

Normal Physiological Function of the Thyroid Gland

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Introduction

- • The thyroid gland is a butterfly-shaped endocrine gland in the neck.
- • It plays a major role in metabolism, growth, and development.
- • Main hormones: T4 (thyroxine), T3 (triiodothyronine), rT3 (reverse T3), calcitonin.

Structure of the Thyroid Gland

- • Two lobes connected by the isthmus.
- • Follicles filled with colloid are the functional units.
- • Follicular cells produce T3 and T4; C cells produce calcitonin.

Thyroid Hormones

- • T4: Major secretory product, prohormone.
- • T3: Biologically active, mainly from peripheral T4 conversion.
- • rT3: Inactive, produced under stress, fasting, or illness.
- • Calcitonin: Lowers plasma calcium via osteoclast inhibition.

Regulation of Thyroid Hormones

- • Hypothalamus releases TRH → stimulates anterior pituitary to secrete TSH.
- • TSH stimulates thyroid gland to produce T3 and T4.
- • Negative feedback: High T3/T4 inhibit TRH and TSH secretion.

Peripheral Conversion and Regulation

- • 80% of T3 is derived from T4 deiodination in liver, kidney, muscle.
- • Type I/II deiodinases: $T4 \rightarrow T3$ (active).
- • Type III deiodinase: $T4 \rightarrow rT3$ (inactive).
- • Fasting, illness, or stress increase rT3 $\rightarrow$ reduces metabolic rate.

Metabolic Effects of Thyroid Hormones

- ****Carbohydrate Metabolism:****
 - • ↑ intestinal glucose absorption, gluconeogenesis, glycogenolysis
 - • ↑ insulin degradation and tissue responsiveness → mild hyperglycemia
- ****Fat Metabolism:****
 - • ↑ lipolysis, fatty acid oxidation, ↓ plasma cholesterol & triglycerides
 - • Hyperthyroidism → fat depletion, weight loss

Physiological Functions

- • CNS: Maturation & neuronal excitability
- • Growth: Skeletal and somatic development
- • Cardiovascular: ↑ heart rate & cardiac output
- • Thermogenesis: Maintains body temperature
- • Overall metabolism: Energy homeostasis

Summary

- • Thyroid gland regulates key metabolic, growth, and developmental processes.
- • Hormones (T3, T4, rT3, calcitonin) maintain homeostasis.
- • Peripheral and central regulation ensure tissue-specific effects.
- • Dysfunction → hypo- or hyperthyroidism with systemic consequences.

Hypothyroidism

Definition and Overview

- • Hypothyroidism is a clinical condition resulting from decreased production of thyroid hormones (T3 and T4).
- • Leads to a generalized slowing of metabolic processes.
- • It can be primary (thyroid gland failure) or secondary (pituitary/hypothalamic dysfunction).

Classification of Hypothyroidism

- • Primary: problem in the thyroid gland itself
- • Secondary: problem in the pituitary gland
- • Tertiary: problem in the hypothalamus

Primary Causes

Quick HIT 

Hashimoto thyroiditis is associated with other autoimmune disorders (e.g., lupus, pernicious anemia). Patients with Hashimoto thyroiditis are at an increased risk of thyroid carcinoma and thyroid lymphoma.

- Hashimoto's thyroiditis: autoimmune destruction of thyroid tissue (most common in developed countries)
- Iodine deficiency: inadequate iodine intake (common worldwide)
- Post-thyroidectomy or radioiodine therapy
- Drugs: amiodarone, lithium, interferon- α , antithyroid drugs

Secondary Causes

- • Pituitary tumors
- • Sheehan's syndrome
- • Pituitary apoplexy
- • Hypophysitis

Tertiary Causes

- • Hypothalamic tumors
- • Trauma or surgery
- • Radiation therapy to head/neck

2. Secondary hypothyroidism (due to pituitary disease; i.e., deficiency of TSH) and tertiary hypothyroidism (due to hypothalamic disease; i.e., deficiency of TRH) account for less than 5% of cases. **Both are associated with a low free T4 and a low TSH level.**

Pathophysiology

- • Low thyroid hormone levels → reduced basal metabolic rate.
- • Accumulation of mucopolysaccharides (myxedema).
- • Affects cardiovascular, gastrointestinal, and nervous systems.

Clinical Features –General

- Fatigue & Weakness: ↓ATP →easy tiring muscles
- Cold intolerance: ↓thermogenesis →feel cold
- Weight gain: ↓metabolism + fluid retention
- Dry skin & hair: ↓protein synthesis & sebum
- Constipation: ↓gut motility

Clinical Features –Cardiovascular & CNS

- Bradycardia: → $\downarrow$ sympathetic activity $\downarrow$ slower heart rate
- Hypotension: $\downarrow$ cardiac output
- Slow reflexes & cognition: $\downarrow$ neuronal metabolism
- Depression: $\downarrow$ neurotransmitter activity

Clinical Features –Skin & Hair

- Dry skin: ↓epidermal proliferation & sebaceous activity
- Hair thinning & brittle nails: ↓protein synthesis
- Puffiness: ↑mucopolysaccharides →fluid retention
- Myxedema: glycosaminoglycan accumulation →thickened skin

Clinical Features –GI & Reproductive

- Constipation: ↓gut motility
- Menstrual irregularities: ↓thyroid →GnRH-LH/FSH disruption
- Decreased fertility: hormonal imbalance
- Slow digestion: ↓enzymatic activity & motility

Clinical Features – Severe Cases

- Myxedema: non-pitting edema from glycosaminoglycans
- Myxedema coma: hypothermia, bradycardia, hypotension, CNS depression
- Life-threatening → ICU management

Diagnosis

- TSH & Free T4:
 - Primary: $\uparrow$ TSH, $\downarrow$ Free T4 $\rightarrow$ thyroid problem
 - Secondary/Tertiary: $\downarrow$ /normal TSH, $\downarrow$ Free T4 $\rightarrow$ central problem
- Anti-TPO antibodies: detect Hashimoto's
- Other labs: $\uparrow$ cholesterol, mild anemia

Treatment – General Principles

- Goal: restore normal thyroid hormone levels
- Monitor: symptoms + TSH/Free T4 every 6–8 weeks
- Mechanism: ↑ metabolism → improve energy, heart, GI motility, lipid profile, neural & reproductive function

Treatment – Medication

- Levothyroxine (T4): synthetic → converted to T3 → restores metabolic rate
- Effect: resolves fatigue, bradycardia, constipation, dry skin/hair
- Start low dose in elderly/cardiac patients → titrate by TSH
- Combination T4/T3: rarely used, for refractory cases

Special Considerations

- Pregnancy: ↑ hormone demand → increase levothyroxine, monitor TSH
- Cardiac disease: start low dose → avoid cardiac strain
- Myxedema coma: IV T4/T3 + supportive ICU care

Summary

- Hypothyroidism: thyroid hormone deficiency → slowed metabolism
- Clinical features: fatigue, weight gain, cold intolerance, dry skin, bradycardia, constipation, cognitive slowing
- Diagnosis: TSH & Free T4, Anti-TPO antibodies
- Treatment: levothyroxine replacement, dose adjustment, special care in pregnancy, cardiac disease, and severe cases

Hashimoto's Thyroiditis

An overview of the most common
cause of hypothyroidism

Definition

- Hashimoto's thyroiditis (chronic lymphocytic thyroiditis) is an autoimmune disorder in which the immune system attacks the thyroid gland, leading to chronic inflammation and gradual destruction of thyroid tissue, resulting in hypothyroidism.

