Thyroid diseases

The thyroid gland is situated in the anterior portion of the neck just below and bilateral to the thyroid cartilage, it consists of two lateral lobes connected by an isthmus, in some individuals a superior portion of glandular tissue, or the pyramidal lobe, can be identified. Embedded in the thyroid gland are the parathyroid glands. It develops from the thyroglossal duct and portions of the ultimobranchial body, it originates from the posterior portion of the tongue and descends to its final location passing through the developing hyoid bone. Remnants of thyroid tissue may be found anywhere along the path of the thyroglossal duct and they may become cystic or neoplastic.

Function

The thyroid gland secretes 3 hormones:
1. T4 (Thyroxine), Tetraiodothyronine, it has a half life of 1 week and it is converted to
2. T3, Triiodothyronine, which is the active form and has a half life of 1 day.
3. Calcitonin, secreted from the medullary C cells, it is involved, along with parathyroid hormone and vitamin D, in regulating serum calcium and phosphorus levels and skeletal remodeling. The hormones influence the growth and maturation of tissues, total energy expenditure and act on metabolism by regulating protein synthesis.

Blood levels of T4 and T3, are controlled by a feedback mechanism mediated by hypothalamic-pituitary-thyroid axis. The hypothalamus releases thyrotropin releasing hormone (TRH) in response to external stimuli (stress, illness, metabolic demands, low levels of T4 and T3), TRH stimulates the pituitary gland to secrete thyroid stimulating hormone (TSH) which causes the thyroid gland to secrete T4 and T3. High levels of T3 and T4 turn off the release of TSH and low levels turn it on, 95% of the thyroid hormones are bound to plasma proteins, the most important are being thyroxine binding globulin (TBG).

Laboratory tests and imaging techniques

The mostly used include:
1. Radioactive Iodine uptake (RAIU), using $^{131}$I or $^{123}$I, the latter exposes the patient to a lower radiation dose. The uptake varies inversely with plasma iodide concentration and directly with the functional state of the gland. The normal value is 10%-30% RAIU and above this indicates hyperfunction.
2. Measuring serum T4 and T3. Elevated levels indicate hyperthyroidism while low levels indicate hypothyroidism.
3. Measurement of the basal serum TSH concentration, high level indicates hypothyroidism and low level indicate hyperthyroidism.
4. Thyroid scan using $^{123}$I injected and a scanner is used to localize areas of radioactive concentration. It is used to localized thyroid nodules and locate ectopic functional thyroid tissues.
5. Ultrasonography to detect the lesions, differentiate solid and cystic lesions and guide needle for aspiration of cyst or biopsy.
6. CT scan and MRI.

**Thyroid diseases**

Thyroid diseases are:
- Hyperthyroidism.
- Hypothyroidism.
- Thyroiditis.
- Thyroid neoplasm.

**Hyperthyroidism (Thyrotoxicosis)**

Excess of T4 and T3 in blood stream.
Causes:
- Thyroid diseases; like Graves’ disease, ectopic thyroid tissue, multinodular goiter, thyroid adenoma and thyroiditis.
- Ingestion of thyroid hormone or food containing thyroid hormones.
- Pituitary diseases involving the anterior lobe.

These conditions result in similar clinical manifestations.

**Clinical presentation**

**Skin:** is warm and moist, rosy complexion and the patient blushes easily, palmar erythema, profuse sweating, excessive melanin pigmentation of the skin, thin fine hair with areas of alopecia and soft nails.
Dermopathy; result from increased concentration of hyaluronic acid and chondrotin sulfate in the dermis in focal areas of the skin causing compression of dermal lymphatics and non-pitting edema.

**Cardiovascular:** palpitation, tachycardia, arrhythmia, angina, myocardial infarction and congestive heart failure. These are caused by increased metabolic activity caused by excessive hormone secretion which leads to increased circulatory demand resulting in increased stroke volume and heart rate.
Thyroid hormones have a profound effect on the sensitivity of tissues to catecholamines like epinephrine and these agents must not be administered to them.
Gastrointestinal; there is weight loss even with increased appetite, anorexia, nausea and vomiting are rare, many patients have Achlorhydria and about 3% develop pernicious anemia.

Central nervous system; nervousness, anxiety, sleep disturbance, emotional lability, impaired concentration, fatigue, weakness and tremor (hands, fingers and tongue).

Eyes; ophthalmopathy, it may produce the greatest long term disability, there is eyelid retraction, periorbital edema, chemosis and bilateral exophthalmos or proptosis due to enlargement of the extraocular muscles and fat within the orbit as a result of mucopolysaccharide infiltration. It may progress to visual loss due to exposure keratopathy or compressive optic neuropathy.

Skeletal; there is bone loss with increased excretion with calcium and phosphorus into the urine.

Others; there is glucose intolerance and rarely diabetes, decreased serum cholesterol, thrombocytopenia also develop, increase in RBCs total number to carry the additional oxygen needed for increased metabolic activity.

