



Role of Oral Physician in Diagnosis of Hyperparathyroidism

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Abstract

Oral physician has an important role in diagnosing patients with systemic diseases which can save the patient's life, one such systemic disease which is diagnosed on incidental findings of oral manifestations is hyperparathyroidism. Parathyroid hormone plays an important role in the metabolism of calcium and phosphorus; influencing the mineralization of bone and teeth. Parathyroid disorder may lead to hyper or hypo secretion of hormone, which results in various oral manifestations. Common oral manifestations in patients with hyperparathyroidism (HPT) are brown tumor, loss of bone density, soft tissue calcification, and dental abnormalities. Dental management of patients with HPT involves a higher risk of bone fracture, whereas in hypo parathyroidism the caries control is the main concern. It is the important that the dentist be aware of the risks and difficulties that may arise during the dental management of these patients.

Earlier the diagnosis better is the treatment. As rightly said mouth is mirror of the body many diseases are diagnosed with oral and maxillofacial manifestations which can be the initial or the only signs of the disease. Thus, emphasizing the role of oral physician in diagnosis and further management of the patient.

Keywords: Hyperparathyroidism; Oral Physician; Oral Manifestations

Introduction

Parathyroid consists of four small glands (3 mm wide × 6 mm long × 2 mm thick), with dark brown in color, which are paired and located behind the thyroid gland in the neck. Parathyroid glands produce and release parathyroid hormone (PTH), which is involved in regulating the metabolism of calcium and phosphorus [1]. So, it plays an important role in tooth and bone mineralization,

increases the bone resorption, stimulates formation of active metabolite of Vitamin D in the kidneys, which promotes the intestinal absorption of calcium and decreases renal re absorption of phosphate. Normal serum PTH is about 10- 65 ng/L [2].

Management of medically compromised patients is challenging, which requires early diagnosis and prompt treatment [2]. Oral physician is the health care professional who takes immense pride

in this situation where on the basis of oral and maxilla-facial manifestations the disease is diagnosed [3]. The present case report is a hyper parathyroid patient who was diagnosed with oral clinical features and incidental radiological features [2]. Search with Pub Med keywords are Hyperparathyroidism oral manifestation/clinical features, Role of dentist/case report articles between years 2009-2016 were used for the review.

Case Report

18 years old male patient came to department of Oral Medicine and Radiology, with chief complaint of pain and obliteration of buccal vestibule in mandibular right posterior teeth region. Patient initially went to the local dentist for the complaint and he took IOPA for the same and gave history of root canal treatment with diagnosis of periapical infection. Unfortunately pain did not subside and hence the patient reported to our Institute. Past Dental history revealed of the endodontic treatment, extraction of tooth and restoration. No relevant medical history was given by patient. Family and habit history were not contributory.

History of tiredness, lethargy, bone pain and mild state of confusion was given by the patient. Physical examination shows normal vital signs. His neck examination show no goitre/nodule. No pulsations or fluctuant areas were noted.

Intra-oral examination shows discolored tooth 46 (Figure 1) obliteration in the buccal vestibule as well as temporary filling associated with 46 was noted. To know the pathology associated with the tooth, we took IOPA (Figure 2) which shows indistinct loss of lamina dura, periapical radiolucency extending from distal aspect of second premolar to mesial aspect of the second molar with altered bony trabeculae throughout and showing ground glass appearance [7]. The orthopantomogram (OPG) was taken and showed in (Figure 3) and it was noted that generalized loss of lamina dura with thinning of inferior border of mandible bilaterally at angle region. Multiple radiolucencies in mandibular anterior and posterior region with corrugated margins with no root resorptions at periapical region with 46, altered bony trabeculae and inferior alveolar nerve canal is also not traceable bilaterally. With missing left mandibular first molar, erupting all four third molars and radiopaque filling noted in right mandibular first molar (Figure 3). A hand- wrist radiograph was taken which showed signs of subperiosteal erosion (Figure 4).

Cone-beam computed tomography (CBCT) examination was performed as shown in (Figure 5) CBCT examination showed, bicortical expansion; however the cortical outlines were hypo calci-



Figure 1: Findings on intraoral examination.



Figure 2: Findings on intra oral periapical radiograph.