Epidemiology

- - Most common cause of hypothyroidism in iodine-sufficient areas.
- - More common in women (female:male ratio $\approx 10:1$).
- - Typically occurs between ages 30–50.
- - Often associated with other autoimmune diseases.

Etiology and Pathogenesis

- - Genetic predisposition (HLA-DR3, DR5, and CTLA-4 polymorphisms).
- - Autoimmune destruction mediated by:
 - • Anti-thyroid peroxidase (anti-TPO) antibodies
 - • Anti-thyroglobulin (anti-Tg) antibodies
 - • Cytotoxic T-cell–mediated apoptosis
- - Leads to gradual thyroid failure.

Clinical Features

- - Painless, firm, and enlarged thyroid (goiter)
- - Symptoms of hypothyroidism:
 - • Fatigue, weight gain, cold intolerance
 - • Constipation, dry skin, hair loss
 - • Menstrual irregularities, depression
- - May rarely present initially with transient hyperthyroidism (Hashitoxicosis).

Investigations

- - Thyroid function tests:
 - Elevated TSH, low free T4 (hypothyroid pattern)
- - Autoantibodies:
 - Anti-TPO and anti-Tg antibodies positive
- - Ultrasound: diffuse hypoechogenicity and heterogeneity.
- - Fine-needle aspiration (FNA): lymphocytic infiltration.

Complications

- - Progression to hypothyroidism.
- - Association with other autoimmune disorders (e.g., type 1 diabetes, Addison's disease).
- - Rare risk of thyroid lymphoma.

Management

- - Lifelong thyroid hormone replacement (Levothyroxine).
- - Monitor TSH regularly to adjust dosage.
- - No specific treatment to stop autoimmunity.
- - Selenium supplementation may be beneficial in some cases.

Summary

- Hashimoto's thyroiditis is an autoimmune destruction of the thyroid gland.
- It is the leading cause of hypothyroidism in iodine-sufficient areas.
- Diagnosis: high TSH, low T4, positive anti-TPO/anti-Tg.
- Treatment: lifelong levothyroxine replacement.

Hyperthyroidism

Introduction

Hyperthyroidism, also referred to as thyrotoxicosis, represents a hypermetabolic clinical state resulting from excessive circulating thyroid hormones (triiodothyronine (T₃) and thyroxine (T₄)). This hormonal excess leads to exaggerated tissue responsiveness to catecholamines and an enhanced systemic metabolic rate, affecting multiple organ systems and clinically manifesting as symptoms like weight loss, tachycardia, and heat intolerance. Globally, the most common etiology is Graves' disease, an autoimmune disorder . Other significant causes include toxic multinodular goiter and toxic adenoma.

Thyrotoxicosis

- **Thyrotoxicosis means thyroid hormone excess.**
- **Hyperthyroidism- means excessive secretion of thyroid hormones by the thyroid.(over active thyroid)**

Causes of thyrotoxicosis

Thyrotoxicosis without hyperthyroidism

- Subacute thyroiditis
- Silent thyroiditis
- Amiodarone
- Ingestion of excess thyroid hormone (thyrotoxicosis factitia), or tissue e.g (Hamburger thyrotoxixcosis)

Causes of Thyrotoxicosis

Primary Hyperthyroidism:

- Grave`s Disease
- Toxic multinodular Goiter
- Toxic adenoma
- Activating mutation of the TSH receptor
- Struma ovarii

Causes of Thyrotoxicosis

Secondary hyperthyroidism:

- **TSH secreting pituitary tumor**
- **Thyroid Hormone resistance syndrome**
- **Chorionic Gonadotropin secreting tumor**

Grave`s Disease

- ❖ **Account for 60-80% of thyrotoxicosis**
- ❖ **Occur in 2% of women,o.2% in men**
- ❖ **More prevalent in areas with high iodine intake.**

Pathogenesis of Grave`s Disease

- ❑ **Combination of genetic factors and environmental factors esp.stress,high iodine intake.**
- ❑ **Caused by Thyroid stimulating Immunoglobulin-TSI: produced by thyroid and lymph-nodes, cross the placenta→neonatal hyperthyroidism.**
- ❑ **TPO antibodies are positive in 80% of patients with Grave`s disease.**
- ❑ **TSH receptor antibodies are not measured routinely, but are commonly present: thyroid-stimulating immunoglobulin (TSI) 80% positive, TSH-binding inhibitory immunoglobulin (TBII) 60-90% in Graves' disease.**

Clinical Manifestations of Grave's

Symptoms:

- **Hyperactivity, irritability**
- **Heat intolerance and sweating**
- **palpitation**
- **Fatigue and weakness**
- **Wt loss with increased appetite**
- **Diarrhea**
- **Amenorrhea, loss of libido**

Signs:

- **Tachycardia, palpitation, atrial fibrillation**
- **Tremors**
- **Goiter**
- **Warm moist skin**
- **Muscle weakness**
- **Lid lag, Lid retraction**
- **Gynecomastia**

Graves Ophthalmopathy

- Onset occur within 1 yr of the appearance of symptoms in 75%.
- May occur in the absence of Graves disease in 10%(pts have +ve thyroid antibodies).