Laboratory findings
Classically there is decreased TSH and increased free T4 concentration, sometimes there is low TSH and normal free T4 and increased free T3, others may show increased TSH and increased T4 especially in pituitary adenoma secreting TSH or in thyroid hormone resistance syndrome.

Thyrotoxic crisis (thyroid storm)
It is a rare complication that occur in 1% of the patients, most of them have a long history of thyrotoxicosis. Precipitating factors include; infection, trauma, surgical emergency and operation. Signs and symptoms extreme restlessness, nausea, vomiting, abdominal pain, fever, profuse sweating, marked tachycardia, arrhythmia, pulmonary edema and congestive heart failure. Coma may follow severe hypotension and death.

Medical management of hyperthyroidis
1. Antithyroid agents; like Carbimazole and Propylthiouracil. These may have adverse effects like Agranulocytosis, Thrombocytopenia and Aplastic anemia.
2. Radioactive iodine; it is contraindicated in pregnancy and during breast feeding, it may cause hypothyroidism.
3. Subtotal thyroidectomy; in large goiter or thyroid nodules, but the patient is given antithyroid drugs before the operation until he/she
becomes euthyroid. Complications include hypoparathyroidism and recurrent laryngeal nerve injury.

4. Beta-adrenergic blockers like Propranolol to alleviate sympathetic overactivity.

**Hypothyroidism**

It can be congenital or acquired. The acquired adult type (Myxedema) follows thyroid gland failure, pituitary gland failure, radiation of the thyroid gland (radioactive iodine), autoimmune diseases (Hashimoto’s thyroiditis), surgical removal and excessive antithyroid drug therapy. It is more common in women than in men and occurs between 30-60 years old individuals.

Most infants with congenital hypothyroidism (Cretinism) have thyroid dysgenesis that is ectopic, hypoplastic or thyroid agenesis.

**Clinical presentation**

**Neonatal Cretinism** characterized by:
Dwarfism, overweight, a broad flat nose, wide set eyes, thick lips, large protruding tongue, poor muscle tone, pale skin, retarded bone age, delayed eruption of the teeth, malocclusion and mental retardation.
These features can be prevented by early detection and treatment.

**Adult hypothyroidism (Myxedema)**
Dull expression, puffy eyelids, alopecia of the outer third of the eyebrows, dry rough skin, dry brittle and coarse hair, increased size of the tongue, slowing of mental activity, slurred hoarse speech, anemia, constipation, weight gain, muscle weakness and deafness. Congestive heart failure may occur in patients with severe Myxedema.

Untreated patients with severe Myxedema may develop hypothyroid (Myxedema) coma, which can be fatal, it occurs in elderly patients during winter months, it is precipitated by stressful conditions like cold, operations, infections or trauma.

**Medical management of hypothyroidism**

Synthetic preparations containing sodium Levothyroxine (T4) or sodium Liothyronine (T3). T4 potentiates the action of Warfarin and may cause further prolongation of prothrombin time. Administration of T4 for diabetics may cause hyperglycemia.

**Dental management**

- Obtaining thorough history.
- The clinician should include the thyroid gland in examination.
- The anterior neck region should be inspected for the presence of goiter or the presence of an old surgical scar indicating previous
thyroid surgery, the gland should be palpated, when the patient swallows the gland moves superiorly.

- Thyroglossal duct cyst moves upward when the patient swallows or protrudes the tongue.
- The posterior dorsal region of the tongue should be inspected for the presence of any nodule that may indicate lingual thyroid.
- Auscultation over the enlarged gland may reveal systolic or continuous bruit as a result of engorgement of the gland’s vascular system.

**Hyperthyroidism (Thyrotoxicosis)**

1. Beware of the clinical manifestations of thyrotoxicosis, so that undiagnosed or poorly treated patients are identified and referred for medical evaluation and treatment. Those patients may develop thyrotoxic crisis (thyroid storm) which can be precipitated by a surgical procedure or acute infections, so we avoid surgical procedures acute infections in such patients. Management of thyrotoxic crisis include:
   - Recognize signs and symptoms.
   - Seek medical assistance immediately.
   - Cold wet packs and ice packs to cool the patient.
   - 100-300 mg hydrocortisone injection.
   - I.V. infusion of hypertonic glucose solution.
   - Cardiopulmonary resuscitation and monitoring of the vital signs.
   - Antithyroid drugs and potassium iodide.
   The use of vasoconstrictor agents in LA or retraction cord or to control bleeding should be avoided in such patients.

2. In well controlled patients, normal procedure and management is implemented, acute and chronic infections should be treated. Those patients can be given normal concentrations of vasoconstrictor agents.

3. In patients taking non-selective beta blockers, epinephrine may cause an increase in blood pressure through inhibition of the vasodilatory action of epinephrine attained through blocking beta2 receptors.

