Extraoral examination revealed gross facial asymmetry with a well-defined swelling involving the middle third of the face on the right side. There was obliteration of nasolabial fold on involved side of face [Figure 1]. Intraoral examination revealed a well-defined solitary swelling measuring about 4 × 4 cm on the right side of hard palate not crossing midline. Posteriorly the lesion was seen extending upto the edentulous region of 15. Mucosa over the swelling was normal with no secondary changes [Figure 2]. The swelling was nontender to palpation and was hard in consistency. No lymphadenopathy was seen. Occlusal radiograph revealed well-defined solitary radiolucent lesion involving the right side of anterior hard palate. Slight root resorption of 11 and 12 was seen. Loss of lamina dura in relation to 21, 22 and 23 region was seen [Figure 3]. Panoramic radiograph (OPG) showed findings consistent with occlusal radiograph although the lesion is superimposed with ghost image of spine. OPG also revealed generalised loss of lamina dura and thinning/loss of cortices of inferior border of mandible, walls of maxillary antrum and inferior alveolar canal suggestive of generalised rarefaction of jaw bones [Figure 4].

Coronal CT showed a solitary hypodense mass in the maxillary anterior region measuring approximately 3 × 4 cm, spherical shaped and encroaching the nasal fossa/cavity [Figure 5]. The radiological differential diagnosis included giant cell lesion associated with HPT. The patient was then subjected to incisional biopsy and gross examination revealed multiple friable soft tissue bits that were brown in colour.
Histopathological examination revealed numerous multinucleated giant cells set in a background of fibrocellular connective tissue stroma showing numerous blood vessels, blood-filled spaces, suggestive of central giant cell lesion [Figure 6].

In view of the radiological and histological evidence of giant cell lesion, the patient was further subjected to blood
examination which included estimation of PTH, serum calcium, alkaline phosphatase and renal functional test. Blood examination revealed abnormally elevated PTH levels of 1810.9 pg/ml, increased Alkaline phosphatase 431 U/l, increased serum creatinine 2.5 mg/dl, elevated blood urea nitrogen (BUN) 35 gm/dl, increased serum phosphatase levels 7.5 mg/dl, decreased serum calcium 7.6 mg/dl, decreased haemoglobin 9 gm%. Based on clinical, radiographical, histopathological and lab findings the patient was diagnosed as Brown tumour resulting from secondary HPT due to underlying chronic renal failure. The patient was then referred to endocrinologist for further management.

DISCUSSION

Brown tumours are non-neoplastic lesions resulting from abnormal bone metabolism in HPT. Bone involvement is a late manifestation of HPT and includes classic skeletal lesions such as bone resorption, bone cysts, brown tumours and generalised osteopenia. Brown tumour is a kind of giant cell lesion and is so named because of its friable red-brown appearance resulting from vascularity, haemorrhage and haemosiderin deposits. They usually present as painless enlargement with slow growth rate occurring with the frequency of 4.5% in primary HPT and 1.5% to 1.7% in case of secondary HPT with overall incidence of 0.1%.

The commonly involved sites are facial bones, clavicle, ribs, pelvis and femur. They are more common in mandible compared to maxilla and are three times more frequent in women aged over 50 years. The present case occurred in maxilla in 42-year-old female and was asymptomatic. Secondary HPT represents a frequent complication in patients with chronic renal failure. Hypocalcemia, hyperphosphatemia may be significant in chronic renal failure and may stimulate increased PTH secretion, which in turn alters intra and extracellular calcium ratio thereby causing increased bone resorption, decreased bone density as seen in the present case.

The histopathological features of Brown tumour are suggestive but not sufficient enough to establish the diagnosis or differentiation from other giant cell lesion such as central giant cell granuloma, giant cell tumour, etc.

In the present case the histopathological features resembled central giant cell granuloma but the diagnosis of Brown tumour associated with secondary HPT was established in view of the clinical, radiographical and laboratory reports.

They are often asymptomatic as in the present case and sometimes may be, the first impending sign of an underlying previously undiagnosed endocrine disorder.

Treatment should commence with treatment of underlying HPT which in turn results in complete resolution. Controlling HPT is mandatory and may be done by carrying out total parathyroidectomy.

CONCLUSION

Although rare, Brown tumours of jaws should be considered in the differential diagnosis of giant cell lesions of bone and the dental surgeon should be aware of oral manifestations associated with underlying systemic disease and need for careful clinical, radiological, serological examination cannot be over-ruled.

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How to cite this article: Thankappan P, Chundru NS, Amudala R, Kuppusamy A. Maxillary tumour as first sign of endocrine abnormality: A report of a rare case. Niger Med J 2015;56:77-9.

Source of Support: Nil, Conflict of Interest: None declared.