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Oral Manifestations of Hyperparathyroidism and Its Management-A Review

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Abstract

Endocrine system is the important system of human body responsible for maintaining the homeostasis and some other physiological functions by releasing hormones from the endocrine gland. Parathyroid gland is one of the important endocrine glandproducing parathyroid hormone. Parathyroid hormone plays an important role in maintaining the calcium and phosphorous metabolism and has a direct influence over the mineralization of bone and teeth. Disorders in parathyroid gland such as hyperparathyroidism and hypoparathyroidism causes altered levels of secretion of parathormone which may cause various symptoms including the oralmanifestations such as brown tumor, loss of bone density, increased caries occurrence rate, high risk of bone fracture etc. The aim of this article is to creating the awareness among the dental professionals about the risk and preventive measures in treating the patients with hyperparathyroidism.

Key Words: *Parathyroid hormone, hypercalcemia, Brown tumor, chronic renal failure, bone fracture.*

Introduction

Parathyroid gland is one of the endocrine gland which produces parathyroid hormone (PTH). Parathyroid gland consists of two pairs of four glands which are located in the back side of the thyroid gland, two numbers in one side and two on the other side. Parathyroid hormone functions to maintain the homeostasis of calcium and phosphorous metabolism. Hence it plays an important role in mineralization of bone and teeth, regulates bone resorption, activate synthesis of active form of vitamin D in kidneys which makes the intestinal epithelium to absorb the calcium more into the blood [1].

Disordersofparathyroidincludehyperparathyroidism and hypoparathyroidism. Hyperparathyroidism (HPT) was first described by Von Recklinghausen in the year 1891. Its normal prevalence rate is 0.05-0.1% of the common population [1]. It is more common in middle aged female than male. Hyperparathyroidism (HPT) is the abnormal production of parathyroid hormone which causes unusual effects such as hypercalcemia and increased osteoclastic resorption. Hypoparathyroidism is the decreased or no production PTH which results in symptoms such as dental abnormalities, paraesthesia of tongue etc.

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HYPERPARATHYROIDISM:

Hyperparathyroidism is characterized by oversecretion of PTH. HPT is divided into three categories[2-5]:

1.Primary hyperparathyroidism

2.Secondary hyperparathyroidism

3.Tertiary hyperparathyroidism

Primaryhyperparathyroidism:Normally brought about by a tumor (adenoma in 85% of all cases) or hyperplasia of the organ that creates an increased activityof PTH discharge bringing about hypercalcemia and hypophosphatemia.

Secondaryhyperparathyroidism:At the point when the parathyroid organs are stimulated to secreteexpanded amount of hormones to equalise abnormal low serum calcium levels in various physiologic or pathologic conditions like renal dysfunction, intestinal malabsorption condition, decreased Vitamin D synthesis, in this manner coming about in parathyroid hyperplasia.

Tertiary hyperparathyroidism:At the point when long-standing secondary hyperplasia becomes self-ruling despite rectification of the underlying stimulant(renal transplant)[2].

SIGNS AND SYMPTOMS:

In Primary HPT, about half of patients have no indications and the issue is gotten as a coincidental finding (through raised calcium or characteristics X ray appearances [subperiosteal resorption of the phalanges of the index and middle fingers]). Numerous different patients just have vague symptoms.[3] These are musculoskeletal issues (weakness, back pain, muscle soreness), gastrointestinal disorders, e.g., vomiting, nausea, constipation, loss of appetite.

One of the principle clinical appearances of HPT is bone sickness. The ribs, clavicles, pelvic support and mandible are the bones generally included. A pathologic fracture might be the primary indication of the infection. Bone tenderness and joint firmness may be present. Renal calculi are a typical finding in this condition. Practically all patients with HPT have skeletal disorders in the serious stages. The loss of calcium in this condition brings about summed up osteoporosis.

ORAL MANIFESTATIONS:

In the oral cavity, the most well-known clinical signs of HPT isbrowntumor; loss of bone thickness, mobile teeth, floating of teeth, unclear jaw bone pain, sensitive teeth during occlusion and percussion, soft tissue calcifications and dental defects, for example, developmental defects and changes in dental eruption[3,6-10]. Malocclusion because of floating of teeth, with definite spacing of the teeth might be one of the principal indications of the disorder. Pseudocysticlesions can also present, radiolucentlesion at the apex of the tooth misdiagnosed as periapical cystor granuloma[11,12]. It doesn't appreciably affect periodontal records and radiographic bone height[13].

Chronic renal failure and secondary HPT additionally show infrequently, as maxillary hyperplasia and hyperostosis cranialis, progressive augmentation of the facial bones over a period. These facial amplifications cause an unusual facial deformation and dental malocclusion[14-17]. Primary HPT likewise seldom presents in the mandible as an enormous exophytic mass or painless swelling[18], and furthermore as a giant cell epulis as an underlying element of primary HPT[19]. Primary HPT patients were bound to have tori[20].

BROWN TUMOR:

Browntumorlesions[17,21-23] might be the soonest appearance of this undiscovered HPT in 6% of the cases. Browntumor presents as osteolytic injury (which might be related withpain and swelling) that creates because of changesin bone metabolism brought about by high serum level of PTH. It is an erosive bony lesions brought about by quick osteolysis and peritrabecular fibrosis bringing about a neighbourhood destructive process.

