Thyroid Dysfunction and the Eye

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Learning Objectives

- Enhance clinical understanding of thyroid dysfunction and the ocular associations
- Enhance clinical diagnosis of ocular manifestations of thyroid disease
- Enrich clinical management and treatment of ocular manifestations thyroid eye disease
- Increase comfort level when ordering or interpreting laboratory tests in thyroid diseases
- Gain confidence in working closer with the endocrinologist, oculoplastic surgeon and orbital specialist

Thyroid

- Thyroid is an endocrine gland
- Two types of glands
  - Endocrine
  - Exocrine
- Endocrine system is a central system of ductless endocrine glands that secrete hormones (chemical messengers) that circulate within the body via the bloodstream or lymph system to affect distant organs
  - Hypothalamus
  - Pituitary gland
  - Thyroid
  - Parathyroid glands
  - Pineal gland

Disclosures

- Greg A. Caldwell, OD, FAAO will mention many products, instruments and companies during our discussion, I don’t have any financial interest in any of these products, instruments or companies.
- In the past 12 months I have lectured or participated in a focus group which I received a honorarium for:
  - Allergan, Alcon, Valeant and SARcode Bioscience
- All of these cases have entered/referred to my practice

Thyroid Disease and Thyroid Eye Disease

- Exocrine glands contain ducts. Ducts are tubes leading from a gland to its target organ
  - Digestive glands have ducts for releasing the digestive enzymes
  - Salivary glands, sweat glands and glands within the gastrointestinal tract
- Pancreas is both endocrine and exocrine
  - Exocrine (ducted gland) secreting digestive enzymes into the small intestine.
  - Endocrine (ductless gland) in that the islets of Langerhans secrete insulin and glucagon to regulate the blood sugar level.

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The thyroid is the largest endocrine gland in the body. It is butterfly shaped, with two lobes located on either side of the trachea in the lower portion of the neck. It lies just below the skin and muscle layers surface. The thyroid is controlled by the hypothalamus and pituitary gland. The primary function of the thyroid is the production of the hormones thyroxine (T4), triiodothyronine (T3), and calcitonin.

The thyroid regulates heart rate, ventilation rate, metabolic rate, and development of cells. Thyroid disorder is approx 1 in 13 or 7.35% or 20 million people in USA, estimated 2 million undiagnosed. Diabetes is approx 1 in 13 or 7.8% or 17.9 million people in USA, 5.7 million undiagnosed.

Thyroid dysfunction can be primary, secondary, or tertiary. Primary thyroid gland failure is the most common cause of thyroid dysfunction. In autoimmune disease, the body typically produces antibodies that attack itself, this can be systemic or organ specific.

Discussion: Thyroid dysfunction can be caused by:
A. Cancer
B. Surgically induced
C. Medication toxicity or side effect
D. Pregnancy
E. Autoimmune disease

In autoimmune disease, the body typically produces antibodies that attack itself, this can be systemic or organ specific.
Antibodies of Thyroid Dysfunction

- TSH Receptor Antibodies
  - Stimulating TSH receptor antibody
  - Thyroid stimulating immunoglobulin (TSI)
  - Thyroid blocking antibody (TBAb)

- Thyroid Peroxidase Antibodies (TPOAb)
  - TPO is found in thyroid follicle cells where it converts the thyroid hormone T4 to T3
  - TPOAb contributes to thyroid cellular destruction

Most autoimmune thyroid dysfunctions have a combination of thyroid antibodies, however depending on which AB is more abundant results in the outcome of the disease.

