

Non-neoplastic thyroid diseases

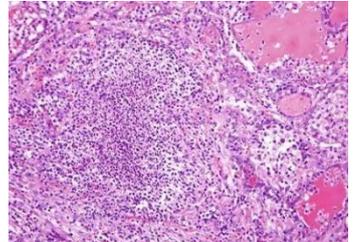
	Hyperthyroidism	Hypothyroidism
Primary	<ul style="list-style-type: none"> -Graves' disease -Toxic multinodular goiter -Toxic follicular thyroid adenoma 	<ul style="list-style-type: none"> -Iodine deficiency -Hashimoto thyroiditis -Post ablative -Genetic defects in thyroid development -Thyroid hormone resistance
Secondary	TSH-secreting pituitary adenoma	Pituitary failure

Thyroiditis

I Types

- Acute / infectious thyroiditis
- Autoimmune thyroiditis (Hashimoto's thyroiditis).
- Riedel thyroiditis
- Subacute granulomatous thyroiditis

Acute Infectious Thyroiditis

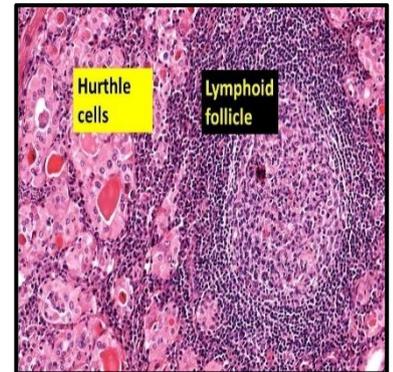
Risk Factors	- Malnourished infants, Debilitated elderly, Immunosuppression and trauma
Etiology	<ul style="list-style-type: none"> - Often Strept haemolyticus, Strept pneumoniae, Staph aureus - Via blood or direct seeding from upper respiratory infections, causes sudden onset of pain and glandular enlargement
N/E	<ul style="list-style-type: none"> - Normal or slightly enlarged thyroid gland. - May have suppurative areas 
M/E	<ul style="list-style-type: none"> - Neutrophils, possibly micro abscesses and tissue necrosis - Fungi are associated with necrosis, acute inflammation and granulomas 



Hashimoto Thyroiditis

- The **most common cause of Hypothyroidism** after iodine deficiency.

- **More common in females**



I

Pathogenesis

- **Auto-immune thyroiditis**

- **Activation of helper** (CD4+) T lymphocytes sensitized to thyroid antigens.

- **These helper T lymphocytes** stimulate proliferation of cytotoxic (CD8+) T lymphocytes, which attack thyroid follicular cells.

- **Activated helper T lymphocytes** also recruit reactive B lymphocytes to produce antibodies against thyroid antigens as anti-thyroglobulin and anti-thyroid peroxidase.

II

Gross description

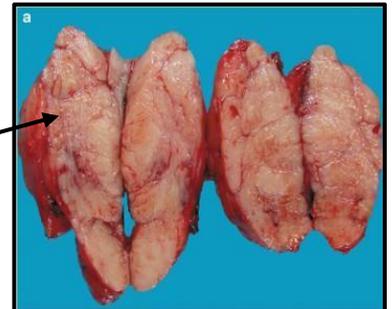
1- Early:

- There is symmetrical diffuse enlargement of the thyroid gland with vague nodularity

- The affected areas are pale (gray or yellow) and firm

- They lack the glistening appearance of colloid

2- Late: The gland becomes symmetrically atrophic



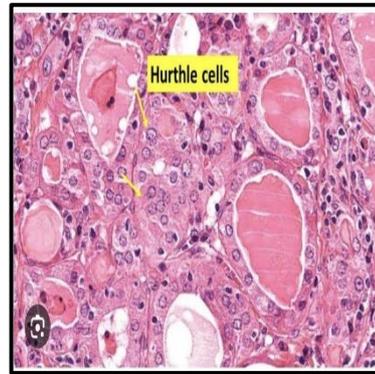
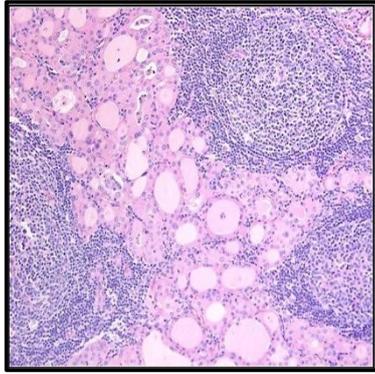
III

Microscopic description

- **Follicles are atrophied.** Follicles are lined by large cubical cells with eosinophilic granular cytoplasm (**Hurthle cells**).

- **Inflammatory cells** (Lymphocytes, plasma cells, macrophages).

- **Reactive lymphoid follicles.**



Riedel Thyroiditis

- **Rare** chronic disease of **unknown cause**.
- **Riedel thyroiditis** may be part of multifocal idiopathic fibrosclerosis.

