Brown Tumour of Jaw Bone: A Diagnostic Attribute of Hyperparathyroidism

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ABSTRACT

Brown tumour is an uncommon focal giant cell lesion that arises as a result of direct effect of parathyroid hormone on bone tissue in patients with hyperparathyroidism. The lesion is characterized by extensive bone resorption which is replaced by fibrovascular tissue and giant cell with abundant deposit of haemosiderin. A rare case of Brown tumour in maxilla and mandible in 57-year-old female patient is reported. This case report highlights the importance of detailed systemic investigation for all the maxillofacial lesions since it could be the diagnostic attribute of the systemic diseases.

Keywords: Brown tumour; diagnostic attribute; hyperparathyroidism; jaw bone.

INTRODUCTION

Brown tumour is an unusual focal giant cell lesion arising as a result of direct effect of parathyroid hormone on bone tissue in patients with hyperparathyroidism (HPT).[1] Brown tumour usually occurs as a consequence of primary hyperparathyroidism and rarely secondary hyperparathyroidism.[2] The ribs, clavicles, pelvic girdle and mandible are the bones involved in Brown tumour. However, clinically significant lesion in craniofacial bone is not common.[3] The name of this metabolic bone lesion is derived from its characteristic dark brown color. Brown color is caused by abundant haemorrhage and deposition of haemosiderin pigments within the lesion.[1]

CASE REPORT

A 57 years old female patient presented with swelling in right lower back region and upper front region of jaw since 4 months. The swelling was gradual on onset and associated with dull aching pain and tendency of bleeding on minor trauma. Patient also gave history of lethargy, weakness and weight loss since past few months. There was no history of trauma. Patient was hypertensive. The incisional biopsy of the lesion was previously diagnosed as Central giant cell lesion.

On extra oral examination, bilateral submandibular lymph nodes were tender and palpable. Intraoral examination revealed nodular and lobulated mass of about 3 cm X 1.5 cm in size extending from first molar to the retromolar region in the right side of the mandible. The overlying mucosa was bright red in color and no surface ulceration was noticed. Similarly, nodular lobulated exophytic mass about 1 cm X 1 cm in size on the right side of anterior region of maxilla was present. The overlying mucosa was bright red in color (Figure 1). On palpation, both the lesions were firm and tender and showed tendency of bleeding on probing.

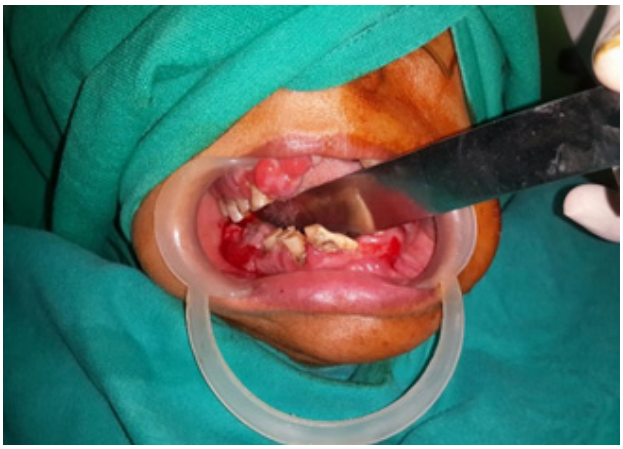

Figure 1: Clinical presentation with nodular lobulated lesion on right side of posterior mandible and right side of anterior maxilla.

Cone beam computed tomography revealed multiple lytic lesions extending from 43 to 48 regions on right posterior mandible. Perforation of buccal and lingual cortical plates at different levels was present (Figure 2a). Presence of soft tissue shadow of medium density in the lytic area was also noticed. Posterior teeth 46 and 47 were missing. Similarly, bone loss in the anterior maxilla involving buccal and palatal cortical plates with loss of alveolar bone support was seen (Figure 2a and 2b). Radiographic examination also revealed generalised loss of alveolar bone support, drifting of teeth (14, 23, 41), floating teeth (31, 45) and multiple missing teeth (11, 12, 13, 21, 22, 34).