Hyperthyroid

- TSI attacks the thyroid
- T3 and T4 increase
- TSH decreases

Hypothyroid

- TBAb attacks the thyroid
- T3 and T4 decrease
- TSH increases

Thyroid Dysfunction

**Hyperthyroidism** (Hyperfunction)

- Primary-autoimmune
  - Graves
  - Graves-Basedow or von Basedow’s
- Secondary/Tertiary
  - Excess thyroid medication for treatment of hypo or goiter
  - Toxic multinodular goiter
  - Toxic adenoma
  - Excess iodine
  - Thyroiditis (inflammatory induced)
  - Excess hormone production ectopic tissue
  - Thyroid carcinoma

**Hypothyroidism** (most common organ-specific autoimmune disorder)

- Primary-autoimmune
  - Chronic autoimmune thyroiditis
  - Hashimoto’s thyroiditis
  - Autoimmune atrophic thyroiditis
  - Primary myxedema (opposite of Graves disease)
- Secondary/Tertiary
  - Lithium medication
  - Pregnancy
  - Surgically induced
  - Disorders of the pituitary gland or hypothalamus

GRAVES’ (Hyperthyroidism)

- A multisystem disorder consisting of a triad
  - Hyperthyroidism with diffuse hyperplasia of the thyroid gland
  - Infiltrative dermopathy
  - Infiltrative ophthalmopathy

- Prevalence:
  - 20-40 year old female (F:M = 7:1)
- Genetic link

- Etiology:
  - Autoimmune disease: hypersensitivity reaction with thyroid stimulation by the circulation of abnormal thyroid-stimulating immunoglobulins (TSI)

Hashimoto’s Thyroiditis (Hypothyroidism)

- The most common cause of hypothyroidism in the United States
- It is named after the first doctor who described this condition, Dr. Hakaru Hashimoto, in 1912
- Autoimmune disease
- Goiter formation
- 5-10 times more common in women than in men
- The underlying cause of the autoimmune process still is unknown
  - Anti-TPO ab and Anti-TB recp ab present
Autoimmune atrophic thyroiditis (Hypothyroidism)
- Atrophic thyroiditis is similar to Hashimoto's thyroiditis
- A goiter is not present

Postpartum Thyroiditis (Hypothyroidism)
- These women develop antibodies to their own thyroid during pregnancy, causing an inflammation of the thyroid after delivery

Systemic Manifestations of Hyperthyroid (Primary or Secondary)
- Symptoms
  - Nervousness
  - Heat intolerance
  - Ictusus
  - Fatigue
  - Palpitation
  - Insomnia
  - Early waking
  - Alopecia
  - Brittle nails
- Signs
  - Sweating
  - Muscle weakness
  - Emotionally labile
  - Tremor
  - Tachycardia
  - Arrhythmia
  - Hypertension
  - Brisk tendon reflex
  - Diabetes
  - ↑ Triglycerides & Ca,
  - ↓ CHO
  - Microcyticanemia
  - Possible goiter
  - Myxedema

Systemic Manifestations of Hypothyroid (Primary or Secondary)
- Symptoms
  - Cold intolerance
  - Weakness
  - Reduced energy
  - Lethargy
  - Muscle cramps
  - Constipation
  - Increased sleeping
  - Weight gain
  - Reduced appetite
  - Joint stiffness
- Signs
  - Cool, scaling skin
  - Puffy hands and face
  - Deep voice
  - Deep voice
  - Myotonia
  - Delirium
  - Bradycardia
  - Slow reflexes
  - Obesity
  - Hypothermia
  - Myxedema

Thyroid Eye Disease (TED)
- Other names used
  - Grave's disease
  - Grave's ophthalmopathy
  - Grave's orbitopathy
  - Exophthalmos in Graves Disease
  - Thyroid Associated Orbitopathy (TAO)
  - Thyroid Orbitopathy
  - Ophthalmic Graves Disease
  - Inflammatory Eye Disease
  - Endocrine Orbitopathy

Why is this so confusing?
- Thyroid Eye Disease
  - Is often seen in conjunction with Graves' Disease (hyperthyroid)
  - Is seen in people with no other evidence of thyroid dysfunction
  - Is seen in patients who have Hashimoto's Disease (hypothyroid)
- Most thyroid patients, however, will not develop thyroid eye disease
Why is this so confusing?