I

Gross description

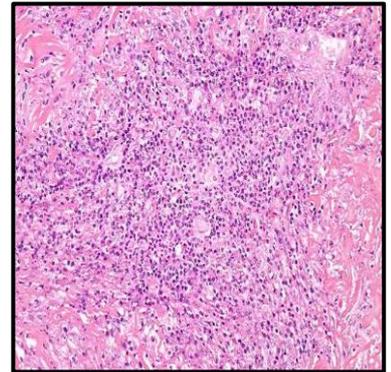
- The gland is stony hard and greyish white
- Adherent to the surrounding structures



II

Microscopic description

- Follicles are atrophied
- Excess fibrous tissue may extend outside the gland
- Lymphocytic infiltrate



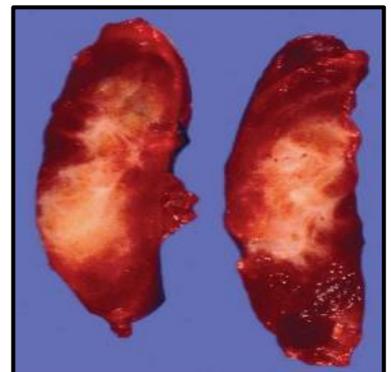
III

Complications

Compression manifestations: dysphagia, dyspnea, recurrent laryngeal nerve paralysis

Subacute Granulomatous Thyroiditis

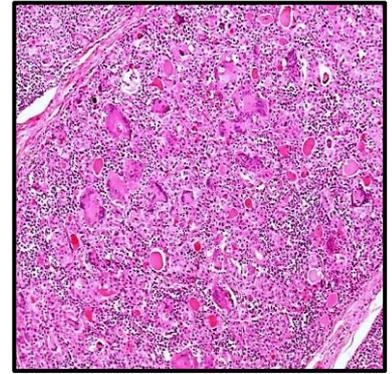
- **A self-limited disorder** in which patients present with a tender thyroid.
- **Etiology is not known** but clinical features of preceding respiratory infection suggest a possible viral etiology





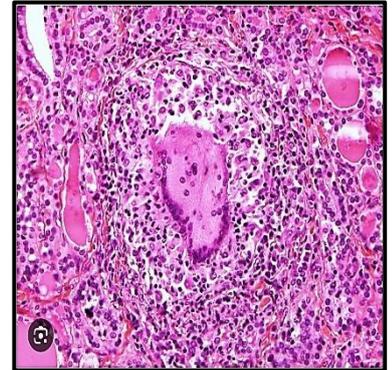
Gross description

- **Moderate enlargement** of the gland which is often focal and asymmetrical.
- **The cut surface** of the involved area is firm and greyish white.



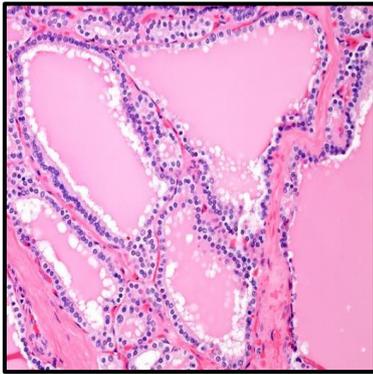
Microscopic description

- **Early:** Acute inflammatory destruction of the thyroid follicles.
- **Late:** Granulomas consist of central colloid material surrounded by histiocytes and scattered multinucleated giant cells.
- **Advanced cases,** Fibrosis

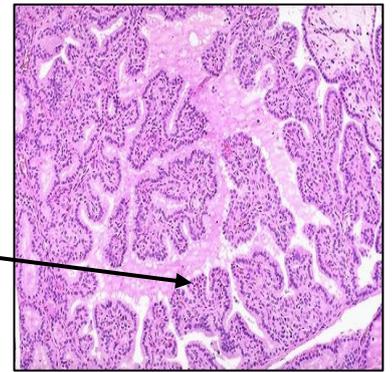


Graves' Disease

Etiology	Autoimmune disease , more common in females.	
Pathogenesis	<ul style="list-style-type: none"> - Due to auto-antibodies. Most common is TSI (thyroid stimulating Ig). - Stimulate TSH receptors leading to diffuse hyperplasia and hyper functioning thyroid follicles with excess thyroid hormone secretion 	
N/E	<ul style="list-style-type: none"> - The thyroid gland is symmetrically enlarged. - The gland appears fleshy. - Cut surface is firm and dark red. - Loss of the normal translucence of stored colloid 	
M/E	<ul style="list-style-type: none"> - The gland is diffusely Hyperplastic and highly vascular. - The follicles are lined with tall columnar cells. -The colloid is depleted and scalloped (moth-eaten) at the periphery. - Hyperplastic papillae project into the lumen of the follicles. - Lymphocytes and plasma cells infiltrate the interstitial tissue and may form reactive lymphoid follicles 	