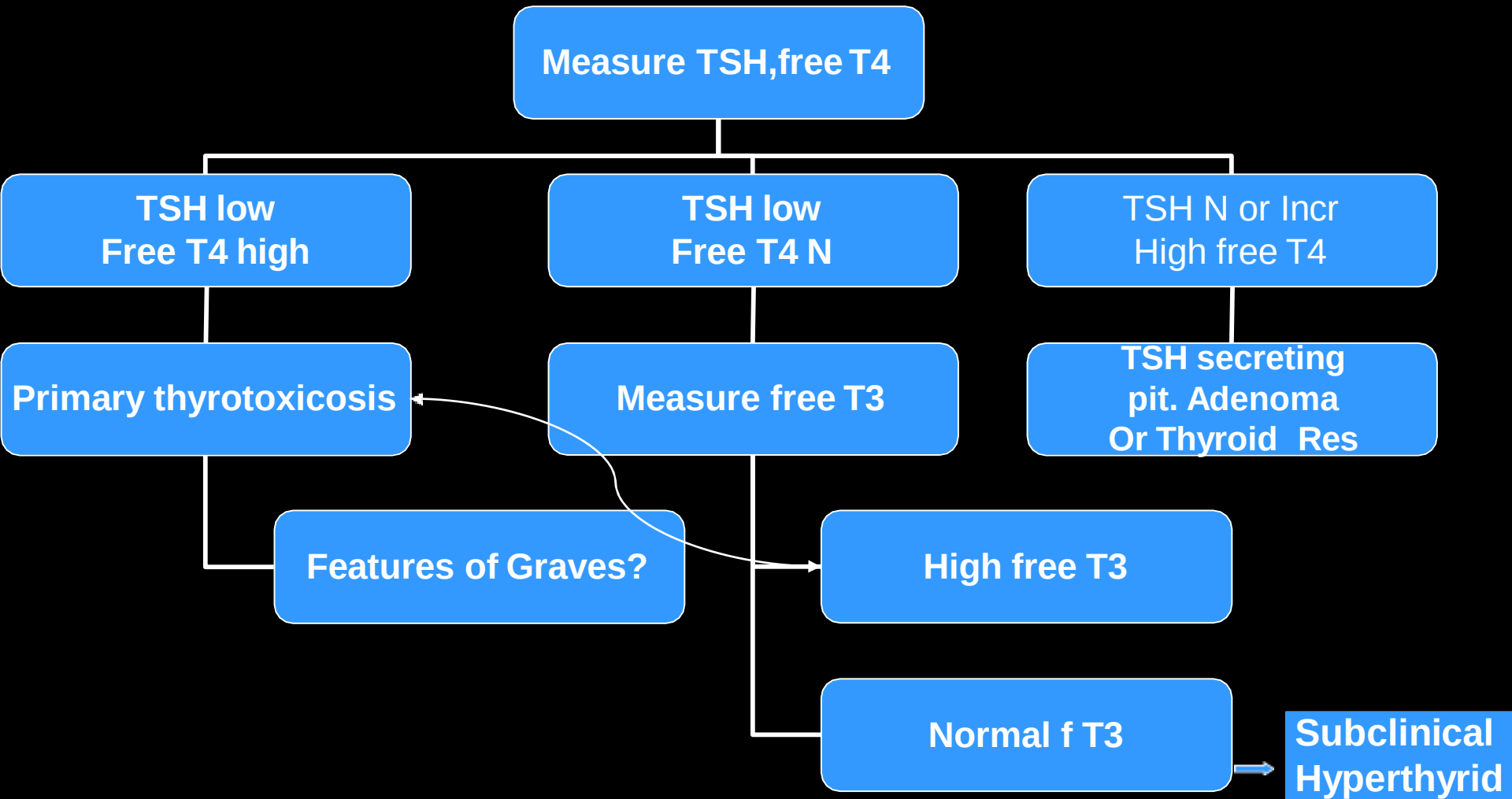
Treatment of G.ophthalmopathy

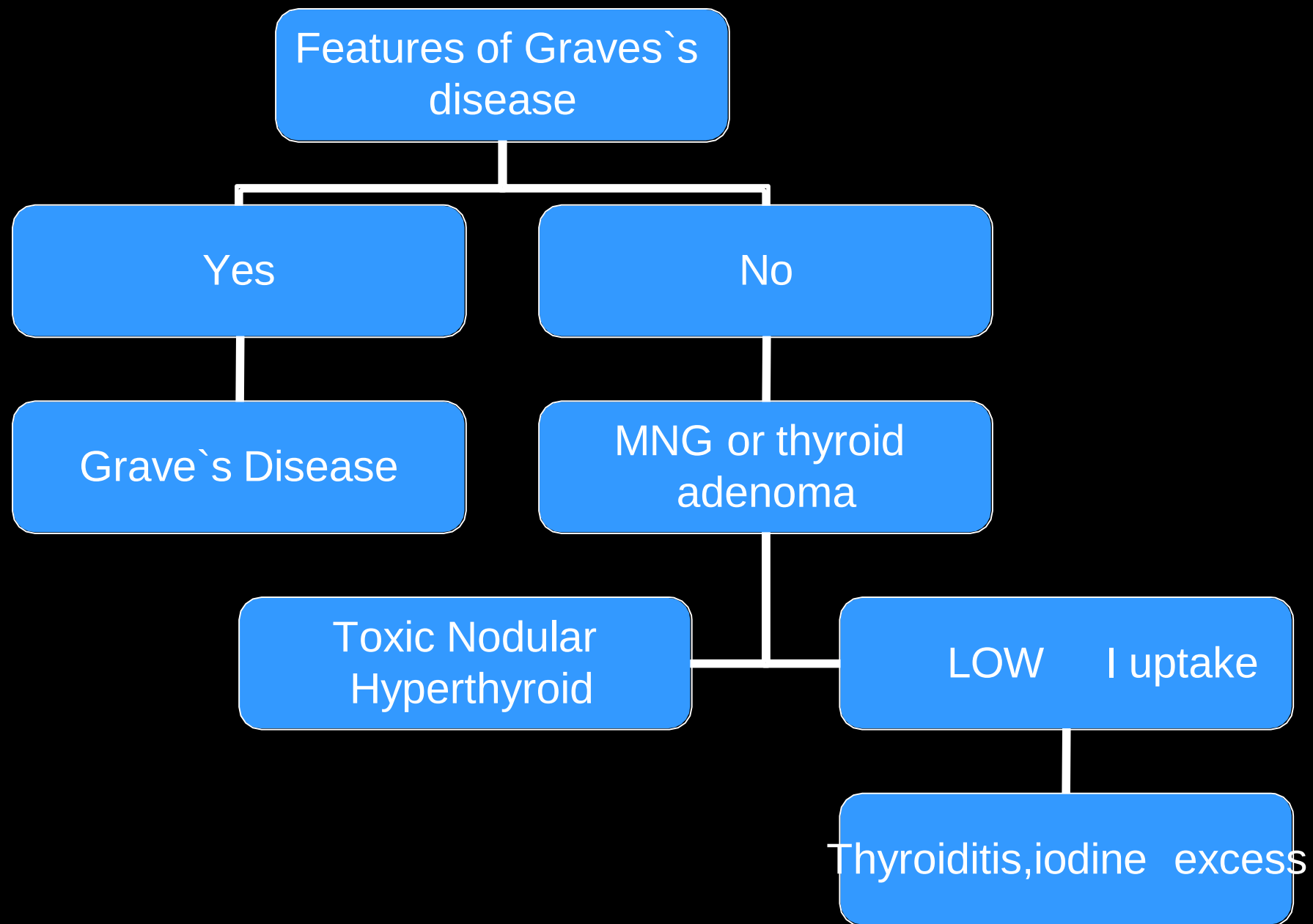
- **When mild give artificial tears (methylcellulose eye drops),elevate head of the bed, advise patient to use sunglasses, and stop smoking.**
- **For moderate disease: as above, plus prednisolone-taper slowly**
- **Severe form refer to ophthalmologist.**

Differential Diagnosis

- **Radionucleotide scan and ^{99}Tc , ^{123}I , ^{131}I will differentiate Graves disease from other causes like thyroiditis and Multinodular goiter.**

Laboratory Evaluation of Thyrotoxicosis





Treatment

- ☐ Anti thyroid Drugs
- ☐ Surgery
- ☐ Radio iodine
- ☐ Propranolol 40-80 mg every 6-8 hours

Anti Thyroid drugs

Thionamides:

- **Propyl thouracil (PTU) and carbimazole and methimazole.**
- **All inhibit TPO, reduce oxidation and organification of iodide, they also reduce thyroid antibodies by unknown mechanisms.**
- **PTU inhibits T4→T3 conversion.**
- **Start treatment with large doses of PTU(100-200 mg TID) or methimazole 10-20 mg every 12 hrs and taper dose gradually depending on response.**
- **Monitor T4 and TSH every 4 wks.**
- **TSH may remain suppressed for several months.**

- **Start carbimazole 20-40 mg daily.**
- **Review after 4-6 weeks and reduce dose of carbimazole depending on clinical state and T_4/T_3 levels. TSH levels may remain suppressed for several months and are unhelpful at this stage.**
- **When clinically and biochemically euthyroid, stop beta-blockers.**
- **Review after 2-3 months and, if controlled, reduce carbimazole.**
- **Gradually reduce dose to 5 mg daily over 6-24 months if hyperthyroidism remains controlled.**
- **When the patient is euthyroid on 5 mg daily carbimazole, discontinue.**

Anti thyroid drugs

Side effects are:

1. Rash-transient, improves with antihistamines.
2. Agranulocytosis :<1 %,pt develop fever and sore throat, the drug should be stopped immediately, and not used again,it is not useful to monitor WBC`s frequently as onset is uprupt.
3. Hepatitis.
- 4, SLE like syndrome.

Radioactive iodine treatment

- **Cause progressive destruction of thyroid gland.**
- **Can be used as initial treatment or after failure of thyroid drugs.**
- **Antithyroid drugs must be stopped at least 3 days before RAI.**
- **Risk of hypothyroidism is 10- 20% in first year, and 5% per year after that.**
- **Pregnancy and lactation are absolute contraindication.**

Surgery

- **Subtotal thyroidectomy is the treatment of choice for patients who relapse on antithyroid drugs.**
- **Careful control of thyrotoxicosis is needed by antithyroid drugs before surgery, followed by Potassium Iodide(sski) 3drops tid, it is needed to prevent thyrotoxic crisis.**
- **Complications of surgery :**
Bleeding, laryngeal edema, hypoparathyroidism, and damage to the recurrent laryngeal nerve are rare when performed by expert surgeon.

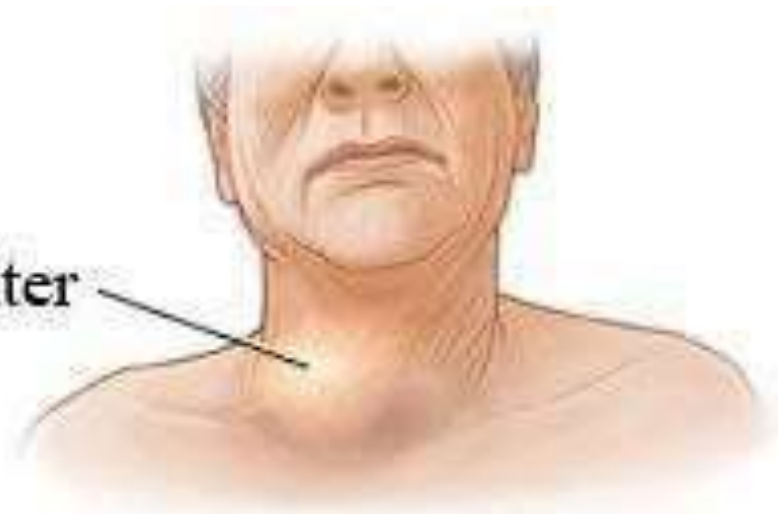
GOITRE (THYROID ENLARGEMENT)

- Goitres are present on examination in up to 9% of the population.
- Most commonly a goitre is noticed as a cosmetic defect by the patient or by friends or relatives.
- The majority are painless, but pain or discomfort can occur in acute varieties.
- Large goitres can produce dysphagia and difficulty in breathing, implying oesophageal or tracheal compression. medication, especially iodine-containing preparations, and possible exposure to radiation.

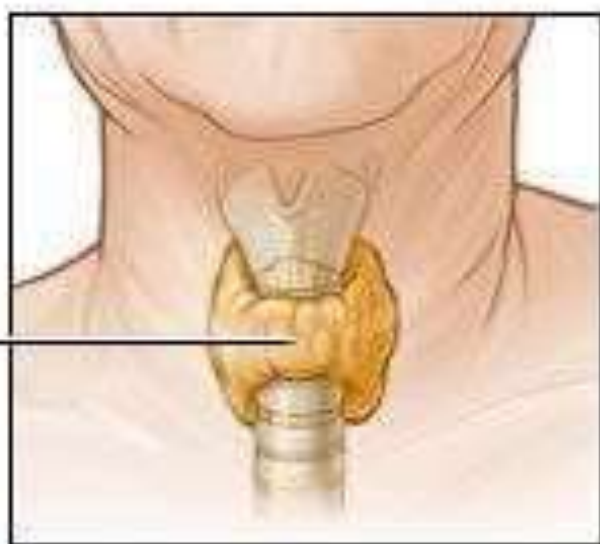


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Goiter



Thyroid
gland



Normal

Enlarged
thyroid
gland



Goiter

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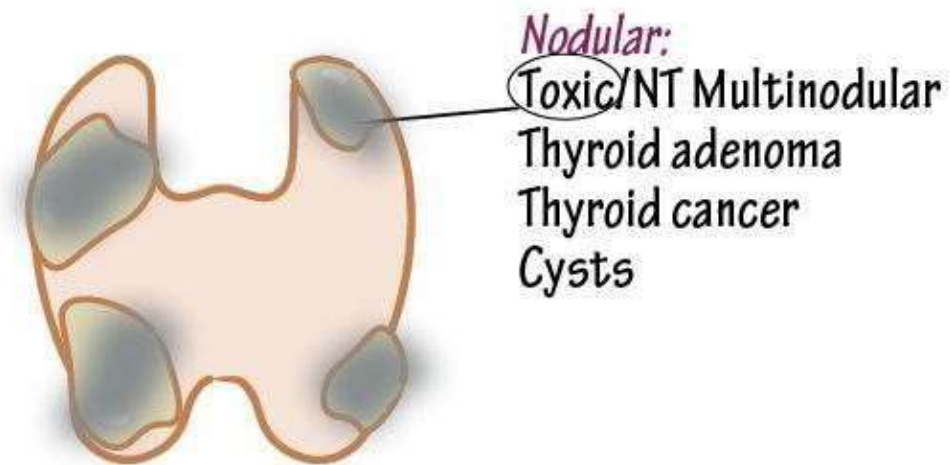
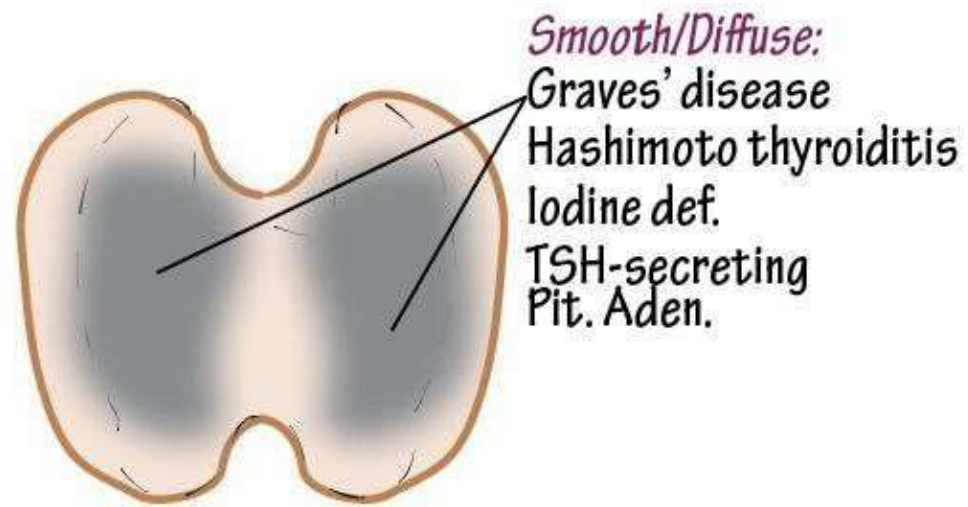
- **Clinical examination**
- **Size, shape, consistency and mobility of the gland as well as whether its lower margin can be demarcated (thus implying the absence of retrosternal extension).**
- **A bruit may be present.**
-
- **Associated lymph nodes should be sought and the tracheal position determined if possible.**
-
- **Examination should never omit an assessment of the patient's clinical thyroid status.**



