THYROID DISORDERS

435 medicine teamwork

[Important | Notes | Extra | Editing file]

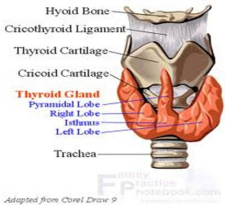
lecture objectives:

- Thyroid anatomy and physiology
- Action of thyroid hormones
- Thyroid function Tests
- **Thyroid disorders:**
  - – Function disorders:
    - Hypothyroidism
    - Hyperthyroidism
  - – Structure disorders:
    - Goiter
    - Nodule

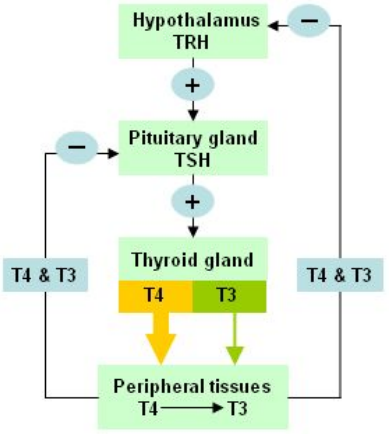
Done By: Lama AlZamil, Munira Alhussaini
& Abdulrahman Alshammari
Edited by: Rahaf Binabbad
Revised By: Ahmed Al Yahya
References: Slides - Davidson

Basic Review of Thyroid Gland

Anatomy & Histology:

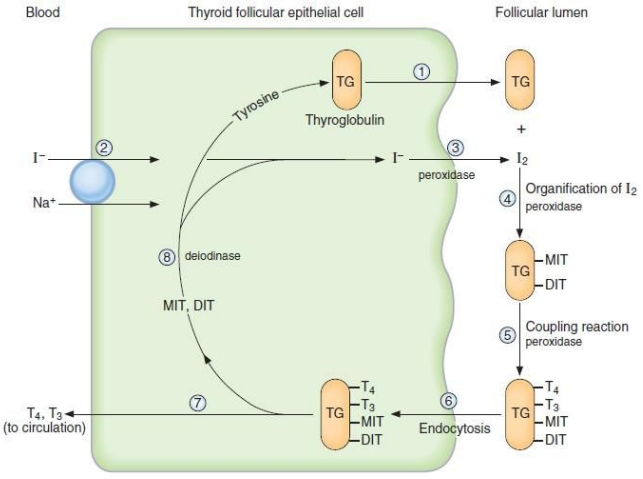
Anatomy	Histology
• Thyroid gland is an endocrine, butterfly shaped gland. • It is located in front of the larynx and consists of 2 lobes (Right thyroid lobe, Left thyroid lobe) connected by an isthmus. • what are the anatomical landmarks of the thyroid? 1- hyoid bone 2- thyroid cartilage 3- cricoid cartilage 4- isthmus 5- right + left lobe  • The two recurrent laryngeal nerves pass underneath the thyroid gland to innervate the larynx. • The thyroid gland weighs 25-30g in adults. Bigger in men. Increases with age and bodyweight and decrease with iodine intake	• Thyroid Gland is covered by fibrous capsule that extends septa into gland (these Septa carry blood vessels, Nerves, and Lymphatics.) which divides it into lobules. Each lobule contains thyroid Follicles which are the structural and functional units of the thyroid gland. • The thyroid tissue is made up of two types of cells: follicular cells and parafollicular cells: 1) Follicular cells are cuboidal in shape and has a central lumen called "colloid". It synthesizes and stores thyroid hormones. 2) Parafollicular or C cells secrete "calcitonin"whenever calcium levels are elevated and suppress bone resorption by osteoclasts.

Physiology:

Regulation of thyroid hormones		
Hypothalamus	The hypothalamus releases Thyrotropin-releasing Hormone (TRH) which acts on the anterior pituitary.	
Pituitary	Upon being stimulated by TRH, the anterior pituitary synthesizes and secretes Thyroid Stimulating Hormone (TSH) which acts directly on the thyroid gland to increase the production of thyroid hormones. Thyroid hormones exert negative feedback on TSH & TRH, when there are little thyroid hormones this negative feedback is inhibited.	
Thyroid	Upon being stimulated by TSH the thyroid gland releases thyroid hormones.	
Target tissue	Many physiological effects follow once thyroid hormones act on target cells.	