Hyperplastic papillae



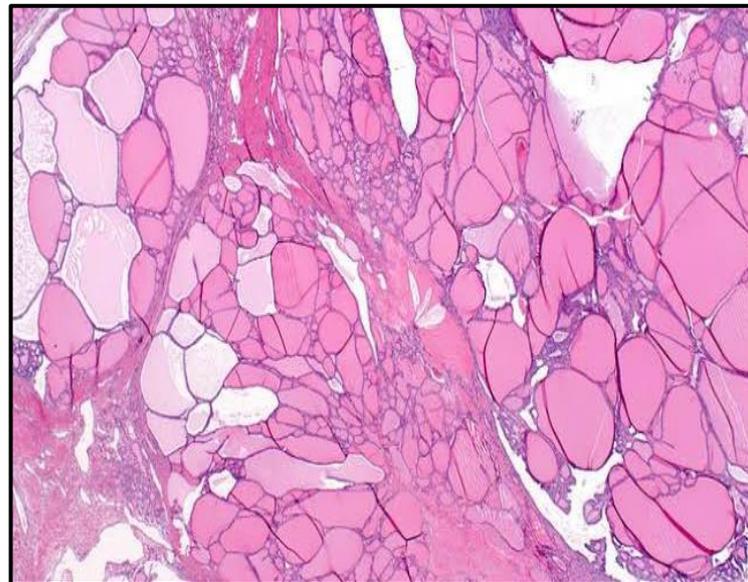
Goiter

Goiter: Non inflammatory, non-neoplastic enlargement of the thyroid gland

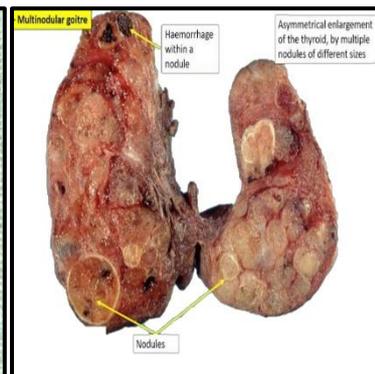
Goitrogen: agents that **suppress thyroid hormone synthesis**, and so elevating TSH levels and leads to hyperplastic changes of thyroid (goitrous enlargement) as cabbage and cauliflower.

Classification

- Simple (non-toxic):** Diffuse or Multinodular
- Toxic** (associated hyperthyroidism)



M/E of Simple (Nontoxic) Goiter



N/E of simple multinodular goiter

	Diffuse Simple (Nontoxic) Goiter	Simple Multinodular (Nontoxic) Goiter
Site & Incidence	<ul style="list-style-type: none"> - Diffusely involves the thyroid gland without producing nodularity. - Common in females 	---
Causes/Results	<p>Physiologic Goiter: Usually seen at times of increased physiological demands as puberty, pregnancy.</p> <p>Forms:</p> <ul style="list-style-type: none"> • Endemic form: occurs in geographic areas where the soil, water, and food supply contain low levels of iodine • Sporadic form 	<ul style="list-style-type: none"> - Repeated cycles of stimulation and involution of a diffuse goiter lead to rupture of some follicles followed by fibrosis, hemorrhage, scarring, and sometimes calcification. - This produces an irregular enlargement of the thyroid gland (variable nodularity and scarring)
Phases	<p>1-Hyperplastic stage: there is diffuse and symmetric enlargement of the thyroid gland. There is hypertrophy and hyperplasia of the follicular epithelium with scant colloid.</p> <p>2-Colloid involution stage: if iodine correction and decreased demand to thyroid hormones occur after a short period of hyperplasia, there is colloid accumulation and atrophy (involution) of follicular epithelium. Most patients are clinically euthyroid.</p>	---
Gross	---	<ul style="list-style-type: none"> - The thyroid gland is asymmetrically enlarged with nodular surface. - The cut surface shows numerous irregular nodules contain glistening colloid. - Fibrosis, calcification, hemorrhage & cystic degeneration may be present.
Microscopic	<ul style="list-style-type: none"> - Nodular thyroid gland separated by fibrous tissue. - Hyperplastic nodules, tall columnar follicular epithelium, papillary structures, formation of small new follicles. - Involuted nodules, large follicles distended by colloid and lined by flat follicular epithelium. Large colloid-containing follicles may fuse to form colloid cysts. - Calcification, chronic inflammation, hemorrhage & cystic degeneration are common in the old standing cases 	
Complications	<ul style="list-style-type: none"> - Pressure effects: on trachea, esophagus and recurrent laryngeal nerve - Some of these nodules may become hyper functioning and cause hyperthyroidism (no exophthalmos) 	<ul style="list-style-type: none"> - Malignancy: rare in 2% of cases