4. Propylthiouracil (antithyroid drug) can cause agranulocytosis and leukopenia which may run the risk of infection, the dentist can consult the patient’s physician or order screening tests to rule out the presence of these complications before surgical procedures. This drug can also increase the anticoagulant effect of Warfarin. Aspirin and NSAIDs increase the amount of T4 and make control of thyroid diseases more difficult.

5. Consultation with the physician is recommended as part of the management program.
**Hypothyroidism**

1. Identification and recognition of the patients through history and clinical examination, referral of the untreated patients for diagnosis and treatment.
2. In patients who are diagnosed but untreated or poorly treated, we should avoid surgical procedures, infections and CNS depressants and narcotics, those patients especially with severe symptoms of hypothyroidism may develop Myxedematous coma.

Management of Myxedematous coma:
- The dentist must be able to recognize the signs and symptoms.
- Seek immediate medical aid.
- Cover the patient to conserve heat.
- 100-300 mg Hydrocortisone.
- Cardiopulmonary resuscitation as needed.
- Administration of i.v. hypertonic saline and glucose.
- Parenteral Levothyroxine.

3. In well controlled patients we implement normal procedure and management and avoid infections, in addition to management of malocclusion and enlarged tongue.

**Oral complications and manifestations**

**Thyrotoxicosis**
- Osteoporosis of alveolar bone.
- Dental caries and periodontal diseases appear rapidly.
- Premature loss of deciduous teeth and early eruption of permanent teeth.
- Presence of lingual thyroid in few patients with thyrotoxicosis, such patients should have thyroid scan to make sure that this is not the only thyroid tissue present before surgical removal.

**Hypothyroidism**

**Infants with Cretinism**
- Thick lips.
- Enlarged tongue.
- Delayed eruption of teeth.
- Malocclusion.
- Skeletal retardation.

**Adults with Myxedema**
- Enlargement of the tongue.
- Generalized swollen gingiva.
**Thyroiditis**

It is inflammation of the thyroid gland that occurs for a variety of reasons; five types have been identified:

- **Hashimoto’s tyroiditis** (Hakaru Hashimoto a Japanese surgeon 1881-1934). It is an autoimmune thyroid disorder. It may be associated with other autoimmune diseases such as pernicious anemia and type 1 diabetes mellitus. The thyroid becomes enlarged and firm. Most patients develop hypothyroidism, its treatment is thyroid hormone and in some cases surgery is indicated.

- **Subacute painful.** It often follows upper respiratory tract viral infections; the gland is enlarged, tender with pain that may radiate to the ear, jaw or occipital region. There is hyperthyroidism which returns to euthyroid state. Treatment includes aspirin, prednisone, and Propranolol for symptoms of thyrotoxicosis.

- **Subacute painless.** It may be related to autoimmune disease, the gland is enlarged and not tender, and there is hyperthyroidism for 5-6 months that return to euthyroid state. Propranolol is used in treatment of thyrotoxicosis.

- **Acute suppurative.** Caused by bacterial infection, the gland is tender, with fever, malaise and the skin over the gland is red and warm. The patient is euthyroid. Treatment is by incision and drainage and antibiotics.

- **Riedel’s thyroiditis** (after Bernhard Moritz Carl Ludwig Riedel a German surgeon 1846-1916). Or chronic fibrosing thyroiditis, it is of unknown origin, the gland is enlarged, hard and fixed to the surrounding tissues, the patient usually remains euthyroid but some patients may become hypothyroidism, treatment is usually not required but sometimes surgery and thyroid hormone may be needed.

**Thyroid Cancer**

The main histological types of thyroid cancer include:

1. Differentiated cancers which include; papillary (75%-80%), follicular (8%-10%), mixed, and Hürthle cell 1% (after Karl Hürthle, a German histologist 1860-1945) carcinomas.
2. Anaplastic (1%-5%).
3. Medullary (5%-8%).
4. Lymphoma (1%-5%).
5. Metastases to the thyroid gland (less than 1%).

In many cases no risk factors can be identified but the main suggested etiological factors involved include:
• External radiation.
• High or low dietary iodine intake may increase the risk for thyroid cancer.
• Genetic factors may be indicated.

**Signs and symptoms** may include:

• A lump or hard mass with possible fixation to the surrounding tissues and rapid growth.
• Enlarged cervical lymph nodes.
• Hemoptysis.
• Dysphagia.
• Stridor.
• Hoarseness of voice.

**Treatment**

Treatment modalities include surgery which ranges from lobectomy to total thyroidectomy with or without cervical lymph nodes dissection, external beam radiotherapy and chemotherapy. Radioiodine ablation may be indicated in some cases. Suppression of thyrotropin through levothyroxine replacement therapy may be indicated.

Overall 10-year survival rates for papillary carcinoma are (80% to 90%), follicular carcinoma (65% to 75%), and medullary carcinoma (60% to 70%). Involvement of cervical nodes predicts recurrence in older patients. The prognosis for anaplastic carcinoma is very poor, and 5-year survival is rare.