Recall synthesis of thyroid hormones (or don't)

1. Endoplasmic reticulum synthesizes thyroglobulin (contains tyrosine), Golgi apparatus packages and sends the thyroglobulin to the colloid.
2. In the basolateral surface, iodide(-) is picked up from blood and trapped into the follicular cell via NIS (sodium-iodine-symporter).
3. On the apical surface, (Pendin) transporter transports iodide into the colloid, in the colloid iodide(-) is oxidized into iodine via Thyroid Peroxidase.
4. Now iodine will bind to the Tyrosine ring on the thyroglobulin. When;
5. One iodine binds to tyrosine --> MIT (monoiodotyrosine)
6. Two iodine bind to tyrosine --> DIT (diiodotyrosine)
7. After that, MIT & DIT or DIT & DIT can form bonds with each other;
8. MIT + DIT = T3 (Triiodothyronine)
9. DIT + DIT = T4 (Thyroxine)
10. Not done yet -_- because T3 & T4 are still attached to the thyroglobulin. The compound gets endocytosed back into the follicular cell. Then it combines with a lysosome to hydrolyze the peptide chain & release the thyroid hormones (T3& T4).
11. T4 & T3 are sent to the circulation bounded to thyroid binding proteins (Thyroxine binding globulin TBG). (Because they're lipid hormones)



Note that:

- T4 is more secreted from the thyroid than T3.
- However T3 is 5x more active than T4.
- Once reaching the target cells T4 is converted to T3 via iodinase enzyme.
- Because thyroid hormones are lipid soluble, they enter the nucleus of target cells to initiate their actions.

Physiological effects of thyroid hormones

Target Tissue	Effect	Mechanism
Heart	Chronotropic	Increase number and affinity of beta-adrenergic receptors.
	Inotropic	Enhance responses to circulating catecholamines. Increase proportion of alpha myosin heavy chain (with higher ATPase activity).
Adipose tissue	Catabolic	Stimulate lipolysis.
Muscle	Catabolic	Increase protein breakdown.
Bone	Developmental	Promote normal growth and skeletal development.
Nervous system	Developmental	Promote normal brain development.
Gut	Metabolic	Increase rate of carbohydrate absorption.
Lipoprotein	Metabolic	Stimulate formation of LDL receptors.
Other	Calorigenic	Stimulate oxygen consumption by metabolically active tissues (exceptions: adult brain, testes, uterus, lymph nodes, spleen, anterior pituitary). Increase metabolic rate.

Thyroid Disorders

- ➔ **Function Disorders:** Hyperthyroidism Or Hypothyroidism
- ➔ **Others:** Subacute Thyroiditis - Multiple Thyroid Nodules - Thyroid Cancer
- ➔ **Structure Disorders:** Goiter or Nodule

Hyperthyroidism:

- **Hyperthyroidism:** Overproduction (increase synthesis and release) of thyroid hormone, due to primary (thyroid itself) or secondary (pituitary) cause.
- **Thyrotoxicosis:** is a metabolic state caused by elevated circulating FT4 & FT3
- **Subclinical Hyperthyroidism:** Asymptomatic/symptomatic patient with low TSH & normal FT4
- **Apathetic Hyperthyroidism:** Symptoms of hyperthyroidism are blunted in elderly people. they don't present with ideal symptoms of hyperthyroidism. some present with only depression, Afib, CHF, or weakness. وممكن المريض يجيك خالي من التعابير ، تقوله نكتة ما يضحك.
- **Thyroid storm:** abrupt onset of thyrotoxicosis (**Emergency endocrine**). very severe symptoms. critical care needed!

Hyperthyroidism

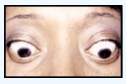
Causes:

Etiology Of Hyperthyroidism غالبًا لمّا نلقى زيادة بالثايرويد هورمون وما كان التشخيص واضح اكلينيكيًا, الخطوة الثانية إننا نطلب Radioiodine uptake test	
Normal Or High Radioiodine Uptake Thyroid gland increases synthesis → increases uptake of iodine	**Low Radioiodine Uptake** Doesn't increase synthesis → doesn't increase uptake of iodine
- Graves Disease TSH receptor antibodies - Hashitoxicosis thyrotoxic phase of hashimoto's - Toxic adenoma & toxic multinodular goiter - Iodine-induced hyperthyroidism ADR of Amiodarone. والمناطق الي عالساحل بسبب تغذيتهم على السمك - Trophoblastic disease & germ cell tumor Molar pregnancy → High levels of hCG → stimulation of thyroid hormones (it resembles the structure of TSH) - TSH-mediated hyperthyroidism (TSH secreting Tumor) i.e Secondary Hyperthyroidism → pituitary secretes too much TSH (central hyperthyroidism) → The only case in this table where the TSH is not suppressed in response to increased thyroid hormones! - Epoprostenol A drug used to treat pulmonary hypertension.	- Thyroiditis inflammation leads to destruction of follicular cells > leakage of all thyroxine> no radioiodine uptake! - Exogenous hyperthyroidism e.g. factitious hyperthyroidism مثال: وحدة سمينة راحت العطار تاخذ شيء ينزل وزنها.. that's why herbal medication history is very important. How to diagnose it? LOW THYROGLOBULIN → precursor of endogenous thyroid hormone (low means source is not endogenous) - Ectopic hyperthyroidism ex: struma ovarii Extra thyroid hormones coming from other than the thyroid gland OSCE STATION : give me 3 causes of low uptake?

Thyroid function test:

Hyperthyroidism			
Types	**Clinical Hyperthyroidism**	**Subclinical Hyperthyroidism**	**TSH Mediated Hyperthyroidism** Secondary hypothyroidism
Blood Test	FT4 high FT3 high TSH low وفي حالات خاصة يكون بس T3 مرتفع! لأن T4 بيتحول لT3 بكثرة فيظهر لنا T4 طبيعي	FT4 normal FT3 normal (upper borderline!) TSH low the pituitary is sensing the overproduction of thyroid hormones so TSH decreases before the level T3, T4 get high	FT4 high FT3 high **TSH high** TSH secreting tumor (not responding to - feedback)

Clinical Features:

Clinical Manifestation Of Hyperthyroidism (↑ Thyroxine)	
skin	Sweating, Moist warm skin, palmar erythema, thin hair
Brain	Hyperthermia, Heat intolerance, Increase appetite, Anxiety, Hand tremor
GIT	Hyperdefecation, Loose bowel motion, Increase gluconeogenesis (Failure of controlling a known DM)
Renal	Urinary frequency
Heart	Palpitation, Sinus tachycardia, Atrial fibrillation
Eye 	Eyelid lag, Eyelid retraction (staring gaze) Why? because thyroid hormones potentiate the effect of sympathetic innervation on the eyelid muscle (levator palpebra) → contraction.
Bone	Bone fracture, Osteoporosis, Hypercalcemia
Muscles	Muscle wasting & weakness, Hyperreflexia, Weight loss
Reproductive	Female: Menstrual cycles disturbances (Oligo-or amenorrhea)/ Male: ED

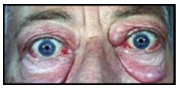
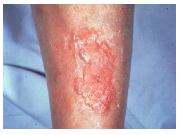
What is Grave's Disease?

- Most common cause of hyperthyroidism (up to 80% of all cases of hyperthyroidism).
- Autoimmune disease. Women are 9 times more affected. Typical age 30-50 yrs (younger if family history).

➔ **Pathophysiology:** The thyrotoxicosis in Grave's disease results from the production of IgG antibodies directed against the TSH receptor (TSH receptor antibodies, TSH stimulating antibodies), stimulation of TSH receptor on the thyroid follicular cells increases thyroid hormone production & cell proliferation (Diffuse goiter in most cases).

➔ **Clinical features:**

- General manifestations of hyperthyroidism (mentioned in table above)
- Specific symptoms seen ONLY in Grave's:

Grave's ophthalmopathy:	Exophthalmos (proptosis) 	Conjunctival irritation & periorbital edema لو ضغطت عليه بتطلع معك دموع وهذا نسميه chemosis 	Diplopia (double vision)
Grave's dermopathy:	Pretibial myxedema (red, raised patches) thick, hard, indurated skin. usually in elderly		
Grave's acropachy:	Similar to "clubbing"		

For your information: Both Grave's ophthalmopathy & dermopathy share similar pathogenesis, which is immunologically mediated → antibodies stimulate fibroblasts → secretion of glycosaminoglycans → swelling → inflammation & fibrosis → behind the eye, pushing it outward (Exophthalmos). On skin, forming inflamed patches (Pretibial myxedema)

NOTES FROM MALE'S DOCTOR:-Neonatal graves disease: it occurs when the mother has or had graves disease. TSH receptor antibodies cross through placenta and affect the thyroid gland in growing baby. A pediatrician should be involved in labor room.

