Revised & Approved

Bassam Alasmari
Rania Almutiri

1

Any future corrections will be in the editing file , Click

Pathology

Pathology of thyroid gland

439

Color index

- Important
- Doctor's note
- Extra info
- Main text
- ★ Male's slide
- ★ Female's slide

"اللهم لا سهل الا ماجعلته سهلا و انت تجعل الحزن إذا شئت سهلا"

Objective

01 To know the ways in which thyroid disorders present.

02 To understand the major causes and clinical manifestations of hypo, hyperthyroidism and thyroiditis .

03 To recognize the pathophysiology, gross and microscopic morphology and clinical manifestations of Graves disease and Hashimoto thyroiditis. (Male Dr: Important to know)

04 To Know the causes of the thyroid goiter and its pathology.

Overview

Thyroid diseases

- Hyperthyroidism
  - **Main cause:** Graves diseases
- Hypothyroidism
  - **Main cause:** Hashimoto thyroiditis
- Mass lesions

Thyroiditis

- Hashimoto thyroiditis
- Subacute granulomatous
- Subacute lymphocytic thyroiditis
- Riedel thyroiditis

Goiter: Male Dr (General word. mostly benign)

Enlargement of the thyroid, is the most common manifestation of thyroid disease

Pathology of Thyroid Gland

Thyroid disease

Clinical recognition of diseases of the thyroid is important, because most are amenable to medical or surgical management.
Diseases include:

- **Hyperthyroidism**: Excessive release of thyroid hormones
- **Hypothyroidism**: Thyroid hormone deficiency
- **Mass lesions**

Hyperthyroidism

Thyrotoxicosis	is a hypermetabolic state due to elevated circulating levels of free T3 and T4. **Thyrotoxicosis** means increase in the circulating levels of thyroid hormones whatever the cause is, but **hyperthyroidism** it is related to the thyroid gland (the problem is in the gland itself) Because it is caused most commonly by hyperfunction of the thyroid gland, thyrotoxicosis often is referred to as hyperthyroidism

Other causes : in certain conditions, however, the oversupply either is related to excessive release of pre-formed thyroid hormone (e.g., in thyroiditis) or comes from an extrathyroidal source, rather than a hyperfunctioning gland

The causes of thyrotoxicosis

ASSOCIATED WITH HYPERTHYROIDISM	NOT ASSOCIATED WITH HYPERTHYROIDISM
▪ **Primary:** 1. **Diffuse toxic hyperplasia** =increased number of cells **(Graves disease)** 2. **Hyperfunctioning ("toxic") multinodular goiter** **Hyperfunctioning ("toxic") adenoma** 3. Iodine-induced hyperthyroidism 4. Neonatal thyrotoxicosis associated with maternal Graves' disease ▪ **Secondary :** TSH-secreting pituitary adenoma (rare)	1. Granulomatous (de Quervain) thyroiditis (painful*) 2. Subacute lymphocytic thyroiditis (painless*) 3. Struma ovarii (ovarian teratoma with ectopic thyroid) 4. Factitious thyrotoxicosis (exogenous thyroxine intake) * Clinically important to differentiate between these two types of thyroiditis

Clinical manifestation of thyrotoxicosis

Hypermetabolic state induced by excessive amounts of thyroid hormone ,overactivity of the sympathetic nervous system:

- ❖ Constitutional symptoms (Fever, change in appetite)
- ❖ Neuromuscular(Tremors, increased irritability, overactivity)
- ❖ Gastrointestinal (Diarrhea)
- ❖ Cardiac
- ❖ Ocular
- ❖ Apathetic hyperthyroidism
- ❖ Thyroid storm (medical emergency): Sudden increase in thyroid hormones

Diagnosis of hyperthyroidism

- ❖ The diagnosis of hyperthyroidism is based on clinical features (Excessive sweating, anxiety, overactivity, stressed all time, heat intolerance, weight loss) and laboratory data.
- ❖ The measurement of serum **TSH** is the most useful single screening test for hyperthyroidism (**low** TSH due to the negative feedback of thyroid hormones)
- ❖ Free thyroid hormone assays (**High** T_3 and T_4)
- ❖ Measurement of radioactive iodine uptake by the thyroid gland and Ultrasound

Hypothyroidism

is (low levels of thyroid hormones) caused by any structural or functional derangement that interferes with the production of adequate levels of thyroid hormone .