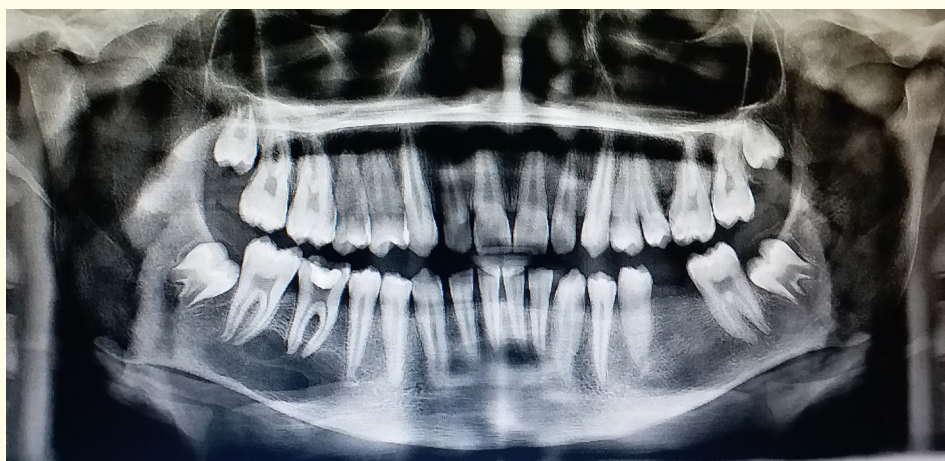


Figure 3: Orthopantomograph findings



Figure 4: Hand wrist radiograph showing sub periosteal erosions.

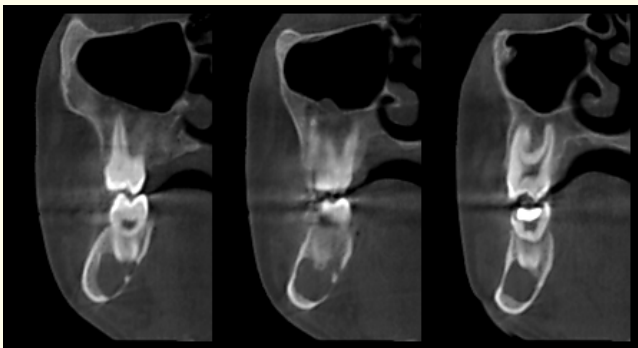


Figure 5: Advanced imaging by CBCT appreciating the changes.

fied with perforation in the lingual cortical plate, showing ground glass appearance and loss of lamina dura. Decrease in trabecular density can be appreciated.

The CBCT details were conclusive and considering patients affordability further radiographic evaluation using CT and DEXA were not undertaken.

After clinical and radiographic examination and correlating it with the history of patient (no history of chronic kidney disease and dialysis), a provisional diagnosis of primary HPT was made with differential Diagnosis of Fibrous Dysplasia, Paget's disease.

To confirm the provisional diagnosis patient was referred for biochemical investigations as mentioned.

Tables 1 shows the reports of the investigations and were positive for the hyperparathyroidism status. It shows marked increase of parathormone level, normal calcium level and alkaline phosphatase on the higher side albeit within normal limits as shown in the (Table 1-3) Provisional diagnosis was confirmed as hyperparathyroidism. Thus the final Diagnosis of Primary hyperparathyroidis was made as there was no kidney involvement detected. The patient was refereed to General Physician for further investigations and management.

	Calcium	Phosphate	Alkaline phosphate	Urea
Primary without bone lesion With bony lesion	Increased	Decreased	Normal	Normal
	Increased	Decreased/Normal	Increased	Normal/Increased
Secondary Hyperparathyroidism Due to Renal failure Due to Malabsorption	Normal/Decreased	Normal /Increased	Normal/Increased	Increased
	Normal/Decreased	Decreased	Increased	Normal
Tertiary hyperparathyroidism with bone lesion without bone lesion	Increased	Normal/Decreased	Increased	Normal/Increased
	Increased	Decreased	Normal	Normal/Increased

Table 1: showing Biochemical reports.

Test	Patients value	Normal Values	Interpretation
Serum Calcium	10.6	8-11 mg/dl	Normal
Alkaline phosphates	155	53-155 IU/L	Borderline
Parathormone	277.1	14-72 pg/mL	Increased

Table 2: Parathyroid Function Test.