- **Complications:** #someone who has graves and developed an infection on top, they'll have thyrotoxic storm. it's a medical emergency! patient may present with : fever - Agitation - tremor - Afib - HF.

- **treatment of the crisis:**
  1- Large dose of anti-thyroid medications
  2- Dexamethasone. why? to stop the conversion of T4 to T3. (active form)
  3- IV fluids
- **thyrotoxic periodic paralysis:** Abrupt onset of paralysis due to hypokalemia! it results from an intracellular shift of potassium induced by the thyroid hormone. most commonly seen in asian men.

Diagnosis:

Diagnostic approach of Hyperthyroidism	
Clinically	- History*, clinical symptoms, & signs *when taking history never forget to ask about: 1- exposure to ionizing radiation 2- iodine ingestion(iodine containing cough preparations-iodine containing contrast material-Amiodarone) 3- Residency(poor iodine ingestion) 4- family history of thyroid disorders or any autoimmune diseases. - History of anemia they can present with any type of anemia. Normal, low, and high MCV - other autoimmune diseases - History of recent pregnancy - Family history
Bio-chemically	- **Clinical Hyperthyroidism:** FT4 high, FT3 high, TSH low - **Subclinical Hyperthyroidism:** FT4 normal, FT3 normal, TSH low - **TSH Mediated Hyperthyroidism**: FT4 high, FT3 high, TSH high - **Thyroid antibodies:** TPO(Thyroid peroxidase antibody), Anti TG (Anti-Thyroglobulin) - **SH Receptor antibodies (Specific for Graves)** - **Other blood test:** CBC for anemia, LFT high ALP, ESR
Radiologi-cally	- **Thyroid Scan:** to differentiate b/w etiology - **Thyroid Ultrasound with doppler** → high flow (Graves) do US especially if you find nodules during examination

Treatment:

Treatment Of Hyperthyroidism	
Normal Or High Radioiodine Uptake	**Low Radioiodine Uptake**
1. **Anti-thyroid medications:** - Methimazole, - PTU Propylthiouracil very dangerous, can cause liver damage, only used in 1st trimester of pregnancy) - Carbimazole. most commonly used & for pregnancy after 1st trimester #what's the most common complication of anti-thyroid medications? Agranulocytosis. 2. **Beta Blockers:**Propranolol, Metoprolol to calm the sympathetic activity 3. **RAIRx** destroy cells > so we give thyroxine 4. **Thyroidectomy** rarely performed, if large goitre or many side effects with medications. Most common complications are : 1- Numbness around face and in hands from hypocalcemia 2- change in voice (recurrent laryngeal nerve damage)	1. **Stop exogenous intake** 2. **Beta Blockers:** Propranolol, Metoprolol 3. **Monitor** every 2-3 months to exclude development of hypothyroidism **#Note**:NO anti-thyroid used !

▸**MALE'S DR. NOTES REGARDING THE RX:**
- how to treat exophthalmus?

1- if active > steroids
2- inactive > Refer to ophthalmology for decompression.

- if someone is taking Amiodarone and developed hyperthyroidism, Do not stop the medication! you should treat the complications. treatment depend on the type of hyperthyroidism he developed:
  1- if graves type → treat with anti-thyroid.
  2- if thyroiditis type → symptomatic treatment (Beta-blockers, Aspirin, paracetamol)

- If i ask you how to treat toxic adenoma or toxic multinodular goitre, is it by medication, surgery or RAI ? you should answer ALL. medications to start with, then surgery or RAI .

