Parathyroid Disorders

435 medicine teamwork

[Important | Notes | Extra | Editing file]

lecture objectives:

- Understand Calcium and related hormones physiology.
- Understand hyperparathyroidism.
- Understand hypoparathyroidism.

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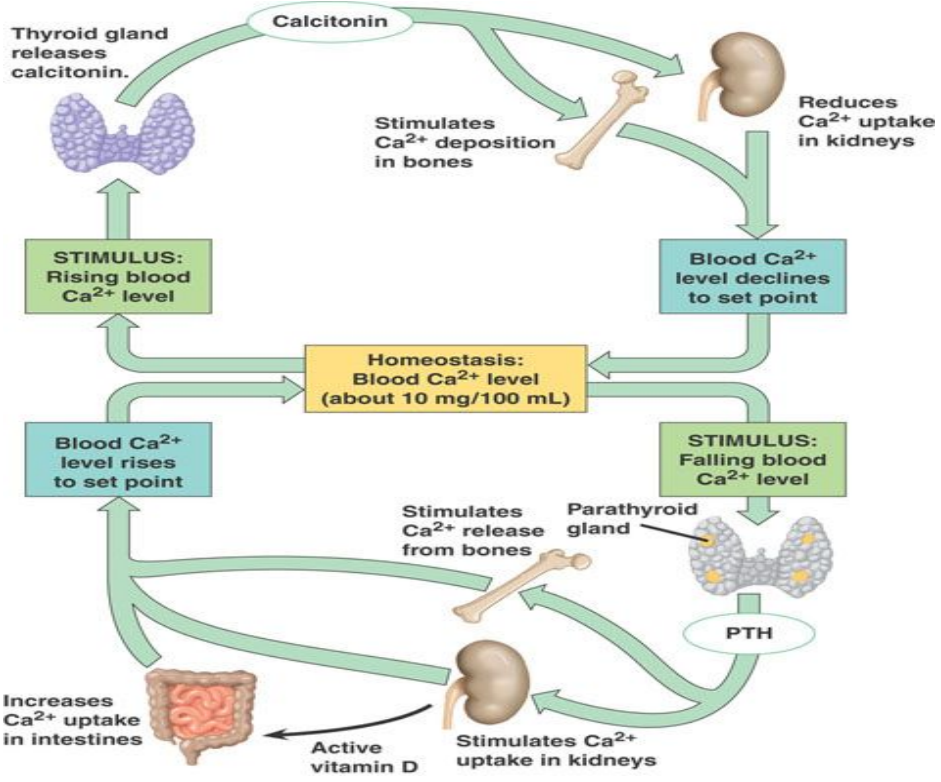
References:
Dr.mona & Dr.Riyadh Slides + Davidson + Kumar+step up

Basic Review of Calcium physiology

Calcium homeostasis is maintained by 3 hormones: PTH(main hormone), Calcitonin and Vitamin D.		
Hormone	**1. Parathyroid hormone (PTH)**	**2. Calcitonin**
Origin	Secreted from Parathyroid gland	Secreted by the parafollicular cells (C cells) of the thyroid gland
its effect	↑ plasma calcium concentration. ↓ plasma phosphate concentration	↓ plasma calcium concentration. ↑ plasma phosphate concentration
Hormone physiology	- it's released in response to hypocalcemia. - The PTH acts directly on the bones and kidneys and **indirectly** on the intestine through its effect on the synthesis of 1,25 $(OH)_2D_3$ (in the kidney) - Its production is regulated by the concentration of serum ionized calcium. (serum ionized calcium has a very narrow normal window from 2.15 to 2.25)	- it's released in response to small increases in plasma ionic calcium. - Calcitonin acts on the kidney and bones to restore the level of calcium to just below a normal set point which in turn inhibits secretion of the hormone. - Calcitonin is the **physiological antagonist of PTH**.
3. Vitamin D (Sterol hormone)		
- Synthesized by the body or taken in food with help of PTH. - Produced in the skin by 7-dehydrocholesterol under the effect of direct UV sunlight - Increases Renal and intestinal calcium and Phosphate absorption. - Most abundant form of Vit.D is: 25-hydroxyvitamin D. It is the one measured in lab tests - Most potent form of Vit.D is: 1,25-dihydroxyvitamin D3. ✓ Best time for sun exposure in Riyadh: Winter(10 am -2 pm)_ Summer (9 am -10:30 & 2-3 pm)		



