

Non-Neoplastic Thyroid

Normal Findings

Follicular epithelium: flat to columnar with round nuclei with finely granular chromatin.

IHC: (+) PAX8, TTF1, Thyroglobulin, CK7, etc..

Colloid: Bright pink. Frequent calcium oxalate crystals (seen best by polarization)(less common in parathyroid so potentially helpful during frozen section analysis)

Frequent metaplasia (often seen with chronic thyroiditis):

Oncocytic/Hürthle cells: larger cells with abundant eosinophilic granular cytoplasm and large nuclei with prominent nucleoli

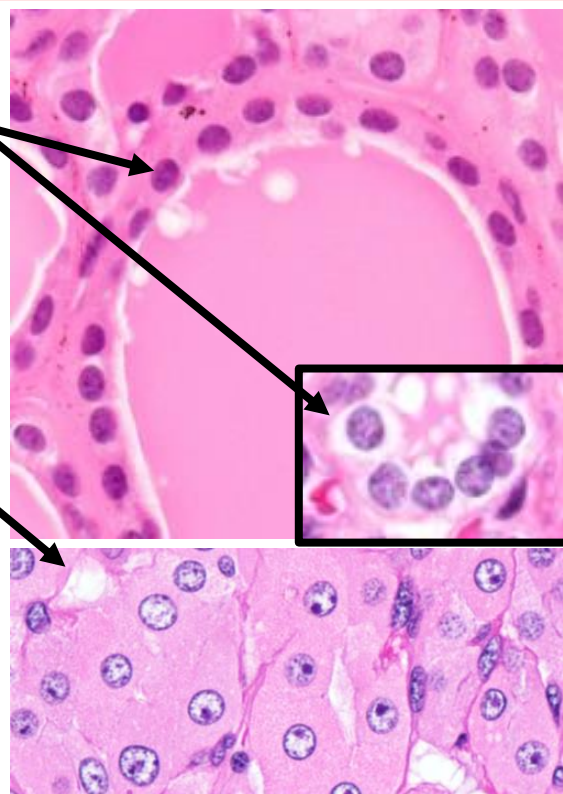
Squamous metaplasia

C cells: often hard to see on H&E.

Single cells or small clusters between follicles.

Stain with calcitonin and neuroendocrine markers

Release calcitonin (helps regulate calcium levels)



Incidental findings

Solid Cell Nests

Solid nests of cells with ovoid nuclei, finely granular chromatin, and frequent grooves. No keratinization.

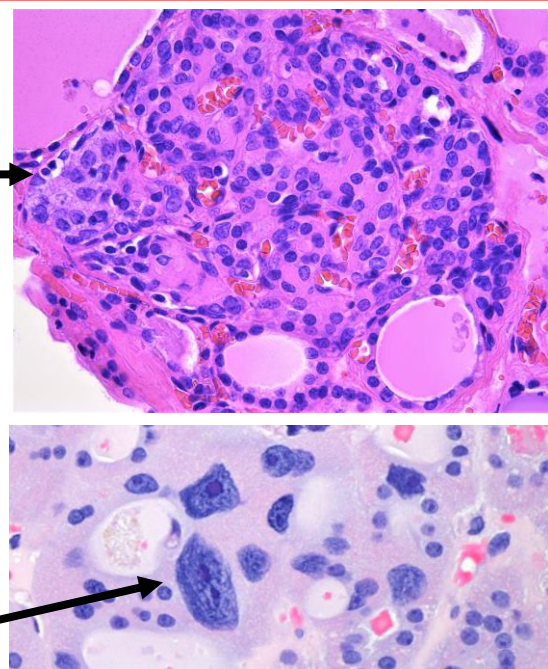
Remnants of the ultimobranchial bodies. (+)p63, (-)TTF1

Ectopic thyroid: can be seen in any location from the tongue to the suprasternal notch, usually in the midline. Inclusions in lymph nodes are somewhat controversial, and most instances likely represent metastases.

Thymus, Parathyroid

Skeletal Muscle: particularly around the isthmus, benign thyroid can intermingle with the skeletal muscle.