Hypothyroidism

Causes:

Etiology of hypothyroidism		
Primary Causes primary = source of problem = thyroid itself	**Secondary hypothyroidism** Secondary = Source of problem = pituitary	**Medications**
Inflammation of Thyroid gland (Hashimoto's Thyroiditis) most common cause	Pituitary → decreased TSH → decreased thyroid hormones Tertiary causes: hypothalamus fails to secrete TRH.	e.g. Lithium → inhibits thyroid hormones release → hypothyroidism
Enlargement of Thyroid Goiter - Endemic Colloid Goiter caused by Dietary Iodide deficiency No iodide > No Thyroid Hormones (T4, T3) > Increase release TSH > Goiter - Idiopathic Non-toxic colloid goiter inflammation > TSH release > goiter		
Destruction of Thyroid gland by irradiation.		
Surgical Removal of Thyroid gland.		
other causes:		
peripheral resistance of thyroid: mutation in thyroid hormone receptors in cells leading to High hormones in blood but symptoms of hypothyroidism		

Thyroid function test:

Hypothyroidism			
Types	**Clinical Hypothyroidism**	**Subclinical Hypothyroidism**	**Secondary Hypothyroidism**
Blood Test	- FT4 Low - FT3 Low - TSH High	- FT4 (Low normal) - FT3 (Low normal) - TSH Raised Treat if symptomatic, or if: 1- autoimmune causes(it progress with time) 2- pregnancy 3- someone with dyslipidemia 4- psychiatric illness 5- goiter	- FT4 Low - FT3 Low - **TSH Low or Normal**

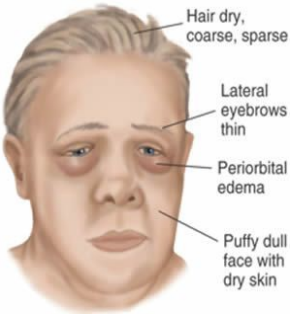
What is Hashimoto's Thyroiditis?

- Most common cause of hypothyroidism.
- Autoimmune disease. (affects women more)
  - ➔ **Pathophysiology:** Hashimoto's thyroiditis is characterized by formation of antibodies against Thyroglobulin (thyroid hormone precursor) and Thyroid peroxidase (important thyroid enzyme) destructive lymphoid infiltration of the thyroid, ultimately leading to a varying degree of fibrosis and thyroid enlargement. (Diffuse goiter)
  - ➔ **Course of the disease:** initially might cause hyperthyroidism (hashitoxicosis), then euthyroidism (most patients stop here) others progress to hypothyroidism.
  - ➔ **Complication:** There is an increased risk of thyroid lymphoma.

Antibody test: Anti-TPO (highly elevated in several forms of thyroiditis, mildly elevated in normal person)
Confirmation of the diagnosis done via FNA ⟶ lymphoid infiltration.

Clinical Features:

Clinical Manifestation of Hypothyroidism ***DECREASE FUNCTIONS OF THE ORGANS "except menstrual flow"***	
General	Myxedematous Appearance (caused by infiltration of mucopolysaccharides) pericardial/pleural effusion , carpal tunnel syndrome. Weight gain
skin	Scaliness of skin, Brittle hair and loss of outer eyebrow
Brain	Cognitive dysfunction, Hypothermia, Cold Intolerance, Extreme Somnolence (sleepiness), Decrease Appetite مريض كبير في السن جاي بس عنده كنفيوجن ناسي وين ملبق سيارته. وفيه بروفسر ماقدر يرجع بيته لأنه ضيع الطريق
GIT	Constipation
Renal	Oliguria electrolyte abnormality seen is hyponatremia
Heart	Bradycardia
Eye	Periorbital Edema (Non-pitting & again caused by infiltration of mucopolysaccharides)
Muscles	Proximal Myopathy, Fatigue, Delayed Relaxation Reflexes.
Reproductive	Male ⟶ Loss of libido. Women ⟶ Menorrhagia.



Hypothyroidism patients are prone to develop Atherosclerosis. Why?Because lack of Thyroid hormone ⟶ decrease liver uptake of LDL and decrease secretion of cholesterol in bile ⟶ Increase blood cholesterol ⟶ Atherosclerosis

▸ **NOTES REGARDING MYXEDEMA COMA :**

- Myxedema coma is defined as severe hypothyroidism leading to decreased mental status, hypothermia, and other symptoms related to slowing of function in multiple organs. It is a medical emergency with a high mortality r**ate.**
- treatment:
  1) Steroids. why? it could be due to pituitary causes, therefore ACTH secretion may be affected!
  2) IV thyroxine

Diagnosis:

Hypothyroidism		
Biochemical	**ECG**	**Serology**
- Primary Hypothyroidism:High TSH, Low T4 - Secondary Hypothyroidism: Low TSH, Low T4	In severe hypothyroidism; ECG shows sinus bradycardia, low-voltage complexes, and ST segment and T wave abnormalities.	- Thyroid Peroxidase autoantibodies. - Anti-Thyroglobulin Antibodies.

