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Thyroid disorders. Part II: hypothyroidism and thyroiditis

James W. Little, DMD, MS, Minneapolis, MN
UNIVERSITY OF MINNESOTA

Part II of the series on thyroid disorders discusses hypothyroidism and thyroiditis that may be found in dental patients. An overview of the conditions is presented. Presenting signs and symptoms, laboratory tests used to diagnose hypothyroidism and thyroiditis, and their medical management is discussed. The dental management of patients with hypothyroidism is discussed in detail. The dentist by detecting the early signs and symptoms of hypothyroidism and thyroiditis can refer the patient for medical diagnosis and treatment and avoid potential complications of treating patients with uncontrolled disease. Patients with thyroiditis may have a short period of being hyperthyroid and it may be best to avoid routine dental treatment during that period. Patients with suppurative thyroiditis should not receive routine dental treatment during the acute stage of the disease. The end stage of Hashimoto's thyroiditis results in hypothyroidism. Central nervous system depressants, sedatives, or narcotic analgesics must be avoided in patients with severe hypothyroidism because significant respiratory depression may occur. In addition, myxedematous coma, particularly in elderly hypothyroid patients, can be precipitated by central nervous system depressants, infection, and possibly stressful dental procedures. In medically well-controlled patients the dental treatment plan is not affected and most dental procedures can be offered to these patients.
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The purpose of this paper (part II in the series on thyroid disorders) is to discuss the dental management of patients with hypothyroidism and thyroiditis. The dentist may detect early signs and symptoms of these thyroid disorders and refer the patient for medical evaluation and treatment. In some cases, this may be lifesaving, whereas in others the quality of life can be improved and complications of certain thyroid disorders avoided.[1]

The dentist by history and clinical examination may detect evidence that may be associated with one of these disorders. Patients found to have signs and symptoms of thyroid enlargement or dysfunction should be referred for diagnosis and possible treatment.[1-5]

HYPOTHYROIDISM

Enlargements of the thyroid gland, termed a *goiter,* can be diffuse, nodular, singular, functional, or nonfunctional.[5,7] Simple goiter accounts for about 75% of all thyroid swellings.[5] Hashimoto's thyroiditis leads to hypothyroidism.[5,7] In contrast, patients with subacute thyroiditis may develop a transient period of hyperthyroidism (Table I).[5,7,8] Hypothyroidism can occur as a congenital or acquired condition.[5,7] Worldwide, the most common thyroid disorder is iodine deficiency (diet-related) goiter. In some of these cases, hypothyroidism develops, and in some, hyperthyroidism results. This type of goiter is called *endemic* if more than 10% of a local population is affected.[11-14] Subclinical hypothyroidism is a common, well-defined condition that often progresses to overt disease.[15-17] In addition, concerns are evident that the subclinical states may contribute to hyperlipidemia and other complications.[6,18,19]

Incidence, Prevalence, and Demographics

Hypothyroidism in Great Britain occurs at a rate of 3 cases per 1000 women per year. The number of established cases was reported to be 14 per 1000 women. The number of established cases in men was 1 per 1000. The mean age at diagnosis was 57 years. About one third of all cases resulted from surgical or radiation treatment for hyperthyroidism.[20]

In the United States hypothyroidism occurs in about

Professor emeritus, University of Minnesota, Minneapolis, MN.
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Table I. Classification of hypothyroid disorders and thyroiditis[8-10]

Disorder	*Causes*
Hypothyroidism (cretinism, myxedema)	Primary
	Idiopathic (end-stage Hashimoto's disease)
	Iatrogenic (^{131}I, surgery, radiation)
	Agenesis or dyplasia
	Goitrous
	Hashimoto's thyroiditis
	Iodine deficiency
	Antithyroid agents
	Insufficient stimulation of the thyroid
	Secondary (pituitary)
	Isolated TSH deficiency
	TSH synthesis defect
	Defect in TSH receptor
Thyroiditis	Hashimoto's
	Subacute
	Pyogenic
	Chronic fibrosing (Riedel's)
	Chronic thyroiditis with transient thyrotoxicosis

TSH, thyroid-stimulating hormone.