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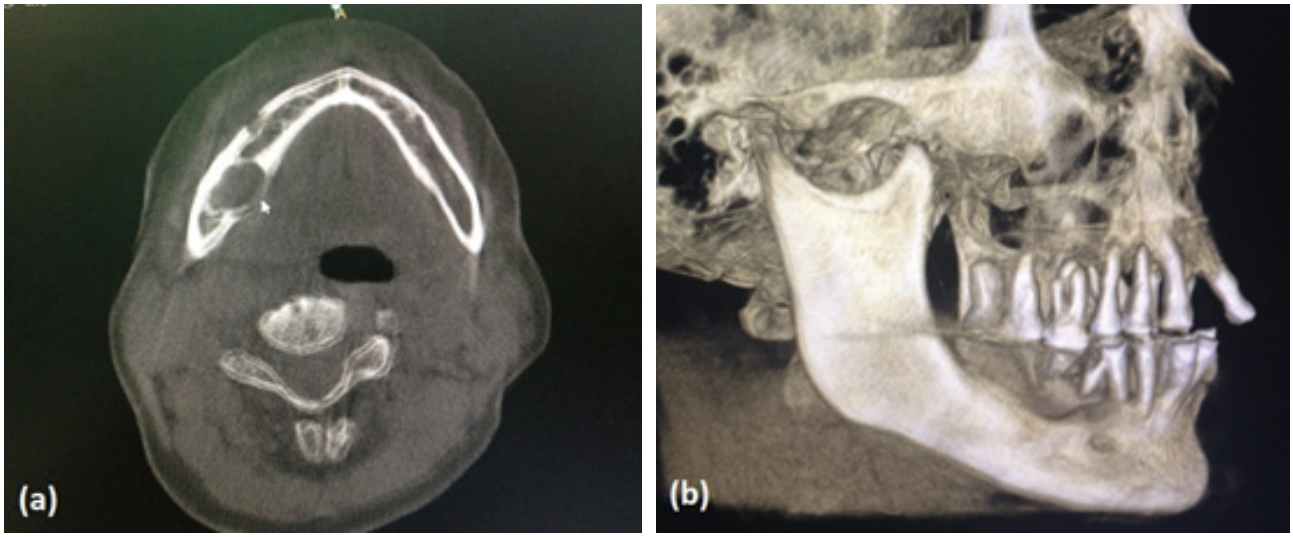

Figure 2: Radiographic presentation (a) axial view (b) 3D reconstruction view.

Table 1: Findings of blood investigation

	Result	Reference Range
PTH	275.5 pg//ml	10-65 pg/ml
Serum calcium	12mg/dl	8.4-10.2 mg/dl
Serum phosphorus	2.5mg/dl	2.5-4.5 mg/dl
Alkaline phosphatase	124IU/L	44-147 IU/L

Table 2: Findings of renal function test

Renal Function Test	Result	Reference Range
Urea	17.4 mg/dl	15-45 mg/dl
Creatinine	0.4 mg/dl	M: 0.6-1.2 mg/dl F: 0.5-1mg/dl
Sodium	143 mEq/dl	135-146 mEq/dl
Potassium	3.5 mEq/dl	3.5-5.2 mEq/dl

Excisional biopsy of both the maxillary and mandibular lesion was done. Histopathological examination revealed proliferation of the spindle cells with variably sized multinucleated giant cells (Figure 3). Areas of haemorrhage and deposition of the haemosiderin pigments could also be appreciated in lesions from both the sites (Figure 4). The blood investigations revealed elevated serum parathyroid hormone (PTH) level (Table 1). Renal function test was also performed to rule out secondary hyperparathyroidism in which urea, creatinine, sodium and potassium all were within normal limits (Table 2).

Correlating clinical presentation, blood investigation and histopathological finding final diagnosis of Brown tumour of hyperparathyroidism was given. Patient was further advised for Ultrasonography (USG) of neck, USG of abdomen, Chest X-ray, X-ray of pelvis and hip. USG of the neck revealed parathyroid adenoma, chest X-ray revealed lytic lesion on left clavicle, USG abdomen did not reveal any significant pathology and X-ray of pelvis was also normal. Excision of parathyroid tumour under general anaesthesia was planned.

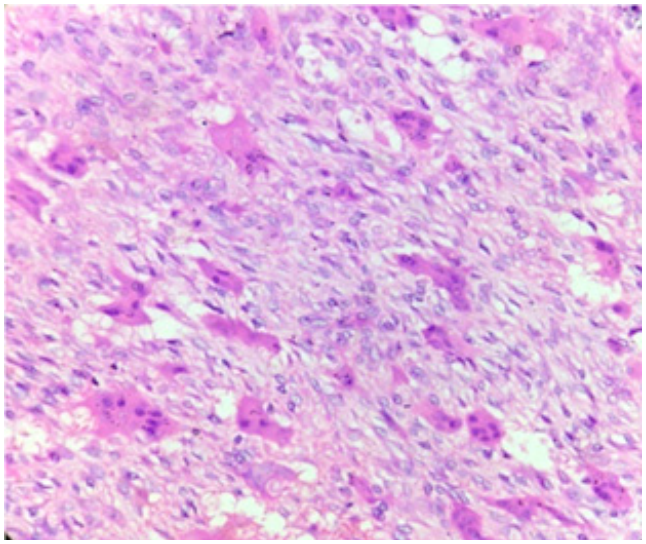

Figure 3: Photomicrograph showing proliferations of the spindle cells with variably sized multinucleated giant cells. 400X Magnification [H&E].

