433 Teams

# MEDICINE

## 10|Thyroid Disorders

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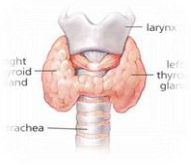
# Objectives:

| |
|---|
| 1. Describe the anatomy and physiology related to thyroid gland |
| 2. Know the Hypothyroidism: presentation, diagnosis and management |
| 3.Define the Goiter and thyroid nodules |

# Thyroid gland



Thyroid gland is made up of follicles and has 2 lobes and connected by the isthmus

- more volume in men, increase with age and bodyweight and decrease with iodine intake
- Located in front of larynx

99.9 % of T4 and T3 are bound to protein in the blood: TBG, albumin,

## Thyroid hormones:

Follicular cells of the thyroid is the main site of hormones synthesis

- Mainly T4 and small amount of T3
- T4 and T3 synthesis and secretion is regulated by pituitary TSH.
- TSH is inhibited by T4 and T3, stimulated by TRH

## Thyroid hormone action:

Thyroid hormones act on the bone and bone development

In children: delayed growth and epiphyseal growth

In brain: cognitive impairment

Regulate metabolic rate and little change in bodyweight

Act on cardiac muscle: tachy and bradycardia

## Goiter:

Chronic enlargement of thyroid gland not due to neoplasm

## What causes goiter:

Goiters can occur when the thyroid gland produces either

- too much thyroid hormone (hyperthyroidism)
- not enough (hypothyroidism)
- Normal production (nontoxic multinodular goiter)
- Sporadic: Diet (cabbage, Caulifower)

Assess thyroid function by :
Free T4, FT3
TSH
Ultrasound neck

# HYPOTHYROIDISM:

## Etiology

Failure of the thyroid to produce sufficient thyroid Hormone.
This accounts for about 95% of all cases.

| Primary | Secondary<br>↓TSH and ↓ freeT4 |
|---|---|
| **Hashimoto's thyroiditis:**<br>**(chronic thyroditis most common cause)** | Hypopituitarism due to:<br>a- Pituitary adenoma<br>b- pituitary ablative therapy<br>c- pituitary destruction |
| **Neonatal hypothyroidism due to placental transmission of TSH-R blocking antibodies** | **Tertiary** |
| **Iatrogenic: 2nd most common cuases**<br>**Radioactive iodine therapy**<br>**Subtotal thyroidectomy** | Hypothalamic dysfunction (rare) |
| **Iodide deficiency**<br>**Drugs:**<br>**as lithium, amiodarone, antithyroid drug therapy** | **Peripheral resistance of the action of thyroid hormone.** |

## Pathogenesis

-Thyroid hormone deficiency affects every tissue in the body, so that the symptoms are multiple

-Accumulation of glycosaminoglycans-mostly hyaluronic acid- in interstitial tissues Increase capillary permeability to albumin Interstitial edema (skin, heart muscle, striated muscle)

Hashimoto's thyroiditis is associated with other autoimmune disorders
(e.g., lupus, pernicious anemia).

Hashimoto's Thyroiditis starts with hyperthyroidism then hypothyroidism.

## Clinical features:

-Common feature: easy fatigability, coldness, weight gain, constipation, Menorrhagia, and muscle cramps.

-Physical findings: cool rough dry skin, puffy face and hands, hoarse husky voice, and slow reflexes, yellowish skin discoloration and non-pitting edema.

| Cardiovascular | Anemia | Neuro-muscular |
|---|---|---|
| -Bradycardia<br>-Decreased cardiac output<br>-Low voltage ECG (due to pericardial edema)<br>-Cardiomegaly<br>-Pericardial effusion<br>-Pulmonary function<br>Shallow and slow respiration<br>-Respiratory failure | Impaired hemoglobin synthesis<br>Iron deficiency<br>Folate deficiency<br>Pernicious anemia, with B12 deficient megaloblastic anemia | -Severe muscle cramps<br>-Paresthesias<br>-Muscle weakness<br>-Carpal tunnel syndrome |
| **GIT** | **Renal** | **CNS** |
| -Chronic constipation<br>-Ileus | -Impaired GFR<br>-Water intoxication | -Chronic fatigue<br>-Lethargy<br>-Decreased concentration<br>-Anovulatory cycles and infertility<br>-Menorrhagia<br>-Depression<br>-Agitation<br>-Diminished hearing |
| | | |

1-Severe hypothyroidism causes myxedema, but hyperthyroidism is associated with pretibial myxedema.