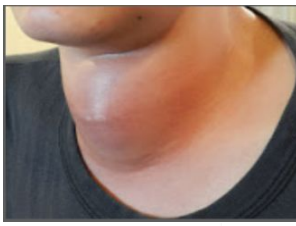
Treatment:

- Levothyroxine Replacement.
  #Every morning 30 min. before breakfast. dose depend on dysfunction but max. is 1.7 microg./kg/d.
  #When to measure the efficacy of the drug? 6-8 weeks after starting treatment.
  #Suppose you gave a woman thyroxine. after 3 months TSH level was still high although she's compliant and correctly treated! what could be the cause? 1- Absorption. patient may took other drug that affected the absorption of thyroxine ex: IRON!! 2- PREGNANCY (increase the dose in pregnant women)

Subacute Thyroiditis (De Quervain)

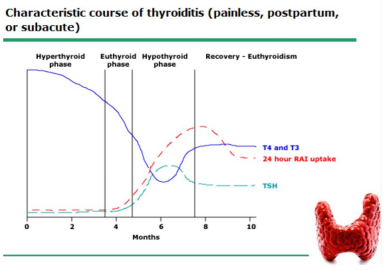
What is it ?

- **Subacute granulomatous thyroiditis** is characterized by:neck pain or discomfort, **a tender diffuse goiter**, and a predictable course of thyroid function evolution.
- **Etiology: viral** infection ex: 1-2 week history of URTI. now presented with neck pain



Clinical Presentation:

1. **Painful, tender and swollen gland**
2. Malaise, fever, chills, and night sweats
3. On Examination: tender, warm, goiter
4. Hyperthyroidism is typically the presentation followed by euthyroidism, hypothyroidism, and ultimately restoration of normal thyroid function thyroid regress to its normal size



Diagnostic Approach:

Clinically	History (of viral infection), clinical symptoms, & signs.
Biochemically	- **Hyperthyroidism**: FT4 high, TSH low - **Hypothyroidism**: FT4 low,, TSH high - **Other blood test;** CBC, LFT, ESR, C-RP
Radiologically	- **Thyroid Ultrasound with dollar low flow** نعمله خصوصا اذا فيه pus - **Thyroid scan → low uptake**

Treatment:

1. **Pain control:** Aspirin, other anti-inflammatory drugs NSAIDS, or Glucocorticoids
2. **Hyperthyroidism:** Beta-adrenergic blockers for **thyrotoxic symptoms**
3. **Hypothyroidism:** L-thyroxine for hypothyroidism

- follow up is very important in these patients due to fluctuating hormone levels

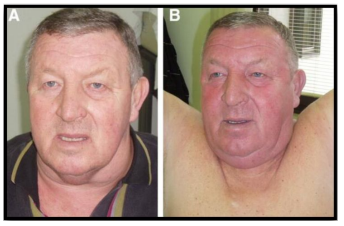
Thyroid enlargement

Clinical history & signs:

- Neck mass, the onset, the growth of the mass, how fast?
- Change in the skin over it can be underlying TB

  Pressure Symptoms:
  - Choking symptoms
  - Swallowing difficulties
  - Hoarseness of the voice
  - Stridor
- History of head & neck irradiation during childhood, whole body irradiation for bone marrow transplant hx of: lymphoma, leukemia
- Family history of thyroid nodules or thyroid cancer, family history of other endocrine tumors; MEN1,2.

Pemberton sign[1]



Endemic Iodine deficiency area

A) Goiter:

Causes:

- **Endemic Goiter: common in china and central africa.** As a result of the iodine deficient soil.
- **Sporadic Goiter: multinodular goiter.**
- **Familial**
- **Hashimoto's Thyroiditis:** **in early stage**
- **Graves' disease: due to chronic stimulation of TSH receptor**
- **Diet: cabbage,Cauliflower**
- **Chronic Iodine Excess**
- **Medication: lithium in 6%**
- **Neoplasm**

B) Thyroid Nodules:

Needs clinical attention when noted:

- By the patient.
- By a clinician during routine physical examination.
- During a radiologic procedure.

