Oral Manifestation of Secondary Hyperparathyroidism : A Case Report

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Abstract

Systemic diseases have been frequently known to cause a variety of signs and symptoms in oral cavity. One such systemic disease causing bony changes in the oral cavity is secondary hyperparathyroidism. This disease occurs in any condition that causes chronic hypocalcaemia; commonest causes being vitamin D deficiency and chronic kidney diseases. This article will be discussing a case of a 25 year old male with chronic renal failure and presenting with bony changes in maxilla and mandible.

Key words: Secondary hyperparathyroidism, Chronic renal failure, Oral manifestations of renal disorder.

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Introduction

Serum calcium and phosphorus levels are regulated by parathyroid glands and kidneys. Low levels of serum calcium, stimulates the release of Parathyroid hormone which causes an efflux of calcium ions from the bones and facilitates calcium reabsorption by the kidneys thereby maintaining levels[1].Hyperparathyroidism [HPT] is a disorder characterized by an excessive production of parathyroid hormone. Depending on the cause, HPT can be characterized into primary, secondary, tertiary and quaternary forms[2].Secondary Hyperparathyroidism results when there is increase in secretion of parathormone in response to lowered serum calcium level, due to some predisposing systemic condition, most commonly renal failure and Vitamin D deficiency[3].

Here, we present a case of secondary hyperparathyroidism as a corollary to chronic renal failure with emphasis on its oral manifestations.

Case Report

A 25-year-old male reported to Department of Oral Medicine and Oral Radiology, Government Dental College and Hospital, Mumbai, with the chief complaint of gradually enlarging, painless swelling on both sides of the upper jaw since 2 - 3 months. The patient had h/o hypertension since 8 years for which he was taking beta blockers-Metoprolol 25mg once daily. He was also suffering from chronic renal failure and was under regular dialysis since 2 years. He had no habits, and his family history was unremarkable.

GENERAL EXAMINATION: The patient had a normal built and gait. There was absence of lunula on all his finger nails.

EXTRA-ORAL EXAMINATION: Slight midfacial expansion was noted bilaterally extending from outer canthus of the eyes upto the ala-tragus line on both sides. **[Figure I &II]**.

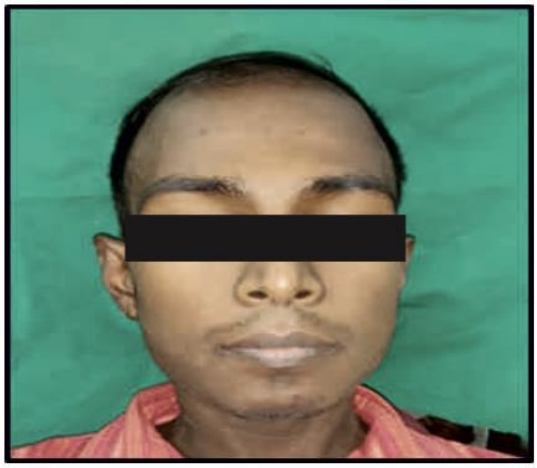

Figure I : Mild mid-facial expansion of the face

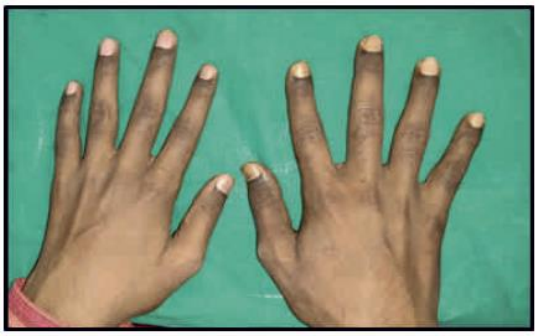

Figure II : Absence of lunula of nails

INTRAORAL EXAMINATION: A bony hard, non-tender, expansile swelling was noted bilaterally in the palatal region extending from behind the naso-palatine foramen upto the mesial aspect of the first molars. Mandibular arch showed slight expansion of the buccolingual cortical plates in posterior regions bilaterally **[Figure III].**

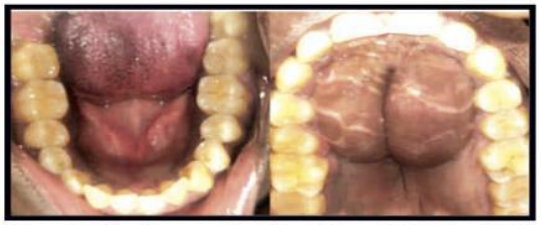

Figure III : Showing expansion of maxillary palatal region (right) and mild expansion of mandibular posterior region(left)

After evaluating the history and clinical examination, provisional diagnosis of benign tumor/cyst was made and following differential diagnosis were considered - Secondary hyperparathyroidism and Central giant cell granuloma.

