Oral Manifestations of Hypothyroidism

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ABSTRACT
The thyroid is a bilobular glandular structure which acts as a major regulator of metabolism regulating all the bodily functions. The oral cavity is prone to fluctuations in the hormone produced by the thyroid. The oral findings and its complications should be understood by the dentist and endocrinologist before a treatment is initiated. Patients with thyroid dysfunction also need proper risk management before certain dental procedures are performed to avoid medical emergencies. This review focuses on the pathophysiology of hypothyroidism and its implication in oral health.

Keywords: endocrine, dysfunction, thyroid.

INTRODUCTION
The thyroid is the principal endocrine gland that regulates body functions, growth and metabolism. Amongst all the endocrine disorders, thyroid diseases are the most common glandular disorder. Various studies have estimated that around 42 million people suffer from thyroid diseases in India 1 with a significantly higher prevalence in women.2 Most cases of thyroid dysfunction go undiagnosed, which indicates that routine dental treatment can lead to adverse effects in the patient.3 Oral health care professionals need to familiarize with the oral manifestations that they may come across in practice, for early diagnosis and control of disease. Modifications in dental treatment and consultation with the patient’s primary care physician are necessary when a thyroid disease is suspected. This review will focus on the oral manifestations of hypothyroidism and the dental management for the same.

Anatomy and Pathophysiology: The thyroid gland is a bilobular structure composed of a right and a left lobe, connected by an isthmus. The main arteries supplying the gland are the superior thyroid, middle and inferior thyroid arteries and a smaller thyroid ima artery. The gland produces two important hormones, triiodothyronine or T3 and thyroxine or T4, from a basic protein thyroglobulin, which is present in the thyroid follicles. Iodine is also essential for the formation of these hormones. Thyroid hormone production is controlled by feedback mechanisms, and a deficiency of T3 or T4 can adversely affect growth and development of the infant and will decrease metabolic functions in adults. An excess or over production can lead to serious and life threatening complications if not discovered and treated on time.3 Hypothyroidism: Hypothyroidism is a condition wherein thyroid gland function and production of the thyroid hormones is reduced. The causes of hypothyroidism can range from severe iron deficiency anemia, insufficient stimulation of the gland, inflammatory (commonly Hashimoto’s thyroiditis), radioactive iodine, surgery, pregnancy (postpartum thyroiditis) and use of pharmacological agents such as lithium and amiodarone.5-6 Hypothyroidism can be primary, where the defect is intra-thyroid, or secondary, where lowering of hormone levels are due to other causes such as surgery. Of all the forms of hypothyroidism, the congenital form of hypothyroidism is the most important as it requires early diagnosis and treatment to prevent brain damage.1 studies have shown that in comparison to the world wide value of 1 in 3800, congenital hypothyroidism in much more common in India, with 1 in 2640 neonates affected.7 This can occur due to dysgenesis, agenesis of the gland, inborn defect in hormone production or defects in the pituitary or hypothalamic metabolism.

In cases of acquired hypothyroidism, it can be childhood hypothyroidism (cretinism), idiopathic (no known cause can be established), iatrogenic (due to radiation therapy or surgical procedures), autoimmune -Hashimoto’s thyroiditis, or endemic to certain areas or populations associated with a high-iodine diet.3 Clinical Manifestations: The features of hypothyroidism include anemia, cardiomegaly, intolerance to cold, constipation, dry and brittle hair, elevation in aspartate transaminase, alanine transaminase and lactate dehydrogenase, elevation in creatine, goiter, hyperlipidemia, lethargy, weight gain, tachycardia, seizures, reduce in cardiac output, reduced respiratory rate, myxedema, paraesthesia.5,8 Oral Manifestations: Childhood hypothyroidism known as cretinism is characterized by thickening of lips, large protruding tongue (macroglossia), malocclusion and delayed eruption of teeth. Thickening of the lips and macroglossia is due to increased accumulation of subcutaneous mucopolysaccharides i.e., glycosaminoglycans due to decrease in the degradation of these substances.
Table 1: Oral Manifestations of Hypothyroidism

<table>
<thead>
<tr>
<th>Oral Manifestations of hypothyroidism</th>
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<tbody>
<tr>
<td>Enamel hypoplasia in both dentitions</td>
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<tr>
<td>Anterior open bite</td>
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<tr>
<td>Macroglossia</td>
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<td>Micrognathia</td>
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<td>Periodontal disease</td>
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<td>Thick lips</td>
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<td>Dysgeusia</td>
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<td>Mouth breathing</td>
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<td>Delayed eruption of teeth</td>
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The long-term effects of severe hypothyroidism on craniofacial growth and dental development have also included impaction of the mandibular second molars. This seems to be caused by a dissociation of ramus growth and failure of normal resorption of the internal aspect of the ramus, resulting in insufficient space for proper eruption of these teeth. The other oral findings in hypothyroidism include dysgeusia, poor periodontal health, delayed wound healing, enamel hypoplasia, anterior open bite.