Cause of hyperparathyroidism	Parathyroid adenoma parathyroid hyperplasia Parathyroid carcinoma	Parathyroid hyperplasia	Autonomous module on top of hyperplasia Chronic kidney disease.
	Result from benign tumor from one of parathyroid gland	Conditions like renal failure, intestinal malabsorption.	When long-standing secondary hyperplasia becomes autonomous in spite of correction of the underlying stimulant (renal transplant)
Sign and symptoms	Bone pain, muscle weakness, long bone fracture, swollen joints, nephrolithiasis, occasionally peptic ulcers, pancreatitis, hypertension.	Abdominal groans, stones, tender bones, psychic moans, and fatigue overtones.	Bone pain, muscle weakness, long bone fracture, swollen joints, nephrolithiasis, occasionally peptic ulcers, pancreatitis, hypertension
Radiological findings	Obliteration of pulp chamber Alteration of Dental eruption Loosing and Drifting of teeth Loss of lamina dura Periodontal ligament widening Chondrocalcinosis ground glass appearance	obliteration of pulp chamber Alteration of Dental eruption Loosing and Drifting of teeth Loss of lamina dura periodontal ligament widening chondrocalcinosis ground glass appearance	obliteration of pulp chamber Alteration of Dental eruption Loosing and Drifting of teeth Loss of lamina dura periodontal ligament widening chondrocalcinosis ground glass appearance
Laboratory Investigations	Increased level of PTH, hypercalcemia, elevated alkaline phosphates levels and decreased phosphorus levels.	Serum calcium ⁺ PTH ⁺ Elevated PTH and Alkaline phosphates with normal or low serum calcium levels confirm the diagnosis and also help in differentiating it from primary variant.	Serum calcium ⁺ PTH ⁺
Management	Of Acute hypocalcaemia medical line- avoid thiazide diuretics Maintain oral fluid intake Surgery Surgical excision of adenoma.	Correcting vit D deficiency Treatment of renal disease. Active Vitamin D A combination of dietary phosphorus restriction, phosphate binders, calcimimetics, and Vitamin D analogs can be used	Total or subtotal parathyroidectomy Auto transplantation of parathyroid tissue.
Diagnostic imaging Technique	Ultrasonography, MRI or CT have sensitivity about 52-75%. Highest sensitivity in localization of ectopic parathyroid adenoma has sestamibi scintigraphy with technetium-99.	Ultrasonography, MRI or CT have sensitivity about 52-75%. Highest sensitivity in localization of ectopic parathyroid adenoma has sestamibi scintigraphy with technetium-99.	Ultrasonography, MRI or CT have sensitivity about 52-75%. Highest sensitivity in localization of ectopic parathyroid adenoma has sestamibi scintigraphy with technetium-99.

Table 3: Summary and differentiating feature of the types of hyperparathyroidism.

Discussion

Hyperparathyroidism (HPT) was first described by Von Recklinghausen in 1891, reported patients with a condition called osteitis fibrosis cystica [4]. It affects 0.05-0.1% of general population. The prevalence is 1 in 400 female and 1 in 1000 male [2]. It usually affects middle aged person [5]. The term hyperparathyroidism was coined by Henry Dixon and colleagues [6]. It was realized that HPT has a effect on multiple system of the body such as renal system, skeletal system, early effect on bone such as subperiosteal erosions and loss of lamina dura [7]. sometimes cortisone test was used to differentiate between hypocalcaemia and HPT, which was not performed in the present case [8]. Diagnosis usually made by increase levels of Serum parathyroid hormone (normal range 14-72pg/ml), Alkaline phosphatase (normal range 53-155 IU/L) and normal levels of calcium (normal range 8-11mg/dl) [3].

The oral physician plays important role in the detection of HPT [3]. Occasionally, the first sign of the disease may be a cyst in the jaw. The disease should be considered by the dentist whenever single or multiple radiolucencies are observed on radiographs of the jaw. It is important for an oral physician to be aware of the various oral and extra oral findings so that the disorder can be diagnosed correctly followed by a precise and prompt treatment. There is higher risk of bone fracture, before providing endodontic treatment [9].

Conclusion

This report also stresses upon taking good past history as radiolucency at periapical region was initially misdiagnosed as endodontic pathology. This warrants for being suspicious with the teeth with failed treatment [9]. Management of medically compromised patients is challenging and requires a multidisciplinary approach. Oral physician is the health care professional who plays a vital role in diagnosing the hidden diseases which are manifested by oral maxillofacial manifestations.

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