Common Parathyroid Disorders in Children

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Agenda

- Calcium homeostasis
- Causes of hypocalcaemia
- Rickets
- hypercalcaemia

Key-players of calcium metabolism

- Calcium & Phosphates
- Parathyroid hormone (PTH).
- Cholecalciferol (Vit.D3) and Calcitriol
- Estrogen and other Sex hormones.
- Calcitonin.

PARATHYROID HORMONE

Function of PTH

1-raises the level of calcium in the blood
2-decreases levels of blood phosphate.
3-Partially antagonistic to calcitonin

PARATHYROID HORMONE

- Secreation stimulated by fall in serum Ca.
- mobilize calcium from bone
- Increases renal reabsorption of ca
- decreases renal clearance of calcium
- increase calcium absorption - intestine

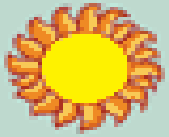
Calcium homeostasis

Vitamin D

- **Fat soluble ‘vitamin’**
- **Synthesised in skin**
- **Food sources include fish oils**

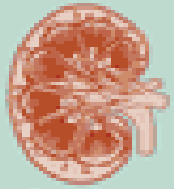
Vitamin D Metabolism

7-dehydrocholesterol



cholecalciferol (vitamin D_3)

calcidiol (25[OH] D_3)



calcitriol
(1α,25[OH]$_2D_3$ and 24R,25[OH]$_2D_3$)

Effects of Calcitriol

Intestines
- Increased calcium absorption
- Increased phosphorus absorption
- Decreased magnesium absorption

Parathyroid gland
- Increased mineralization indirectly via increased calcium absorption in intestinal lumen
- At high doses, increased osteoclastic bone

Kidneys
- Autoregulation of calcitriol production

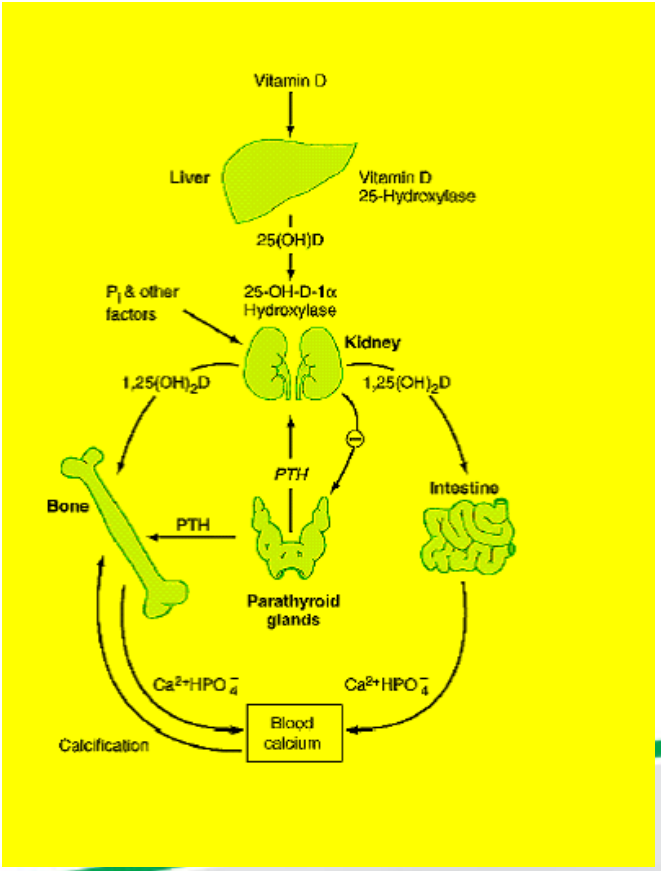
Years of research have led to an in-depth understanding of the metabolism of vitamin D. But the moniker of "vitamin" is not correct in the classic sense. In reality, vitamin D is a prohormone that has many effects.

Vitamin D

The active hormone is $1,25(OH)_2D_3$

It increases absorption of calcium from gut.

It increases reabsorption of ca from kidney.



