ISSN 2822-2032 (Print) 2822-2040 (Online)

Medical Journal of Eastern Nepal

Volume 02, Number 02, Issue 04, July-December 2023, 32-34

Case Report

BROWN TUMOR, A DECEPTIVE AND RARE ENTITY: A CASE REPORT

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Submitted: 1st – June – 2023, Revised: 20th -July-2023, Accepted: 5th – August - 2023

DOI:

ABSTRACT

A case of 27 years old male from Jhapa district, who presented to Oral and Maxillofacial department of B&C hospital with firm, non-tender swelling over buccal vestibule for 2 months. Computed tomography (CT) revealed an expansile lytic bony lesion with soft tissue component at the right hemimandible with cortical destruction. Provisional diagnosis of ameloblastoma was made.

Incisional biopsy done and specimen was sent to the Pathology department. Haematoxylin and Eosin- stained slides revealed disorderly, fascicular architecture exhibiting clustered distribution of osteoclastic giant cells, surrounded by fibroblastic stromal cells along with reactive new bone formation. The differentials considered were CGCG (Central giant cell granuloma), GCT (Giant Cell tumor), ABC (Aneurysmal Bone Cyst) and Brown tumor. Serum Calcium and Serum PTH (Parathyroid hormone) were advised to rule out brown tumor in histopathology report and for rest of the differentials excisional biopsy was suggested.

Serum calcium and serum PTH were found to be raised. On further probing, the patient has been having right lower back pain and thin urine stream on and off for few months. USG abdomen was done for the same which revealed bilateral nephrolithiasis with left sided PUJ calculus causing gross hydronephrosis along with bilateral medullary nephrocalcinosis. DJ stenting was thus advised by the nephrologist.

On identifying the primary cause, hemithyroidectomy with parathyroid removal was done. The decrease in parathyroid hormone level was drastic after the surgery. His urine stream was normal during his post –op stay at the hospital. The patient was advised for regular follow up.

Keywords: Brown tumor, Hyperparathyroidism, expansile lytic bony lesion.

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Citation
Vaidya M, Rai U, Gautam P, Yadav R, Sah. BP., Brown Tumor, A Deceptive And Rare Entity: A Case Report MJEN. 2023 July-December; 2(4):32-34

INTRODUCTION

A brown tumor is a bone lesion that results from bony resorption by excess osteoclastic activity, and replacement by fibrous tissue and giant cells. Brown tumor is an uncommon pathognomonic sign of hyperparathyroidism[1]. The incidence of primary hyperparathyroidism is reported as approximately 22 per 100,000 persons per year. Primary hyperparathyroidism is more common in woman than man, and peak incidence occurs in the 6th to 7th decade of life[2-3].

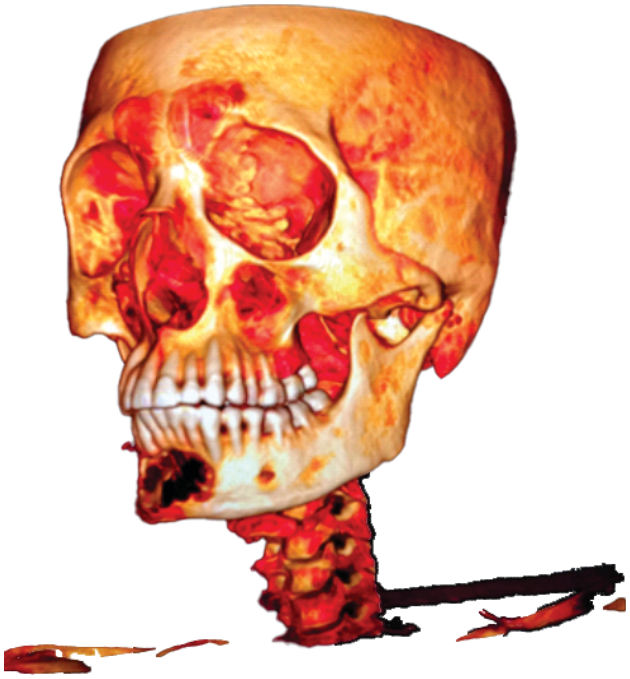

Figure 1: Skull showing mandibular lesion. 3D (VRT) image of CT.