Brown tumorpresents itself as a friable red-brown mass. Its name is because of shading that it takes from the haemorrhagic infiltrates and hemosiderin stores that are frequently found inside. Bone injury may create huge cortical development.Mandible contribution is normal, particularly in the region of premolars and molars, and it is uncommon in the maxilla. It happens mostly in secondary HPT patients with chronic renal failure or renal osteodystrophy[24], yet it has additionally been depicted as a uncommon indication of calcium malabsorption, a few types of osteomalacia and of primary HPT[25,26].

Radiographically, lesions are portrayed by all around characterized unilocularor multilocular radiolucent regions. The trademark radiographicfindings show anwidespread loss of the lamina dura and changes in thetrabecular bone pattern of the jaws. Long-standing injury regularly creates a vital extension of cortical bone, root resorption and displacement of roots.

Histologically, it is portrayed by aabundant stroma, comprising of packs of spindle or oval cells, and a few multinucleated osteoclast-like giant cells. Calcified material can be found, just as regions with

extravasations of red blood cells and pigmentation by hemosiderin.Differential diagnosis must be made with other lesions[24], for example, an aneurysmal bone sore, cherubism, centralgiant cell granuloma.Sagliker disorder is realized that skeletal changes due to secondary HPT can be serious in chronic kidney disease [27,28].

Hyperparathyroidism-jaw tumorsyndrome is an uncommon disease, different individuals from the family are likewise included, and there is danger of harmful illness. The Patient gave bilateral or recurrent mandibular radiolucenciesanalyzedhistopathologically as cemento-ossifying fibromas. It is expected to be due tomutation of gene[29].

RADIOGRAPHIC FINDINGS:

Oral Radiographs of hyperparathyroid patient shows [20,30-33] as loss of medullary trabecular example, jaw shows up finely radiopaque portrayed as clear "ground glass" appearance and almost as summed up rarefaction.

DIAGNOSIS:

Serum parathyroid hormone (normal value 15-65 pg/ml) and calcium (Normal value 9-11 mg/dl) :

When a raised PTH has been affirmed, objective of determination is to decide if the HPT is primary or secondary in beginning by acquiring a serum calcium level. High serum calcium level due show primary HPT. Though, low or ordinary calcium level may demonstrate secondary HPT. Tertiary HPT has a high PTH and high serum calcium. It is separated from primary HPT by a background marked by chronic kidney failure and secondary HPT.

Serum phosphate (Normal value: 2.4-5 mg/dl):

In Primary HPT, serum phosphate levels are very low because of diminished renal tubular phosphate reabsorption.Nonetheless, this is just present in about half of cases, as dietary admission likewise impacts phosphate level. This differences with secondary HPT, in which serum phosphate levels are large as a result of renal disease.

Alkaline phosphatase (Normal value: 500-750 IU/L):

Alkaline phosphatase levels are generally raised in HPT. In primary hyperthyroidism, levels may remain within normal, notwithstanding, this is "improperly typical" given the expanded degrees of plasma calcium.

DENTAL MANAGEMENT OF THE PATIENT WITH HYPERPARATHYROIDISM:

The dental management of these patients doesn't need any exceptional thought. We should realize that there is a higher danger of bone fracture, so we should avoid potential risk in surgical treatments. It is essential to perceive the presence of the brown tumor and to play out a right differential analysis so as not to direct a deficient treatment.

The treatment of HPT is the initial phase in the management of the brown tumor, as unconstrained relapse of the lesion regularly happens. In any case, a few instances of browntumor that didn't vanish or even developed after standardization of PTH level have been accounted for. In these cases, browntumor resection ought tobe the favourable treatment[2,3,34-36].

Jaw amplification is treated by recontouring of the maxilla and mandible. A three-dimensional recreation of the computed tomography (CT) filter was useful in assessing the facial deformations and in treatment planning[14].

A careful clinical and radiographic assessment is fundamental before giving endodontic treatment. The investigations or diagnostic procedures must start with an efficient survey of the clinical history. There are various systemic diseases that can cause bone injuries all through the body. In a few cases, these lesions show up in the periapical area of teeth and can prompt a misdiagnosis of an lesion of endodontic origin.[1,2,12] The finding of periapical radiolucency on a radiograph ought not consequently lead to access opening, root canal treatment by the dental specialist.

ROLE OF DENTIST:

The dentist assumes significant part in the diagnosis of HPT. Occasionally, the main indication of the illness might be a growth in the jaw. The disease ought to be considered by the dentist at whatever point single or multipleradiolucencies are seen on radiography of the jaw. Oral manifestations and dental radiographs of jaws are of incentive in early recognition of secondary HPT

because of renal osteodystrophy.

Concluision

For a dental health professional, having knowledge about the oral and systemic manifestations of parathyroid disorders and precautions need to be taken while treating the parathyroid disorder patients are of utmost important. The lack of knowledge for dentists about the parathyroid disorders results in the serious complication of the patients. The aim of this article is to review the facts about hyperparathyroidism their oral manifestations and management of those patients in dental office.

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