2-Almost all bodily processes are being slowed except the menstrual flow

## Diagnosis:

**All thyroid disorders are best tested first with a TSH**

- **Elevated serum TSH**
- **Low serum FT4 (or normal)**

High TSH (double normal) +normal T4 = treatment

Antithyroid peroxidase antibodies tell who needs thyroid replacement when T4 is normal and TSH is high,

If the TSH level is suppressed, measure free T4 levels.

TSH levels are markedly elevated if the gland has failed.

-Thyroid antibodies

-TRH stimulation test

## Complications:

1- Myxedema coma

-The end stage of untreated hypothyroidism

-Progressive weakness, stupor, hypothermia, hypoventilation, hypoglycemia, hyponatremia, water intoxication, shock, and death.

-Associate illnesses and precipitating factors: pneumonia, MI, cerebral thrombosis, GI bleeding, ileus, excessive fluid administration, and administration of sedatives and narcotics.

-Three main issues: CO2 retention and hypoxia, fluid and electrolyte imbalance, and hypothermia.

2- Myxedema and heart disease

3- Hypothyroidism and neuropsychiatric disease

## Treatment:

Replacing thyroid hormone with thyroxine (synthroid) is sufficient.

### Subclinical Hypothyroidism

• Thyroid function is inadequate, but increased TSH production maintains T4 level within the reference range
of normalcy; therefore, TSH level is elevated and T4 level is normal.
• Look for nonspecific or mild symptoms of hypothyroidism, as well as elevated serum LDL levels.
• Treat with thyroxine if patients develop goiter, hypercholesterolemia, symptoms of hypothyroidism, or significantly
elevated TSH level (>20 ⌈U/mL).

Indication to start treatment in sub-clinical hypothyroidism: pregnancy, huge goiter, elderly patient, psychotic patient, and adolescents.

## Treatment of Myxedema coma:

-Acute medical emergency

-Monitor blood gases

-Patient may need intubation and mechanical ventilation

-Give IV levothyroxine

-Asses adrenal function and treat if needed

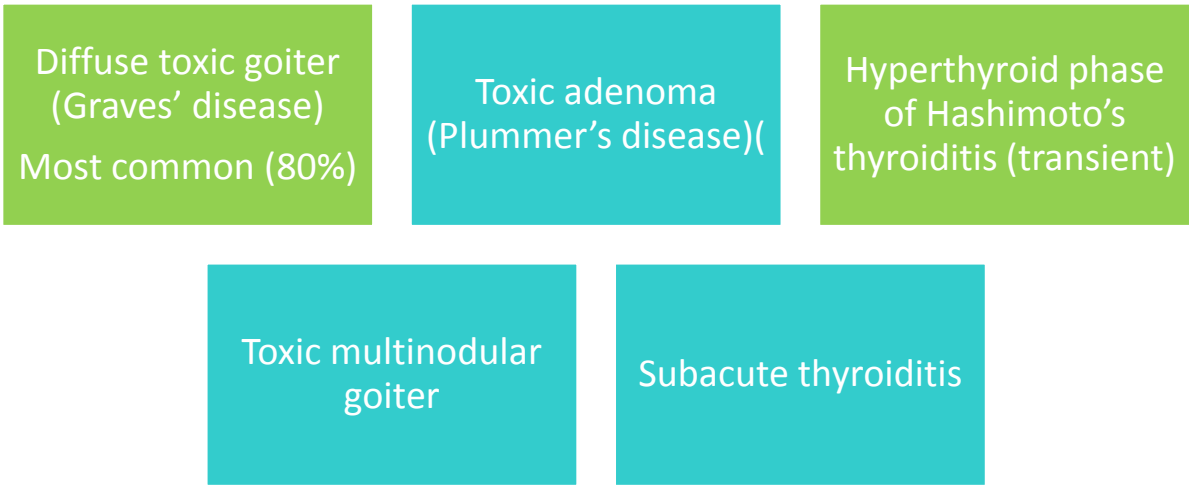
-In pituitary myxedema, glucocorticoid replacement is essential

## HYPERTHYROIDISM & THYROTOXICOSIS

-**Thyrotoxicosis**: is the clinical syndrome that results when tissues are exposed to high levels of circulating thyroid hormone

-**Hyperthyroidism**: is the hyperactivity of the thyroid gland

## Conditions associated with thyrotoxicosis:




## Diffuse Toxic Goiter (Graves' disease)

-Females > Males

(more often in younger women& associated with other autoimmune disorders)

## Etiology

-Autoimmune disease of unknown cause

-There is a strong familial predisposition

-Peak incidence in the 20- to 40- year age group

## Pathogenesis

-Local viral infection inflammatory reaction leading to the production of IFN-g and other cytokines by non-thyroid-specific infiltrating immune cells

will induce the expression of HLA class II molecules on the surface of thyroid follicular cells.