Radiographic Findings

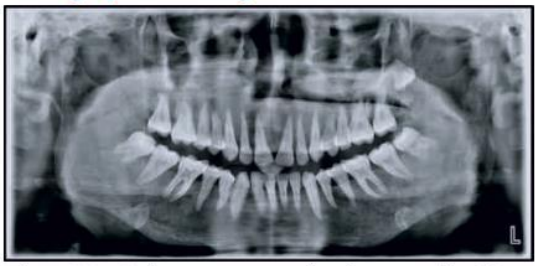

Figure IV : Orthopantomogram showing generalized loss of lamina dura and generalized haziness due decalcification

The orthopantomogram revealed generalized loss of lamina dura**[Figure IV]** reduced bone density, haziness and alteration of normal trabecular pattern ('ground glass appearance'), loss of cortication of the inferior border of the mandible, thinning of cortical boundary of inferior alveolar nerve canal, loss of cortication in the medial and posterior walls of maxillary sinus and floor of the maxillary sinuses was not traceable bilaterally.

The posterior- anterior skull view revealed typical, 'salt and pepper' appearance over calvarium. Hand and wrist radiograph revealed subperiosteal erosion over tips of phalanges **[Figure V]**.

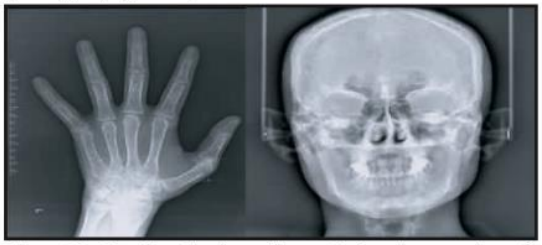

Figure V : Hand and wrist radiograph showing erosion of terminal phalanges (left) & PA skull view showing 'Salt and pepper' appearance (right)

Cone Beam Computed Tomography [CBCT] examination revealed generalized resorption of the visualized skull bones **[Figure VI & VII]**, bicortical

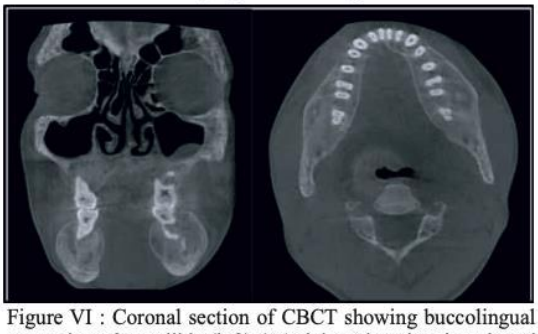

Figure VI : Coronal section of CBCT showing buccolingual expansion of mandible (left) & Axial section showing altered trabecular pattern (right)

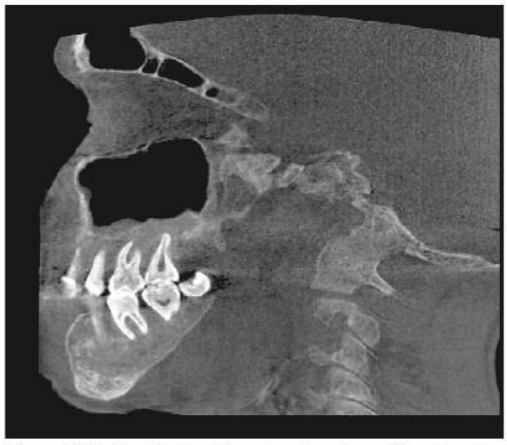

Figure VII : Sagittal section showing loss of lamina dura and osteoporotic changes of bone

expansion of mandibular posterior region with thinning of the cortical outlines, interior of the lesion showed mixed radiodensity and sagittal section showed generalized osteoporotic changes in the base of the skull, vertebrae and mandible.