- The eye symptoms usually occur at the same time as the thyroid disease
- However, they may precede or follow the obvious symptoms of the thyroid abnormality
- The incidence of thyroid eye disease associated with thyroid dysfunction is higher and more severe in smokers
- There is no way to predict which thyroid patients will be affected

- While eye disease may be brought on by thyroid dysfunction
- Successful treatment of the thyroid gland does not guarantee that the eye disease will improve
- No particular thyroid treatment can guarantee that the eyes will not continue to deteriorate
- Close-informed, the eye disease may remain active from several months to as long as three years
- There may be a gradual or, in some cases, a complete improvement

Thyroid Eye Disease

- Commonly known as Graves' ophthalmopathy
- About 80% of all patients with TED have the autoimmune hyperthyroid disorder known as Graves' disease
- Another 10% of all cases are seen in patients with autoimmune hypothyroidism, either Hashimoto's thyroiditis, atrophic thyroiditis or Hashitoxicosis
- Another 10% of all cases are seen in people with normal thyroid function
  - When thyroid function is normal, the eye condition is referred to as euthyroid Graves' disease
  - Euthyroid is a term meaning that thyroid function tests are normal. Most people with euthyroid Graves' disease develop a thyroid disorder within eighteen months of the emergence of the eye disorder
  - But some people with euthyroid Graves' disease never develop thyroid dysfunction.

Thyroid Eye Disease

- What causes the Thyroid Eye Disease signs and symptoms?
- The high and low levels of T3 and T4
- The antibodies that are attacking the thyroid gland

Thyroid Eye Disease

- Thyroid Eye Disease has 2 phases
  - A phase secondary to abnormal thyroid hormone levels
    - Increased or decreased FT3 and FT4 levels
    - Once these levels are normalized, ocular symptoms will resolve
  - Congenital Autoimmune form of Thyroid Eye Disease
    - Active phase-stimulating or blocking TRAb are causing ocular activity
    - Plateau phase-reduced activity
    - Resolution phase-symptoms regress and eyes return to normal.

Thyroid Eye Disease

- Hyperthyroid eye symptoms
  - Excess hormone acting on the nerves that supply the eye
  - Usually spastic and include staring
  - Dryness
  - Eyelid retraction

- Hypothyroidism eye symptoms
  - Deficient hormone causing venous congestion, impaired circulation, and fluid stagnation
  - Periorbital edema

- This form of TED resolves within a few weeks after thyroid hormone levels (FT4 and FT3) are corrected and brought back into the normal range.
- The pituitary hormone TSH can stay low or suppressed for many months during the course of treatment for hyperthyroidism and doesn’t mean that the patient is still hyperthyroid.
- TSH also lag at least 6 weeks behind thyroid hormone levels and often remains elevated longer in people who have been hypothyroid.
- Relying on the TSH level can be misleading in treating TED.
Congestive Autoimmune form of Thyroid Eye Disease
(Active phase, Plateau phase, Resolution phase)
- Caused by both stimulating and blocking TSH receptor antibodies (TRAb) and also immune system chemicals known as cytokines
- Secondary targets appear to be TSH receptor antigens (epitopes) located on orbital fibroblasts as well as dermal fibroblasts
- Active “inflammatory” phase of TED varies
  - Symptoms resolve quickly although on average this active phase lasts about 12-18 months
  - TRAb levels are high, patients are smokers, nutrient deficiencies are present, or the patient continues to be exposed to environmental triggers such as excess dietary iodine, the active phase can last as long as 5 years
  - Avoid any lid, muscle or orbital surgery
- Plateau phase and Resolution “passive” phase
  - An individual may be left with structural changes, such as eye protrusion, eyelid retraction, and in some cases, double vision
  - There are corrective procedures that can be performed to address these problems.