Dental Management of patients with Hypothyroidism: The dentist should be familiar with the systemic manifestations so as to avoid complications during treatment. Before initiating treatment for a patient, complete medical history with past medical complications should be obtained. In patients with controlled hypothyroidism, dental treatment like restorations can be done but dental surgical procedures should be avoided in the patients with infection or severe stress. Although patients with hypothyroidism do not have any increased susceptibility to infection, They are susceptible to cardiovascular disease from arteriosclerosis and elevated LD levels. Before treating such patients, a medical evaluation of their cardiovascular statuses is indicated. Patients who have atrial fibrillation may be on anticoagulation therapy and might require antibiotic prophylaxis before invasive procedures, depending on the severity of the arrhythmia. If valvular pathology is present, the need for antibiotic prophylaxis must be assessed.

It is important to highlight the possibility of an iatrogenic hyperthyroid state caused by hormone replacement therapy used to treat hypothyroidism, as in the case of Hashimoto’s thyroiditis, the patients who have Diabetes Mellitus (DM) become hyperglycemic when treated with T4. When providing dental care to patients who have DM, attention should focus on complications associated with poor glycemic control. Another aspect that requires attention is the lethargy in hypothyroid patients, which can lead to possible aspiration of dental materials or lowering of respiratory rate when in the dental chair. As shown by Johnson et al., epinephrine usage does not affect patients with controlled hypothyroidism and those with minimal cardiovascular involvement. In those with uncontrolled disease or any cardiovascular involvement, epinephrine and retraction cord should be used cautiously. Achieving hemostasis is not a concern unless the patient is on any anticoagulation therapy. Patients with long standing hypothyroidism may have increased subcutaneous mucopolysaccharides which may decrease the ability of small blood vessels to constrict when cut and may result in increased bleeding from infiltrated tissues, including mucosa and skin. Local pressure for an extended time will help control the bleeding from the small vessels.

Drug Interactions: Hypothyroid patents are sensitive to central nervous system depressants and barbiturates; hence these medications should be used with caution. Patients on propylthiouracil treatment must be monitored for possible agranulocytosis, hypoproteinaemia or bleeding, and a complete blood count including prothrombin time must be performed before doing any invasive procedures. It has been found that recent exposure to a surgical antiseptic that includes iodine can increase the risk of thyroiditis or hypothyroidism. Patients with underlying thyroid antibodies and a tendency toward autoimmunity appear to be at more risk.

Drug interactions of l-thyroxine include increased metabolism due to phenytoin, rifampicin and carbamazepine, as well as impaired absorption with iron sulfate, sulrafate 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 hypoglycemic agents must be increased. Concomitant use of tricyclic antidepressants elevates l-thyroxine levels. The use of Narcotic drugs used for post-op pain management should be limited owing to heightened susceptibility to these agents.

The presence of oral infection, central nervous depressants and surgical procedures can precipitate a myxedematous coma. Myxedematous coma comprises of hypothermia, bradycardia, severe hypotension and epileptic seizure. If that occurs, dental treatment should be discontinued and emergency medical services should be provided.

**CONCLUSION**

In conclusion, the dental treatment modifications are indicated in patients reporting with hypothyroidism. A complete blood picture should be advised before start of dental surgery. In patients with cardiovascular disease, consultation with primary care physician and cardiologist is essential. Limit or avoid use of epinephrine, CNS depressant drugs. The risk of oral infection should be controlled and dentist should be aware of the drug interactions of thyroxine. Good glycemic control in patients with diabetes mellitus is required before initiating dental treatment. Treatment should not be continued if myxedematous coma develops. Thus, dentist play a vital role in screening dental patients with undiagnosed thyroid functions.

**REFERENCES**


