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PREVENTION AND MANAGEMENT OF ENDOCRINE RELATED EMERGENCIES IN DENTAL OFFICE

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ABSTRACT

Life threatening emergencies may occur in the practice of dentistry. Although the occurrence of life threatening emergencies in dental offices is infrequent, a number of factors exist today that can increase the likelihood of such incidents. These includes

i. The increasing number of older persons seeking dental care.
ii. Medically compromised patients.
iii. Growing trend towards longer dental appointment.
iv. Increasing use and administration of drugs in dentistry.

Fortunately other factors exist to minimize the development of life threatening situations. These include *pre-treatment* physical evaluation of each patient which includes medical history of patient, physical examination and possible modifications in dental care to minimize medical risks. **Objectives:** The aim of this article is to focus the management of patients with endocrine disorders in dental practice. **Results and discussion:** The article describes the nature of endocrine disorders, their manifestations in oral cavity and the special dental management that should be carried out in these patients.

KEYWORD:- dentistry, pre-treatment, endocrine disorders.

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 is responsible for adaptation to environmental changes. Also, a function of the nervous system is to provide acorrect organic response. Its response may be primary, with the release of neurotransmitters, or if the stimulus prevails, the endocrine system secretes hormones. This is especially important in dentistry because many of the patients attending the dental clinics face stressful situations. Awareness is therefore necessary of the risks and difficulties that may arise during the dental management of patients with endocrine disorders.[1,2]

ENDOCRINE RELATED DISORDERS

1) Acute adrenal insufficiency
2) Diabetes Mellitus
3) Thyroid and parathyroid Gland Dysfunction

ACUTE ADRENAL INSUFFICIENCY

Adrenal gland is an endocrine gland composed of cortex and medulla. Adrenal cortex produces and secretes more than 30 steroid hormones, of which Cortisol is widely considered the most important. Cortisol helps the body adapt to stress.[3,4]

Hypersecretion of Cortisol leads to increased fat deposition in certain areas, such as lace and back, elevates blood pressure, and alters blood cell distribution. Clinically Cortisol hypersecretion is referred to as Cushing's syndrome, a condition usually corrected through surgical removal of part of adrenal gland.[3-5]

Addison's disease is a rareendocrine disorder in which the adrenal gland does not produce enough steroid hormones. It is also known as adrenocortical deficiency. Clinical manifestations of adrenocortical insufficiency usually do not develop until atleast 90% of the adrenal cortex is destroyed and several months pass before a diagnosis is made and therapy is instituted. During this time, patient is capable of maintaining levels of endogenous Cortisol adequate to meet the requirements of day today living, however, in stressful situations, adrenal cortex cannot produce the additional Cortisol required to adapt to stress.[3-5]

The administration of exogenous glucocorticoids to a patient with functional adrenal cortices may produce a second form of adrenocortical hypofunction. Glucocorticosteroid drugs are widely prescribed for symptomatic relief of a wide variety of disorders. When used in this manner, exogenous glucocorticosteroid administration produces a disease atrophy of adrenal cortex, decreasing the ability of adrenal cortex to increase corticosteroids levels in response to stressful situations. This in turn leads to development of acute adrenal insufficiency.[3-5]

Prevention: Acute adrenal insufficiency can best be managed through its prevention, which is based on medical history. Medical history should include details of drugs taken by patient during past 2 years. If patient gives a history of glucocorticoid drugs then the dosage and route by which drug taken is important and also duration of therapy. Although exact length of time required for development of significant cortical suppression varies from patient to patient, it has been demonstrated net uninterrupted glucocorticosteroid therapy for as little as 2 weeks may induce suppression. The length of time required for full regeneration of normal cortical Junction varies according to the dosage and length of therapy but is normally atleast 9-12 months.[3-5]

Dental therapy considerations

1) **Addison's disease**: Patients who are currently receiving glucocorticoid therapy or recently have received such therapy and meet thecriteria of rule of two may required dental treatment modifications.

