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Endocrine Disorders in Head Neck Region: A Radiographic Perspective in Dental Clinic

Diş Hekimliği Kliniğindeki Radyografik Bakış Açısıyla Baş Boyun Bölgesindeki Endokrin Bozukluklar

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ABSTRACT

Endocrine diseases may have manifestations locally or in a generalized manner. When an imbalance in normal bone homeostasis occurs, regulated by various hormones such as parathyroid hormone, growth hormone it results in conditions like hypercalcemia, hypophosphatemia etc. which may affect bone density in a generalized manner. The condition cause: excessive resorption of bone or excessive deposition of bone. Intraoral or extraoral radiographs are thus useful in detecting diseases like hyperparathyroidism, hyperpituitarism, hypoparathyroidism, and hypopituitarism which are subclinical and are detected accidently on routine radiographic examination.

Key words: Hyperparathyroidism, hypoparathyroidism, hypopituitarism, radiography.

ÖZET

Endokrin hastalıkları bölgesel veya genel belirtiler gösterebilirler. Normal bir kemikteki paratiroid hormonu ve büyüme hormonu gibi çeşitli hormonlar ile düzenlenen homeostazda herhangi bir dengesizlik meydana geldiği zaman hiperkalsemi ve hipofosfotemi gibi durumlar ortaya çıkar ki bu durumda kemik yoğunluğunun geneli etkilenir. Bu durumun ise; ya kemikte aşırı rezorpsiyona ya da depozisyona yol açar. Dolayısıyla, normal rutin radyograf taramalarda subklinik veya tesadüfi tespit edilen hiperparatiroidizm, hiperpituitarizm, hipoparatiroidizm, ve hipopituitarizm gibi hastalıkların belirlenmesinde intra veya ekstra oral radyograf yöntemi oldukça kullanışlıdır.

Anahtar kelimeler: Hiperparatirodizm, hipoparatirodizm, hipopituitarizm, radyografi.

Introduction

The endocrine system is responsible for hormonal secretion and is closely related to the central nervous system, as it diversifies its functions through the hypothalamus and pituitary. It controls physiological processes and maintains homeostasis. The neuroendocrine system plays a significant role for adaptation to environmental changes[1]. These changes are manifested in skull and jaws too. Many diseases affect bone in similar ways and create similar lesions, thus making it difficult to determine the exact disease represented. The development of the radiographic techniques has improved the assessment of radiological findings that systemic diseases produce to the skull and jaws[2]. Thus skull and jaw radiographs remain invaluable as a first step in the diagnosis of many endocrine disorders especially in hyperparathyroidism, hypoparathyroidism, hyperpituitarism and hypopituitarism[3].

Hyperparathyroidism

Hyperparathyroidism is a generalized disorder of calcium, phosphate and bone metabolism due to increase in parathyroid hormone secretion[4]. Hyperparathyroidism is characterized by hypersecretion of parathyroid hormone which occurs in three categories[1]:

Table.1. Stages of Chronic Renal Disease According to Glomerular Filtration Rate (GFR) [5].

Stage	GFR cc/min	Manifestation
1	>90n	Asymptomatic, may have haematuria or proteinuria
2	60-90	Asymptomatic, may have haematuria or proteinuria
3	30-60	May develop anaemia, Secondary hyperparathyroidism
4	15-30	Start to prepare for dialysis or transplantation
5	<15	Initiate dialysis (<15cc/min in diabetic and <10cc/min in non-diabetic patients)

Primary hyperparathyroidism which occurs with hyperfunction of one or more parathyroid glands, usually caused by adenoma in 85% of all cases or hyperplasia of the gland that produces an increase in parathyroid hormone secretion resulting in hypercalcemia and hypophosphatemia[1].

Secondary hyperparathyroidism which is normally related to patients with intestinal malabsorption syndrome or chronic renal failure, occurring in a decrease of vitamin D production or with hypocalcemia causing the glands to produce a high quantity of parathyroid

hormone. There is also hyperphosphatemia. The parathyroid hormone-related signs are brown tumors and osteitis fibrosa cystica, which is referred as renal osteodystrophy or Von Recklinghausen's disease[1]. (Table. 1][5]

Tertiary hyperparathyroidism is an uncommon condition, affecting up to 8% of patients with secondary hyperparathyroidism after a successful renal transplant. It occurs when the parathyroids activity becomes autonomous and excessive, leading to hypercalcemia[1].