The causes of hypothyroidism

Worldwide, the most common cause of hypothyroidism is **dietary deficiency of iodine** , while in most developed nations, autoimmune causes predominate

★**Primary:**

1. Postablative: Surgery, radioiodine therapy, or external irradiation [1].
2. Autoimmune hypothyroidism: **Hashimoto thyroiditis**.
3. Iodine deficiency.
4. Drugs (lithium, iodides, p-aminosalicylic acid).

▪ **Rare causes:**

5. Congenital biosynthetic defect (dyshormonogenetic goiter).
6. Genetic defects in thyroid development. (PAX8, FOXE1, TSH receptor mutation)
7. Thyroid hormone resistance syndrome. (THRB mutations)

▪ **Secondary :**

Pituitary failure or Hypothalamic failure.

Clinical manifestations of hypothyroidism

Cretinism	myxedema
Is hypothyroidism developing in **infancy or early childhood:** - Endemic cretinism, sporadic cretinism - Clinical features of cretinism include : impaired development of the skeletal system and central nervous system, severe mental retardation, short stature, coarse facial features, a protruding tongue, umbilical hernia. **Easy to treat (replace thyroid hormones) and avoid all these complications**	Hypothyroidism developing in **older children and adults.** Manifestations of myxedema include : 1-generalized apathy (خمول وكسل) and mental sluggishness that in the early stages of disease may mimic depression. 2-Cold intolerance, obesity. 3-The skin is cold and pale 4-Shortness of breath. 5-Broadening and coarsening of facial features (matrix substance) 6-enlargement of the tongue, and deepening of the voice. 7-Constipation. 8-Pericardial effusions are common In later stages, if stayed untreated, the heart is enlarged, and heart failure may supervene.

Diagnosis (Laboratory evaluation)

❖ Measurement of serum **TSH** is the most sensitive screening test for this disorder : The serum TSH is **increased** in primary hypothyroidism

❖ Serum T4 is **decreased** in patients with hypothyroidism of any origin.

1. A patient with any pathology in the thyroid gland that had to be removed surgically, he has to be on continuous supply of thyroxine. So, non compliance with the treatment will lead to hypothyroidism.

Thyroiditis

Clinically significant types of thyroiditis

Female Doctor: You have to know all the names because any of these names may come in the exam.

- **Hashimoto thyroiditis** (chronic lymphocytic thyroiditis)
- **Subacute granulomatous** (de Quervain) thyroiditis
- **Subacute lymphocytic thyroiditis**

Chronic Lymphocytic (Hashimoto) Thyroiditis

- Hashimoto thyroiditis is the most common cause of hypothyroidism in areas of the world where iodine levels are sufficient.
- It is characterized by gradual thyroid failure secondary to autoimmune destruction of the thyroid gland.
- It is most prevalent between the ages of 45 and 65 years and is more common in women than in men, with female predominance in a ratio of 10:1 to 20:1.
- It has a significant genetic component.
- Increased susceptibility to Hashimoto thyroiditis is associated with polymorphisms in multiple immune regulation–associated genes, the most significant of which is the linkage to **cytotoxic T lymphocyte–associated antigen-4 gene (CTLA4).**
- Male Doctor: تلقى الام عندها ووحدة من بناتها عندها (be careful if it comes in a case).
- **Female Doctor:** Multiple family members maybe affected.
- **What is the pathogenesis of hashimoto thyroiditis? Autoimmune destruction of thyroid gland**

Clinical features

Painless enlargement of the thyroid, usually associated with some degree of hypothyroidism.
It may be preceded by **transient thyrotoxicosis** caused by disruption of thyroid follicles, with secondary release of thyroid hormones (hashitoxicosis). **[IMP]**

As hypothyroidism supervenes, T4 and T3 levels progressively fall, accompanied by a compensatory increase in TSH.

Patients with Hashimoto thyroiditis often have other autoimmune diseases and are at increased risk for the development of **B cell non-Hodgkin lymphomas.** (**Most common complication**)

Male Doctor: By transient thyrotoxicosis we mean that since its an inflammatory process, at the start of the inflammatory process (during the first week) the hormonal reserve will be released all at once (due to the inflammation taking place and destroying the thyroid follicles releasing the reserve) this will create the clinical picture of hyperthyroidism with investigations showing an increase in TH levels and the patient presenting with symptoms of hyperthyroidism (lasting for the first month or so until the reserves get used up) and then TH levels decrease so the patient gives a presentation of hypothyroidism.