Endocrine Atypia: Random, usually focal nuclear pleomorphism. Thought to be degenerative. Not indicative of malignancy



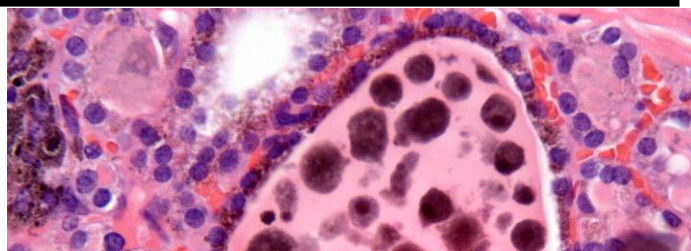
"Black Thyroid"

Abundant black pigment

Visible grossly and microscopically

Usually from minocycline/tetracycline administration

No impact on thyroid function



Thyroiditis

Chronic Lymphocytic Thyroiditis

Most common autoimmune thyroiditis.

Hypothyroidism frequent. More common in women.

Autoantibodies (e.g., anti-thyroglobulin)

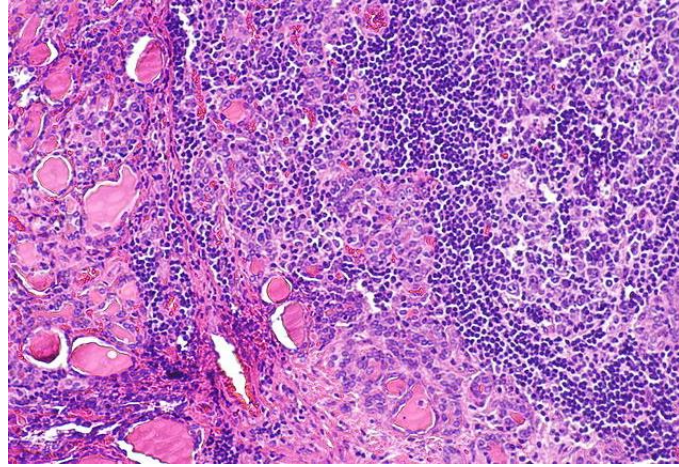
Treat with hormone replacement

Diffuse infiltration by lymphocytes, often with germinal centers.

Frequent Hürthle (oncocytic) cell change

Fibrosing variant: Dense fibrosis with keloid-like bands. Confined to thyroid (unlike Riedel's).

aka "Hashimoto Thyroiditis"



Subacute Granulomatous Thyroiditis

aka "de Quervain disease"

Self-limited inflammation.

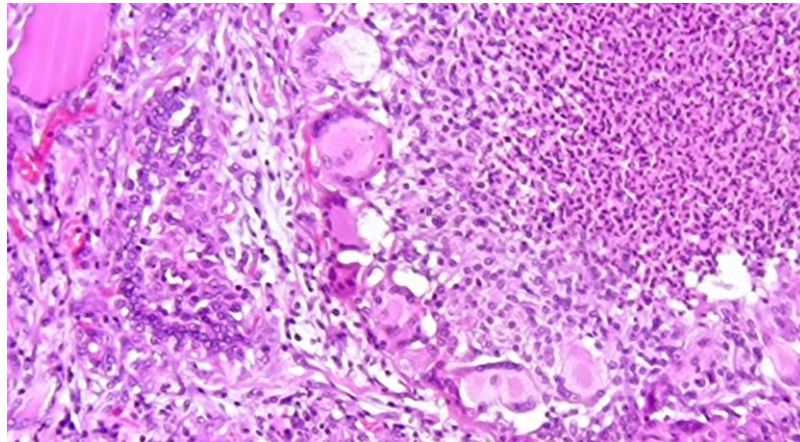
More common in women, present with prodrome → **painful** thyroid gland

Can occur after viral infection.