(Calcium goes up leading to a regulatory response that will suppress the action of PTH on the bone. and increase the kidney excretion of calcium and stimulate calcitonin and vice versa)

(Calcium is very important for neuromuscular junction all over the body and important for cardiac function as well)

Hypoparathyroidism

Deficient secretion of PTH which manifests itself biochemically by ↓ Ca^{2+} (hypocalcemia), ↑ PO_4 (hyperphosphatemia) diminished or absent circulating iPTH (immunoreactive parathyroid hormone). and clinically the symptoms of **neuromuscular hyperactivity**. (usually patients are symptomatic. Why do we have hyperactivity if the calcium is low? because the excitatory threshold gets lower due to the hypocalcemia)

Causes:

1- Hypocalcemia with Hypoparathyroidism causes:

1- Surgical hypoparathyroidism: (the commonest cause)	2- Idiopathic hypoparathyroidism:	3- Functional hypoparathyroidism:
Head and neck surgeries: a. thyroidectomy. b. parathyroidectomy c. Radical surgery for head and neck malignancies. - After anterior neck exploration for thyroidectomy, abnormal parathyroid gland removal, excision of a neck lesion. It could be due to the removal of the parathyroid glands or due to interruption of blood supply to the glands. - a good surgeon will let the nurse check the calcium levels postoperatively every 4 hours to see if the patient's calcium level drop or not.	most of the time we think it happens because of autoimmune at a younger age or old age "autoimune is the 2nd common cause" (*Don't forget the autoimmune diseases cluster together so whenever you have autoimmune disease look for other autoimmune diseases) - a form occurs at an **early** age (genetic origin) with autosomal recessive mode of transmission "Multiple Endocrine Deficiency Autoimmune Candidiasis (MEDAC) syndrome". - Juvenile familial endocrinopathy. - Hypoparathyroidism, Addison's disease, mucocutaneous candidiasis (HAM) syndrome,AKA Polyglandular autoimmune syndrome Type 1. هذا المرض مره انترستنق تتأثر فيه الغدد الصم الواحدة تلو الأخرى وعادة اول مانيفيستيشن تظهر يكون مالها علاقه بالغدد ,الي هي candidial infection (monilia) تظهر في سن الاربع سنوات يعدها في سن 6-8 سنوات الغدة الجار درقيه تروح بعدها بسنتين الغدة الكظريه تروح,هذي السيندروم ,دائما تحدث بهذا النمط مايصير العكس - Circulating antibodies for the parathyroid glands and the adrenals are frequently present. - Other associated disease: ○ Pernicious anemia ○ Ovarian failure ○ Autoimmune thyroiditis ○ Diabetes mellitus - The late onset form occurs sporadically without circulating glandular autoantibodies.(without autoimmunity)	**Hypomagnesemia**: Magnesium is necessary for PTH to be released from the gland and also for the peripheral action of the PTH. Low magnesium levels ⟶ lead to **increased urinary loss of calcium** ⟶ it is reversed by magnesium replacement.

1- Hypocalcemia with Non-Hypoparathyroidism causes:

1- **Chronic Renal Failure**	2- Vitamin D deficiency	3- Others
- The **most common cause** of hypocalcemia. - The kidney converts 25 hydroxy-D to the more active 1,25 hydroxy-D⟶ So Renal failure = Loss of vitamin D - Hyperphosphatemia in CKD lower Ca conc.	- Vitamin D and Calcium deficiency. - Vitamin D resistance. - Decreased intestinal absorption of vitamin D or calcium due to **primary small bowel disease**, short bowel syndrome, and post-gastrectomy syndrome. - Drugs that cause rickets or osteomalacia: such as phenytoin, phenobarbital, **cholestyramine**, and laxative.	- **Acute pancreatitis** (quite common). - Citrated blood in massive transfusion (not uncommon) - Low plasma albumin, e.g. malnutrition, chronic liver disease. because Calcium bound to albumin. - Pseudohypoparathyroidism (syndrome of end-organ resistance to PTH) - Hyperphosphatemia:in phosphate therapy.