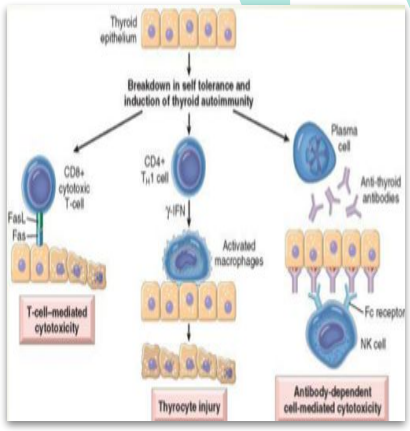
Female Doctor: So, the patient comes complaining of swelling only, we determine if it is a thyroid gland problem by asking the patient to swallow (i.e drinking water), if the swelling moves during swallowing we confirm the diagnosis (thyroid gland problem)

Chronic Lymphocytic (Hashimoto) Thyroiditis

Pathogenesis

Hashimoto thyroiditis is caused by a breakdown in self-tolerance to thyroid autoantigens. Thus, circulating autoantibodies against thyroid antigens are present in the vast majority of patients

Female Doctor: So what's the pathogenesis? Autoimmune disease with AI destruction (involving antigen-antibody reaction with circulating autoantibodies acting on thyroid gland receptors/antigens leading to the destruction of receptors) which might either increase or decrease the thyroid gland activity.
Female Doctor: So we should order TSH, T3, T4, and Circulating antibody tests.



Morphology

1

Diffuse and symmetrical enlargement [IMP]
The cut surface is pale and gray-tan in appearance, and the tissue is firm and somewhat friable.
Male Doctor: there might a slight nodularity but nothing well defined.

2

ˇnfiltration of the parenchyma by a mononuclear inflammatory infiltrate containing small lymphocytes, plasma cells, and well-developed germinal centers.

3

The thyroid follicles are atrophic and are lined in many areas by epithelial cells distinguished by the presence of abundant eosinophilic (pink), granular cytoplasm, termed Hürthle, or oxyphil, cells.

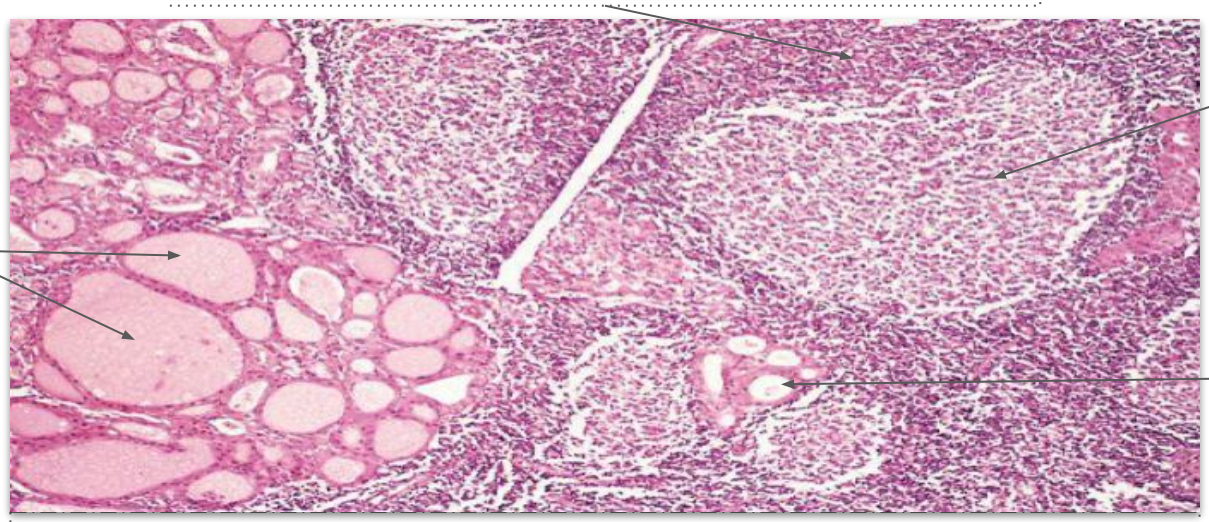
4

Interstitial connective tissue is increased and may be abundant. Less commonly, the thyroid is small and atrophic as a result of more extensive fibrosis **(fibrosing variant)**
Unlike in Reidel thyroiditis, the fibrosis does not extend beyond the capsule of the gland.