Diffuse:

- **Simple**
- **Physiological (puberty, pregnancy)**
- **Autoimmune**
- **Graves' disease**
- **Hashimoto's disease**
- **Thyroiditis**
- **Acute (de Quervain's thyroiditis)**
- **Iodine deficiency (endemic goitre)**
- **Dyshormonogenesis**
- **Goitrogens (e.g. sulfonylureas)**

Goiter - Enlarged Thyroid



● = hot nodule on iodine uptake test

Nodular

- **Multinodular goitre**
- **Solitary nodular**
- **Fibrotic (Reidel's thyroiditis)**
- **Cysts**
- **Adenomas**
- **Carcinoma**
- **Lymphomas**

Miscellaneous

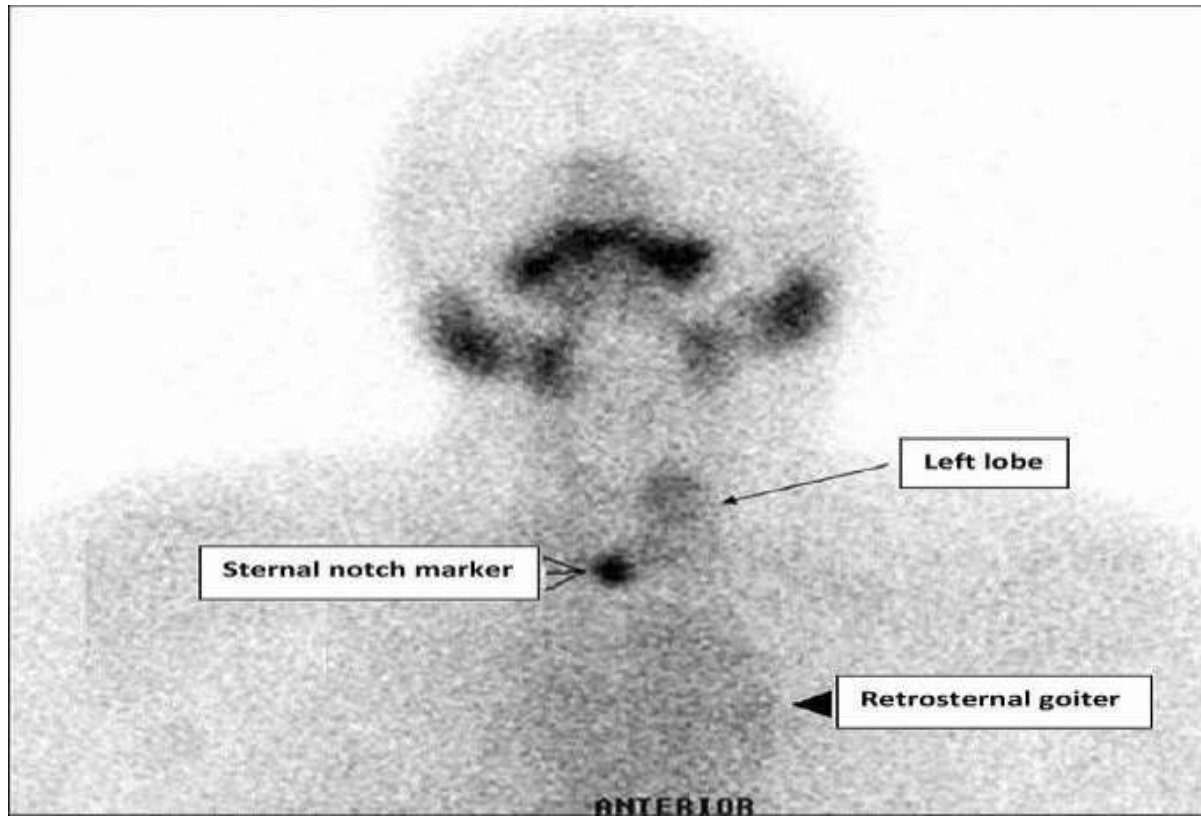
- **Sarcoidosis**
- **Tuberculosis**

Particular points of note are:

- **Puberty and pregnancy may produce a diffuse increase in size of the thyroid.**
- **Pain in a goitre may be caused by thyroiditis, bleeding into a cyst or (rarely) a thyroid tumour.**
- **Excessive doses of carbimazole or propylthiouracil will induce goitre.**
- **Iodine deficiency and dyshormonogenesis can also cause goitre.**

Investigations

- 1. Thyroid function tests - TSH plus free T₄ or T₃ .**
- 2. Thyroid antibodies - to exclude autoimmune aetiology.**
- 3. Ultrasound. Ultrasound with high resolution is a sensitive method for delineating nodules and can demonstrate whether they are cystic or solid. In addition, a multinodular goitre may be demonstrated when only a single nodule is palpable. Unfortunately, even cystic lesions can be malignant and thyroid tumours may arise within a multinodular goitre; therefore fine-needle aspiration is often required and performed under ultrasound control at the same time as the scan.**
- 4. Chest and thoracic inlet X-rays to detect tracheal compression and large retrosternal extensions in patients with very large goitre or clinical symptoms.**

