Parathyroid Disturbance and related Minerals

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Parathyroid Disturbance and related Minerals

General information

- The parathyroid gland is very close to thyroid gland.
- The parathyroid glands are four pea-sized glands located behind the thyroid gland in the neck.
- Parathyroid are NOT related to the thyroid (except they are neighbors in the neck).
- the parathyroid glands control body calcium.

CALCIUM

- Normal concentration in extracellular fluid (ECF) is 2.4 mmol/L. Distribution of Ca in the body: 1% in ECF, 2% in ICF and 97% in the bone.
- In plasma: Ca present in three forms:
- 41% : combined with plasma proteins (globulin and albumin), non-diffusible through capillary membrane.
- 9%: Ca combined with anionic substances (citrate and phosphate), diffusible.
- 50% : Ionized Ca+2, diffusible.

Calcium is essential to life, and is used primarily for three things:

- To provide the electrical energy for our nervous system i.e. transmission of signals, nerve impulses and excitation of neurons.
- To provide the electrical energy for our muscular system i.e. for contraction of skeletal, cardiac and smooth muscles.
- To provide strength to our skeletal system i.e. bone formation.
- Blood clotting mechanism.
- Many enzymatic pathway.

Important values on Ca

- Calcium intake20 [12–35] mmol •
- Calcium excretion in feces18mmol/day
 - in urine2mmol/day /day* •

PHOSPHATE

- 85% in bones, 14% in ICF and 1% in ECF. Serum level 3-5 mg/dl in adults and 4-5 mg/dl in children.
- Increase or decrease phosphate level 2-3 times the normal does not cause major immediate effect on the body.

HYPERPHOSPHATEMIA

In adults, hyperphosphatemia is defined • as a level 5.5 mg/dL. The most common causes are acute and chronic renal failure •

TREATMENT dietary phosphorus intake should be limited.

HYPOPHOSPHATEMIA (1.5 mg/dL)

Mild hypophosphatemia is not usually associated with clinical symptoms. In

severe hypophosphatemia, pts may have • muscle weakness,

Respiratory insufficiency can result from diaphragm muscle weakness.

TREATMENT

hypophosphatemia can be • replaced orally with milk

Hypocalcemia should be corrected first

Calcitonin

 As the level of calcium in the blood rises, the amount of calcitonin secreted by the C cells of the thyroid increases. Calcitonin stimulates osteoblasts to form bone taking calcium out of the circulation. At the same time, calcitonin inhibits the mobilization of bone (and calcium) by osteoclasts. The end result is a decrease in the level of calcium in the blood thus helping to maintain proper blood calcium levels.



Vit D:

- Increase absorption of Ca+2 and phosphate from the gut.
- Decrease renal Ca+2 and phosphate excretion.
- On bone: increase vit D lead to absorption of bones because Ca+2 mobilized to the blood.

- Vit D important for parathyroid hormone function. Absence of vit D result in poor function of parathyroid hormone
- Persons with a vitamin D deficiency suffer from rickets, characterized by soft, poorly calcified bone, along with poor absorption of calcium. Calcitriol or any of its precursors promotes a dramatic increase in the absorption of calcium by the intestine and a prompt repair. of the diseased bone.

Hyperparathyroidism

 Primary hyperparathyroidism is the result of parathyroid gland disease, most commonly due to a parathyroid tumor (adenoma) which secretes the hormone without proper regulation.
Common manifestations of this disorder are chronic elevations of blood calcium concentration (hypercalcemia), decalcification of bone.

HYPERCALCEMIA

occur at levels of serum calcium _2.9 mmol/L

Causes of Hypercalcemia

- I. Parathyroid-related •
- II. Malignancy-relatedA. Tumor B.Hematologic malignancies

- III. Vitamin D–related •
- IV. Associated with high bone turnover
 - V. Associated with renal failure •

TREATMENT

- The type of treatment is based on the severity of the hypercalcemia and the
 - nature of the associated symptoms.
 - Mild hypercalcemia [_3 mmol/L •
- (can usually be managed by hydration may not require surgery
 - Severe hypercalcemia with more than 3.7 mmol/L •
- In pts with severe primary hyperparathyroidism, surgical parathyroidectomy
 - should be performed

Secondary hyperparathyroidism

 is the situation where disease outside of the parathyroid gland leads to excessive secretion of parathyroid hormone.

A common cause of this disorder

 is kidney disease - if the kidneys are unable to reabsorb calcium, blood calcium levels will fall, stimulating continual secretion of parathyroid hormone to maintain normal calcium levels in blood. Secondary hyperparathyroidism can also result from inadequate nutrition - for example, diets that are deficient in calcium or vitamin D, or which contain excessive phosphorus .
A prominent effect of secondary hyperparathyroidism is decalcification of bone, leading to pathologic fractures or "rubber bones". Excess PTH can cause a number of problems. For example, the bones may lose calcium, and get thinner or more porous. This condition is called osteopenia and osteoporosis. When bones are exposed to high levels of parathyroid hormone for several years they become brittle and much more prone to fractures.

Hypoparathyroidism

- Inadequate production of parathyroid hormone- hypoparathyroidism - typically results in decreased concentrations of calcium and increased concentrations of phosphorus in blood.
- Treatment focuses on restoring normal blood calcium concentrations by calcium infusions, oral calcium supplements and vitamin D therapy.

HYPOCALCEMIA

Chronic hypocalcemia is less common than hypercalcemia but is usually symptomatic and requires treatment

symptoms

- burning of the hands
 - loss of memory
- spasms of the face, wrists and feet
 - muscle spasms,
 - laryngeal spasm,
 - Increased intracranial pressure
 - irritability,
 - depression,
 - chronic malabsorption
 - hypocalcemia may be lethal

Treatment

Symptomatic hypocalcemia may be treated with intravenous calcium gluconate

Management of chronic hypocalcemia requires administration of calcium according to serum calcium levels and urinary excretion. with a vitamin D therapy).