Asymmetric, uneven inflammation

Early: Acute inflammation (hyperthyroid)

Later: **Epithelioid histiocytes, multinucleated giant cells**, chronic inflammation, and fibrosis (hypothyroid)



Riedel Thyroiditis

aka "Invasive Fibrous Thyroiditis"

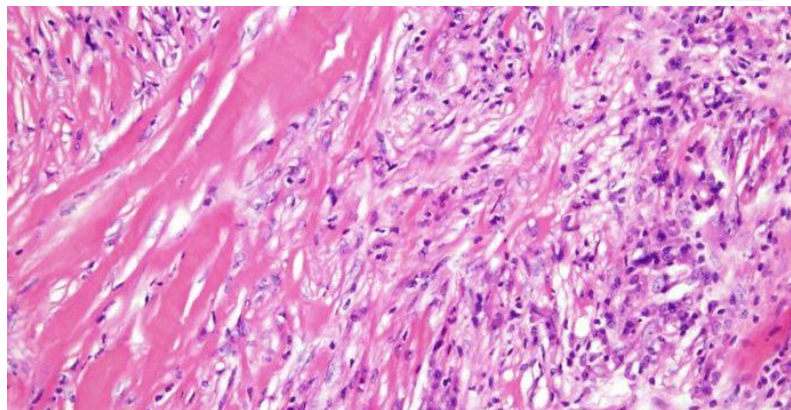
Fibrosclerosing inflammation of the thyroid and adjacent soft tissues

Usually, **IgG4-related**

Destruction/replacement of gland by: dense collagen with keloidal-like bands.

Increased plasma cells, and phlebitis.

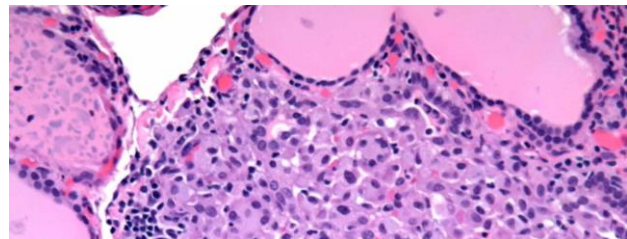
Very rare. Hard "wooden" thyroid.



Palpation Thyroiditis

Scattered foci of giant cells, granulomas, and/or foamy macrophages with other inflammatory cells near ruptured follicles. Presumed to be due to vigorous **manipulation**.

Asymptomatic. **Incidental**



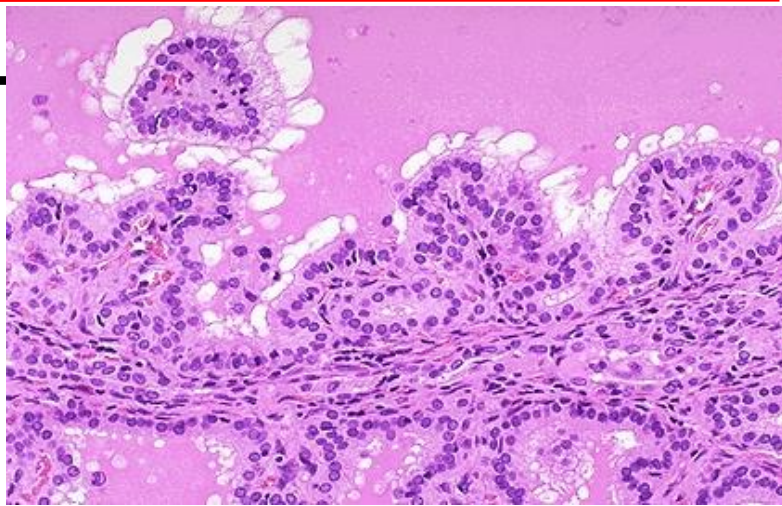
Hyperplasia

Graves Disease

Thyroid stimulating antibodies → stimulates thyroid hormone synthesis → diffuse proliferation → **hyperthyroid**

More common in women.

Diffuse follicular epithelium hyperplasia → non-branching papillary projections
Scalloping of colloid.
Often tall, pink cells.
Frequent lymphocytic infiltration.