Similar receptors are found in the skin, fat and muscle of the orbit

General Ocular Symptoms
- Prominent eyes, stare
- Pain
- Lacrimation
- Eyelid swelling
- Foreign-body sensation
- Double vision
- Photophobia
- Decreased vision in one or both eyes

NOSPECS: Grading System
1969 by S.C. Werner
- Class 0: No signs or symptoms
- Class 1: Only signs, upper lid retraction
- Class 2: Soft Tissue involvement with symptoms
- Class 3: Proptosis
- Class 4: EOM involvement
- Class 5: Corneal Involvement
- Class 6: Light Loss

Within classes 2 to 6 the investigator has to differentiate the severity grades A, B, C.
NOSPECS classifies severity but not the activity or stage (active/inflammatory or passive/congestive)

Euthyroid Graves’ disease
- If thyroid function is normal, how does one develop thyroid eye disease?

Similar receptors are found in the skin, fat and muscle of the orbit

General Ocular Symptoms
- Prominent eyes, stare
- Pain
- Lacrimation
- Eyelid swelling
- Foreign-body sensation
- Double vision
- Photophobia
- Decreased vision in one or both eyes

NOSPECS: Grading System
- Class 0: No signs or symptoms
- Class 1: Only signs (upper lid retraction without lid lag or proptosis)
- Class 2: Soft Tissue involvement with symptoms (excess lacrimation, sandy sensation, retrobulbar discomfort)
  - Grade 0: absent
  - Grade A: minimal: 21mm -23mm
  - Grade B: moderate: 24mm -27mm
  - Grade C: marked: 28mm or more
- Class 3: Proptosis associated with classes 2-6 only
  - Grade 0: absent
  - Grade A: minimal: 3mm -6mm
  - Grade B: moderate: 6mm -9mm
  - Grade C: marked: 9mm or more
- Specify if inequality of >3 mm between eyes, or if progression of >3 mm under observation
NOSPECS: Grading System

- 4: EOM involvement (usually with diplopia)
  - 0: absent
  - A: minimal (limitation of motion, patient reports diplopia but no obvious restriction
  - B: moderate (evident restriction of motion)
  - C: marked (position of globe is fixed)
- 5: Corneal involvement (due to proptosis, incomplete closure, lagophthalmos)
  - 0: absent
  - a: minimal (staining)
  - b: moderate (ulceration)
  - c: marked (clouding, necrosis, perforation)
- 6: Sight loss (due to optic nerve involvement)
  - 0: absent
  - A: minimal (disc pallor or edema, or VF defect, vision 20/20-20/60)
  - B: moderate (same as A but VA 20/70-20/200)
  - C: marked (blindness, VA < 20/200)

LEMO Classification

- 1991-Boergen and Pickardt
- Complements NOSPECS
- 4 finding-categories
  - Lid
  - Exophthalmos
  - Muscular
  - Optic nerve
- Grade between 0 and 4 depending on severity
- LEMO: classifies severity but not the activity or stage
  (active/inflammatory or passive/congestive)

Grading Scales

- New grading scales are trying to be developed to not only grade the severity but also help to determine if inflammatory or passive stage

Lid Involvement

- Lid Retraction
- Lid Lag
- Lagophthalmos
Thyroid Dysfunction and the Eye  

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Lid Retraction

- Scleral show in primary gaze
- Occurs in ~90% of Grave’s patients
  - Excess innervation of Müller’s muscle
  - Fibrosis inferior rectus
  - Mechanical restriction or infiltration of levator
  - Increased orbital volume: excess exophthalmos

Normal Lid Position

- Upper lid intersects cornea at the 2 and 10 o’clock position
  - ~2 mm below the limbus
- Lower lid coincident or 1-2 mm below the limbus

Eyelid Lag: von Graefe’s Sign

- Immobility or lagging of upper eyelid on downward gaze
- Fibrosis of the inferior rectus muscle may induce lower lid retraction

