NOTES HYPERPARATHYROIDISM & HYPOPARATHYROIDISM

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

 An imbalance of parathyroid hormone (PTH) due to overproduction or underproduction by the parathyroid gland resulting in impaired regulation of calcium and other electrolytes

Hyperparathyroidism

 ↑ PTH → ↑ bone resorption and ↑ renal reabsorption of calcium → ↑ serum calcium levels → asymptomatic or symptomatic hypercalcemia

Hypoparathyroidism

• \downarrow PTH $\rightarrow \downarrow$ serum calcium \rightarrow symptomatic hypocalcemia

RISK FACTORS

- Hyperparathyroidism
 - Genetic mutations, chronic kidney disease, ↓ vitamin D intake/absorption, hyperplasia of parathyroid glands
- Hypoparathyroidism
 - Most commonly iatrogenic cause due to accidental removal or damage to parathyroid blood supply during thyroid surgery

COMPLICATIONS

- Hyperparathyroidism
 - Osteoporosis, osteitis fibrosa cystica, nephrolithiasis, keratopathy, symptomatic hypercalcemia (e.g. hypertension, cardiac arrhythmias)
- Hypoparathyroidism
 - Symptomatic hypercalcemia (e.g. respiratory paralysis, cardiac arrhythmias)

SIGNS & SYMPTOMS

See individual disorders

DIAGNOSIS

LAB RESULTS

 Measure serum PTH, calcium, phosphate, magnesium, 25-hydroxyvitamin D, urine calcium

OTHER DIAGNOSTICS

Genetic testing

TREATMENT

MEDICATIONS

- Hyperparathyroidism
 - Vitamin D analogs, calcimimetics, bisphosphonates
- Hypoparathyroidism
 - IV calcium gluconate (acute), vitamin
 D analogs, synthetic PTH, thiazide
 diuretics (↓ renal calcium excretion)

SURGERY

- Hyperparathyroidism
 - Partial/complete parathyroidectomy; radiofrequency ablation

OTHER INTERVENTIONS

- Hyperparathyroidism
 - Physical activity to ↓ bone resorption, maintain hydration to ↓ nephrolithiasis, vitamin D supplements
- Hypoparathyroidism
 - Calcium, magnesium, and vitamin D supplements

HYPERPARATHYROIDISM

osms.it/hyperparathyroidism

PATHOLOGY & CAUSES

TYPES

Primary

 Parathyroid gland creates PTH independently of calcium levels, does not respond to normal feedback mechanisms

Secondary

- Parathyroid gland hyperplasia, excess parathyroid hormone secreted in response to chronic hypocalcemia
- Impaired kidney function; kidneys do not filter phosphate properly into urine, make insufficient calcitriol
 - AKA renal osteodystrophy (bone pain, fracture)
- Altered calcium, phosphate levels → increased parathyroid hormone levels → bone resorption

Tertiary

- Develops in individuals with secondary hyperparathyroidism for many years, often due to hyperplasia of parathyroid glands
- Autonomous secretion of PTH separately from blood calcium levels
 - Even if causes of secondary hyperparathyroidism (e.g. renal transplant) corrected, increased PTH persists

RISK FACTORS

Primary

- Genetic mutations
 - Multiple endocrine neoplasia (MEN) syndrome

COMPLICATIONS

Primary

 Brown tumors, large bone cysts (due to high osteoclast activity)

SIGNS & SYMPTOMS

• "Stones, thrones, bones, groans, and psychiatric overtones"; see mnemonic

Primary, tertiary

 Slower muscle contractions caused by less excitable neurons secondary to hypercalcemia

Secondary

 Bone resorption/renal osteodystrophy; calcification of blood vessels, soft tissues



MNEMONIC

- Signs and symptoms of hyperparathyroidism
- Stones: calcium-based kidney stones, gallstones
- **Thrones:** toilet; polyuria (frequent urination) from impaired sodium, water reabsorption
- **Bones:** pain from chronic hormone-driven demineralization
- **Groans:** constipation, muscle weakness
- Psychiatric overtones: depressed mood, confusion

