groans, and moans.’’[7] These include symptomatic nephrolithiasis (stones), bone pain (bones), nausea and vomiting (gastrointestinal groans), and stupor (psychiatric moans). Fewer than 5% of cases are recognized as the result of osteolytic giant cell lesions, and even fewer present with a peripheral giant cell lesion as the initial finding.[8]

Aims and Objectives:

1. To report a case of brown tumour in the mandible, which was also a concomitant case of long standing primary hyperparathyroidism.
2. To discuss the literature published on studies of brown tumours in maxillofacial region due to primary hyperparathyroidism.
3. To highlight the importance of a thorough systemic diagnostic workup in susceptible patients of radiolucent jaw lesions and exhibiting classical signs and symptoms of Calcium and Parathormone dysfunction.

Material and Methods:

- A computerized literature search was conducted at our Institute in the Department of Oral and Maxillofacial Surgery between July 2015 and October 2015 using PubMed for published English articles on brown tumour and hyperparathyroidism.
- MeSH phrases used in the search were: “brown tumour” and “parathyroid”.
- The search yielded 552 articles, of which 38 were chosen for review.
- The full texts of all these articles were thoroughly examined by the authors.
- Inclusion criteria included:

- Age : 13-80 years
- Case reports, review studies of brown tumour of Hyperparathyroidism (BTHPT) in the jaws.
- Studies involving Primary hyperparathyroidism.
- Articles published from 2004-2015.
- Exclusion criteria:
- Secondary and tertiary hyperparathyroidism
- Brown tumour lesions not involving the maxilla or mandible

Case Report:This study was carried out following the approval of the Ethics Committee of our institution and informed consent was obtained from the patient. A 32 year old female patient presented to our Outpatient Department of Oral and Maxillofacial Surgery at Government Dental College & Hospital, Ahmedabad with a swelling on the right side of the face since 2 months. Patient gave history of generalized weakness and lethargy since 6 months. Her family history, past medical and dental history was insignificant.

On extra-oral examination, a single well defined swelling was noticed on right body region of mandible, measuring approximately 3 cm × 4 cm, extending anteriorly from the right corner of mouth to angle of mandible. The skin over the swelling was shiny and stretched. There was no sinus, fistula, ulceration or erythema noticed over the swelling. On palpation, swelling was soft to firm, tender and non fluctuant.

On intraoral examination,(Fig.1) an expansion of the buccal cortex was noted from the mesial aspect of lower right canine upto right second molar region posteriorly. Obliteration of buccal vestibule was observed. On palpation, swelling was soft and tender. No associated discharge or bleeding was noted. Right submandibular lymph nodes were palpable, tender and firm in consistency.

Radiographic examination with orthopantamogram(Fig.2) and CT scan showed large unilocular radiolucency on the right side of mandible, extending from right canine to second molar without involving the lower border of mandible. There was generalized decrease in density of mandible with no resorption of teeth. An incisional biopsy report revealed hemorrhagic fibrovascular connective tissue with multinucleated giant cells consistent with the diagnosis of a giant cell lesion.

Patient was advised other blood investigations that revealed alkaline phosphatase and Parathormone levels of 310 IU/L (Normal: 28-78 IU/L) and 1421.3pg/ml (Normal: 12-65 pg/ml) respectively. Serum calcium level was 14.11 mg/dl (Normal: 8.5-10.5 ml/dl).

USG Neck revealed a well-defined heterogeneously hypoechoic mass with internal and peripheral vascularity, arising from the lower pole of left lobe of thyroid gland and measuring 10 X 10 X 24 mm., suggestive of parathyroid adenoma. Radionuclide scanning with ^{99m}Tc- sestamibi showed focal area of increased tracer uptake in left inferior parathyroid region. Scan findings were suggestive of adenoma involving left inferior parathyroid gland.

Patient underwent parathyroid adenoma excision. The resected gland was histologically suggestive of hyperplasia of parathyroid gland. Patient was given oral calcium supplementation in addition to vitamin D3 for possible post-operative hypocalcemia. The deranged serum values normalized expediently to Serum Parathormone- 56.6 pg/ml and Serum Calcium- 9.8 mg/dl, within 30 minutes after excision of adenoma. The clinical outcome of serum values being normalized resulted in diminished feeling of weakness and lethargy, as noted by patient herself. On the post-op follow-up after 6 months OPG showed calcification of the lesion (Fig.3) and after 1 year clinical and OPG showed the regression of the lesion (Fig.4).

Result: Brown tumour of hyperparathyroidism (BTHPT) is more common in developing countries, where fewer patients have access to health care and hyperparathyroidism goes untreated.[9] A review of the literature over the last 12 years (2004-2015) identified a total of 64 patients exhibiting primary hyperparathyroidism with brown tumour in maxilla or mandible in 38 reports. Analysis of this data is provided in *Table 1*. The highest number of BTHPTs was reported from Mexico (22) and 10 cases were reported from India.

Of the total 64 patients identified with BTHPT, 49 were females and 15 were males (3.26:1). This finding correlates with the study that stated BTHPT is three times more common in women than in men.[10] The mean age was 44.48 years (13 to 77), which is in accordance with a study by Bringhrust[11] stating that majority of lesions occurred between third to fifth decade of life. 51.5% brown tumours occurred in the mandible, 35.9% occurred in the maxilla, and 12.6% involved both jaws. This inference supports the study by Keyser et al[10] stating mandible to be the commonest among head bones

as a preferential site for development of brown tumour.