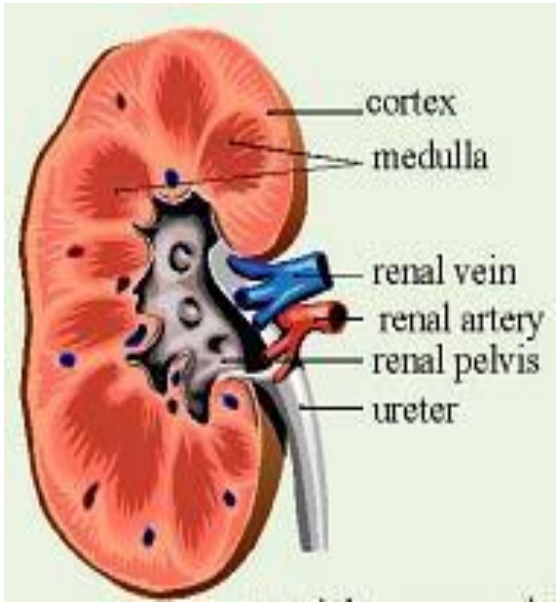
Calcitonin

- It is a calcium lowering hormone
- Secreted by Thyroid C cells

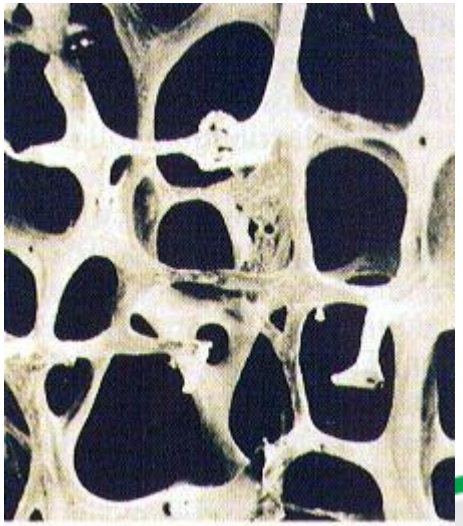
Anti - PTH

Target Organs

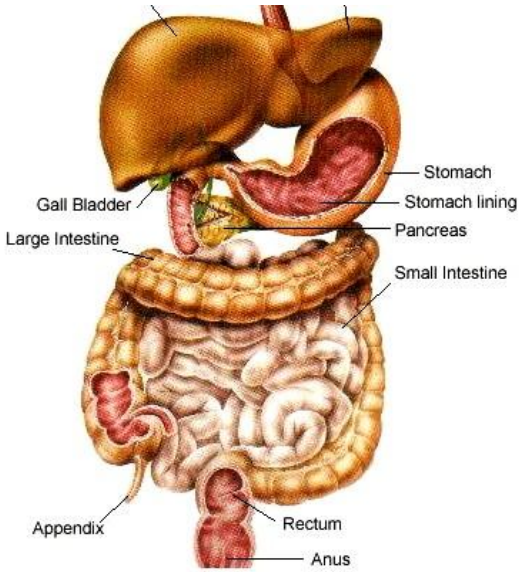
Kidney



Bone



G.I.Tract



Dysfunction of parathyroid Gland

1. Too little parathyroid hormone – hypoparahypothyroidism causes low serum calcium and high phosphate

2. Too much parathyroid hormone– hyperparahyperthyroidism causes high calcium and low phosphate

Calcium profile

- To diagnose a metabolic bone disease
  - calcium
  - Phosphate
  - Alkaline phosphatase
  - Parathyroid hormone
  - Vitamin D
  - Urinary calcium and phospherus

Causes of hypocalcaemia

- Rickets
- Hypopararthyroidism
- Psuedohypopararthyroidism
- Familial hypocalcaemia
- Renal failure
- Drugs: phenytoin
- Maternal diabetes
- Premarurity
- DiGoerge syndrome

Rickets

- Reduced **mineralization** of bone matrix due to calcium deficiency.

rickets results when the osteoid does not have mineral.

Calcium deficiency/Vit D deficiency

Deficiency of Vit. D

- Dietary lack of the vitamin
- Insufficient ultraviolet **skin exposure**
- Malabsorption of fats and fat-soluble vitamins- A, D, E, & K.
- Abnormal metabolism of vitamin D chronic renal failure.

Rickets: Non renal causes –

- Nutritional
- Intestinal – malabsorption
- Hepatobiliary
- Metabolic – anticonvulsant therapy
- Rickets of prematurity

Renal causes

- Renal osteodystrophy:CRF
  Familial hypophosphataemic rickets
  Renal tubular acidosis
  Fanconi syndrome
    Primary
    Secondary - cystinosis, wilsons disease,lowe syndrome,tyrosinemia
  Vitamin D dependent type 1 rickets
  Vitamin D dependent type 2 rickets

Rickets:Effect at growth end plate

- Inadequate growth plate mineralization.
- Defective calcification in the interstitial regions
- The growth plate increases in thickness.
- The columns of cartilage cells are disorganized.

Rickets

- Cupping of the epiphyses.
- Bones incapable of withstanding mechanical stresses and lead to bowing deformities.
- Eventual length of the long bones is diminished. (short stature)

Age of presentation

- **VITAMIN D DEFICIENCY RICKETS –**

  6 to 18 months.

- **NON NUTRITIONAL RICKETS**

  Beyond this age group.