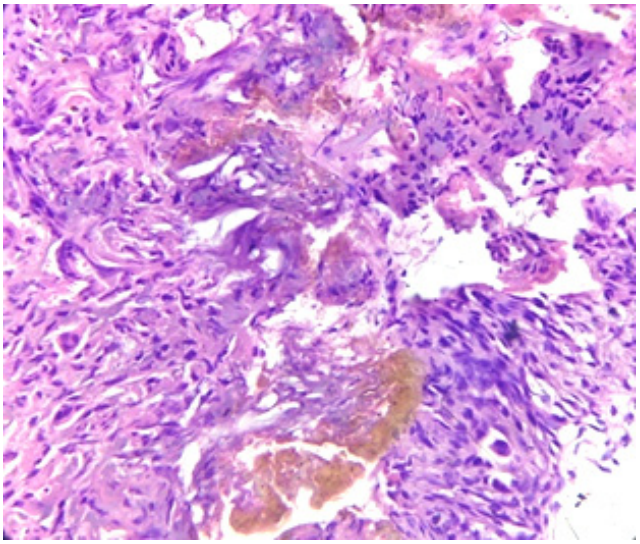

Figure 4: Photomicrograph showing areas of deposition of haemosiderin pigment. 400 X Magnification [H&E].

DISCUSSION

Hyperparathyroidism and hypoparathyroidism are two well-defined entities associated with parathyroid gland. In primary HPT, there is autonomous secretion of PTH, usually by a single parathyroid adenoma varying in size. Secondary HPT results from increased PTH secretion to compensate for prolonged hypocalcaemia and is associated with hyperplasia of all parathyroid tissue. In a very small proportion of cases of secondary HPT, continuous stimulation of the parathyroid glands results in adenoma formation and autonomous PTH secretion which is known as tertiary HPT.[2]

Primary hyperparathyroidism is the most common of the parathyroid disorders with prevalence of about 1 in 800. It is two to three times more common in female than male with 90% of patients over 50 years of age. It also occurs in all of the familial Multiple endocrine neoplasia syndromes.[4] Clinical features of HPT are described as "bones, stones and abdominal groans." The ribs, clavicles, pelvic girdle and mandible are the bones commonly involved. The maxilla, mandible and clavicle were the bones involved in the present case. A pathologic fracture may be the first symptom of the disease. Fifty percent of the patients with primary HPT are asymptomatic.[5] Hypertension is common in HPT which was also present in this case.4 The bones of affected persons with HPT show a general radiolucency as compared with those of normal people. In the jaws, the bone radiograph in HPT has been described as having a 'ground-glass' appearance.[4,6] Other dental manifestations includes mobile teeth, drifting of teeth, complain of vague jaw pain, sensitive teeth in mastication and percussion which were also present in our case.[7]

Osseous tumour which develops with the persistence of HPT is called Brown tumour of HPT. It is so called because of its characteristic dark brown colour. Brown colour is caused by abundant haemorrhage and deposition of haemosiderin pigments within the tumorous tissue.[8] Degeneration of brown tumour in long standing cases leads to osteoclastic bone resorption, cystic lesions and replacement of the lesion with vascularized fibrous tissue containing giant cells which is known as Osteitis fibrosa cystica.[8] Similar histopathological finding was present in our case. Jaw bones are commonly affected by Brown tumour in primary HPT.[6] In the areas of bone resorption one also finds many plump osteoblasts lining islands of osteoid.[8,9] Diagnosis is further confirmed by hypercalcemia, hypophosphatemia, elevated serum PTH level, hypercalciuria, hyperphosphaturia and elevated serum alkaline phosphates level.[8,10] Diagnosis of present case was also confirmed by elevated serum PTH level, calcium and alkaline phosphatase level which differentiates brown tumour from central giant cell lesion.[8]

Treatment involves control of HPT and for primary HPT, partial parathyroidectomy is required. Small osteolytic jaw lesions may regress spontaneously, however, with large disfiguring and symptomatic lesions, excisions may be indicated. Postoperative hypocalcemia may occur in patients who undergo partial parathyroidectomy, therefore, calcium supplements could be required.[10]

Brown tumour is indistinguishable microscopically from the central giant cell granuloma of bone. Patients diagnosed as a central giant cell lesion should be evaluated medically to rule out the possibility of hyperparathyroidism. Diagnosis of brown tumour is readily confirmed by establishing elevated serum calcium and PTH levels.

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