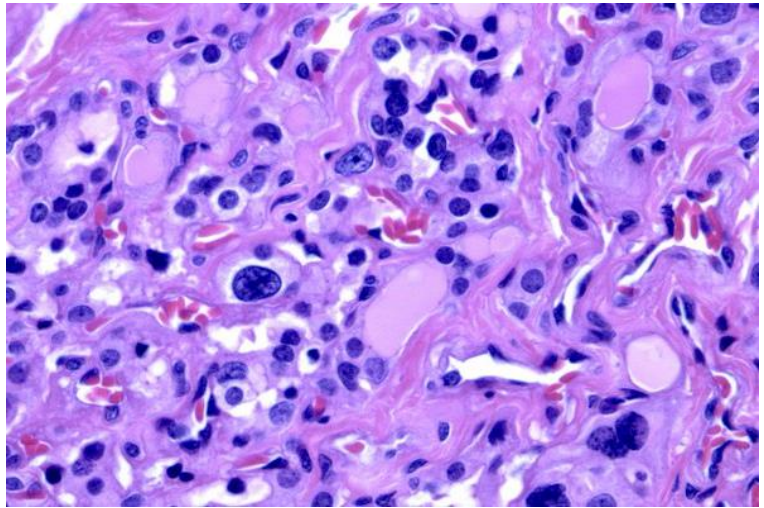


Dyshormonogenetic Goiter

Genetic defect in thyroid hormone production
→ Elevated TSH via feedback loop → causes **gland hyperplasia** (but no increase in production). Autosomal recessive.

ALL thyroid tissue is abnormal.
Scant to **absent colloid**. Hypercellular nodules.
Fibrosis. **Prominent cytologic atypia.**
IHC: (-) Thyroglobulin (+) PAX8, TTF1

Medically treat with hormone replacement, surgery for symptomatic goiter.



“Endemic” Goiter

Refers to thyroid hyperplasia in certain geographic areas, usually secondary to **dietary iodine deficiency** (Iodine is needed to make thyroid hormone).
Pathologically, resembles adenomatous hyperplasia due to other causes.

Adenomatous Hyperplasia

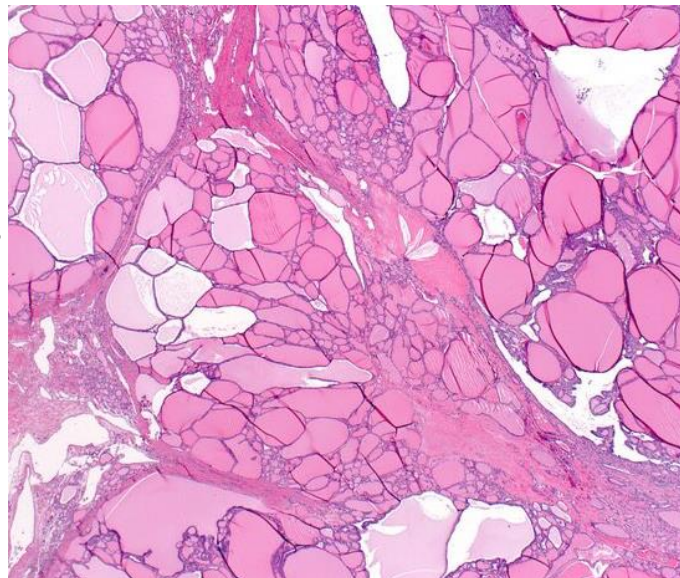
Multinodular thyroid gland enlargement due to follicular epithelial hyperplasia.

Very common. More common in females.

Mostly Unencapsulated nodules with pushing borders
Most nodules contain **abundant colloid**
Variably sized nodules, Some may be dominant
Epithelial metaplasia and hyperplasia common.
Can see cystic change, hemorrhage, calcifications.

“Goiter” is generally a clinical term, not a surgical pathology diagnosis.

Total thyroidectomy for symptomatic disease



Other Lesions

Post-FNA Changes

Hemorrhage and hemosiderin-laden macrophages. Granulation tissue.