Their clinical importance:

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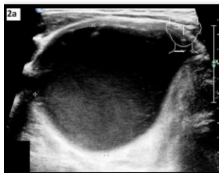
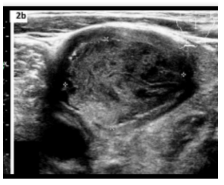
[1] what is it ?click here

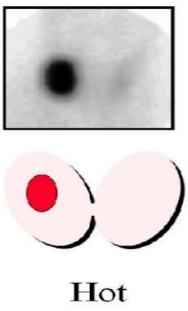
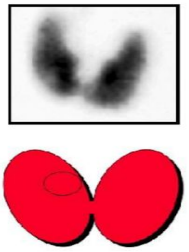
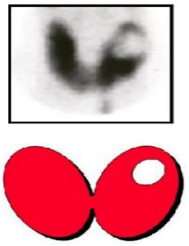
- The need to **exclude thyroid cancer**, which accounts for 4 - 6.5 % of all thyroid nodules in nonsurgical cases.
- The presence of **pressure symptoms**.

Causes:

- **Thyroid cyst**
- **Follicular adenoma**
- **Prominent nodule in multinodular goiter**
- **Cancer**

Thyroid enlargement	
Physical examination + US (the best to assess suspected structural thyroid disorder) **→ The cause is either:**	
Diffuse thyroid enlargement	**Thyroid nodules**
Investigations	
• **TFT (is it hypo- eu- or hyperthyroidism)**	
-Radioactive uptake scan if TSH low **-?Antibodies** **-CT (when pressure symptoms or retrosternal goiter)** **-Biopsy, FNA**	**-FNA (when > 1 cm)** **-Thyroid scan (is it hot,cold or warm) if FNA is indeterminate (can't tell if benign or malignant) or if low TSH.**
Management	
Treat underlying cause. **Surgery (thyroidectomy) if:** -Pressure symptoms -Malignancy -Cosmetic	**Observation if:** -Less than 1 cm -More than 1 cm & FNA showed benign pattern **Surgery if:** -Suspicious (malignant) -> as seen on FNA -Pressure symptoms

Radiological investigations of thyroid nodule		
	Low risk of malignancy	**High risk of malignancy (high**
Thyroid US	- Cystic no solid component (pure cystic). - Spongiform Thyroid Cyst 	- Solid with irregular margins. - Presence of microcalcification. - Hypervascularity. Thyroid Nodule 

Thyroid scan (scintigraphy)	Nodules detected by thyroid scans are classified as cold, hot, or warm. If a nodule is composed of cells that do not make thyroid hormone (don't absorb iodine), then it will appear "cold". A nodule that is producing too much hormone will show up darker and is called "hot." If appearing “warm” it’s producing normal amount of hormones.		
	Hot: low risk of malignancy.  Hot	Warm: low risk of malignancy.  Warm	Cold:Higher risk of malignancy (although most cold nodules are benign).  Cold

Thyroid Cancer

دكتور الاولاد ماتكلم عنها

Overview: Types Of Thyroid Cancers (enjoy this part in details when studying surgery)			
Papillary	**Follicular**	**Medullary**	**Anaplastic**
- Most common - Best Prognosis - Rx:Total Thyroidectomy, Radioactive-Iodine (When radioactive iodine (RAI), also known as I-131, is taken into the body in liquid or capsule form, it concentrates in thyroid cells. The radiation can destroy the thyroid gland and any other thyroid cells (including cancer cells) that take up iodine, with little effect on the rest of your body).	- Good Prognosis - Rx:Total Thyroidectomy, Radioactive-Iodine	- Affect C cells - Bad prognosis - Rx:Total Thyroidectomy - Risk of Lymph vascular invasion and Distant metastasis > Chemotherapy	- Worst Prognosis - Rapid Progression - High Risk of Lymphovascular invasion and Distant metastasis - No surgery is advised

Complication of Post Thyroidectomy:

- Hypothyroidism :Needs Thyroid hormone replacement
- Hypocalcemia:Secondary to parathyroidectomy
- Hoarseness:due to Injury to recurrent laryngeal nerve
- Infection
- Bleeding
- Needs for Tracheostomy