The clinical finding of hyperparathyroidism has been described by Jackson and Frame in 1972 as the tetrad of 'bones, stones, abdominal groan and psychic moans with fatigue overtones'[4]. In their review of literature Terezhalmy and colleagues found that this condition occurs mostly in women, with a peak incidence between the ages of 40 and 50 years[6]. The primary involvement is in the kidneys and skeletal system and the 80% of the cases are asymptomatic. Stones refer to the increased deposition of calcium in renal parenchyma and a tendency to develop recurrent nephrolithiasis (renal calculi) with resulting complications like urinary tract obstruction, infection, loss of renal function and uremia. Metastatic calcifications are also seen in blood vessel walls, subcutaneous soft tissues, dura and the region around the joints. Band keratopathy, where calcification occurs as a narrow band at the limbic margin of the cornea of the eye is also seen[7].

Bones refer to the distinctive involvement of the bone. Osteitis fibrosa cystica, bone lesions which on histopathological examination show multinucleated giant osteoclasts in scalloped areas of the bone surface (Howship's lacunae) and replacement of normal cellular and marrow elements with fibrous tissue. Abdominal groans refer to subtle vague gastrointestinal (GI) disturbances such as nausea, vomiting, anorexia, pancreatitis, duodenal and peptic ulcers.

Psychic moans may be due to central nervous system (CNS) manifestation ranging from mild personality problems to severe psychiatric disorders due to hypercalcemia[7]. Other manifestations include neuromuscular problems with proximal muscle weakness, easy fatigability and muscle atrophy which are differentiated from other neuromuscular disorders by regression after surgical removal of glands. One of the first signs is development of malocclusion because of drifting of teeth. Giant cell tumours and pseudocysts of the jaws are the other possible lesions found. Hyperparathyroidism leads to the preferential loss of cortical bone and preservation or increase in trabecular bone.

It has been proposed that the presence of hypercalcemia in hyperparathyroidism patients is preceded by longer periods of elevated parathyroid hormone levels. It could be that the tori represent an expansion of trabecular bone at the expense of cortical bone in response to elevated parathyroid hormone levels with possible contributions from the mechanical forces present in oral cavity. Furthermore, parathyroid hormone mediated endosteal resorption has been shown to be compensated by parathyroid hormone-mediated periosteal apposition with an increase in overall bone size[4].

Radiographic Features

In intraoral periapical radiographs, orthopantamograms, maxillary and mandibular true occlusal radiographs the density of the jaws is decreased, resulting in a radiolucent appearance that contrasts with the density of the teeth. The teeth stand out in contrast to the radiolucent jaws. A change in the normal trabecular pattern may occur, resulting in a 'Ground-glass appearance' of numerous, small, randomly oriented trabeculae. Demineralization and thinning of cortical boundaries often occur in jaws in cortical boundaries such as inferior border, mandibular canal. Another reliable change is subtle erosions of the bone from the subperiosteal surfaces of the mandibular angle.

Chondrocalcinosis or calcification of articular cart observed in hyperparathyroidism[8]. Intraoral periapical radiographs and paranasal sinus view shows thinning of cortical outlines of maxillary sinus and nasal cavities. Orthopantamograms, lateral oblique and postero-anterior views of body of mandible shows brown tumors of the jaws.

Brown tumours of hyperparathyroidism may appear in any bone but are frequently found in the facial bones and jaws, particularly in long-standing cases of disease. These lesions may multiple with in the single bone. They have variably defined margins and they may produce cortical expansion[8]. Depending on duration and severity of the disease, intraoral periapical radiographs shows loss of lamina dura around one tooth or all the remaining teeth due to increased uptake of calcium from bone as a result of reduction in vitamin D production. The loss may be either complete or partial around a particular tooth. The result of lamina dura loss may give the root a tapered appearance because of loss of image contrast. Submentovertex, postero-anterior skull and true lateral skull view, shows pepper-pot skull, the entire calvarium has a granular appearance caused by the loss of central (diploic) trabeculae and thinning of cortical tables[8].

Hypoparathyroidism

Hypoparathyroidism is an uncommon endocrine-deficiency disease characterized by low serum calcium levels, elevated serum phosphorus levels, and absent or inappropriately low levels of parathyroid hormone in the circulation[9] (Table 2)[10].

Dental manifestations comprises of enamel hypoplasia caused by insufficient quantities of vitamin D resulting from a lack of proper calcification of enamel matrix, widened pulp chambers, pulp stones, shortened roots, delayed eruption and hypodontia[11].

Table.2. Clinical Features of Hypocalcaemia [10].