Pseudohypoparathysoidism and Pseudopseudohypoparathyroidism: for extra reading

- A rare familial disorders with target tissue resistance to PTH.(here in pseudohypoparathyroidism: high PTH and low Ca while in post surgical and autoimmune hypothyroidism everything is low)
- There is hypocalcemia, hyperphosphatemia, with increased parathyroid gland function.
- There is also a variety of congenital defects in the growth and development of skeleton including:
  - Short stature
  - Short metacarpal and metatarsal bones.
- In pseudopseudohypoparathyroidism they have the **developmental defects** without the biochemical abnormalities.
- The diagnosis is established when low serum calcium level with hyperphosphatemia is associated with increased serum iPTH as well as **diminished nephrogenous CAMP** and phosphature response to PTH administration

Clinical features:

A. Neuromuscular:

- one of the classic symptoms is a **carpopedal spasm** could be spontaneous or you can induce it.

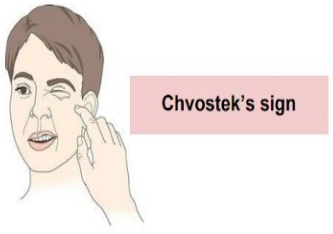
–The rate of decrease in serum calcium is the major determinant for the development of neuromuscular complications. "sudden drop" (the signs and symptoms depends on how quick the calcium levels go down for example somebody with a calcium of 2.3 and suddenly becomes 1.3 they will have major symptoms but if it's like over 10 years going down from 2.2 to 2.1 they'll probably not present with symptoms)

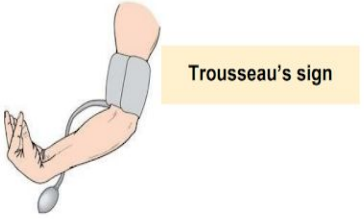
When nerves are exposed to low levels of calcium they show abnormal neuronal function which may include decrease threshold of excitation, repetitive response to a single stimulus and rarely continuous activity.

- Paresthesia, circumoral numbness.
- Tetany
- Hyperventilation
- Adrenergic symptoms
- Convulsion (More common in young people and it can take the form of either generalized tetany followed by prolonged tonic spasms or the typical epileptiform seizures)

Signs of latent tetany:

- **Chvostek sign** (causes twitching of the ipsilateral facial muscle)
- **Trousseau sign** (induces tetanic spasm of the fingers & wrist)





- Extrapyramidal signs like resting tremor(due to **basal ganglia calcification** تظهر على المدى البعيد)

why? because of something in physiology called the calcium phosphorus product[Ca x Phosphorous بضرب المركب هذا ذائبية يحسبون] and here the calcium is low the phosphorus is high [Ca x Phosphorus =will be high]if calcium phosphorus very high this predisposes to ca deposition in soft tissue and blood vessels تصلب الشرايين !!كيف كذا يادكتور يصير كالسيفيكشن و عندنا نسبة الكالسيوم منخفضه؟

B. Other clinical manifestation:

(more with CHRONIC(long term) or congenital or juvenile cases)

1. Posterior lenticular cataract. على المدى البعيد

2. Cardiac manifestation:

- **Prolonged QT interval** in the ECG.very classic sign لازم تعرفوها not good can cause arrhythmia.
- Resistance to digitalis
- Hypotension
- Refractory heart failure with cardiomegaly can occur.

3. Dental Manifestation:
Abnormal enamel formation with delayed or absent dental eruption and defective dental root formation.

4. Malabsorption syndrome:
Presumably secondary to decreased calcium level and may lead to steatorrhoea with long standing untreated disease.