Hx & Physical Exam of Thyroid

Do not skip it

C: Clinical (History)	**1. Sympathetic Activation** - Nervousness, Anxiety, Increased perspiration, Heat intolerance, Hyperactivity, Palpitations **2. Cardiovascular Symptoms:** - (SOB, atrial fibrillation) and unexplained weight loss **3. Ophthalmopathy (Graves disease)** **4. Autoimmune disease** **5. Radiation exposure** **6. family history** **7. medications and dietary**
C: Clinical (Examination)	1. Tachycardia or atrial arrhythmia ,Systolic hypertension with wide pulse pressure ,Warm, moist, smooth skin 2. Lid lag ,Stare 3. Hand tremor, Muscle weakness **4. Thyroid examination:** - diffusely enlarged and slightly **firm** + bruit → Grave's Disease (GD) - enlarged (2 - 3 x) + **soft** → MNG - enlarged and painful → ○ Subacute painful or granulomatous thyroiditis ○ degeneration or hemorrhage into a nodule and suppurative thyroiditis 5. **Ophthalmologic examination:** - 50% Grave's Disease (GD) have Graves' orbitopathy (GO): ○ periorbital edema ○ conjunctival edema (chemosis) ○ Injection ○ poor lid closure ○ extraocular muscle dysfunction (diplopia) ○ Proptosis **6. Dermatologic examination:** - Pretibial myxedema - deposition of glycosaminoglycans in the dermis of the lower leg - non- pitting edema, erythema and thickening of the skin, without pain or pruritus - orange peel in color and texture.
B: Biochemical	**1. Thyroid function tests** - Free T4 - TSH **2. Autoantibody tests:** - Anti- TPO ○ (most specific autoantibody test for autoimmune thyroiditis), high titer ○ Could be mildly elevated in normal person - TSI (TSH receptor Ab) ○ GD
A: Anatomical	**Thyroid uptake scan:** - Technetium-99m (99m Tc) - Iodine-123 (123 I)

MCQs

1) A 35 years female presented with a neck swelling for 3 months. On palpation thyroid was soft and enlarged. Her free T4 was 56.2 (High) and TSH <0.01 (Low). What is the best radiological modality to assess her condition ?

a. Chest X-ray of the neck and chest
b. CT of the neck and upper chest
c. Thyroid ultrasound
d. Radioactive uptake

2) A 38-year-old female presented with weight gain and fatigue for the past 3 months. She had small goiter. Her free T4 is 7.2, and TSH is 40. What is the most likely differential diagnosis ?

a. Grave's Disease
b. Hashimoto's thyroiditis
c. Secondary hypothyroidism
d. Sheehaan's Syndrome

3) 25-year-old female presented with palpitation, weight loss, exophthalmos and toxic goiter on examination. What is the diagnosis ?

a. Graves' disease
b. Hashimoto's thyroiditis
c. thyroxine use or overdose
d. adenoma

4) A pregnant lady came with eye exophthalmos and tachycardia. later on she was diagnosed with grave's diseases, what is the best management in her case ?

a. Propylthiouracil
b. Surgery
c. Radioactive iodine
d. None of them

5) A 35 year old female presented to you with a 6 month history of palpitation, tremor, heat intolerance, and weight loss. which one of the following results of investigation is consistent with the patient's symptoms?

a. TSH ↓ Free T4 ↓ Free T3 ↓
b. TSH ↓ Free T4 ↑ Free T3 ↑
c. TSH ↑ Free T4 ↑ Free T3 ↓
d. TSH ↑ Free T4 ↓ Free T3 ↓

6) 25 years old female complaining of palpitation, oligomenorrhea, lack of concentration for last 8 months. On physical examination she had normal BMI, heart rate of 120 beats/min, exophthalmos, conjunctival congestion, large diffuse firm goiter and bruit, increase reflexes in the upper and lower limbs, pretibial myxedema. which one of the following is most likely the cause of her symptoms??

a. Hyperthyroidism due to toxic Adenoma
b. Hyperthyroidism due to exogenous thyrotoxin
c. Hyperthyroidism due to grave's disease
d. Hyperthyroidism due to follicular thyroid carcinoma

7) A 45 year old lady presented to the clinic with 4 month history of fatigue, muscle pain, progressive increase in her weight and tendency to sleep longer time. she also noticed occasionally abdominal pain and constipation. O/E found to be obese, dry skin, and peripheral weakness. what is the appropriate next step to reach diagnosis?

a. Calcium level
b. Thyroid function test
c. Colonoscopy
d. Ultrasound abdomen

Answer key:

1 (D) | 2 (B) | 3 (A) | 4 (A) | 5 (B) | 6 (C) | 7(B) |