Of the 38 articles reviewed, 17 (44.73%) articles reported on followup and recurrence of brown tumour lesions. Reséndiz-Colosia et al.[12] studied the highest number of patients (22) in this review, all of which were followed up for 2 years and found to be recurrence free. Among the articles reviewed, only one study by Triantafillidou et al.[13] reported a case of recurrence in a 71 year old male, who presented with a secondary lesion of brown tumour in mandible, one year after surgical removal of right parathyroid adenoma. Patient was then managed by parathyroidectomy of three and one half of the fourth parathyroid glands and kept on close follow up.

Review of the treatment protocols in the evaluated studies shows that parathyroidectomy was the most commonly performed procedure in 40 out of the 65 cases (60.65%) followed by parathyroidectomy with removal of brown tumour. *(Table 2)*

The treatment of BTHPT varies from case to case. However, the treatment of choice of primary HPT is to regularise the hormone levels by excising the offending parathyroid gland.[14] Many authors report that, after parathyroidectomy, tumour regression and healing occurs spontaneously.[15] The most common approach to identifying the offending parathyroid gland is imaging using technetium (^{99m}Tc) sestamibi scan.

Although removal of the parathyroid gland in HPT has been shown to lead to regression of the brown tumour and osseous remodeling, the patient's age can be a factor in the rate of regression.[12] When regression is slow, surgical removal of the tumour is warranted.[49] Also in cases of huge tumours, with subsequent risk of fracture or excess morbidity to the tissues, excision of the brown tumour should be undertaken concurrently with parathyroidectomy. In the present review containing the sole case of intra lesional steroid injection by Wilson et al[40] triamcinolone acetonide was used to salvage an involved second molar and to perform a limited residual tumour excision in the pregnant female patient. A series of 10 monthly injections of intralesional Triamcinolone acetonide were employed to promote tumour regression.

Table 2 summarizes the methods of treatment for the 60 cases of maxillofacial BTHPT identified in the last 12 years. Majority of patients were treated with parathyroidectomy (61.66%). The rest had a combination of treatment modalities, although the extent and decision of surgical curettages and excisions were difficult to assess from reviewing these reports, as these factors are case specific, lesion dependent and based on the systemic values of hormones. Parathyroidectomy was attempted in our patient, which is also in accordance with the findings from the review.

Discussion: Brown tumour should be differentiated from other true giant cell tumours of bone, and it represents reparative granuloma rather than a true neoplastic process.[29,50] with an imbalance of osteoblastic and osteoclastic activities due to hormonal disturbances.[6] Macroscopically, these tumours are red or brown because of hemosiderin deposits caused by bleeding within these spaces, hence the term brown tumour. These tumours can occur in any bone of the skeleton and are representative of the later stages of hyperparathyroidism dependent bone pathology.[51] The most commonly areas affected include the ribs, clavicle, and pelvis.[15]

In a study involving 220 cases of hyperparathyroidism, only 4.5% of cases showed facial bone involvement.[52] Likewise, it is extremely rare for a brown tumour occurring within the facial bones to be the first manifestation of the systemic condition.[8] Primary hyperparathyroidism is far more commonly diagnosed as the result of routine serologic testing or symptomatic hypercalcemia.

Treatment of the brown tumour of hyperparathyroidism depends on the response after normalization of the PTH. In the present case, intraoperatively, PTH($t^{1/2}$=3–5 min)[1] normalized 30 minutes after the adenoma excision, which indicated successful removal of the affected gland. Lytic bony lesions tend to regress spontaneously if the calcium and PTH levels normalize.[53] According to Pellegrino[54], surgical excision of the brown tumour is indicated if the lesion is large and disfiguring, or if the affected bone is weakened and prone to pathologic fracture. In addition to surgery, several medical therapies for giant cell lesions have been reported, including systemic calcitonin, interferon, osteoprotegerin, receptor activator of nuclear factor-kβ ligand (AMG 162), imatinib, and intralesional corticosteroids.[55]

Despite naturally expected regression, several cases of brown tumour progression have been reported despite correction of hyperparathyroidism[56,57]

Authors such as Scott et al.[32] believe that bone lesions reappear spontaneously following removal of the diseased parathyroid gland; others such as Martinez-Gavidia et al.[15] recommend initial treatment with systemic corticosteroids in order to reduce the tumour size, followed by surgical removal. Jacoway et al[10] in 1988 described the rationale for steroid injections as histologic similarity between giant cell lesions and sarcoidosis, another granulomatous disease, which is effectively treated by corticosteroids. Studies by Flanagan et al[58]. indicated that multinucleated cells in giant cell granulomas of the jaws are osteoclasts and dexamethasone's inhibition of osteoclast-like cells in marrow cultures supports the use of intralesional corticosteroid.

In the case of large destructive tumours, the amount of tissue damaged may be so great that there are few possibilities of remodeling once normocalcemia has been achieved[26.] In these situations, or in cases where the lesions continue for more than 6 months, or there is disruption of the function of the affected organ, or tumour growth despite adequate metabolic control, Yamazaki et al[59] recommend curettage and enucleation.

Conclusion: Brown tumours are rare in developed countries and occur more frequently in developing countries because they are usually the result of uncontrolled and untreated HPT, perhaps due to lack of access to care. Prompt and thorough systemic and clinical workup with adjunctive use of radiography and appropriate treatment modality coupled with long-term follow-up of patients should be adopted as a basic standard protocol for management of brown tumour patients.

Conflict of interest: The authors hereby wish to state that this paper does not have any financial and personal relationships with other people or organisations that could inappropriately influence (bias) their work.

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