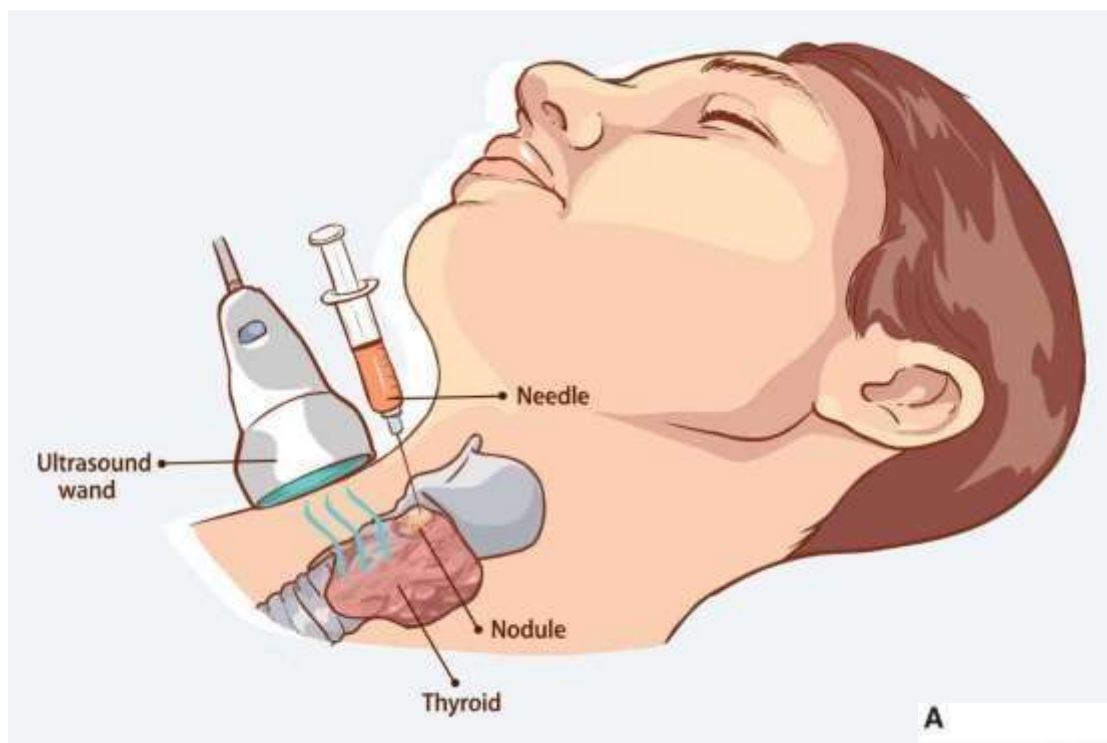


TSH	T4	T3	Interpretation
High	Normal	Normal	Hypothyroidism
Low	Normal	Normal	Hyperthyroidism
Normal	Normal	Low	Low T3 syndrome

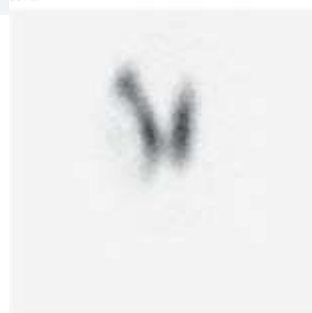
5. Fine-needle aspiration (FNA).

In patients with a solitary nodule or a dominant nodule in a multinodular goitre, there is a 5% chance of malignancy; in view of this, FNA should be performed. This can be done in the outpatient clinic. Cytology in expert hands can usually differentiate the suspicious or definitely malignant nodule. FNA reduces the necessity for surgery, but there is a 5% false-negative rate which must be borne in mind.

6. Thyroid scan (^{125}I or ^{131}I) can be useful to distinguish between functioning (hot) or non-functioning (cold) nodules. A hot nodule is only rarely malignant; however, a cold nodule is malignant in only 10% of cases and FNA has largely replaced isotope scans in the diagnosis of thyroid nodules



A



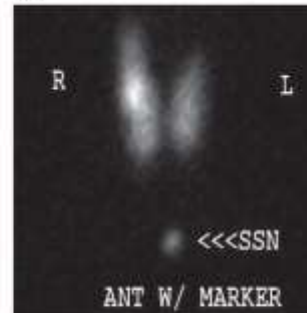
Right mid to lower pole cold nodule

B



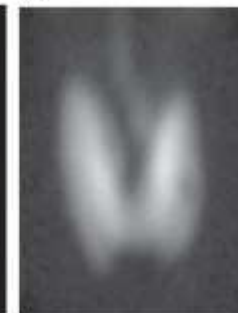
Right pole hot nodule

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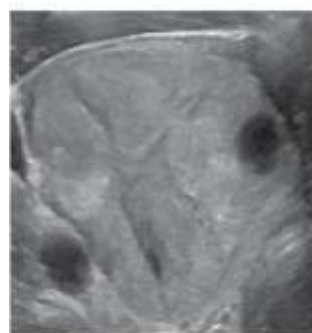


Right lateral mid pole nodule hot on thyroid scan

D



Left mid pole (LMP) cold thyroid nodule



Right mid to lower pole nodule benign by FNA



Ultrasound appearance benign right pole nodule



Right lateral mid pole nodule with benign ultrasound appearance



LMP nodule suspicious by US and PTC proven by cytology

Toxic Adenoma

Toxic Adenoma

Also called 'Hot Nodule'

A benign tumor of the thyroid that produces excess thyroid hormones

Definition

- A single thyroid nodule that autonomously secretes thyroid hormones
- Leads to hyperthyroidism
- Usually not cancerous

Causes / Risk Factors

- Genetic mutations (activating TSH receptor mutations)
- More common in older adults
- More frequent in women

Symptoms

- Weight loss despite normal/increased appetite
- Heat intolerance & sweating
- Palpitations / fast heart rate
- Tremors
- Nervousness / irritability

Signs

- Single palpable nodule in the thyroid
- Goiter may be absent or mild
- Signs of hyperthyroidism:
 - Fast heart rate
 - Warm, moist skin
 - Hand tremor

Investigations

- Blood tests:
 - Increased Free T4, Increased T3
 - Decreased TSH
- Scintigraphy (radioactive iodine scan): 'Hot nodule' takes up iodine
- Ultrasound: solid, well-defined nodule

Treatment

- Radioactive iodine therapy: destroys the adenoma
- Surgery (thyroidectomy): if large or compressive symptoms
- Beta-blockers: to control symptoms temporarily

Case 1

A 28-year-old woman presents with weight loss despite increased appetite, palpitations, heat intolerance, and anxiety. On examination, she has a diffusely enlarged, non-tender thyroid, exophthalmos, and a fine tremor. Laboratory results show suppressed TSH and elevated free T4. Thyroid-stimulating immunoglobulins (TSI) are positive.

Questions:

- What is the most likely diagnosis?
- What is the underlying pathophysiology of her thyroid disorder?
- What are the first-line treatment options?

Case 2

A 65-year-old man presents with long-standing neck swelling and recent onset of palpitations, fatigue, and shortness of breath. On examination, his thyroid is irregular, nodular, and firm. Lab results reveal low TSH and elevated T3 and T4. Thyroid scintigraphy shows multiple hyperfunctioning nodules with suppressed uptake in the rest of the gland.

Questions:

- What is the most likely diagnosis?
- How does this condition differ from Graves' disease in terms of etiology, clinical presentation, and management?

Case 3

A 50-year-old woman presents with unintentional weight loss, increased sweating, and occasional palpitations. On examination, she has a solitary palpable nodule on the right lobe of her thyroid. Laboratory tests show suppressed TSH and elevated free T4. Thyroid scintigraphy reveals a single "hot" nodule with suppressed activity in the rest of the thyroid.