5. Papilloedema.

Diagnosis of hypoparathyroidism:

1. Low serum calcium ↓ Ca^{2+} (after correction for any albumin abnormalities)
2. High serum phosphate ↑ PO_4
3. Serum PTH inappropriately low. (undetectable serum iPTH confirms the dx)
4. Low urine cAMP. (The parathyroid hormone works on Gs G protein which then leads to decreased levels of cAMP (as second messenger).

Treatment:

Mainstay of treatment:

- Combination of:
  1- Oral calcium.
  2- Pharmacological doses of **active** form of vitamin D its potent analogues(calcitriol or alfacalcidol).(why active? bc we don't have PTH to activate VitD2 in the kidney!!)
  3- Phosphate restriction in diet may also be useful with or without aluminum hydroxide gel to lower serum phosphate level.

Emergent treatment:

- only give in case of symptomatic and present of neural irritability(like in hungry bone syndrome* or in tetany): **IV calcium gluconate** till adequate serum calcium level is obtained and then vitamin D supplementation with oral calcium should be initiated.

*(what is Hungry bone syndrome??in state of severe hyperparathyroidism ,after total parathyroidectomy :the bone is hungry for ca "it was starving for years" & the stimulus of ca resorption from bone (PTH) is gone→thus the bone will cause sudden & sharp uptake of ca resulting in severe hypocalcemia, severe hypocalcemia عندنا وينتج الكالسيوم بيمص العظم ؟ايش حيصير طيب اذا شلنا الادينوما . they get suppressed؟اذا وحده منها فيها ادينوما other parathyroid glands بالمناسبه ايش يصير بال نسمي هالحاله hungry bone syndrome)

Hyperparathyroidism

Primary hyperparathyroidism:

General characteristics:

- Primary hyperparathyroidism is due to excessive production of PTH by one or more hyperfunctioning parathyroid glands.
- This leads to hypercalcemia which fails to inhibit the gland activity in the normal manner.
- Most common cause of hypercalcemia in the outpatient setting.while in the inpatient: Malignancy particularly breast cancer with metastases
- The incidence of the disease increase dramatically after the age of 50 and it's 2-4 folds more common in women (almost like all endocrine diseases most common in women).

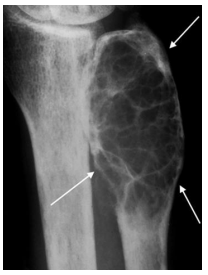
Causes of primary hyperparathyroidism:

1. **Single adenoma** (> 80%) of patients with primary hyperparathyroidism (one abnormal parathyroid takes over and disrupt it all)
2. Four glands hyperplasia account for 15%-20% of cases.
3. Parathyroid carcinoma could be the etiology in a rare incidence of less than 1%.

Clinical features:

- The two major sites of the potential complications are the bones and the kidneys.
  - Nowadays these complications are seen less commonly and around 20% of patients or less show such complications because we are picking up hyperparathyroidism **earlier** than before, even if the patient was asymptomatic)

Most common presentation: is asymptomatic hypercalcemia
(classic features of severe hyper PTH and hypercalcemia: Bones, Stones*renal*, abdominal moans and cyclic groans)

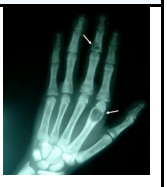
Kidney	Bones
1. Nephrolithiasis. (due to Ca deposition while the kidney tries to get rid of it) 2. Nephrocalcinosis (diffuse deposition of **Ca^{2+} PO_4 complex** in the parenchyma)	1. Bone aches and pains. 2. osteoporosis and fractures. 3. In skeleton a condition called **osteitis fibrosa cystica** (**classic** feature of hyperparathyroidism but rarely seen) could occur with subperiosteal resorption of the distal phalanges, distal tapering of the clavicles, a "**salt and pepper**" appearance of the skull as well as bone cysts and **brown tumors** of the long bones. زياده الباراثايروويد تؤدي الى زيادة امتصاص الكالسيوم من العظام فعشان كذا بتقل كثافة العظام ▸**NOTE:**Such overt bone disease even though typical of primary hyperparathyroidism is very rarely encountered. 