Female Doctor : the Highlighted in red is the most IMP.
Male Doctor: Hashimoto's is most likely with the above morphology.
Male Doctor: Reidel Thyroiditis is malignant, meaning that it is diffuse and extending and so it might give the impression of cancer.

Mononuclear infiltrate (plasma cells + Lymphocytes)

Follicular epithelium



Germinal center formed by the infiltrate

Follicular cells undergoing metaplasia to become Hurthle cells (pinkish in color)

Hashimoto thyroiditis. The thyroid parenchyma contains a dense lymphocytic infiltrate with germinal centers. Residual thyroid follicles lined by deeply eosinophilic Hürthle cells also are seen.

Subacute Granulomatous (de Quervain) Thyroiditis

- De Quervain thyroiditis is most common between 30 and 50 years of age and, like other forms of thyroiditis, occurs more frequently in women than in men.
- Subacute thyroiditis is believed to be caused by a viral infection or an inflammatory process triggered by viral infections. [IMP]
- A majority of patients have a history of an upper respiratory infection just before the onset of thyroiditis.
- The onset of this form of thyroiditis often is acute (2 days), characterized by pain in the neck (particularly with swallowing), fever, malaise, and variable enlargement of the thyroid. [IMP]
- **Transient hyperthyroidism** may occur, as in other cases of thyroiditis, as a result of disruption of thyroid follicles.
- The leukocyte count and erythrocyte sedimentation rates are increased.
- With progression of disease and gland destruction, a transient hypothyroid phase may ensue.
- The condition typically is **self-limited**, with most patients returning to a euthyroid state within 6 to 8 weeks. [IMP]
- Male Doctor: self-limiting as in 2 months and it goes away.
- Male Doctor: Transient hyperthyroidism in this case means hyperthyroidism followed by hypothyroidism.
- Male Doctor: cough + sore throat which extends to the thyroid and effects its and its painful.

Morphology

The gland is firm, with an intact capsule, and may be unilaterally or bilaterally enlarged.

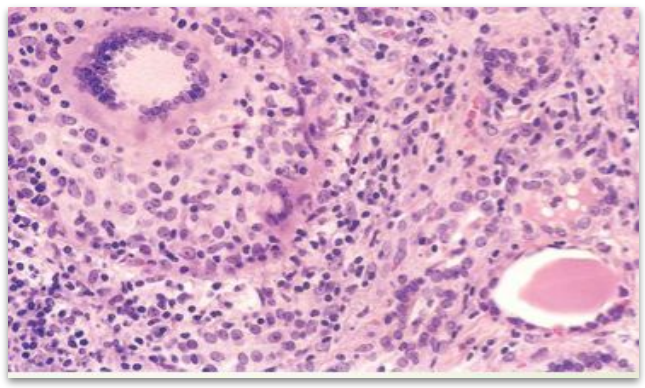
Histologic examination reveals disruption of thyroid follicles, with extravasation of colloid leading to a polymorphonuclear infiltrate (**neutrophils**), which is replaced over time by lymphocytes, plasma cells, and macrophages.

The extravasated colloid provokes an exuberant granulomatous reaction with giant cells.

Healing occurs by resolution of inflammation and fibrosis.

Male Doctor: If we were able to diagnose clinically then there is no need for a biopsy.

Granuloma formation is seen here, which is a collection of histiocytes (Macrophages) with some forming giant cells and this reaction is surrounded by a rim of lymphocytes.



Subacute Lymphocytic Thyroiditis

In a subset of patients the onset of disease **follows pregnancy (postpartum thyroiditis)**. This disease is most likely to be autoimmune in etiology.

The histologic features: consist of lymphocytic infiltration and hyperplastic germinal centers within the thyroid parenchyma.

Painless neck mass or features of thyroid hormone excess.