Rule of two: Adrenocortical suppression should be suspected if patient has received glucocorticosteroid therapy through two of the following methods.

1) In a dose of 20 mg and more of cortisone or its equivalent

2) Via oral or parenteral route for a continuous period of 2 weeks orlonger. Within 2 years of dental therapy.

A patient with Addison's disease or a patient receiving long term pharmacologic doses of glucocorticosteroid therapy is defined asASA II or III risk. Since patients with adrenocortical insufficiency are less able to adapt tostress they require administration of glucocorticosteroid before toincrease blood steroids levels. In an extremely fearful patient with Addison's disease, the patient may be hospitalized and receive 200-500 mg cortisone / day which is equivalent to maximal response of HPA system to extreme stress. In case of moderately

fearful patients, usually two fold or four fold increase in glucocorticosteroid dosage on the day of dental treatment prepares the patient adequately.[3,5,6]

2) Cushing's syndrome:It most commonly arises due to administration of exogenous glucocorticoids. The second most common cause is a high production of ACTH as a result of a pituitary corticotroph adenoma or less frequently by an extrapituitarytumor or very rarely by a tumor secreting CRH. Hypertension, glucose intolerance, menstrual irregularity, osteoporosis and pathological fractures, delayed healing, increased risk of infection and neuropsychological disturbances including depression, emotional irritability are observed. Purple striae and muscular atrophy are particularly positive in adults, whereas in child growth retardation is frequently present. Dental management in these patients consists in prevention of infections, hypertension and pathological fractures during surgical treatment.[4]

Management: Prevention is the best management approach for Addison's crises. We should perform a detailed clinical history and a consultation with the specialist. However if crises takes place during procedure then dental treatment should be interrupted immediately. Individuals should be placed in supine position with legs slightly elevated. Vital signs should be monitored. Summon for medical assistance. If patient is known to suffer from chronic adrenal insufficiency then 100 mg hydrocortisone sodium succinate is administered IV/IM every 6-8hours. However, if patient does not have a prior history of adrenal insufficiency and immediate diagnosis of acute adrenal Insufficiency is empiric, it is recommended that corticosteroids therapy initiated immediately with4mg dexamethasone phosphate administered IV every 6-8 hours.[2-4,6]

DIABETES MELLITUS

It represents a syndrome of disordered glucose metabolism and inappropriate hyperglycemia that results from an absolute deficiency in insulin secretion, a reduction in the biologic effectiveness of insulin or both.[2,6,7]

Type1 - Insulin dependent diabetes mellitus (IDDM): This is a more severeform. In type I, circulating insulin is essentially absent, plasma glucagon levels are elevated and pancreatic alpha-cells do not respond, to insulinogenic stimuli.[3,7]

Type2 - Non insulin dependent diabetes mellitus (NIDDM): It is the milder forms of diabetes that occurs most frequently in adults and only occasionally in children. Most type II doesn't require exogenous insulin therapy to sustain life.[3,7]

Hyperglycemia: It is the condition in which excessive amount of glucose circulates in blood plasma.Any of the following factors which increase the body's demand for insulin may precipitate hyperglycemia:-Weight gain, Pregnancy, Hyperthyroidism, Corticosteroid therapy, Acute infection.[3,7]

Hyperglycemia usually by itself does not lead to an acute, life threatening emergency. If untreated, it progresses to diabetes ketoacidosis and subsequently diabetic coma both of which are life threatening. Diabetic ketoacidosis most often occurs in type I diabetes and is associated with inadequate administration of insulin.[3,7]

Hypoglycemia: Unlike hyperglycemia, this condition may develop rapidly, especially in patient receiving injectable insulin therapy who may lose consciousness within minutes after insulin administration patients receiving oral hypoglycemic agents; the onset of symptoms is slower. Common causes of hypoglycemia include omission or delay of meals, excessive exercise before meals and insulin over dose. Dental treatment is a potential threat to diabetics and to control of disease state. Stress physiologic and psychologic increases the body's requirement for insulin, which increases the chance for developing hyperglycemia. Prolonged anesthesia and extensive dental procedures, may delay patients next meal, increasing the risk of hypoglycemia.[2,3,7]