Soft Tissue Involvement

- Conjunctiva
- Chemosis
- Periorbital edema

Conjunctiva

- Conjunctival and epibulbar injection
  - Especially near the horizontal recti insertions
- Chemosis
  - Edema of the conjunctiva and eyelids
- Superior Limbus Keratoconjunctivitis
  - 65% correlation between HK and systemic thyroid disease
  - Reiter’s arthritis
  - Trigeminal neuralgia

Lagophthalmos

- Inability to form a complete lid closure with a normal blink due to Exophthalmos/Proptosis
- Often leads to corneal exposure

"If it is Red think TED"

Dr. Andy Morgenstern 12-7-2013 OMS-Contemporary Resort
**Periorbital Edema**
- Inflammation of the subcutaneous connective tissue
- May be first sign of thyroid eye disease
- Greatest in the morning

**Infiltrative Orbitopathy (Exophthalmos/Proptosis)**
- Thyroid Eye Disease is most common cause of unilateral and bilateral exophthalmos
- The term exophthalmos is reserved for prominence of the eye secondary to thyroid disease
- May need MRI to determine or obvious exophthalmos may be present
- It is permanent in 70% of cases
- Caused by increased volume of the extra ocular muscles:
  - Lymphocytic infiltration
  - Proliferation of fibroblasts
  - Edema within the interstitial tissue of the muscle

**Exophthalmometry**
- Is race dependent (Asian versus Black men is statistically significant)
- Hertel or Luedde results
- Adults
  - Average reading 17 mm
  - 95% of population have readings between 13-21 mm
- General concern:
  - A difference of 2 mm or more between the eyes
  - A measurement of more than 24 mm

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<td>Asians</td>
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Restrictive Myopathy

- Secondary to edema and fibrosis of EOM’s
- Inferior Rectus (IR) muscle is most commonly involved
- Occurs in 30-50% of patients
- Diplopia may be transient but in 30% it’s permanent

IOP in Thyroid Eye Disease

- A rise in IOP has been reported with TED
- I would have higher suspicion when you see:
  - Periorbital edema
  - Exophthalmos, proptosis
  - Restrictive myopathy
- Some literature reports IOP in up gaze to be part of the diagnoses of thyroid dysfunction

Restrictive Myopathy

Obvious restrictive myopathy but also note the periorbital edema, and conjunctival hyperemia

Corneal Exposure

- Exposure keratopathy, secondary to exophthalmos and lagophthalmos
- Significant threat to visual function

Optic Neuropathy

- Affects 5% of patients
- Usually mild to moderate exophthalmos and shallow orbits
- Enlargement of the rectus muscles compresses ONH or its blood supply at the apex of the orbit
- Compression MAY occur without significant proptosis
- Compresive and/or ischemic and/or toxic

Treatment of Thyroid Eye Disease

- Depends on what phase of the disease we are in:
  - Phase secondary to abnormal thyroid hormone levels
  - Active “inflammatory” phase
  - Plateau phase and Resolution “Passive” phase
- Depends on what orbital tissue or structures are involved
- Depends on the risk of vision loss
- Depends if primary, secondary or tertiary thyroid dysfunction
- Management consists of:
  - Control of inflammation
  - Prevention of outer and visual damage
  - Addressing ocular motort disorder
  - Improving cosmetic disfigurement
- Patient education is essential
- Communication with an endocrinologist or internist will ensure proper patient care
**Treatment of Thyroid Eye Disease**

- **Palliative (hormone imbalance, active, passive)**
  - Lubricants
  - Topical anti-inflammatory (Lotemax/Restasis)
- **Prisms**
- **Steroids (active phase)**
  - Oral
  - Periocular injection
- **Oral steroids (active phase)**
- **Orbital radiotherapy (active phase)**
- **Orbital Decompression (passive phase)**
  - Fat removal orbital decompression (FROD)
  - Bone removal orbital decompression (BROD)

**Smoking causes the thyroid eye disease to be more severe**
**Smoking causes treatments to be less effective**

**Paradigm shifts**
- Decrease in orbital radiotherapy
- Waiting for passive stage but doing surgery
- Increase usage of fat removal orbital decompression as first approach
- Peri-orbital injection of steroids for recurrent disease after orals

**Future**
- Looking for better or different ways to treat the active phase of this disease

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**Lid Retraction, Eyelid Lag, Lagophthalmos**

- Must treat underlying thyroid dysfunction.
- Treat the exposure keratitis with lubricants.
  - Tape eyelids shut at night
  - Lid weights
  - Moisture chamber at night
  - Antibiotic ointment.