DIAGNOSIS

LAB RESULTS

Primary

- High total serum calcium (hypercalcemia), low phosphate (hypophosphatemia), high PTH valve during diastole
- Hypercalciuria from excess calcium loss through urine, may cause dehydration
- Serum 25-hydroxyvitamin D
 Determine type

Secondary

Low calcium, high phosphate, low vitamin D

Tertiary

• Normal-high calcium, high PTH, low vitamin D

TREATMENT

MEDICATIONS

Primary, tertiary

- Calcimimetics
 - Drugs that imitate calcium by attaching to CaSR on parathyroid cells
 - If surgery not an option

Secondary

- Hyperphosphatemia
 - Phosphate binders
- Vitamin D supplements
 - Increase calcium absorption, reduce PTH synthesis
- Calcitriol, vitamin D analogs (doxercalciferol, paricalcitol)
 - Suppress PTH levels
- Calcimimetics
 - \circ Modulate CaSR \rightarrow increase sensitivity of serum calcium \rightarrow decrease PTH levels

Tertiary

SURGERY

Remove abnormal parathyroid glands



Figure 17.1 An ultrasound of the neck demonstrating a large parathyroid adenoma situated posteriorly and to the right of the right thyroid lobe. The skin surface is at the top of the image.



Figure 17.2 A X-ray image of the forearm demonstrating a brown tumor of the distal radius in an individual with hyperparathyroidism.

HYPERPARATHYROIDISM LABS

	SERUM CALCIUM	SERUM PHOSPHATE	PARATHYROID HORMONE
PRIMARY HYPERPARATHYROIDISM	High	Low	High
SECONDARY HYPERPARATHYROIDISM	Low	High	High
TERTIARY HYPERPARATHYROIDISM	High	Varies	High

HYPOPARATHYROIDISM

osms.it/hypoparathyroidism

PATHOLOGY & CAUSES

- Underproduction of parathyroid hormone (PTH); hypo- = under/low
- No parathyroid hormone → ↓ bone resorption, ↓ renal calcium reabsorption, ↓ intestinal calcium reabsorption → hypocalcemia, hyperphosphatemia → ↑ cell excitability → tetany, paresthesias, seizures, arrhythmias

CAUSES

Autoimmune disorders

Magnesium deficiencies

Latrogenic

- Most common
- Thyroid/parathyroid surgery/radiation

Hereditary abnormalities

- DiGeorge syndrome (DGS)
- Autosomal dominant hypoparathyroidism
- Albright hereditary osteodystrophy (pseudohypoparathyroidism)
 - Kidney resistance to PTH, increased PTH

SIGNS & SYMPTOMS

- Asymptomatic/life-threatening
 - Degree, duration of hypocalcemia
 - \circ Muscular dysfunction \rightarrow respiratory paralysis \rightarrow death
- ECG changes
 - Prolonged QT, ST
 - Torsades des pointes
 - Atrial fibrillation

Acute

- Muscular spasms/cramps → tetany → Chvostek, Trousseau signs
- Perioral numbness, paresthesias, seizures

Chronic

- Extrapyramidal movements → basal ganglia calcifications
 - Dystonias, parkinsonism, athetosis, hemiballismus, oculogyric crisis
- Cataracts
- Dermatologic manifestations
 Dry, coarse skin; brittle nails; patchy alopecia



Figure 17.3 Dry, brittle nails are a dermatologic manifestation of chronic hypoparathyroidism.

DIAGNOSIS

LAB RESULTS

- Hypocalcemia, low serum PTH
- Hypercalciuria

OTHER DIAGNOSTICS

Medical history of thyroid surgery/radiation

TREATMENT

MEDICATIONS

- IV calcium gluconate (severe cases)
- Oral calcium (mild-moderate cases)
- Vitamin D supplementation
- Synthetic PTH