Prevention: An adequate preliminary patient evaluation can help to overcome acute complication of diabetes. After completing the medical history questionnaire, diabetic patient should be evaluated carefully for sign and symptoms of secondary disease, particularly of the cardiovascular system. Hyperglycemia patients may appear flushed and their skin dry; whereas hypoglycemia patients appear wet and cold and clammy. Patients with diabetic ketoacidosis exhibit characteristic smell of acetone on the breath.[3,7]

Dental therapy considerations: If doubts exist about patient medical status, consultation with physician is warranted. Type II diabetic is less prone to acute fluctuation in blood glucose levels and mostly can tolerate all forms of dental treatment, including general anesthesia and local anesthesia without increased concern. In case of type I diabetic individual should be advised be maintain normal dietary habits by taking usual insulin dose and eating normal breakfast before a dental appointment. Scheduling appointments earlier in day helps minimize episodes of hypoglycemia.[2,3,7]

Management: Prompt recognition of diabetes related complication is vital. Equally important is the doctors' ability to differentiate between hyperglycemia and hypoglycemia. Hyperglycemia and ketoacidosis usually develop over a period of many hours or days and the diabetic appears chronically ill. Another important factor in differential diagnosis is hot, dry appearance seen in hyperglycemia with contrast to the cold, wet look of hypoglycemia. When doubt remains as to cause of condition, supportive therapy is indicated until additional medical assistance became available.[2,3,7]

Definitive management of hyperglycemia, ketosis and acidosis consists of administration of insulin to normalize the body's metabolism and restoration of fluid and electrolyte deficiencies. Definitive management of hypoglycemia consist oral carbohydrate in case of conscious patient. Sugar should be dissolved in water and ingested by the patient. In case of unconscious patient administration of parenteral carbohydrates is necessary. 1 mg glucagon maybe administered IM or IV or 50 ml of 50% dextrose solution may be administered IV over 2-3 minutes.[2,3,7]

Patient usually responds with 10-15 minutes after IM-injection of glucagon and within 5 minutes following 50% dextrose IV. Then oral carbohydrates are started as soon as they are tolerated by the patient. If parenteral route is not possible then a thick paste of concentrated glucose may be used or small amount of honey may be placed in the patient a buccal fold. Recovery is slow, usually takes 30-40 minutes.[7]

THYROID GLAND DYSFUNCTION

The thyroid gland secretes three hormones: thyroxine (T4), tri-iodothyronine (T3) and calcitonin. T4 and T3 are hormones that affect metabolic processes throughout the body and are involved in oxygen use. Thyroid stimulating hormone (TSH or thyrotropin), produced by the pituitary gland, regulates the secretion of thyroid hormones (T4 and T3) through a negative feedback mechanism. Calcitonin is involved, with parathyroid hormone and vitamin D, in regulating serum calcium and phosphorus levels and in the skeletal remodelling. Thyroid hormones influence the growth and maturation of tissues, energy metabolism and turnover of both cells and nutrients.[2,3,8]

Hyperthyroidism: This condition arises due to over production of thyroid hormone. It is caused by ectopic thyroid tissue, toxic thyroid adenoma, toxic multinodulargoiter, subacute thyroiditis, and Graves' disease. Clinical manifestations include weight loss, increased

appetite, thin andbrittle hair, warm and moist skin, increased skin pigmentation, heat intolerance. palmar erythema, fine tremor, sweating and clubbing, fatigue, weakness, tachycardia, palpitations, systolic hypertension ,impaired concentration and reduced stress threshold, bilateral exophthalmos, ptosis and periorbital oedema.[2,3,9,10]

Oral manifestations includes accelerated dental eruption in children, Maxillary or mandibular osteoporosis, Enlargement of extra glandular thyroid tissue(mainly in the lateral posterior tongue),Increased susceptibility to caries, Periodontal disease, Burning mouth syndrome, Development of connective-tissue diseases like, Sjogren's syndrome or systemic lupus erythematosus.[2,3,9,11]