Female Doctor: The most important 2 types are Hashimoto's and De Quervain ;)

Riedel thyroiditis

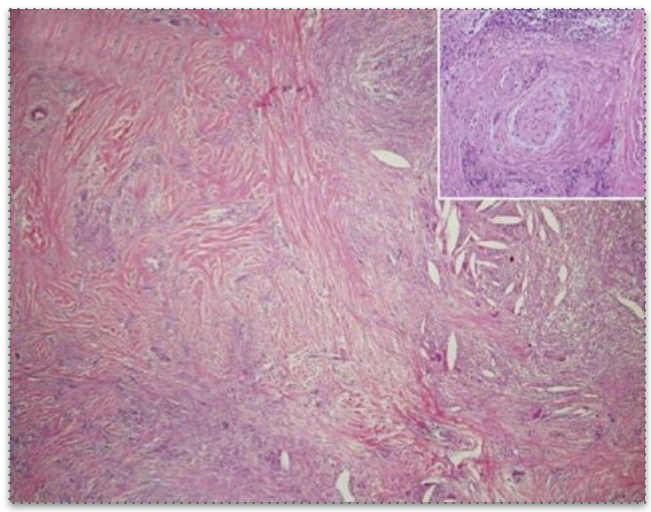
Riedel thyroiditis, a rare disorder that is a manifestation of IgG4-related disease.

Characterized by **extensive fibrosis** involving the thyroid and contiguous neck structures.

Clinical evaluation demonstrates a hard and fixed thyroid mass, simulating a thyroid neoplasm.

It may be associated with idiopathic fibrosis in other sites in the body, such as the retroperitoneum.

Male Doctor: extensive fibrosis here gives the impression of a malignant invasive disease such as a cancer.



Graves disease

Graves disease has a peak incidence between the ages of 20 and 40, with women being affected up to seven times more commonly than men.
Graves disease is the most common cause of endogenous hyperthyroidism. It is characterized by a triad of manifestations:

01 **Thyrotoxicosis**:caused by a diffusely enlarged, hyperfunctional thyroid, is present in all cases.

02 **An infiltrative ophthalmopathy** with resultant **exophthalmos** is noted in as many as 40% of patients.
In Graves ophthalmopathy, a result of several causes:
(1) marked infiltration of the retro orbital space by mononuclear cells (mainly T cells)
(2) inflammatory edema and swelling of extraocular muscles
(3) accumulation of extracellular matrix components (glycosaminoglycan)
(4) increased numbers of adipocytes

03 A localized, infiltrative **dermopathy** (sometimes designated pretibial myxedema) is seen in a minority of cases.
The **dermopathy**, if present, is characterized by thickening of the dermis, as a result of deposition of glycosaminoglycans and lymphocyte infiltration.

Pathogenesis

- Genetic factors are important in the causation of Graves disease
- Graves disease is associated with the presence of certain human leukocyte antigen (HLA) haplotypes, specifically HLA-DR3, and polymorphisms in genes whose products regulate T-cell responses, including the inhibitory T- cell receptor CTLA-4 .
- Graves disease is characterized by a breakdown in self-tolerance to thyroid autoantigens, of which the most important is the TSH receptor. The result is the production of multiple autoantibodies, including:

Thyroid-stimulating immunoglobulin:
An lgG antibody that binds to the TSH receptor and mimics the action of TSH, relatively specific for Graves disease.

Thyroid growth-stimulating immunoglobulins:
Also directed against the TSH receptor.

TSH-binding inhibitory immunoglobulins:
These anti-TSH receptor antibodies prevent TSH from binding to its receptor on thyroid epithelial cells and in so doing may actually inhibit thyroid cell function.

The coexistence of stimulating and inhibiting immunoglobulins in the serum of the same patient is not unusual—a finding that may explain why some patients with Graves disease spontaneously develop episodes of hypothyroidism.

The morphology

Grossly	On microscopic examination
The thyroid gland is enlarged (usually symmetrically) due to diffuse **hypertrophy and hyperplasia of thyroid follicular epithelial cells.** The gland is usually smooth and soft, and its capsule is intact.	- the follicular epithelial cells in untreated cases are tall, columnar, and more crowded than usual. This crowding often results in the **formation of small papillae**. Such papillae **lack fibrovascular cores**, **in contrast with those of papillary carcinoma**. - The **colloid** within the follicular lumen **is pale**, with **scalloped margins** (characteristic feature of graves disease) - Lymphoid infiltrates, are present throughout the interstitium; germinal centers are common.

Diagnosis

Laboratory findings in Graves disease include **elevated serum free T4 and T3 and depressed serum TSH.**
Because of ongoing stimulation of the thyroid follicles by TSIs, radioactive iodine uptake is increased, and radioiodine scans show a diffuse uptake of iodine

Diffuse and Multinodular Goiter

Goiter

Enlargement of the thyroid, is the most common manifestation of thyroid disease. Swelling of the thyroid is called **goiter** whatever the cause is, with/without hyperthyroidism or hypothyroidism or hashimoto disease. Sometimes it is non-functional.