Questions:

- What is the likely diagnosis?
- What is the typical treatment approach for this condition?
- Why is surgery sometimes preferred over medical therapy?

Thyroid Factitious Disorder

Introduction

- • Thyroid factitious disorder is a condition where patients self-administer exogenous thyroid hormones.
- • Often done to lose weight or manipulate lab results.
- • Can mimic hyperthyroidism despite a normally functioning thyroid gland.

Epidemiology & Risk Factors

- • More common in women than men.
- • Often seen in patients with underlying psychiatric disorders.
- • May be associated with prior weight loss attempts or healthcare knowledge.

Clinical Features

- • Signs of hyperthyroidism: palpitations, heat intolerance, tremors, weight loss.
- • May lack goiter or eye signs typical of Graves' disease.
- • Symptoms often fluctuate with hormone ingestion patterns.

Biochemical Findings

- • Suppressed TSH with elevated T4/T3 levels.
- • rT3 levels may vary depending on timing of ingestion.
- • Thyroid antibodies usually negative.
- • Endogenous thyroid function appears normal when tested after hormone washout.

Peripheral & Metabolic Effects

- Mimics hyperthyroid metabolic state:
 - - ↑ basal metabolic rate (BMR)
 - - ↑ carbohydrate metabolism:
gluconeogenesis & glycogenolysis
 - - ↑ lipid metabolism: lipolysis & cholesterol reduction
 - - ↑ protein catabolism: muscle wasting
- Cardiovascular: tachycardia, increased cardiac output
- CNS: anxiety, tremors, hyperactivity

Diagnosis

- • High suspicion based on clinical presentation & lab pattern.
- • Exclude true hyperthyroidism (Graves', toxic nodules).
- • Review medication history and pill counts.
- • TSH suppression with elevated T4/T3 and negative antibodies suggest factitious disorder.

Management & Treatment

- • Gradual withdrawal of exogenous thyroid hormones.
- • Psychiatric evaluation and therapy if underlying disorder present.
- • Patient education on risks of hormone misuse.
- • Monitor thyroid function and metabolic parameters.

Summary

- • Thyroid factitious disorder results from intentional hormone ingestion.
- • Mimics hyperthyroidism clinically and biochemically.
- • Diagnosis relies on lab patterns, history, and exclusion of true thyroid disease.
- • Management includes withdrawal, psychiatric support, and monitoring.

Before we start :

Acute Thyroiditis : sudden , rapid inflammation in thyroid gland , usually caused by bacterial infection (rarely viral), rapid prognosis , come as Severe localized neck pain, redness, and swelling with High fever.

- ESR & CRP are very high.
- T3,T4,TSH normal or slightly change.
- May involve abscess or pus.
- Requires antibiotics ± surgical drainage.
- Rare.

Subacute Thyroiditis:

- ❑ painful (de Quervain)
- ❑ Painless (Silent Thyroiditis)

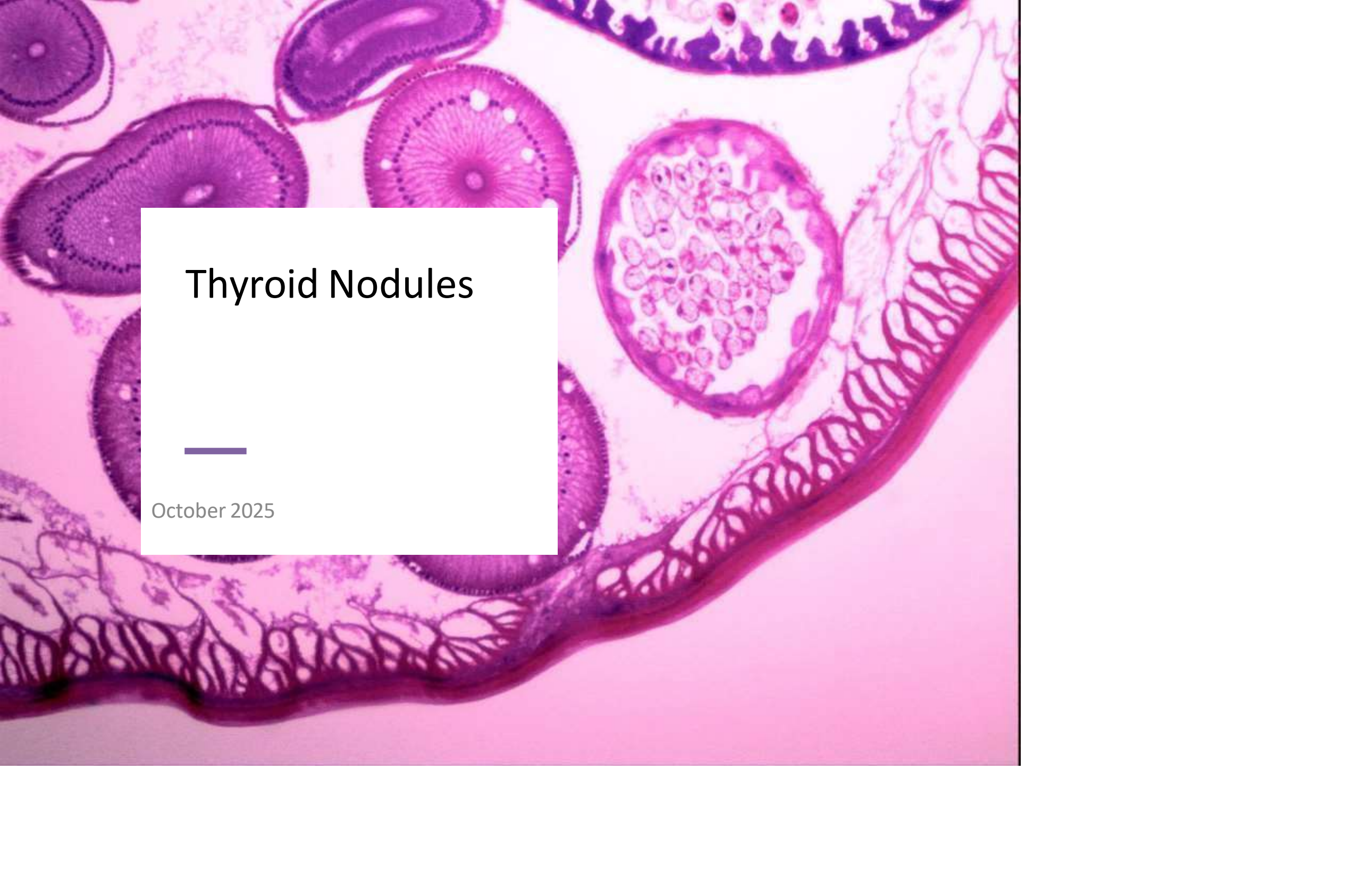
1) Subacute (de Quervain, granulomatous) thyroiditis

- post-viral inflammatory process and is often preceded by an upper respiratory illness.
- characterized by fever, neck pain, and a tender goiter following an upper respiratory illness.
- Thyrotoxicosis in subacute thyroiditis resolves spontaneously within a few weeks and may be followed by a hypothyroid phase lasting a few months. Most patients eventually recover to a euthyroid state.
- Diagnostic testing :
 - RAIU is low.
 - Initial elevation in T4 and T3 (due to leak of hormone from the gland) followed by hypothyroidism as the hormone is depleted.
 - TSH is low.
- Treatment is symptomatic with beta blockers to control thyrotoxic symptoms and nonsteroidal anti-inflammatory drugs (NSAIDs) for pain relief , Glucocorticoids are used for severe thyroid pain not responding to NSAIDs.