Skeletal manifestations of Rickets

- Craniotaes
- Delayed closure of anterior fontanelle
- Frontal and parietal bossing
- Delayed eruption of primary teeth
- Rosary

Skeletal manifestations

EXTREMITIES –

Enlargement of long bones around wrists and ankles

Bow legs, knock knees

green stick fractures

Extra – skeletal manifestations

SEIZURES AND TETANY –

Secondary to hypocalcaemia

HYPOTONIA AND DELAYED MOTOR DEVELOPMENT

In rickets developing during infancy.

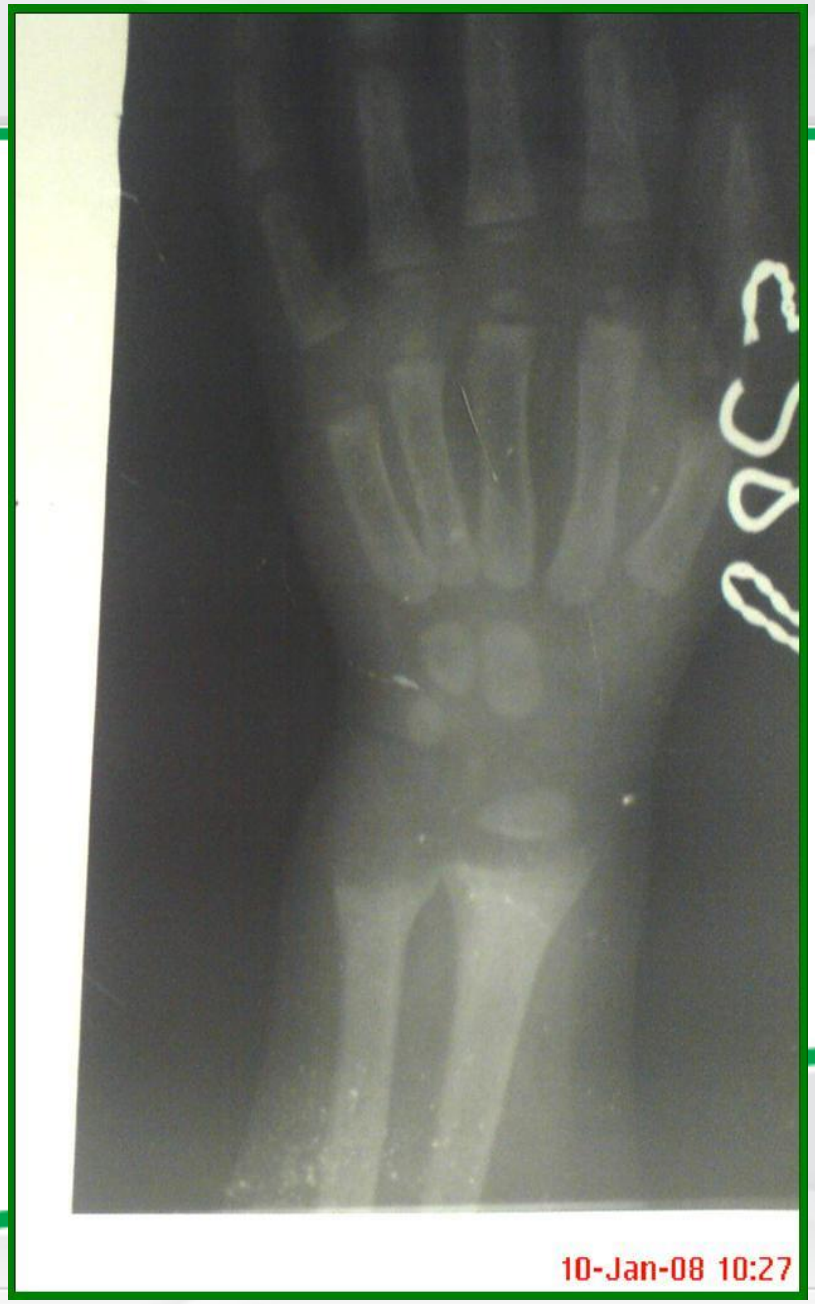
Investigations,

- **BASIC INVESTIGATIONS TO CONFIRM RICKETS**

- Low or normal serum Ca
- Low phospherus
- High alkaline phosphatase
- X rays of ends of long bones at knees or wrists
  - Shows Widening, fraying, cupping of the distal ends of shaft.
- Vit D level low
- Parathyroid hormone high

Rickets

- Radiology changes



Vitamin D Resistant Rickets

- In the renal tubular disorders, rickets develops in the presence of normal intestinal function and are not cured by normal doses of vitamin D.
- Resistant or refractory rickets.

Defective final conversion of Vit. D in to active form or End organ insensitivity.

Treatment of Rickets

- Vitamin D supplement
- Type and dose depens on underline cause of Rickets

Causes of hypercalcaemia

- Hyperparathyroidism
- Vitamin D intoxicity
- William syndrome
- Familial hypocalcuric hypercalcaemia
- malignancy