Hyperthyroidism: Causes, Symptoms and Treatment

Hyperthyroidism is a clinical syndrome resulting from excessive production and release of thyroid hormones (thyroxine, T4, and triiodothyronine, T3) by the thyroid gland, leading to a hypermetabolic state known as thyrotoxicosis.

Pathophysiology

- Excess thyroid hormones increase basal metabolic rate, causing heightened oxygen consumption and heat production.
- When caloric intake does not match increased metabolic demand, catabolism of fat and protein leads to weight loss.
- Muscle protein breakdown causes muscle weakness despite hyperactivity and fine tremors due to increased beta-adrenergic receptor sensitivity.
- Peripheral vasodilation facilitates heat dissipation, resulting in heat intolerance and sweating.
- Cardiovascular effects include increased heart rate, stroke volume, and cardiac output, partly due to heightened sensitivity of cardiac tissue to catecholamines.

Causes of Hyperthyroidism

1. **Graves' Disease (Diffuse Toxic Goiter):**
    - Autoimmune disease with thyroid-stimulating immunoglobulins that activate TSH receptors, causing diffuse thyroid hyperplasia and hormone overproduction.
2. **Toxic Adenoma ("Hot Nodule"):**
    - Autonomous functioning thyroid nodule producing excess hormone independent of TSH regulation.
3. **Toxic Multinodular Goiter (Plummer’s Disease):**
    - Multiple autonomously functioning nodules cause sustained hormone overproduction.
4. **Subacute Thyroiditis:**
    - Viral or postpartum inflammation causes transient release of preformed thyroid hormone, usually self-limiting.
5. **Drug-Induced Hyperthyroidism:**
    - Amiodarone (iodine-rich antiarrhythmic) can cause thyrotoxicosis by excess iodine or destructive thyroiditis.
    - Iodine contrast exposure can precipitate hyperthyroidism in patients with preexisting nodular thyroid disease.

Clinical Features

- Nervousness, irritability, anxiety
- Heat intolerance, increased sweating
- Weight loss despite increased appetite
- Palpitations, tachycardia, atrial fibrillation (especially in elderly)

- Tremors (fine, distal)
- Muscle weakness (proximal)
- Warm, moist skin
- Lid lag and stare (due to sympathetic overactivity)
- Systolic hypertension with widened pulse pressure
- Oligomenorrhea or amenorrhea
- Diarrhea, hyperdefecation

Diagnostic Workup

- **Thyroid Function Tests:**
  - Suppressed TSH (most sensitive marker)
  - Elevated free T4 and free T3 (may see T3 toxicosis if T3 elevated with normal T4)
- **Autoantibodies:**
  - TSH receptor antibodies (TRAb) for Graves' disease
  - Anti-thyroid peroxidase (anti-TPO) antibodies (can be elevated)
- **Imaging:**
  - Thyroid ultrasound to evaluate nodules or goiter
  - Radioactive iodine uptake scan to differentiate causes (diffuse high uptake in Graves, focal in toxic adenoma, patchy in multinodular goiter)

Management

Supportive Therapy

- Beta-blockers (e.g., propranolol) for symptom control of tachycardia, tremors, anxiety.
- Adequate hydration and nutrition.

Antithyroid Medications

- **Methimazole:** Preferred due to potency and longer half-life.
- **Propylthiouracil (PTU):** Reserved for first trimester pregnancy, thyroid storm, or methimazole intolerance.
- Titrate doses every 4-6 weeks based on thyroid function tests.
- Treatment duration often 12-18 months; remission rates vary.

Definitive Therapy

- **Radioactive Iodine (RAI) Therapy:**
  - Oral administration causes gradual thyroid tissue destruction.
  - Contraindicated in pregnancy, lactation, young children, and severe ophthalmopathy.
  - May result in hypothyroidism requiring lifelong levothyroxine replacement.
- **Surgery (Thyroidectomy):**
  - Indicated in large goiters causing compressive symptoms, severe ophthalmopathy, pregnancy intolerance, or patient preference.
  - Requires careful perioperative management.

Special Considerations

- **Ophthalmopathy:**
  - Mild: Supportive care with lubricants, sunglasses.
  - Severe (vision-threatening): High-dose corticosteroids, orbital decompression, radiotherapy