After correlating history of systemic condition of renal failure with clinical and radiographic examination, a provisional diagnosis of secondary hyperparathyroidism was made. The following radiographic differential diagnosis of secondary hyperparathyroidism, polyostotic fibrous dysplasia and Paget's disease were considered.

The laboratory investigations confirmed blood biochemistry changes **[Table 1]**, which could be correlated

Tests	Normal values	Patient's values	Interpretations
Serum Calcium (mgs%)	8.5-11	9.20	Normal
Serum Phosphorus (mgs%)	2.3-4.7	5.40	High
Alkaline Phosphotase (IU/L)	25-147	566	High
Paratharmone levels (pg/mL)	10-69	>2500	Significantly increased
Vitamin D levels (ng/ml)	30-100	19.7	Insufficiency

to those associated with secondary hyperparathyroidism. The patient was referred to department of Endocrinology for further management and currently is under follow-ups.

Discussion

Hyperparathyroidism was first described by Von Recklinghausen in 1891, presenting its systemic effects. Its oral manifestations were reported in 1945 by Weinmann in the mandible[4]. Hyperparathyroidism occurs due to increased activity of the parathyroid glands, either from an intrinsic abnormal change or due to extrinsic factors affecting calcium homoeostasis. Depending on the cause, it can be characterized into following forms – a] Primary Hyperparathyroidism- Usually caused by a tumor (adenoma in 85% of all cases) or hyperplasia of the gland[5], b] Secondary Hyperparathyroidism- Low serum calcium levels in different physiologic or pathologic conditions like renal failure, intestinal malabsorption syndrome, decrease of Vitamin D production, trigger release of PTH[6], c] Tertiary Hyperparathyroidism- When long-standing secondary hyperplasia becomes autonomous in spite of correction of the underlying stimulant[3] and d] Quaternary Hyperparathyroidism- When long standing primary adenoma after surgical removal relapses again with adenoma formation[2].

HPT is generally seen in middle-aged patients (30–60 years) with female predominance and male: female ratio of 1:1.7. In the present case, the patient was a 25-year-old male. Symptoms and clinical signs often relate with chronic hypercalcaemia rather than to increased PTH. Signs and symptoms are classically described as abdominal groans, kidney stones, tender bones, psychic moans, and fatigue overtones[7]. In the systematic review done by Palla et al including 254 patients, facial asymmetry was the primary presenting feature in 78.0% of the cases. 40.8% of the cases presented with bony pathologies in the mandible, while in the maxilla it was 29.4%, and 29.8% included both the jaws[8]. This case presented with chief complaint of gradually enlarging swelling leading asymmetry of face.

Radiographic changes commonly observed includes osteopenia, loss of cortication of mandibular canals and blurring of normal trabecular pattern causing ground glass appearance[7]. All of these features were appreciated in the presented case. In a study by Silverman et al of 42 patients with HPT, only five cases were found with partial loss of lamina dura and not a single case with a complete loss[9]. Lamina dura was completely absent in the present case.

Brown's tumor, also called osteitis fibrosa cystica, was observed in the present case and is seen in approximately 13% cases of (renal osteodystrophy) secondary HPT caused by end-stage renal disease[7,10] Diagnosis of secondary Hyperparathyroidism was confirmed and differentiated from its primary variant by blood chemistry analysis which showed elevated PTH and Alkaline phosphatase with normal or low serum calcium levels[11].

Treatment is generally aimed at reducing the level of PTH by various medical and surgical means. Treatment of the underlying renal disease or kidney transplantation has been shown to produce marked improvement in the condition. Regression or resolution of Brown's tumour occurs following either oral/iv administration of vitamin D supplements such as calcitriol and paricalcitol[12,13].

Conservative surgical debridement of Brown's tumour though not common has also been shown to yield acceptable results. Parathyroidectomy can also be considered as a viable option in extreme and recalcitrant cases.

Conclusion

In patients with chronic renal failure the likelihood of developing secondary hyperparathyroidism is very high which further increases the complications and morbidity in such patients. Osseous changes in the jaw bones can be the first presenting signs in such cases and this places a dentist at an unique position to be the first one to identify and diagnose the condition thus, it is imperative for a dental practioner to familiarize himself with the variable clinical and radiological features of hyperparathyroidism and therefore facilitate prompt and optimum treatment of such patients.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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