2) Silent Thyroiditis :

- Painless thyroiditis (silent thyroiditis) is a variant of chronic lymphocytic (Hashimoto) thyroiditis.
- Antibodies to thyroid peroxidase and anti-thyroglobulin antibodies may be present.
- characterized by a self-limited hyperthyroid phase due to release of preformed thyroid hormone, followed by a hypothyroid phase or return to a euthyroid state.
- This condition is an autoimmune process with a nontender gland and hyperthyroidism. There are no eye, skin, or nail findings.
- Unlike Graves disease, the radioactive iodine uptake (RAIU) test is low, since this is not a hyperfunctioning gland; it is just “leaking”.
- Treatment: None. It does not require specific therapy, but a beta blocker may be prescribed to control symptoms due to hyperthyroidism (palpitations, tremulousness).

Feature	Painful Subacute Thyroiditis (<i>de Quervain's</i>)	Silent Subacute Thyroiditis *(Painless)
Cause	Viral (post-infection)	Autoimmune
Neck Pain	✓ Present (often severe)	✗ Absent
Onset	Subacute (days to weeks)	Subacute
Fever	✓ Mild fever possible	✗ Usually absent
Thyroid Tenderness	✓ Present	✗ None
Thyroid Function Phases	Hyperthyroid → Hypothyroid → Recovery	Same
ESR / CRP	✓ Elevated	✗ Usually normal or mildly elevated
Thyroid Antibodies	✗ Negative	✓ May be positive (anti-TPO)
Common in	Any adult, especially women	Often postpartum
Treatment	NSAIDs ± Steroids	Supportive (beta-blockers if needed)

A microscopic image of thyroid tissue stained with hematoxylin and eosin (H&E). The image shows several thyroid follicles of varying sizes. Some follicles contain clear, pale-staining colloid, while others are filled with a dense population of small, dark-staining cells, likely lymphocytes, suggesting an inflammatory process. The follicular epithelium is visible as a single layer of cuboidal cells lining the follicles. A white rectangular text box is overlaid on the left side of the image.

Thyroid Nodules

October 2025

Definition & Epidemiology

- Thyroid nodule: a discrete lesion within the thyroid gland that is radiologically distinct from the surrounding parenchyma.

- Prevalence: ~5% of adults by palpation, up to 70% by ultrasound.

- More common in females and with increasing age.

Etiology & Classification

- • Benign causes: colloid nodules, cysts, thyroiditis, adenomas.
- • Malignant causes: papillary, follicular, medullary, anaplastic carcinoma, lymphoma, metastasis.
- • Functional classification:
 - - Non-toxic (euthyroid)
 - - Toxic (autonomous hyperfunctioning)

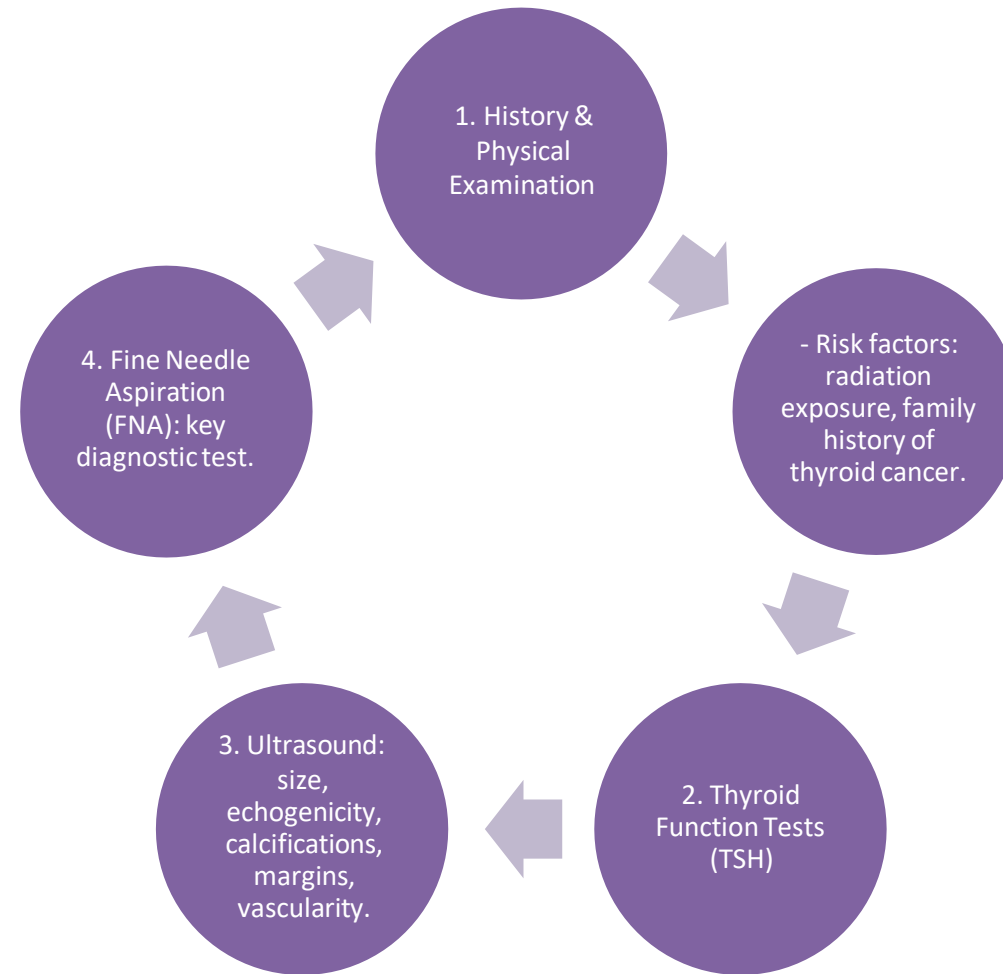
Clinical Features

- Usually asymptomatic, found incidentally.

- Symptoms (if present): neck swelling, dysphagia, dyspnea, hoarseness.

- Signs suggesting malignancy: hard, fixed nodule, rapid growth, lymphadenopathy, vocal cord palsy.

Evaluation



Management

- Benign nodules: observation, follow-up with US.

- Suspicious or malignant: surgical removal.

- Toxic nodules: radioactive iodine or surgery.

- Follow-up with repeat imaging and TSH monitoring.

Summary / Key Points

- Most thyroid nodules are benign.

- TSH and ultrasound are initial key investigations.

- FNA is the gold standard for cytological diagnosis.

- Management depends on malignancy risk and nodule function.

THANK YOU

- Reference
 1. American Thyroid Association Guidelines, 2015.
 2. Harrison's Principles of Internal Medicine, 21st Edition.
 3. UpToDate: Evaluation and management of thyroid nodules.