Neuromuscular irritability
Paresthesias
Laryngospasm
Bronchospasm
Tetany
Seizures
Chvostek sign
Trousseau sign
Prolonged Q-T interval on Electocardiogram (ECG)

Radiographic Features

Postero-anterior skull radiograph shows calcification of basal ganglia, which appear flocculent and paired within cerebral hemisphere. Jaw radiographs may reveal enamel hypoplasia, external root resorption, root dilacerations, abnormal calcifications, blunting of molar roots and enlarged pulp chambers[12]. Hypoparathyroidism requires lifelong therapy with vitamin D or metabolites (Calcitriol 0.25 to 2.0 mg/d)[9, 13].

Hyperpituitarism

Hyperpituitarism results from hyperfunction of the anterior lobe of pituitary gland, which increases the production of growth hormone. An excess of growth hormone causes overgrowth of all tissues in the body still capable of growth. The usual cause of this problem is

a benign, functioning tumour of the acidophilic cells of the anterior pituitary gland. When it occurs in childhood it is termed "Gigantism", and in adults "Acromegaly" [8].

Acromegaly is characterized by progressive cosmetic disfigurement and systemic organ manifestations, including arthropathy, neuropathy and cardiomyopathy. Patients may exhibit coarsened facial features, exaggerated growth of the hands and feet, and soft tissue hypertrophy. Other characteristics may include hyperhydrosis, goiter, osteoarthritis, carpal tunnel syndrome, fatigue, visual abnormalities, increased number of skin tags, colon polyps, sleepapnea and somnolence, reproductive disorders, and cardiovascular disease (congestive heart failure, arrhythmia, and hypertension). Majority of patients manifest a combination of acral changes, arthralgia, increased sweating, and physical weakness[14]. From the dental perspective, these patients have mandibular prognathism as a result of increased growth of the mandible, which may cause apertognathia (anterior open bite). Growth of the jaws may cause spacing of the teeth, resulting in diastema formation. Soft tissue growth often produces uniform macroglossia in affected patients[7].

The cardinal clinical feature of gigantism is growth acceleration. Mild to moderate obesity is common and macrocephaly has been noted to precede linear and weight acceleration in at least one case. All have been noted to have coarse facial features and disproportionately large hands and feet with thick fingers and toes. Frontal bossing and prominent jaw have frequently been present. Organomegaly and deteriorating glucose tolerance were also documented in one patient observed over several years before treatment. Enlargement of facial features, excess acral growth and soft tissue swelling are essentially ubiquitous among these patients. Additional common manifestations include headaches, excessive sweating, peripheral neuropathy and arthritis. Frequently associated endocrinopathies include hypogonadism, diabetes, thyromegaly, and galactorrhea. Enlargement of mandible and macrodontia are the oral manifestations seen in these patients[15].

Radiographic Features

The pituitary tumor responsible for hyperpituitarism often produces enlargement (ballooning) of the sella turcica. Skull radiographs radiographs such as true lateral skull, lateral cephalogram, paranasal view, postero-anterior skull view characteristically reveal enlargement of the paranasal sinuses (especially frontal sinus) and increased pneumatisation of temporal bone. Diffuse thickening of outer table of skull can be seen. Jaw radiographs like

lateral oblique ramus, posterior body mandible, orthopantamograms reveal enlargement of the mandible, excess condylar growth and height of ascending ramus, increased angle between the ramus and body of the mandible with the loss of antegonial notch, spacing and flaring of anterior teeth with anterior open bite, class III skeletal relationship and increase in thickness and height of alveolar process. Intraoral periapical radiograph include hypercementosis and supraeruption of posterior teeth to compensate for the growth of mandible[8]. Ideally, the GH-producing adenoma should be removed completely, with subsequent restoration of pituitary function. Nonsurgical treatment options for acromegaly include medical therapy with somatostatin analogs or dopamine agonists and radiotherapy[5].

Hypopituitarism

Hypopituitarism is defined as the deficiency of one or more of the hormones secreted by the pituitary gland[16]. When it occurs before puberty it is called Dwarfism and when it occurs after puberty it is called Simmond's disease. Reduced energy and vitality, reduced muscle mass and strength, increased central adiposity, decreased sweating and impaired thermogenesis, increased cardiovascular risk, reduced bone mineral density[17]. The most striking feature of pituitary dwarfism is short stature of the affected patient. The maxilla and mandible of affected patients are smaller than the normal and the face appears smaller with the permanent teeth showing a delayed pattern of eruption. Often the shedding pattern of deciduous teeth is delayed by several years, and also the development of roots of permanent teeth appears to be delayed[18]. The dental arches are smaller than the normal and therefore cannot accommodate all the teeth resulting in dental malocclusion. The complete absence of buds of the wisdom tooth even in the patient in fourth decade of life is also reported. Other rare findings such as agenesis of the upper central incisor and solitary maxillary central incisor have been observed. Amelogenesis imperfecta a diverse group of hereditary disorder that is characterized by defect in formation of tooth enamel has also been seen in the patient with reduced amount of growth hormone. In adult hypopituitarism, changes in the head include thin eye brows, loss of eyelashes, sharp features, thin lips, and immobile expression. No specific dental changes have been described in this condition[18].