Subsequently, thyroid specific T-cells will recognize the antigen presented on the HLA class II molecules and will be activated

The activated thyroid-specific T-cells stimulate B cells to produce

**→TSH receptor-stimulating antibodies → hyperthyroidism**

## Clinical features:

| ***Skin*** | **Eyes** | **Cardiac** |
|---|---|---|
| -warm, excessive sweating<br>-Onycholysis, hyperpigmentation<br>-Pruritus, vitiligo, alopecia, thining of the hair<br>-Pretibial myoxedema | Extraocular muscles dysfunction: diplobia, proptosis, lid retraxtion,corneal ulceration, optic neuropathy and blindness<br>- Periorbital and conjunctival odema | -Atrial fibrillation in 10-20 %<br>-High output cardiac failure<br>-Wide pulse pressure, hypertension |
| ***Bone*** | **GIT** | **Respiratory** |
| Bone turnover increased: osteoporosis | Weight loss, diarrhoea, increase liver enzyme | Dyspnoea |

***Neuropsychiatry:***

- *Behavioral and personality changes: irritability, depression*

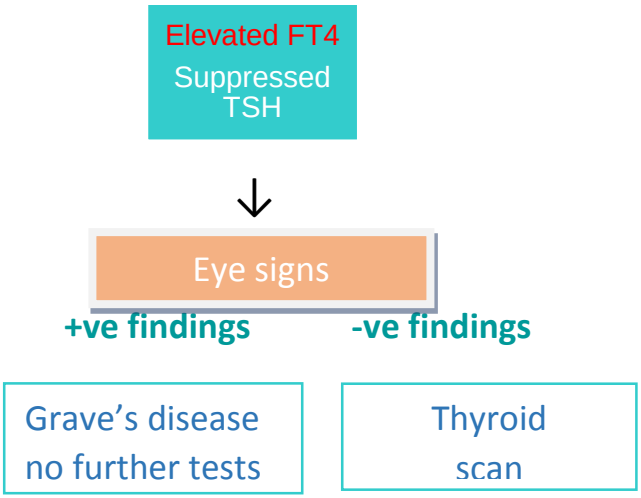
There are three signs of hyperthyroidism specific to Graves' disease:

• Exophthalmos • Pretibial myxedema • Thyroid bruit

## Diagnosis:

All forms of hyperthyroidism have an elevated T4 (thyroxine) level.
Only pituitary adenomas will have a high TSH level. In all the others, the pituitary release of TSH is inhibited.

Apathetic hyperthyroidism: no signs of thyrotoxicosis (in elderly patients presents with weight loss only)




| Diagnosis | TSH | RAIU | Confirmatory |
|---|---|---|---|
| **Graves disease** | low | Elevated | +ve Anti-bodies |
| **Subacute thyroiditis** | low | decreased | tenderness |
| **Painless "silent" thyroiditis** | Low | Decreased | none |
| **Exogenous thyroid hormone use** | low | decreased | History and involuted non-palpable gland |
| **Pituitary adenoma** | high | Not done | MRI of head |

## Treatment:

| Diagnosis | Treatment |
|---|---|
| Graves disease | Radioactive iodine |
| Subacute thyroiditis | Aspirin |
| Painless "silent" thyroiditis | none |
| Exogenous thyroid hormone use | Stop use |
| Pituitary adenoma | Surgery |

## Complications:

Thyrotoxic crisis (thyroid storm)

-Clinical features:

* Fever / Agitation

* Altered mental status

* Atrial fibrillation / Heart failure

**Treatment of Acute Hyperthyroidism and "Thyroid Storm":**

1. Propranolol: blocks target organ effect, inhibits peripheral conversion of T4--7T3
2. Thiourea drugs (methimazole and propylthiouracil): blocks hormone production
3. Iodinated contrast material (iopanoic acid and ipodate): blocks the peripheral conversion of T4 to the more active T3; also blocks the release of existing hormone
4. Steroids (hydrocortisone)
5. Radioactive iodine: ablates the gland for a permanent cure

What to look for in Hypothyroidism and Hyperthyroidism

| Hypothyroid | Hyperthyroid |
|---|---|
| **Bradycardia** | Tachycardia, palpitations, arrhythmia (atrial fibrillation) |
| **Constipation** | Diarrhea (hyperdefecation) |
| **Weight gain** | Weight loss |
| **Fatigue, lethargy, coma** | Anxiety, nervousness, restlessness |
| **Decreased reflexes** | Hyperreflexia |
| **Cold intolerance** | Heat intolerance |
| **Hypothermia (hair loss, edema)** | Fever |

## Thyroid Nodules:

These are incredibly common, and are palpable in as much as 5% of women and 1% of men. Ninety-five percent are benign (adenoma, colloid nodule, cyst).