Psychiatric overtones	Groans	Others
1. Depression. 2. Easy fatigability. 3. Anorexia. 4. Sleep disturbances. 5. Anxiety, lethargy.. dear self, go to check ur parathyroid :"	1. Muscle pain and weakness. 2. Pancreatitis.لاحظوا زيادة الكالسيوم تسبب بنكريتايتس لكن البنكيريتايتس لما تحدث تؤدي الى هايبوكالسيميا 3. Peptic ulcer disease; bc Ca increase gastrin production. 4. Gout or pseudogout. 5. Constipation	1. polydipsia. 2. polyuria.(Hypercalcemia cause a state of ADH resistance→polyuria→severe state of volume depletion) 3. Cardio:**shortened QT interval**(MCQ), HTN(→ventricular hypertrophy) (HTN is bc of the effect of PTH on the endothelium in the vessels) 4. Weight loss. 5. Anemia It is suggested that parathyroid hormone (PTH), when in excessive amounts, interferes with normal erythropoiesis by downregulating the erythropoietin receptors on erythroid progenitor cells in the bone marrow → *normocytic, normochromic anaemia*.

Diagnosis:

✓ In all endocrine pathology to be diagnosed, u have to have high index of suspicion.

1. Nowadays almost 90% of diagnosed cases in the developed countries are picked up by routine screening for calcium level using the new automated machines.(First step when you have hypercalcemia ,measure the parathyroid hormone)

Lab tests	Radiology
In Primary hyperparathyroidism: - Calcium is high (hypercalcemia,hypercalciuria) - Phosphorus is low - PTH is high	Plain X-ray of hands can be diagnostic showing subperiosteal bone resorption usually on the radial surface of the distal phalanx with distal phalangeal tufting as well as cysts formation and generalized **osteopenia**.these're very very rare  **Preoperative Parathyroid scanning in an attempt to localise an adenoma and allow a targeted resection:** (better details in radiology lecture<3) **1.** Us **2.** MRI **3.** CT **4.** Thallium^{201} - technetium^{99} scan (subtraction study) → (Gold standard yet sometimes it might be negative if its a very very small adenoma or sometimes it shows you an adenoma on one side while an ultrasound shows one on the other side and becomes confusing but on the other hand it's great for picking up a fifth ectopic parathyroid in the mediastinum or somewhere else in the neck because we can't pick up those with MRI or CT) (NEWER modality is the dual phase MIBI scan) **6.** Sestamibi scan.

Treatment:

1. A large proportion of patients have “biochemical” hyperparathyroidism but with prolonged follow up they progress to overt clinical presentation.
2. Resection of the parathyroid lesion is **curative** with recurrences observed mainly in the multiple glandular disease.
3. **Medical treatment of the hypercalcemia:** (if a patient comes with very high calcium 3 or 3.5 for example you have to admit them and control the calcium cuz it might be **fatal** especially to the cardiac function, they can have arrhythmia, severe dehydration, hypovolemia so we have to treat them initially with hydration)

Acute severe form	Mainstay therapy is adequate **HYDRATION** with saline and **forced diuresis** by diuretics to increase the urinary excretion of calcium rapidly along with sodium and prevent its reabsorption by the renal (unfortunately this is only written in books but does not work in real life especially with very high calcium but it's a good way to start treatment)
Calcitonin	Inhibits osteoclast activity and prevent bone resorption.
Bisphosphonates	Given intravenously or orally to prevent bone resorption
Phosphate	Oral phosphate can be used as an **anti-hypercalcemic** agent and is commonly used as a temporary measure during diagnostic workup.
Estrogen	Decreases bone resorption and can be given to postmenopausal women with primary hyperparathyroidism using medical therapy
Cinacalcet	acts as a calcimimetic, the calcium-sensing receptors on the surface of the chief cell of the parathyroid gland is the principal regulator of parathyroid hormone secretion (PTH). Cinacalcet directly lowers parathyroid hormone levels by increasing the sensitivity of the calcium sensing receptors to activation by extracellular calcium, resulting in the inhibition of PTH secretion. as a treatment for patients with primary hyperparathyroidism who are unwilling to have surgery or are medically unfit