**Passive Phase**
- Surgical Management
  - Inferior rectus recession
  - Mullerotomy
  - Recession of lower lid retractors

**Infiltrative Orbitopathy**

- Orbital disease consult
  - Systemic steroids to reduce inflammation
  - Low dose radiotherapy
  - Surgical orbital decompression

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**Conjunctiva, Periorbital edema**

- Topical lubricants
  - Artificial tears
  - Ointments at night
  - Topical steroids
  - Keratitis
- Tape eyelids closed at night or use mask
- Elevate head at night to decrease lid edema
- Oral diuretics Aprepitant
- Oral steroids
  - 60-80mg/day for 3 months
- IV steroids
- Peri-orbital steroids
  - Renalog test 1 month
### Restrictive Myopathy
- Non-surgical (while waiting for stability)
  - Teach proper head position to alleviate diplopia.
  - Prism in spectacle correction (fresnel or ground in).
  - Oral steroids.
  - Botulinum toxin injection.
- Surgical Consult
  - Rectus muscle recession.
  - Diplopia in primary and reading gaze.
  - Stable angle of deviation for at least 6 months.
  - No evidence of active disease.
  - Binocular vision in at least primary and reading positions.

### Corneal Exposure
- Manage the corneal defect as first line
  - Lubricating and antibiotic.
  - Lid taping.
  - Moisture barrier.
- Surgical Consult
  - High dose oral steroids
    - 120-140 mg/day x 7 days.
  - Orbital decompression.

### Optic Neuropathy
- Systemic Steroids
  - If rapidly progressive and painful in the early stage of the disease.
  - Only if no contraindications.
  - Prednisolone 80-100 mg, expect results within 48 hours. Taper dose and d/c within 3 weeks.
- IV Methylprednisolone.
- Radiotherapy: if contraindication to steroids.
- Orbital decompression.

### Orbital Decompression
- Not effective if no medical treatment
  - One-wall decompression
    - 3-6 mm retroplacement of the globe.
  - Three-wall decompression
    - 6-10 mm retroplacement.
  - Four-wall decompression
    - 10-15 mm retroplacement.

### Orbital Decompression (Surgical/Cosmetic)

### Thyroid Eye Disease and Depression
- When facial disfigurement occurs, thyroid eye disease is equivalent to the diagnosis of cancer and AIDS.
Orbital Decompression
(Medical/Vision Threatened)

IOP in Thyroid Eye Disease

- A rise in IOP has been reported with TED
- I would have higher suspicion when you see:
  - Periorbital edema
  - Exophthalmos, proptosis
  - Restrictive myopathy
- Some literature reports IOP in up gaze to be part of the diagnoses of thyroid dysfunction. Let’s discuss

IOP in Thyroid Eye Disease

Glaucoma

- PURPOSE: To estimate the prevalence of open-angle glaucoma, including normal-tension glaucoma, in association with Graves’ disease.
- CONCLUSIONS: The prevalence of normal-tension glaucoma as well as open-angle glaucoma and ocular hypertension was significantly higher among patients with Graves’ disease than in the general population.