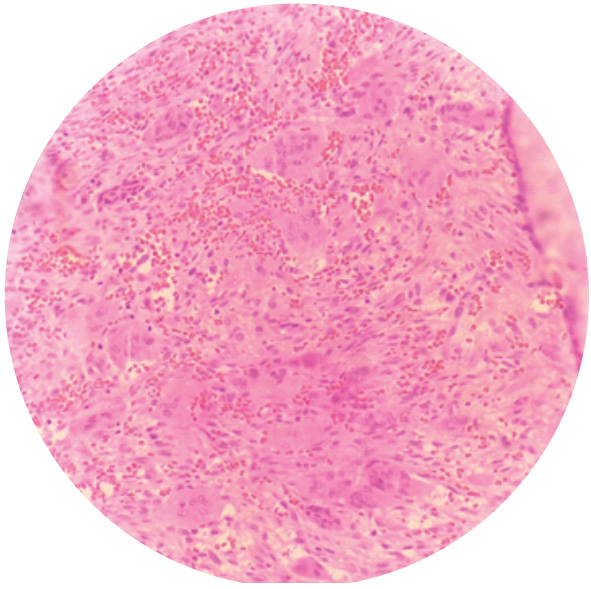

Figure 2: Fascicular growth of osteoclastic giant cells, surrounded by fibroblasts. H&E stain, 400x magnification.

CASE REPORT

A 27 years old male from Jhapa district presented to the Oral and Maxillofacial surgery department of B & C hospital with firm, non-tender swelling over buccal vestibule for 2 months. Plain CT with VRT revealed an expansile lytic bony lesion with soft tissue component at the right hemimandible with cortical destruction. Provisional diagnosis of ameloblastoma was made.

Incisional biopsy was done and the specimen sent to our Pathology department. The clinical diagnosis given was Ameloblastoma. Plain CT with Volume rendered technique (VRT) revealed an expansile lytic bony lesion with soft tissue component at the right hemimandible extending towards left with cortical destruction at both buccal and labial surfaces extending to the inferior margin of the right hemimandible. Radiological differentials considered were Ameloblastoma, Dentigerous cyst, Odontogenic myxoma.

The sample received in the Pathology department were three pieces of greywhite brownish soft tissue ranging in size from 0.5 to 1 cm in greatest dimension. All processed. **Microscopic examination showed** disorderly, fascicular architecture exhibiting clustered distribution of osteoclastic giant cells, surrounded by fibroblastic stromal cells. These giant cells were small, bearing 5 - 12 nuclei within. Extravasation of red blood cells and reactive new bone formation was seen. Mitotic figures were rare. There was absence of foamy macrophages within the stroma. Neither necrosis nor epithelial islands and cords noted. T**he overall clinicomorphological features favoured CGCG (Central giant cell granuloma) over other differentials like GCT (Giant Cell tumor) and ABC (Aneurysmal Bone Cyst).** *Serum Calcium and Serum PTH (Parathyroid hormone) levels were advised to rule out Hyperparathyroidism/* Brown tumor which came out to be raised [Serum calcium (Ca): 14.9 mg/dl (Reference range: 8.8 – 10.6) and Parathyroid hormone (PTH): 712 pg/ml (Ref.: 8.7 – 79.6)]. USG abdomen and pelvis revealed Bilateral nephrolithiasis with left- sided PUJ calculus causing gross hydronephrosis along with bilateral medullary nephrocalcinosis. Hence, Right superior parathyroidectomy along with Right Hemithyroidectomy was done (owing to the enlarged right superior parathyroid gland being adherent to the adjacent thyroid tissue). The right superior parathyroid gland was highly vascular and measured around 2 x 2 x 2 cm.

Figure 3: Gross view of the specimen.

The microscopic diagnosis of **Parathyroid adenoma, Oncocytic type** was made. IHC (KI- 67, PTH) was advised to rule out the rare possibility of carcinoma. However, patient refused. On post op investigation, Both Calcium and PTH levels were drastically reduced (Ca: 11.2 mg/dl, PTH: 46 pg/m).

DISCUSSION

The recorded prevalence of brown tumors is 0.1% [4]. They arise secondary to both primary and secondary hyperparathyroidism[5]. The disease can manifest at any age, but it is more common among persons older than 50 years and the sexual predilection is Female: Male = 3:1 [6]. Both monoostotic and polyostotic forms occur. They commonly affect the mandible, clavicle, ribs, pelvis, and femur[7]. Radiographically, brown tumors appear as well-defined marginated expansile lytic lesions and may cause cortical expansion [7-8].

Skeletal involvement occurs both in primary and secondary hyperparathyroidism. In the areas of bone resorption, the replacing fibroblastic tissue contains numerous osteoclast-like giant cells. If close attention is not paid to the clinical presentation and radiographic features, this may lead to mistaken diagnosis of a giant cell tumor. However, the giant cell tumors, in the typical diagnostic fields, usually lack fibrogenic stroma. Around the periphery may be osteoid-producing areas with an appearance reminiscent of fibrous dysplasia.

CONCLUSION

In our case, the diagnosis of Brown's tumor was established for the mandibular lesion, pathophysiology being Primary hyperparathyroidism due to Parathyroid adenoma. Though a rare entity, Brown tumor must be thought of as a possibility for a bony lesion with giant cells and serum levels of calcium and PTH be sought.

ACKNOWLEDGEMENT

We would like to acknowledge all the related departments and staff for active participation in this research work with special thanks to the Radiology department without whom the study would not have been possible.

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