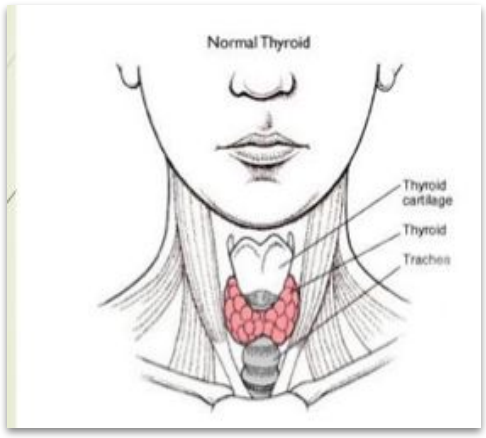
They reflect impaired synthesis of thyroid hormone, most often caused by **dietary iodine deficiency**.

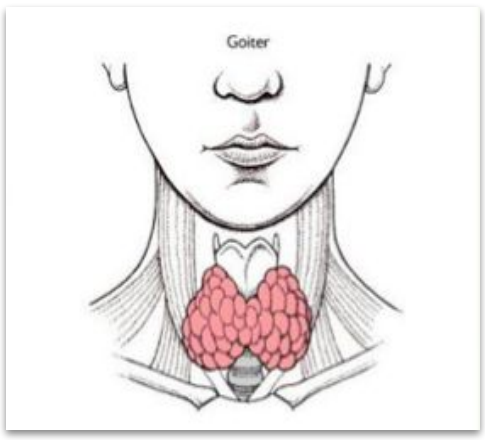
Impairment of thyroid hormone synthesis leads to a compensatory rise in the serum TSH, causes hypertrophy and hyperplasia of thyroid follicular cells and, ultimately, gross enlargement of the thyroid gland.

The compensatory increase in functional mass of the gland is enough to overcome the hormone deficiency, ensuring a euthyroid=normal metabolic state in the vast majority of affected persons.

If the underlying disorder is sufficiently sever the compensatory responses may be inadequate to overcome the impairment in hormone synthesis, resulting in goitrous hypothyroidism.

- Goiters can be endemic or sporadic.
- Diffuse, symmetric enlargement of the gland (diffuse goiter): The follicles are lined by crowded columnar cells, which may pile up and form projections.
- colloid-rich gland (colloid goiter): dilation of follicles filled with colloid.
- All long-standing diffuse goiters convert into multinodular goiters. (Typically Hormonally Silent)
- Multinodular goiters typically are hormonally silent, although a minority (approximately 10% over 10 years) can manifest with thyrotoxicosis secondary to the development of autonomous nodules that produce thyroid hormone independent of TSH stimulation. This condition, known as toxic multinodular goiter or Plummer syndrome. (Some of the nodules may secrete hormones some not)
- Male Doctor: several cases lead to goiter some even with involvement in thyroid function (so its not necessarily due to Hashimoto's or Graves) and independent of hypo or hyperthyroidism.
- Male Doctor: Goiter is not identified as benign or malignant though we can assume its benign, and even though its benign due to its location it may still lead to symptoms other than the neck swelling and might even be fatal (suffocation).
- Male Doctor: those with iodine deficiency are the ones with the greatest degree of thyroid enlargement due to failed compensatory mechanism attempted by the pituitary gland.



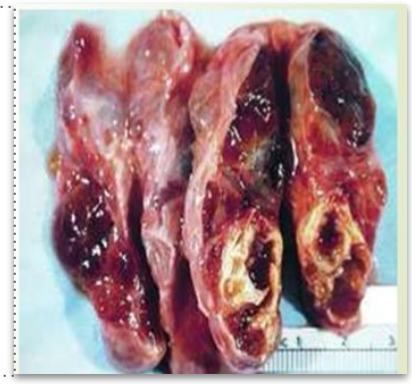


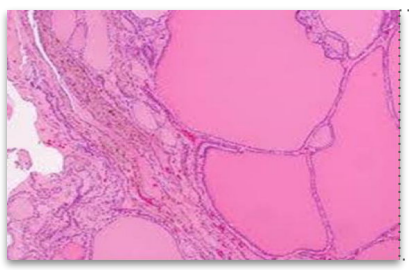
MULTINODULAR GOITER

Multinodular goiters are multilobulated, asymmetrically enlarged glands, which may attain massive size.