4. **Surgery:**
    a. Surgical treatment should be considered(considere doesn't mean have to) in all cases with established diagnosis of **primary hyperparathyroidism**.
    b. INDICATION FOR SURGERY(have to do surgery): ***symptomatic*** patient ‘lithiasis , osteoporosis, pancreatitis’ ,or ***aged less than 50*** ,or ***Asymptomatic*** but with significant hypercalcaemia.
    c. During surgery the surgeon identifies all four parathyroid glands (using biopsy if necessary) followed by the removal of enlarged parathyroid or 3 ½ glands in multiple glandular disease. (when you have hyperplasia which is very rare. They cannot remove all 4 so we don’t get HYPOparathyroidism and the half that is left usually is inserted under the skin in the forearm because if it turns to a hyperfunctioning gland or hyperplastic it will be easy to access because the neck is a very sensitive area)

DDx of hypercalcemia: د.رياض:هذا الجدول مهم جدا جدا وما استبعد اني اكتب سؤال سؤالين عليه

Parathyroid related:	1. Primary hyperparathyroidism: A. Solitary adenomas. B. Multiple endocrine neoplasia 2. Lithium therapy. parathyroid adenomas have been detected in patients on lithium treatment 3. Familial hypocalciuria hypercalcemia.it happens due to mutation in ca sensing receptors on the parathyroid glandsفينقرز الباراثيرويد هورمن بغض النظر عن مستويات الكالسيوم بالدم	**Related with high bone turnover:**	1. Hyperthyroidism is often associated with hypercalcemia which is provoked by osteoclastic activity of the thyroid hormoneS. 2. Immobilization 3. Vitamin A intoxication.stimulates bone resorption
Malignancy related:	1. **Solid tumor**(lung, kidney,commonest cause is breast cancer).How :O? !results from: secretion of parathyroid hormone-related peptide (PTHrP)by cancer cells which increases osteoclast proliferation/activity 2. **Hematologic malignancies:** ○ lymphoma(Hodgkin or non-Hodgkin), leukemia ○ the most classical is **multiple myeloma**’plasma cells tumour’: produce osteoclast activating factor,(the doctor mentioned a case presented with pneumonia and when they checked the CBC they found he had severe anemia with increased plasma cells and had osteolytic lesions in the bone → this is a manifestation of multiple myeloma) [▶NOTE:PTH IS NORMAL in malignancy induced hypercalcemia]	**Vit.D related:**	1. Vitamin D intoxication 2.Produce1,25$(OH)_2$:sarcoidosis &other granulomatous diseases مايخلى اختبار من دون مايسألوها 3.Idiopathic hypercalcemia of infancy
Others	Severe 2ry hyperparathyroidism with renal failure | Aluminum intoxication | Milk alkali syndrome | **Adrenal insufficiency** during the adrenal crisis, but the underlying pathophysiology is unclear | **Thiazides diuretics** enhances ca reabsorption in proximal tubule ,very important & commonly asked in the exam thiazide causes hypercalc :)نوته من الترم الأول في درس الالكترولايت امبالانس| .		

Secondary hyperparathyroidism:

or normocalcemic hyperparathyroidism,i don't want u to know about it ,bc it's pretty confusing

- An increase in PTH secretion which is adaptive and <u>unrelated to intrinsic disease</u> of the parathyroid glands is called secondary hyperparathyroidism. (commonest cause of high PTH with normal Ca is a secondary hyperparathyroidism and the commonest cause of secondary hyperpara specially in our community is vitamin D deficiency. So classically in Vit.D deficiency the Ca should be very low but due to the compensatory mechanism of PTH it can be in the **<u>low normal</u>**)
- This is due to chronic stimulation of the parathyroid glands (which are all perfectly normal) by a chronic decrease in the ionic calcium level in the blood.