Radiographic Features

Lateral cephalogram, true lateral skull and postero-anterior view shows small dimension of skull and facial bones. Orthopantamograms, intraoral periapical radiographs shows retarded

tooth level, over retained deciduous teeth and impacted permanent teeth, complete absence of third molar tooth buds and short roots with open apices[19]. The treatment of hypopituitarism includes therapies directed at the underlying disease process, and endocrine replacement therapy[17].

Conclusion

As dentists we come across specific skull and jaw manifestations of endocrine diseases that are pictured in intraoral and extraoral radiographs which are useful in further verification of the disease process.

References

1. Fabue LC, Soriano YJ, Perez MGS. Dental management of patients with endocrine disorders. J Clin Exp Dent. 2010; 2:196-203.
2. Papagrigorakis MJ, Karamesinis KG, Daliouris KP, Kousoulis AA, Synodinos PN, Hatziantoniou MD. Paleopathological findings in radiographs of ancient and modern Greek skulls. Skeletal Radiol 2012; 41:1605–11.
3. Macaron C, Li YP, Hindo W. Skull X-ray in the evaluation of endocrine diseases. Am Fam Physician. 1978; 17:85-91.
4. Ongole R, Praveen B N. Textbook of Oral Medicine Oral Diagnosis and Oral Radiology. New Delhi, Elsevier India, 2010.
5. Lynch MA, Brightman VJ, Greenberg MS. Buket's Oral Medicine, 11th ed. Philadelphia, Lippincott, 2008.
6. Langlais RP, Langland OE, Nortje CJ. Diagnostic Imaging of the Jaws. Philadelphia, Williams&Wilkins, 1995.
7. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and Maxillofacial Pathology, 3rd ed. Philadelphia, WB Saunders, 2009.
8. White SC, Pharoah MJ. Oral Radiology Principles and Interpretation, 6th ed. Philadelphia, Mosby, 2011.
9. Bilezikian JP, Khan A, Potts JT Jr, Brandi ML, Clarke BL, Shoback D et al. Hypoparathyroidism in the adult: epidemiology, diagnosis, pathophysiology, target-organ involvement, treatment, and challenges for future research. J Bone Miner Res. 2011; 26:2317-37.
10. Rubin M R, Bilezikian J P. Hypoparathyroidism: clinical features, skeletal microstructure and parathyroid hormone replacement. Arq Bras Endocrinol Metab. 2010; 54: 220-6.

11. Nyamati SB, Venkatraman S, Nagaraju K, Annapoorna HB, Prakash S. Role of dental findings in the diagnosis of idiopathic hypoparathyroidism: case report. Journal of Indian Academy of Oral Medicine and Radiology. 2011; 23:462-4.
12. Worth HM. Principles and Practice of Oral Radiologic Interpretation. Chicago, Year Book Medical Publishers, 1963.
13. Schilling T, Ziegler R. Current therapy of hypoparathyroidism: a survey of German Endocrinology Centers. Exp Clin Endocrinol Diabetes.1997; 105: 237-41.
14. Melmed S, Jackson I, Kleinberg D, Klibanski A. Current treatment guidelines for acromegaly. J Clin Endocrinol Metab. 1998; 83:2646-52.
15. Eugster EA, Pescovitz OH. Gigantism. J Clin Endocrinol Metab. 1999; 84:4379-84.
16. Romero CJ, Nesi-Franca S, Radovick S. Molecular basis of hypopituitarism. Trends Endocrinol Metab. 2009; 20:506-16.
17. Prabhakar VKB, Shalet SM. Aetiology, diagnosis, and management of hypopituitarism in adult life. Postgrad Med J. 2006; 82:259–66.
18. Atreja G, Atreja SH, Jain N, Sukhija U. Oral manifestations in growth hormone disorders. Indian J Endocrinol Metab. 2012; 16: 381–3.
19. Karjodkar FR. Textbook of Dental and Maxillofacial Radiology, 2nd ed. New Delhi, Jaypee Brothers Medical Publishers, 2009.

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