Dental therapy considerations

1. In controlled patients, same dental management should be carried out as in healthy patients. We must avoidsevere stress situations and the spread of infectious.[2,3,11]

2. In uncontrolled cases, use of epinephrine or other amines in local anaesthetics and the retraction cords should be avoided because the myocardium of these patients is sensitive to adrenaline and may cause arrhythmias, palpitations and chest pain. If an emergency surgical procedure is required, consultation with the patient's endocrinologist is advisable. Conservative treatment is often preferable. Treatment should be discontinued if signs orsymptoms of a thyrotoxic crisis develop, and accessto emergency medical services should be available. These symptoms include tachycardia, irregular pulse, sweating, hypertension, tremor, nausea, vomiting, abdominal pain and coma.[2,3,11]

3. People who have hyperthyroidism and are treated with propylthiouracil must be monitored for possibleagranulocytosis, hypoproteinemia or bleeding, and a complete blood count including prothrombin time before performing any invasive procedures is usuallyrecommended.[2,3,11]

Hypothyroidism: In this condition there is deficiency of the thyroid hormone. It can be acquired or by congenital defects. When it is present in infancy, it is manifested as cretinism and if it occurs in adults, it is known as myxedema. It is caused by severe iron deficiency, chronic thyroiditis (Hashimoto's disease), lack of stimulation, radioactive iodine that causes follicle destruction, surgery and pharmacological agents such as lithium and amiodarone. This condition can be classified into two categories: primary hypothyroidism, in which the defect is intrathyroid; or secondary hypothyroidism, in which other pathologies can cause an

indirect decrease of circulating hormone. Characteristic signs of cretinism include mental retardation, developmental and growth delay, marked disproportion between the head and body, lack of muscle tone, overweight, less expressive face with a broad and flat nose, hypertelorism, short neck and thick, pale, dry and wrinkled skin. Myxedema is characterized by widespread metabolic slow-down, depression, overweight, diminished cardiac output and respiratory rate, decreased pulse, generalized edema, hoarseness because the edema affects to vocal cords, sinus bradycardia, scalp brittleness, thin or absent eyebrows and decreased sweating. Generalizededema can affect the tongue causing difficulty speaking and swallowing and serrated tongue.[8-9,12,13]

Oral manifestations include delayed tooth eruption, enamel hypoplasia in both dentitions, being less intense in the permanent dentition, micrognathia, open bite due to lack of condylar and mandibular growth, macroglossia, thick lips, and mouth breathing.[9-12]

Dental therapy considerations: In hypothyroidism, there is no heightened susceptibility to infection. Owever They are susceptible to cardiovascular disease from arteriosclerosis and elevated LDL. Before treating such patients, consult with their primary careproviders who can provide information on their cardiovascular statuses. Patients who have atrial fibrillation can be on anticoagulation therapy and might require antibiotic prophylaxis before invasive procedures, depending on the severity of the arrythmia. If valvular pathology is present, the need for antibiotic prophylaxis must be assessed. Drug interactions of l-thyroxine include increase dmetabolism due to phenytoin, rifampin and carbamazepine, as well as impaired absorption with iron sulfate, sucralfate and aluminum hydroxide. When l-thyroxine is used, it increases the effects of warfarin sodium and, because of its gluconeogenic effects, the use of oral hypoglycemicagents must be increased. Concomitant use of tricyclic antidepressants elevates l-thyroxine levels. Appropriate coagulation tests should be available when the patient is taking an oral anticoagulant and thyroid hormone replacement therapy. Patients who have hypothyroidism are sensitive to central nervous system depressants and barbiturates, so these medications should be usedsparingly. In a literature review, the effects of epinephrine in patientswho have hypothyroidism was examined. No significant interaction was observed in controlled patients who had minimal cardiovascular involvement. In patients who have cardiovascular disease or who have uncertain control, local anesthetic and retraction cord with epinephrine should be usedcautiously. People who are on a stable dosage of hormone

replacement for a long time should have no problem withstanding routine dental treatment.[2,3,8-13]