On Cut surface

- On cut surface, irregular nodules containing variable amounts of brown, gelatinous colloid are evident.
- Older lesions often show areas of fibrosis, hemorrhage, calcification, and cystic change.
- Male Doctor: Here we see the cut surface, some nodule appear larger than others, and some are with hemorrhage. The nodules are of different ages some old and with time new nodules appear.





The microscopic appearance includes colloid-rich follicles lined by flattened, inactive epithelium and areas of follicular epithelial hypertrophy and hyperplasia

On Microscopy

Clinical features

1. The dominant clinical features of goiter are those caused by the mass effects of the enlarged gland.
2. A hyperfunctioning (toxic) nodule may develop within a long-standing goiter, resulting in hyperthyroidism.
3. In addition to the obvious cosmetic problem of a large neck mass, goiters also may cause airway obstruction, dysphagia, and compression of large vessels in the neck
4. The incidence of malignancy in long-standing multinodular goiters is low (less than 5%) but not zero, and concern for malignancy arises with goiters that demonstrate sudden changes in size or associated symptoms (e.g.,hoarseness).

Female Doctor: The presentation is often a Euthyroid with goiter leading to hoarseness of voice and difficulty of breathing.

Male Doctor: Further investigations are required in the long stranding potentially malignant cases.

Summary

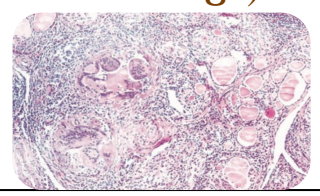
Functional classification		
	Hyperfunction (hyperthyroidism)	**Hypofunction (hypothyroidism)**
Overview	Hypermetabolic state induced by excessive amounts of thyroid hormone and over activity of the sympathetic nervous system	thyroid gland can't make enough thyroid hormone to keep the body running normally
Cause	-Graves disease	Primary : -Postablative: Surgery, radioiodine therapy -Autoimmune: Hashimoto thyroiditis - iodine deficiency -Drugs : lithium Secondary : -pituitary insufficiency
Symptoms	-CVS : Palpitations -CNS : Nervousness, tremor, irritability -GIT : hyper mortality , weight loss despite increased appetite , diarrhea -ocular : Wide, staring gaze and lid lag Skin : Soft, warm, and flushed skin	-in early fetal life : Cretinism 1)severe mental retardation 2)Short stature 3)protruding tongue In adult : Myxedema 1)mental sluggishness 2)Shortness of breath 3)Broadening and coarsening of facial features 4)Enlargement of the tongue , deepening of the voice 5)Cold and pale skin 6)Constipation , obesity 7)In later stages : heart is enlarged, and heart failure may supervene
Diagnosis	-low TSH -high T3 and T4	-high TSH -low T3, T4

Etiological classification

Gravis disease	Overview	The most common cause of endogenous hyperthyroidism
	Pathogenesis	-Thyroid-stimulating immunoglobulin: lgG antibody that binds to the TSH receptor and mimics the action of TSH -Thyroid growth-stimulating immunoglobulins : directed against the TSH receptor -TSH-binding inhibitory immunoglobulins : prevent TSH from binding to its receptor
	Clinical feature	Thyrotoxicosis : Diffusely enlarged, hyperfunctional thyroid Ophthalmopathy : Exophthalmos caused by Accumulation of glycosaminoglycan
	Morphology	Macroscopic : Enlarged gland usually symmetrically Microscopic: columnar cell , colloid is pale (clear vasculated) , Lymphoid infiltrates
	Diagnosis	-decrease TSH -increase T3, T4 -increase radioactive iodine uptake
Thyroiditis — Hashimoto Thyroiditis Chronic Lymphocytic Thyroiditis	Overview	Chronic inflammation of thyroid gland , most common cause of hypothyroidism in areas of the world where iodine levels are sufficient
	Characteristic	gradual thyroid failure secondary to **autoimmune destruction**
	Clinical features	-Painless enlargement of the thyroid -Hashitoxicosis:cycle through an initial period where there is too much thyroid hormone in the body and demonstrate symptoms of hyperthyroidism.
	Pathogenesis	Circulating autoantibodies against thyroid antigens leads to progressive depletion of thyroid epithelial cells associated with lymphocytic infiltrates and fibrosis.
	Risk factor	Mutation of CTLA4 gene
	Morphology	-Mononuclear inflammatory infiltrate (lymphocytes, plasma cells) with germinal centre formation -Hürthle cells : atrophic follicles of thyroid Cytoplasm : eosinophilic, granular
	de Quervain Thyroiditis Subacute Granulomatous Thyroiditis — Cause	-caused by a viral infection
	Characteristic	-Pain in the neck , Fever and malaise -Variable enlargement of the thyroid.
	Clinical features	-Transient hyperthyroidism -Transient hypothyroidism -with most patients returning to a euthyroid (self limited)
	Morphology	-Disruption of thyroid follicles extravasation of colloid polymorphonuclear infiltrate -granulomatous reaction with giant cells -Healing occurs by resolution of inflammation and fibrosis
	Subacute Lymphocytic Thyroiditis — Definition	postpartum thyroiditis : onset of disease follows pregnancy , most likely autoimmune in etiology
	Clinical features	Painless neck mass or features of thyroid hormone excess
	Morphology	lymphocytic infiltration and hyperplastic germinal centers.
	Riedel thyroiditis — Definition	Rare disorder of unknown etiology
	Clinical features	hard and fixed thyroid mass
	Morphology	extensive (sever) fibrosis