Thyroid nodules are rarely associated with clinically apparent hyperfunctioning or hypofunctioning.

## Diagnostic Tests

Thyroid nodules >1 em must be biopsied with a fine-needle aspirate if there is normal thyroid function (T4/TSH). Nodules in those who are euthyroid should be biopsied. There is no need to ultrasound or do radionuclide scanning because these tests cannot exclude cancer.

Needle biopsy is the mainstay of thyroid nodule management.

**When a patient has a nodule:**

1. Perform thyroid function tests (TSH and T4).
2. If tests are normal, biopsy the gland.

# MCQs

**Q1. A 30-year-old woman complains of palpitations, fatigue, heat intolerance, and insomnia. She is otherwise healthy. She and her husband desire children and are not interested in contraception. On physical examination, her extremities are warm and she is tachycardic. There is diffuse thyroid enlargement and proptosis, as well as thickening of the skin in the pretibial area. Laboratory testing reveals a free T4 value of 3.2 ng/dL (normal 0.9-2.4) with an undetectably low TSH level. Radioiodine uptake at 24 hours is 42% (normal 10%-30%). What is the best treatment plan for this patient?**

A. Propylthiouracil

B. Radioactive iodine

C. Propranolol

D. Thyroid surgery

**Q2. A 20-year-old woman presents after recent upper respiratory infection. She complains of neck pain and heat intolerance. The thyroid is tender. Erythrocyte sedimentation rate is elevated; free thyroxine value is modestly elevated. What is the most likely diagnosis?**

A. Subacute thyroiditis

B. Graves disease

C. Factitious hyperthyroidism

D. Struma ovarii

**Q3. A 60-year-old woman comes to the emergency room in a coma. The patient's temperature is 32.2°C (90°F). She is bradycardic. Her thyroid gland is enlarged. There is diffuse hyporeflexia. BP is 100/60. Which of the following is the best next step in management?**

A. Await results of T4 and TSH.

B. Obtain T4 and TSH; begin intravenous thyroid hormone and glucocorticoid.

C. Begin rapid rewarming.

D. Obtain CT scan of the head.

| 1.A | 2.A | 3.B |
|---|---|---|

## Answers

**Q1. The answer is A.** Antithyroid drugs are the treatment of choice in a patient with Graves disease who may become pregnant. Iodine 131 has been used successfully in Graves disease and is a reasonable option if the patient is willing to practice secure contraception for at least 6 months. However, it often causes permanent hypothyroidism and may worsen ophthalmopathy in some patients. The treatment of choice is the oral agent propylthiouracil. Propylthiouracil is chosen in cases such as this owing to low transplacental transfer. Methimazole is preferred in men and non–childbearing women because it can be given once daily. Propranolol relieves the adrenergic symptoms resulting from Graves disease but will not treat the underlying disease. Subtotal thyroidectomy is reserved for thyrotoxic pregnant women who have had severe side effects to medication. Surgical complications include hypoparathyroidism and recurrent laryngeal nerve injury. Corticosteroids are used in thyroid storm but not in the stable patient with Graves disease.

**Q2. The answer is A.** A tender thyroid gland and elevated ESR make subacute thyroiditis a likely diagnosis. Hyperthyroid symptoms are common early in the illness. The condition is self-limited (usually lasting 6-8 weeks), so antithyroid drugs are not used. Beta-blockers can alleviate symptoms until the inflammation resolves.

**Q3. The answer is B.** The clinical picture strongly suggests myxedema coma. Unprovoked hypothermia is a particularly important sign. Myxedema coma constitutes a medical emergency; treatment should be started immediately. Should laboratory results fail to support the diagnosis, treatment can be stopped. An intravenous bolus of levothyroxine is given (500 µg loading dose), followed by daily intravenous doses (50-100 µg). Impaired adrenal reserve may accompany myxedema coma, so parenteral hydrocortisone is given concomitantly. Intravenous fluids are also needed but are less important than thyroxine and glucocorticoids; rewarming should be accomplished slowly, so as not to precipitate cardiac arrhythmias. If alveolar ventilation is compromised, then intubation may also be necessary. Hyponatremia and an elevated PCO2 are laboratory markers of severe myxedema. CT of the head would not be the first choice, since a structural brain lesion would not explain the hypothermia, diffuse goiter, or hyporeflexia seen in this case.

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