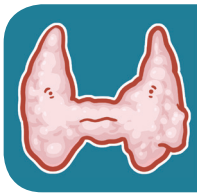

NOTES
THYROIDITIS

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Group of autoimmune disorders resulting in inflammation, destruction, and functional impairment of the thyroid gland

SIGNS & SYMPTOMS

- Hypothyroidism
  - Weight gain despite reduced appetite, constipation
  - Cold intolerance, fatigue, lethargy, weakness
  - Brittle hair and nails, dry skin, hair loss (alopecia)
  - Mental slowness (bradypsychia)
  - Voice hoarseness → compression of recurrent laryngeal nerve
  - Enlarged thyroid gland (goiter)

DIAGNOSIS

- Suspect based on clinical presentation

LAB RESULTS

- Serum antibody levels against thyroid components
- Thyroid biopsy via fine needle aspiration

TREATMENT

MEDICATIONS

- Thyroid hormone replacement → levothyroxine

SURGERY

- Surgical removal if adjacent structures are affected

HASHIMOTO'S THYROIDITIS

osms.it/hashimotos-thyroiditis

PATHOLOGY & CAUSES

- Chronic autoimmune disorder leading to inflammation, gradual destruction and functional impairment of the thyroid gland resulting in hypothyroidism and increased risk of thyroid cancer
- Most common cause of hypothyroidism in areas where dietary iodine is sufficient
- Cause unclear; related to *HLA-DR3* and *HLA-DR5* genes; may occur in combination with Graves' disease; may be influenced by environmental factors
- Hürthle cells
  - Enlarged follicular cells with an eosinophilic, granular cytoplasm
- Gene mutation → B cell dysfunction → B cell thyroid invasion → B cell germinal centers established within thyroid → B cell activation and autoantibody production → NK cells signaled to destroy thyroid follicular cells + $CD4^+$ cells produce inflammatory cytokines + $CD8^+$ cells attack thyroid follicular cells → release of stored T_3 and T_4 → transient hyperthyroidism

→ burnout → hypothyroidism → compensatory increase in thyroid-stimulating hormone (TSH), thyrotropin-releasing hormone (TRH)
- Chronic inflammation → connective tissue buildup → enlarged gland
- B cells have the potential to become malignant → B cell lymphoma of the thyroid
  - Rare; usually in females over 70 with history of Hashimoto's

SIGNS & SYMPTOMS

Hypothyroidism

- Weight gain despite reduced appetite, constipation
- Brittle hair and nails, dry skin, hair loss (alopecia)
- Cold intolerance, fatigue, lethargy, weakness
- Mental slowness (bradypsychia)
- Enlarged, nodular thyroid gland (goiter)
  - Non-tender, firm
  - Voice hoarseness → compression of recurrent laryngeal nerve
  - Stridor → tracheal compression
- Menstrual abnormalities, galactorrhea
- ↑ TRH → ↑ prolactin levels
- Myxedema
  - Nonpitting edema caused by mucopolysaccharide deposition in upper skin layers
  - Most common around tibial area, may also occur around eyes and feet
- Rapidly growing goiter suggests B cell lymphoma of the thyroid

DIAGNOSIS

- Suspect based on clinical presentation

LAB RESULTS

- ↓ T_3 and T_4
- ↑ TSH and TRH
- Autoantibodies against thyroid peroxidase (anti-TPO) and against thyroglobulin (anti-TG)
- Perform fine needle aspiration (FNA) if B cell lymphoma of the thyroid suspected

TREATMENT

MEDICATION

- Thyroid hormone replacement → levothyroxine

SURGERY

- Goiter affecting adjacent structures → surgical removal

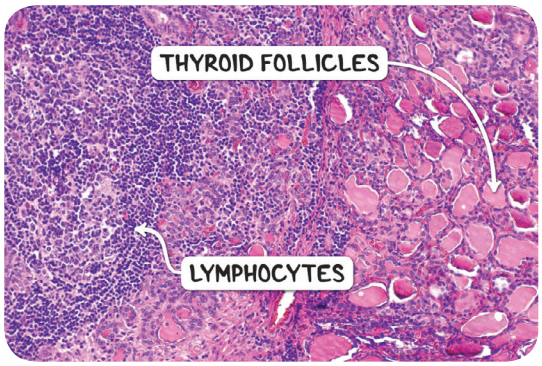

Figure 21.1 The histological appearance of Hashimoto's thyroiditis. The normal thyroid follicles are on the right. The lymphocytic infiltrate has replaced the normal thyroid tissue on the left.