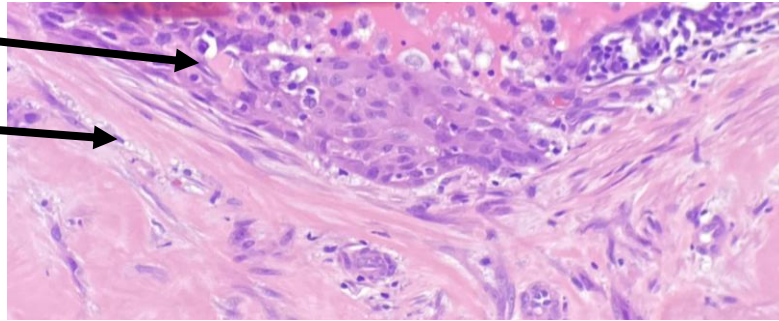
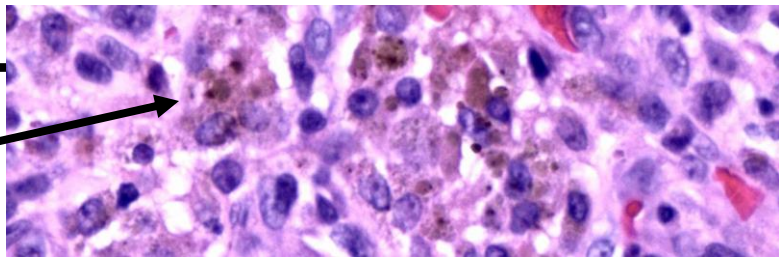
Metaplasia (e.g., squamous)

Infarction

Fibrosis

Nuclear atypia

Capsular alterations—can mimic capsular invasion (hint is other changes above)



Amyloid Goiter

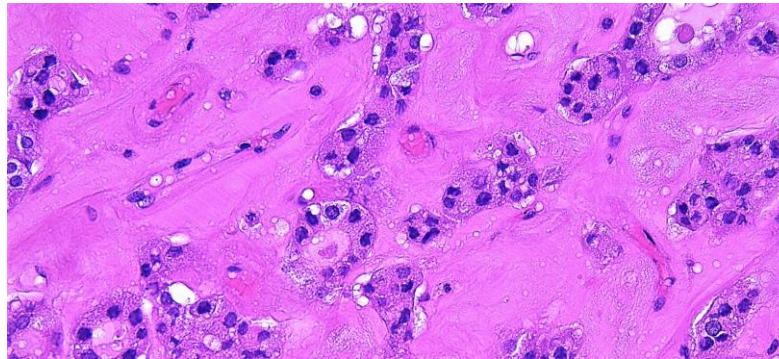
Mass enlargement due to amyloid deposition.

Usually diffuse throughout gland and angiocentric.

Compresses follicles.

Causes: 1° or 2° amyloidosis, medullary carcinoma

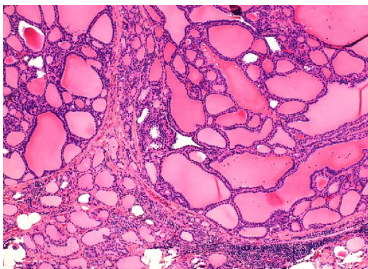
Stains: **(+)** Congo Red with “apple green birefringence”



Adenomatoid Nodule

Asymmetric nodular enlargement—usually part of adenomatous hyperplasia, with multiple nodules, but one nodule may be *dominant*.

Main DDX is a follicular adenoma. Since both are benign, and there is some interobserver variation, some people just say “**Benign Follicular Nodule**”



Adenomatoid nodule	Follicular adenoma
<i>Think: polyclonal growth</i>	<i>Think: monoclonal neoplasm</i>
<u>Un</u> encapsulated. May have variable, incomplete, surrounding fibrosis	<u>Encapsulated</u> . Well-defined, fibrous, often with thick vessels
<u>Multiple</u> patterns of growth, resembling background thyroid	<u>Uniform</u> pattern of growth; distinct from surrounding thyroid
Relatively abundant colloid	Less colloid
Often <u>multiple</u> nodules	<u>Single</u> nodule
Usually Pushing border	Compresses nearby tissue