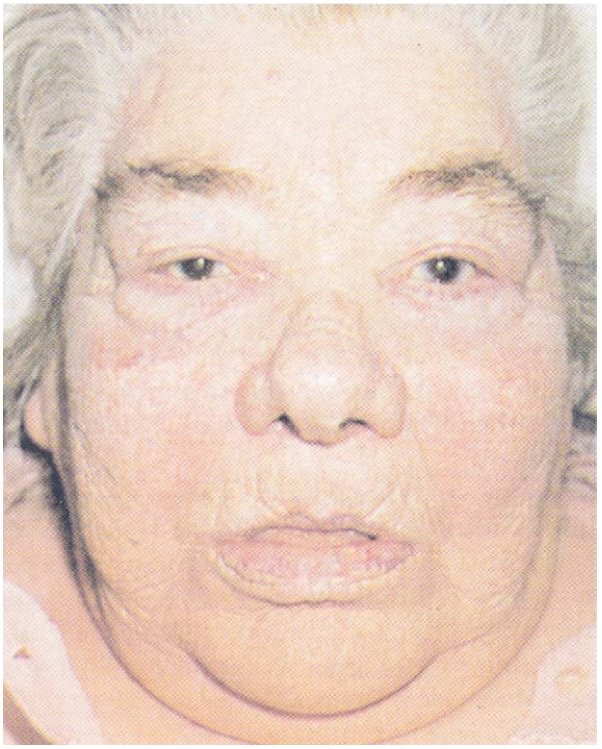

Fig 1. Gross clinical hypothyroidism showing nonpitting edema in the skin of the face. Note the dry, puffy facial appearance and the course hair. The patient had hypothermia, the skin was cold, and she showed mental apathy. (With permission from Forbes CD, Jackson WF. Color atlas and text of clinical medicine. 3rd ed. St Louis: Mosby; 2003. p. 311.)

1% to 2% of the general populaton.[9] It occurs in 3% to 4% of ill older patients admitted to the hospital. It is 5 to 6 times more common than hyperthyroidism. It is estimated that 10% of the women older than the age of 40 years have a thyroid hormone deficiency caused by autoimmune thyroid disease.[14] Both hypothyroidism and hyperthyroidism are 5 or more times common in women than in men in the United States.[20,21]

Acquired impairment of thyroid function affects about 2% of adult women and about 0.1% to 0.2% of adult men in North America. Neonatal screening programs in many areas of the world show that hypothyroidism is present in 1 of every 4000 newborns.[7,9] Permanent hypothyroidism also occurs about once in every 3500 to 4000 live births in the United States.[5,7,22] Transient hypothyroidism occurs in 1% to 2% of newborns.[7,9]

The incidence of hypothyroidism is 10 times higher than average in iodine-deficient areas.[9] The incidence also is increased in areas exposed to waterborne goitrogens or where there is excessive consumption of goitrogens such as cassava.[9] The incidence is increased in areas exposed to excessive radiation.[9]

Etiology and Pathogenesis

The causes of hypothyroidism (Table I) can be divided into 3 main categories: (1) primary, or permanent loss or atrophy of thyroid tissue; (2) goitrous hypothyroidism (hypothyroidism with compensatory thyroid enlargement due to impairment of hormone synthesis); and (3) insufficient stimulation of a normal gland (hypothalamic or pituitary disease or defects in the throid-stimulating hormone [TSH] molecule).[5,7,22] Primary and goitrous hypothyroidism account for 95% of all cases.[9]

Most infants with permanent congenital hypothyroidism have thyroid dysgenesis: ectopic, hypoplastic, or thyroid agenesis. The acquired form may follow thyroid gland or pituitary gland failure. Radiation of the thyroid gland (radioactive iodine), surgical removal, and excessive antithyroid drug therapy are responsible for the majority of these cases of hypothyroidism; however, some cases appear with no identifiable cause.[5,7]

Clinical Presentation

Neonatal cretinism is characterized by dwarfism; overweight; a broad, flat nose; wide-set eyes; thick lips; a large, protruding tongue; poor muscle tone; pale skin; stubby hands; retarded bone age; delayed eruption of teeth; malocclusions; a hoarse cry; an umbilical hernia; and mental retardation.[5,7] All of these characteristics can be avoided with early detection and treatment.[5,7]