Metabolic Bone Diseases

for further information about bone anatomy [link]

Osteoporosis هشاشة العظام | **Osteomalacia** لين العظام

[Note:Dr.Mona didn't explain this part and she said metabolic bone diseases is extra not including in the exam,while Dr.Riyadh explained this part during the lecture and after i contacted Dr.Riyadh,he said he haven't discussed the exam qs with dr.mona ,but metabolic bone diseases is imp regardless to the exam. so pleasssssssse read it just in case]

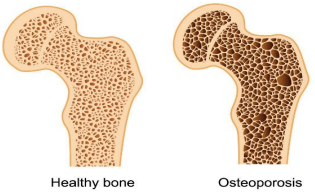
Osteoporosis

Definition:

Low bone mass[↓ bone density]with micro architectural disruption resulting in fracture from minimal trauma.

Causes:

- Menopause(We do not give estrogen unless very severe hot flushes,and it shouldn't given for more than 5 years)
- Old age [2 commonest causes is old age and menopause]
- Calcium and vitamin D deficiency
- Estrogen deficiency in women and androgen deficiency in men
- Use of steroids:!!!من الأدوية الخطيرة
  - Steroids for several days causes bone loss more on axial bones (40 %) than on peripheral bones (20%).
  - Muscle weakness
  - Prednisolone more than 5 mg /day for long time.
- Exclude <u>secondary causes</u> especially in younger individuals and men,(Common secondary causes of bone loss):
  - hyperparathyroidism
  - vitamin D deficiency
  - malabsorption state(e.g.:celiac disease,IBD,short gut syndrome)
  - Hypercalciuria
  - Hyperthyroidism
  - Chronic lung disease(COPD)
  - Malignancy
  - Rheumatoid arthritis
  - Hepatic insufficiency



Diagnosis:

Dual-energy x-ray absorptiometry (DXA) is **the most accurate test** in measuring bone mineral density (BMD):

- it measures the bone density of the lumbar spine and proximal femur and compare it to BMD of a healthy woman.(if the lumbar is normal and the femur is osteoporotic and vice versa I diagnose the patient as osteoporotic. We do not have to find the osteoporosis in both of them)here in saudi السبب مانعرفه the lumbar spine is most affected while the femur is not.

WHO Osteoporosis criteria 1994:

Definition based on BMD:

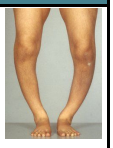
- Normal: BMD within 1 SD(T score above -1).(SD? The standard deviation (SD) is the difference between your BMD and that of the healthy young adults)
- Osteopenia: BMD which lies between <u>1 and 2.5</u> SD "<u>below young normal adult</u>"(T score between -1 & -2.5).
- Osteoporosis: more than or equal to <u>2.5</u> SD "<u>below young normal adult</u>"(T score below -2.5).
- Severe osteoporosis: osteoporosis + with 1 or more fragility fractures. من زحلقه بسيطه يتكسر

Management:	**Prevention:** - Public awareness - Adequate calcium and vitamin D supplements - Physical activity **Pharmacological Intervention:** - FIRST LINE → Bisphosphonates:reducing bone breakdown - Denosumab: reduces bone break down(MOA: RANK ligand (RANKL) inhibitor) - In severe cases or unresponsive to other therapy → IV Teriparatide: anabolic(Recombinant human parathyroid peptide stimulate bone formation)

Osteomalacia

Definition:

Inadequate mineralization of the osteoid frameworkالخرسانه, leading to **SOFT** bones,produces:



- ***rickets*** during bone growth in ***children***
- and ***osteomalacia*** following epiphyseal closure in ***adults***.

Causes:

1. Vitamin D deficiency. (commonest cause)أهم سبب بالسعوديه عدم التعرض لأشعة الشمس
2. Ca deficiency
3. Phosphate deficiency
4. Liver disease
5. Renal disease (renal tubular acidosis type 1 "distal")
6. Malabsorption (celiac disease)leads to Vit D malabsorption (diagnose it with anti-TTG and treat it with a gluten free diet)
7. Hereditary forms
8. (intestinal and gastric surgery): Bariatric surgery.لوحظ في السنوات الاخيرة وجود مرض لين العظام في المريضين و المريضات اللواتي اجرين عمليات تكميم معدة، تصغير معدة او تحويل امعاء. عندما تبدأ المريضة بخسارة وزنها يقل الامتصاص لجميع المعادن والفيتامينات بما فيها فيتامين د
9. Drugs: antiepileptic drugs.