PARATHYROID GLAND DYSFUNCTION

Parathyroid glands secret parathyroid hormone (PTH) involved in regulating the metabolism of calcium and phosphorus. PTH plays an important role in tooth development and bone mineralization and increases bone resorption. In the kidneys, it stimulates formation of active metabolite of vitamin D, which promotes the intestinal absorption of calcium and decreases renal reabsorptionofphosphate. [2,3,14]

Hyperparathyroidism: Hyperparathyroidism is characterized by hypersecretion of parathyroid hormone which occurs in three categories.[2,3,14]

1) Primary occurs with a hyperfunction of one or moreparathyroids, usually caused by a tumour or hyperplasia of the gland that producesan increase in PTH secretion resulting in hypercalcemiaand hypophosthamia.

2) Secondary normally related to patients with intestinalmalabsorption syndrome or chronic renal failure, occurringin a decrease of vitamin D production or withhypocalcemia causing the glands to produce a highquantity of PTH.

3) Tertiary is an uncommon condition, affecting up to 8% of patients with secondary HPT after a successful renal transplant. It occurs when the parathyroids activitybecomes autonomous and excessive, leading to Hypercalcemia.

One of the main clinical manifestations of hyperparathyroidismis bone disease. The ribs, clavicles, pelvicgirdle and mandible are the bones most involved. In the oral cavity, the most common clinical manifestations of HPT are brown tumor, loss of bone density, weak teeth, malocclusions, soft tissue calcifications and dental abnormalities such development defects,alterations in dental eruption and widened pulp chambers. Brown tumor presents itself as a friable red-brownmass. Its name is due to color that it takes from thehaemorrhagic infiltrates and haemosiderin deposits that are often found inside. Brown tumor presents asosteolytic lesion that develops due to changes in bonemetabolism caused by high serum concentration of PTH. Mandible involvement is common, especially in the area of premolars and molars, and it is rare in maxilla. Spontaneous regression of the lesion often occurs. However, several cases of brown tumor that did not disappear or even grew after

normalization of HPT level have been reported. In these cases brown tumor resection should be the preferred treatment.[2,3,14]

Dental therapy considerations: The clinical management of these patients does not requireany special consideration. However there is a higher risk of bone fracture, so we must take precaution in surgical treatments. On the other hand, It is important to recognize the presence of brown tumour and to perform a correct differential diagnosis so as not to conduct an inadequate treatment.[2,3,14]

Hypoparathyroidism: Hypoparathyroidism is a metabolic disorder characterized by hypocalcemia and hypophosphatemia due to adeficiency or absence of parathyroid hormone secretion. Hypoparathyroidism can cause hypocalcemia with consequent paraesthesia's, tetany and seizures. Clinical manifestations include alopecia, scaling of the skin, deformities of the nails and dental abnormalities such as enamel hypoplasia, poorly calcified dentin, widened pulp chambers, dental pulp calcifications, shortened roots, hypodontia and mandibular tori as PTH affects rate of eruption, formation of the matrix and calcification.[2,3,14]

Dental therapy considerations: These patients have more susceptibility to caries because of dental anomalies. Dental management will be the prevention of caries with periodic reviews, advice regarding diet and oral hygiene instructions. Before performing dental treatment, serum calcium levels should be determined. They must be above 8mg/100ml to prevent cardiac arrhythmias, seizures, or bronchospasms.[3,8]

CONCLUSION

Patients with an endocrine disease present a treatment challenge to dentists. Awareness of the condition and current stage of treatment is important in understanding the possible modifications needed for dental treatment. Length and current state of therapy are important in understanding the metabolic control of patient. Consultation with the patient's primary care physician or an endocrinologist is warranted if any sign or symptom of disease is noted on examination. Stress reduction, awareness of drug side effects or interactions, and vigilance for appearance of signs or symptoms of hormone toxicity are among the responsibilities of the oral health care provider.

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