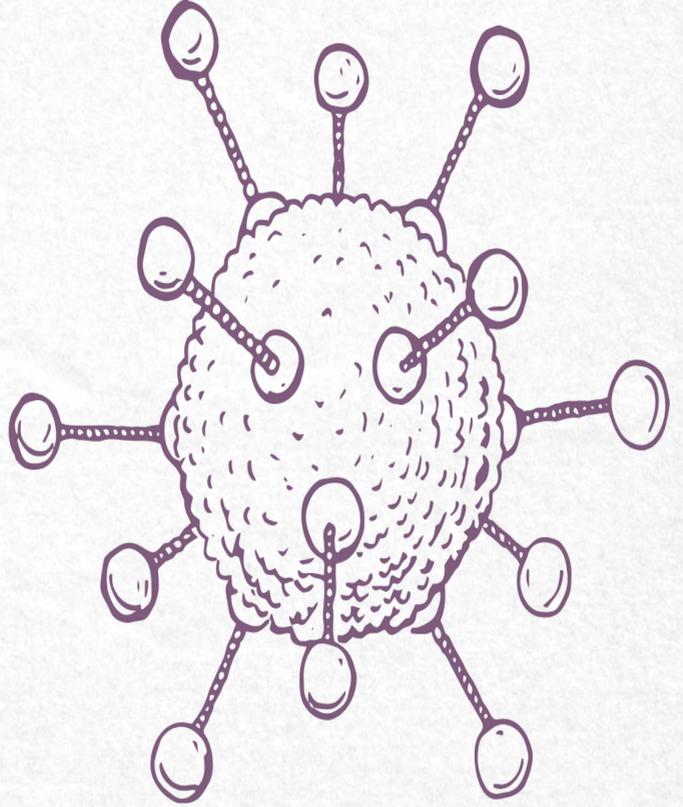


لاخوف عليكم

همة الأسنان لديكم

PATHOLOGY

ENDOCRINE 3



همة الأسنان



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Endocrine Lecture 3

parathyroid diseases

Hyperparathyroidism

- Primary form
- Secondary form
- Tertiary form (less common)
- Primary: autonomous overproduction of PTH
- Secondary & tertiary: due to chronic renal insufficiency

A) Primary hyperparathyroidism

- Common endocrine disorder, major cause of hypercalcemia
- Increase in detection due to routine serum calcium assays in hospitalized patients
- The frequency of occurrence of the various parathyroid lesions underlying primary hyperparathyroidism
 1. Adenoma: 85%-95%
 2. Primary hyperplasia (diffuse or nodular): 5%-10%
 3. Carcinoma: ~1%

Parathyroid carcinomas



- May be circumscribed lesions that are difficult to distinguish from adenomas,
- Or they may be clearly invasive neoplasms.
- These tumors enlarge one parathyroid gland
- Sometimes exceed 10 g in weight.
- The tumor mass is usually enclosed by a dense, fibrous capsule.
- The diagnosis is not based on cytologic detail-unreliable
- Diagnosis: invasion of surrounding tissue and metastasis are the only definitive criteria
- Local recurrence occurs in one third of cases, and more distant dissemination occurs in another one third

Parathyroid Hyperplasia

- Multiglandular process (It usually affects all the parathyroid glands meaning more than one gland, not just a single one).
- In some cases, enlargement may be grossly apparent in only one or two glands, complicating the distinction between hyperplasia and adenoma

B) Secondary Hyperparathyroidism

- Cause: Chronic low serum calcium level , mainly due to renal failure which leads to compensatory overactivity of the parathyroids
- The mechanisms by which chronic renal failure induces secondary hyperparathyroidism are complex :
 - 1) Chronic renal insufficiency is associated with decreased phosphate excretion, which results in hyperphosphatemia , the elevated serum phosphate levels directly depress serum calcium levels **Hyperphosphatemia (↓ phosphate excretion → ↓ calcium)**
 - 2) Loss of renal α -hydroxylase activity, which is required for the synthesis of the active form of vitamin D, reduces the intestinal absorption of calcium ,these alterations cause chronic hypocalcemia, which stimulates the activity of the parathyroid gland **↓ Vitamin D (↓ α -hydroxylase → ↓ calcium absorption)**
 - Leads to hypocalcemia → stimulates parathyroid glands

Morphologic changes in other organs:

I. Skeletal changes :

1. Ostitis fibrosa cystica	2. Brown tumors of hyperparathyroidism
<ul style="list-style-type: none"> - High PTH Increases osteoclastic activity, which results in erosion of bone and mobilization of calcium salts, affecting the metaphyses of long bones. - Bone resorption is accompanied by increased osteoblastic activity and the formation of new bone - In more severe cases, the cortex is grossly thinned and the bone marrow contains increased amounts of fibrous tissue accompanied by foci of hemorrhage and cysts 	<ul style="list-style-type: none"> - Aggregates of osteoclasts, reactive giant cells, and hemorrhagic debris form masses that may be mistaken for neoplasm

II. Renal changes :

1. Nephrolithiasis	2. Nephrocalcinosis
- PTH-induced hypercalcemia favors formation of urinary tract stones calcium contain stones	Calcification of the renal interstitium and tubule

III. Metastatic calcification

- Calcification secondary to hypercalcemia also may be seen in other sites, including the stomach, lungs, myocardium, and blood vessels- Calcification secondary to hypercalcemia also may be seen in other sites, including the stomach, lungs, myocardium, and blood vessels

Clinical Features :

- Affects adults, is much more common in women than in men
- The most common manifestation is an increase in serum calcium
- Primary hyperparathyroidism is the most common cause of clinically silent hypercalcemia.
- Most common apparent hypercalcemia cause: cancer which can cause hypercalcemia
- Cancer causes:
 - A) secretion of PTH-like polypeptides from cancers of other organs, such as lung adenocarcinoma and this is called paraneoplastic syndrome
 - B) osteolytic bone metastases

Other laboratory findings

- a. hypophosphatemia
- b. increased urinary excretion of Calcium and phosphat

Clinical Manifestations :

1. Pain secondary to :
 - a. Fractures (osteoporosis or osteitis fibrosa cystica)
 - b. Renal stones was at one time a prominent manifestation of primary hyperparathyroidism
2. GI: constipation, nausea, ulcers, pancreatitis, gallstones
3. CNS: depression, lethargy, seizures
4. Neuromuscular: weakness, hypotonia
5. Polyuria, secondary polydipsia