QUIZ!

MCQs

01 | A female patient was presented to the hospital with a painless enlargement, a biopsy was taken and the microscopic morphology showed Hurthle cells, which of the following is the most likely diagnosis.			
A) Graves disease	B) Riedel Thyroiditis	C)Hashimoto's Thyroiditis	D) De Quervain Thyroiditis
02 | which of the following is often associated with an acute viral infection.			
A) Graves disease	B) Riedel Thyroiditis	C) Hashimoto's Thyroiditis	D) De Quervain Thyroiditis
03 | increased risk for the development of B cell non-Hodgkin lymphomas is associated with			
A) Riedel thyroiditis	B) Subacute Granulomatous Thyroiditis	C) Hashimoto Thyroiditis	D) Graves Disease
04 | 29-year-old woman complains of nervousness and muscle weakness of 6 months in duration. She is intolerant of heat and sweats excessively. She has lost 9 kg (20 lb) pounds over the past 6 months, despite increased caloric intake. She frequently finds her heart racing and can feel it pounding in her chest. She also states that she has missed several menstrual periods over the past few months. Physical examination reveals warm and moist skin and bulging eyes (exophthalmos). Laboratory studies will likely reveal which of the following?			
A) Anti-thyroid DNA antibodies	B) Anti-TSH receptor antibodies	C) low T3	D) Increased serum TSH
05 | A 43-year-old woman complains of low-grade fever and has a 3-day history of pain in her neck. Physical examination reveals a slightly enlarged thyroid. A CBC is normal. A biopsy of the thyroid reveals granulomatous inflammation and the presence of giant cells (shown in the image). What is the appropriate diagnosis? 			
A) hashimoto thyroiditis	B) Nontoxic multinodular goiter	C) Graves' disease	D)Subacute (deQuervain) thyroiditis

MCQs Answer key	01	02	03	04	05
	C	D	C	A	D

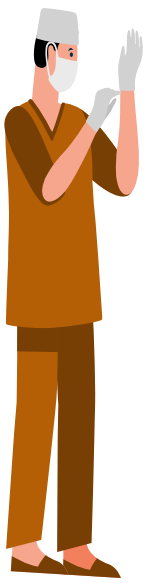
"اللهم علمنا ماينفعنا ، وانفعنا بما علمتنا وزدنا علما يارب العالمين"

Team leaders

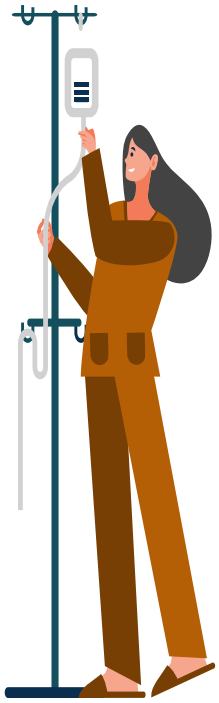
Hamad Almousa

Fatimah Alhilal

Team members



- Faisal Alfadel
- Hadi Alhemsi
- Abdulrahman Barashid
- Mansour Albawardy
- Ghada Alabdi
- Mariam Alruhaimi
- Renad Alhomaidi
- Rania almutiri
- Ghaida Almarshoud
- Ghadah Alsuwailem



This Lecture done by

- Organizer
- Member
- Note taker
- Reviser

Contact us through :
Pathology439@Gmail.com