ICD-9 Codes

- 242.90 or 242.91 Hyperthyroidism: Cause not specified
  - This includes TSH-mediated, Postpartum, Destructive, and Recurrent
- 242.00 or 242.01 Hyperthyroidism: Graves Disease; Autoimmune Stimulatory
  - This includes Postpartum, Toxicosis
- 242.10 or 242.11 Hyperthyroidism: Toxic Nodule (uninodular)
- 242.20 or 242.21 Hyperthyroidism: Toxic Nodular Goiter (multinodular)
- 242.30 or 242.31 Hyperthyroidism: Excessive Thyroid Hormone Ingestion
- 245.1 Thyroiditis, subacute
- 245.2 Thyroiditis, lymphocytic
- 775.3 Hyperthyroidism: Neonatal
- 246.8 Abnormal Thyroid Hormone Binding Protein
- 244.9 Hyperthyroidism: Amy Treatment
- 243 Hyperthyroidism, complicated
- 244.0 Hyperthyroidism, Post-surgical


ICD-9 Codes

- 244.1 Hypothyroidism, Post-obese
- 193 Thyroid Cancer: All Types
- 785.6 Lymphadenopathy
- 246.0 Disorder of Calcitonin
- 240.0 Thyroid Nodule: Nontoxic (uninodular)
- 241.1 Euthyroid Goiter: Multinodular
- 246.2 Thyroid Cyst:
- 246.3 Hemorrhage of thyroid
- 246.4 Thyroiditis: Nontoxic chronic lymphocytic thyroiditis
- 246.5 Euthyroid Goiter: Benign Dystrophy
- 240.0 Euthyroid Goiter: Simple
- 246.7 Euthyroid Goiter: Dystrophy, Dystrophy
- 246.8 Thyroiditis: Amy Treatment
- 227.3 Thryroid Tumor: Includes craniopharyngioma, Rathke’s cleft cyst
- 251.2 Thryroid insufficiency
Laboratory Testing

- Thyroid Hormone Levels
  - Serum TSH concentration
  - Serum total T3 (Triiodothyronine)
  - Estimation of the serum free T4 (or T3) concentration
  - Thyroglobulin (Tg) level
- Anti-thyroid antibodies
  - Thyroglobulin (Tg) Antibodies (TgAb)
  - Anti-TPO antibodies
- Commonly Used Thyroid Tests
  - Basal T4 uptake test
  - Serum TSH test
  - Thyroid hormone suppression test
  - Neutrophils
  - Needle Biopsy
  - Thyroid Echo

Hypothyroid

- Low FT4, High TSH, indicates primary check antibodies
- Low FT4, Low TSH, indicates secondary or tertiary, TRH stimulation
- Hashimoto’s (primary disease)
  - Most common
  - Low FT4, High TSH, High Anti-TPO Abs, High levels of Thyroglobulin (Tg) Antibodies (TgAb), Anti-TB Recp Abs (approx 40% present)
  - Autoimmune atrophic thyroiditis
    - Low FT4, High TSH, Low Anti-TPO Abs, High levels of Thyroglobulin (Tg) Antibodies (TgAb), Anti-TB Recp Abs (approx 50% present)
- Treatment: Levothyroxine (Synthroid, Levothroid, Levoxyl, Unithroid)

Hyperthyroid

- High FT4, Low TSH
- TSH present

Sign’s in Thyroid Eye Disease

- Dalrymple’s sign: Lid retraction
- von Graefe’s sign: Upper lid lag on downward gaze
- Gauthier’s sign: Lower lid lag on downward gaze
- Boston’s sign: Jerky irregular movement of the upper lid on downward gaze
- Bilower’s sign: Increased pigmentation of the lid
- Bell’s sign: Infrequent blinking
- Kosker’s sign: Increased lid retraction with visual fixation
- Brash’s sign: Puffy swelling of the lid
- Bouveret’s sign: Tension of closed lids
- Mollison’s sign: Weakness of convergence
- Batten’s sign: Bulging of one or more extraocular muscles
- Baker’s sign: Weakness of fixation on lateral gaze
- Crouzon’s sign: Jerky papillary constriction to consensual light
- Trigeminal nerve: Unequal distribution of the pupils
- Jeffery’s sign: Absence of forehead wrinkling on upward gaze

Thank-You
and
Hope You Enjoyed
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