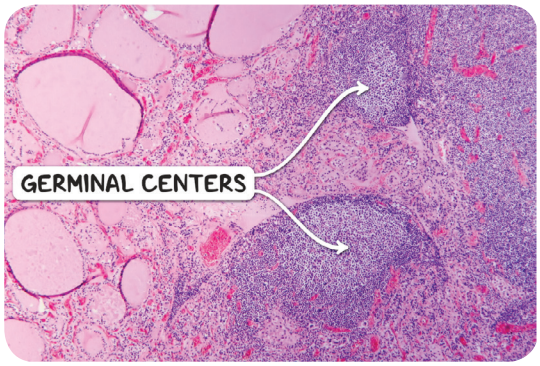

Figure 21.2 A low power image of Hashimoto's thyroditis, showing the lymphoycytic infiltrate forming germinal centres.

POSTPARTUM THYROIDITIS

osms.it/postpartum-thyroiditis

PATHOLOGY & CAUSES

- Autoimmune destruction of the thyroid gland occurring within one year after after parturition, resulting in transient thyroid dysfunction and thyroid hormone imbalance
- Related to normal fluctuations in maternal immune function in the setting of subclinical autoimmune thyroid disease
- Autoimmune-related thyroid inflammation (1–4 months postpartum) → damage to thyroid follicles and thyroglobulin → ↑ ↑ thyroxine (T_4) and triiodothyronine (T_3) release into the blood → hyperthyroidism (last 2–8 weeks)
  - T_4/T_3 stores eventually used up + TSH-induced cessation of new thyroid hormone synthesis → transient hypothyroidism
  - Resolution of inflammation → follicle regeneration → return to normal thyroid levels

RISK FACTORS

- Prior history of postpartum thyroiditis
- Pre-existing hypothyroidism (e.g. Hashimoto's thyroiditis with remaining functional thyroid hormone)
- Type 1 diabetes mellitus
- Familial predisposition (possible inheritance pattern)

COMPLICATIONS

- Chronic hypothyroidism

SIGNS & SYMPTOMS

- Symptoms of hyper- and hypothyroidism are usually mild
- Hyperthyroid phase (↑ metabolic rate)
  - Anxiety
  - Heat intolerance
  - Tachycardia, palpitations
  - Tremor
  - Fatigue
  - Weight loss
  - Diffuse, painless goiter
- Hypothyroid phase (↓ metabolic rate)
  - Impaired concentration
  - Cold intolerance
  - Sluggishness
  - Constipation
  - Dry skin

DIAGNOSIS

DIAGNOSTIC IMAGING

- Radioactive iodine uptake
  - Profoundly suppressed (test is contraindicated if breastfeeding)

LAB RESULTS

- Blood studies
  - *Hyperthyroid phase:* ↑ T_4, T_3; ↓ TSH
  - *Hypothyroid phase:* ↓ free T_4, ↑ TSH
  - ↑ antithyroid peroxidase antibodies
- Thyroid biopsy
  - Lymphocytic thyroiditis - infiltration of lymphocytes, follicular destruction

OTHER DIAGNOSTICS

- History and physical examination

TREATMENT

- Mild symptoms require no treatment

MEDICATIONS

- *Symptomatic hyperthyroidism:* beta-blocker
- *Symptomatic hypothyroidism:* levothyroxine

RIEDEL'S THYROIDITIS

osms.it/riedels-thyroiditis

PATHOLOGY & CAUSES

- Rare autoimmune disorder leading to inflammation, fibrotic infiltration, gradual destruction, and functional impairment of the thyroid gland
- May be related to a systemic autoimmune fibrotic disease process
- Component of IgG_4-related disease
  - May also cause fibrosis of salivary glands, kidneys, pancreas and lungs
- IgG_4 attacks thyroid follicular cells → T cells release inflammatory cytokines → abnormal fibroblast activation within thyroid stroma → stromal fibrosis replaces damaged follicles → gland enlarges and hardens → fibrosis spreads to neck structures (parathyroid glands, blood vessels, trachea, muscles, nerves)

SIGNS & SYMPTOMS

- Hardened, wood-like, fixed, painless and enlarged thyroid gland (goiter)
- Hypothyroidism
  - Weight gain despite reduced appetite, constipation
  - Brittle hair and nails, dry skin, hair loss (alopecia)
  - Cold intolerance, fatigue, lethargy, weakness
  - Mental slowness (bradypsychia)
  - Bradycardia
- Other neck structures affected by compression
  - Tracheal fibrosis → shortness of breath (dyspnea)
  - Recurrent laryngeal nerve fibrosis → voice hoarseness
  - Esophageal fibrosis → dysphagia
  - Parathyroid gland fibrosis → hypocalcemia and tetany (hypoparathyroidism)

DIAGNOSIS

- Suspect based on clinical presentation

LAB RESULTS

- Autoantibodies against thyroid components (anti-TPO)
- ↓ T_3 and T_4
- ↑ TSH and TRH
- Tissue biopsy
  - Predominant fibrous tissue and collagen + lymphocyte infiltration

TREATMENT

MEDICATIONS

- Corticosteroids
- Tamoxifen may decrease goiter size
- Thyroid hormone replacement → levothyroxine

SURGERY

- Debulking or surgical removal if goiter affects adjacent structures