Clinical presentations:

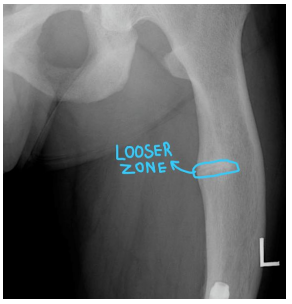
1. two third of patients are asymptomatic.
2. Incidental radiological findings.
3. Unexplained high Alkaline phosphatase.
4. deafness(ear dysfunction was probably due to the low **calcium** level in inner ear fluid and/or the direct effect of vitamin D deficiency on the inner ear)

Severe vitamin D deficiency may present with hypocalcaemia:

5. Bony deformity:Large skull, frontal bossing, bowing of legs.
6. Fracture tendency: vertebral crush fractures, tibia or femur. healing is rapid.
7. bony tenderness ,pains and erythema.
8. Proximal muscle weakness and pain are the common symptoms.

Diagnosis:

1. **Lab:**
   a. Low serum vitamin D
   b. Phosphate and calcium: may be normal or low.
   c. High serum ALP
   d. High PTH
2. **Radiology:**
   a. X-ray:Subperiosteal resorption, looser's zones[see the fig →] (pathognomonic)They develop PSEUDOfractures (CALLED LOOSER ZONE very classic)commonly bilateral لينه عظامهم لأنه فراكتشر ماحيحصلهم بس :O



   b. Bone scan.

Management:

1. Calcium and vitamin D supplements
2. Sun exposure(alone is not enough)
3. Results of treatment is usually very good.لو تقولي يادكتور ايش احسن مرض لما تعالجه المريض سيشعر بالامتنان اليك سأقول لين العظام لدرجة المريض يقولك انت احسن طبيب بالعالم ;D
4. Correcting underlying cause.

خلاص كفاية عليكم الجرعة هذي من المعلومات ;P

MCQs

1) Which one of the following can cause hypercalcemia?

a. Cushing disease
b. Hypothyroidism
c. Loop diuretics
d. Thiazide

2) Which one is favorable Site of calcification in hypoparathyroidism?

a. Cerebellum
b. Basal ganglia
c. Optic chiasm
d. Brainstem

3) Which one of the following ECG changes is likely to be found in case of hypocalcemia?

a. Peaked T wave
b. U wave
c. Depressed PR interval
d. Prolong QT interval

4) A 45 years old patient with a history of recurrent kidney stones. Investigations:
Calcium level: High
Parathyroid hormone level: High
Which one of the following is the next step for management?

A. Observation
B. Hydration
C. Parathyroidectomy
D. Thiazide diuretics

5) A 23-year-old man presented to the ER with right hip fracture, which happened after jogging. Lab results: PTH (high), Ca (high), vitamin D (normal), phosphorus (low), ALP (...). X-Ray of both hips and legs revealed large cystic areas, one of which involving the right femur neck.Which of the following is most likely the diagnosis?

A. Bone metastasis from an unknown primary
B. Primary hyperparathyroidism with brown tumors
C. Severe vitamin D deficiency with secondary hyperparathyroidism
D. Unknown diagnosis of chronic renal failure with renal osteodystrophy

6) A 21-year-old woman complains of urinary frequency, nocturia, constipation and polydipsia. Her symptoms started 2 weeks ago and prior to this she would urinate twice a day and never at night. She has also noticed general malaise and some pain in her left flank. A urine dipstick is normal. The most appropriate investigation is:

A. Serum phosphate
B. Serum calcium
C. Parathyroid hormone
D. Plasma glucose

Answer key:

1 (D) | 2 (B) | 3 (D) | 4 (C) | 5 (B) | 6 (B)