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REVIEW ARTICLE ON HYPERPARATHYROIDISM

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Abstract

Primary hyperparathyroidism has been associated with bone loss, especially at cortical skeletal sites. Results from studies evaluating the mineral density of cancellous bone have been more difficult to interpret. Most densitometry studies support the concept that the parathyroid hormone appears to be catabolic at cortical sites and may have anabolic effects at cancellous bone sites. Studies completed to date, however, have been limited by design, definitions of fracture and inadequate control groups. Primary hyperparathyroidism is now increasingly being detected during the asymptomatic phase. The need for parathyroidectomy has been questioned in such patients because there may be no disease progression in the absence of surgery. Medical management of primary hyperparathyroidism has to date been limited to estrogen replacement therapy in postmenopausal women. Identification of the calcium receptor has improved our understanding of calcium homeostasis, and significant reductions in calcium receptor levels have been detected in parathyroid adenomas. Thus, a new class of therapeutics may include the calcimimetic agents. Bisphosphonates are also currently being evaluated with regard to their impact on fracture prevention and their beneficial effects on bone mineral density. Recently a decline in the incidence of primary hyperparathyroidism has been reported. With the limitation on screening that is now occurring in the United States and Canada, the incidence of primary hyperparathyroidism could decline further. Currently the prevalence rates are about 1 to 4 per 1000, with a female: male ratio of 3:1. In Sweden about 3% of postmenopausal women are affected.

Keywords: parathyroid hormone(PTH),primary hyperparathyroidism (PHPT),GIT(gastrointestinal tract)

Introduction

It is a condition involving an increased secretion of parathyroid hormone(PTH). PTH helps regulate calcium and phosphate levels by stimulating bone reabsorption of calcium, renal tubular reabsorption of calcium, and activation of vitamin D. thus over secretion of PTH is associated with increased serum calcium levels. Hyperthyroidism affects approximately 1% of the general population and is more common in women than men. The most common clinical presentation of primary hyperparathyroidism (PHPT) is asymptomatic hypercalcemia detected by routine biochemical screening. However, the presentation may be atypical and include a spectrum of disturbances in calcium homeostasis, ranging from symptomatic severe hypercalcemia (parathyroid crisis) to normocalcemic PHPT. The clinical manifestations that are directly related to PHPT

will be reviewed here. Symptoms and signs (gastrointestinal, neuromuscular, renal, and psychological) that are likely related to hypercalcemia .

Definition

Hyperparathyroidism is a condition in which one or more of the parathyroid glands become overactive and secrete too much parathyroid hormone (PTH). This causes the levels of calcium in the blood to rise, a condition known as hypercalcemia.

Types

Primary hyperparathyroidism

It is due to an increased secretion of PTH leading to disorders of calcium, phosphate, and bone metabolism. The most common cause is a benign tumour in the parathyroid gland. Primary hyperparathyroidism usually occurs between 30-70 years of age. Peak incidence is in the 40s and 50s. patients who have previously undergone head and neck radiation may have an increased risk of developing a parathyroid adenoma.

Secondary hyperparathyroidism

It appears to be a compensatory response to conditions that induce or cause hypocalcemia, the main stimulus of PTH secretion . disease conditions associated with secondary hyperparathyroidism include vitamin D deficiencies, malabsorption, chronic renal failure, and hyperphosphatemia.

Tertiary hyperparathyroidism

it occurs when there is hyperplasia of the parathyroid glands and loss of negative feedback from circulating calcium levels.

Pathophysiology

- **Primary hyper parathyroidism**

Decreased ECF Ca^{2+} , it leads to Renal Ca^{2+} resorption, renal hydroxylation of 25-hydroxy vitamin D bone resorption and Decreased ECF Ca^{2+}

- **Secondary hyper parathyroidism**

Decreased GFR it leads to Vit.D deficiency/resistance, increase FGF-23 and hyperphosphatemia , it leads to Hypocalcemia, Increase PTH, Bone disease occur

Clinical manifestation

- **Cardiovascular:** dysrhythmias, hypertension
- **GIT:** abdominal pain, anorexia, nausea and vomiting, constipation, pancreatitis, peptic ulcer, cholelithiasis, weight loss.
- **Musculoskeletal system:** skeletal pain, backache, weakness, fatigue, osteoporosis, muscle atrophy .
- **Neurological:** emotional irritability, memory impairment, depression, confusion, coma, headache, paraesthesias
- **Renal:** hypercalciuria, kidney stones, urinary tract infection, polyuria.

Diagnostic studies

- Radioimmunoassay, Urine calcium serum chloride, uric acid, creatinine, bone density measured, dual-energy x-ray absorptiometry (DEXA), MRI, CT.

Collaborative care

The treatment objectives are to relieve symptoms and prevent complications caused by excess PTH . the choice of therapy depends on the urgency of the clinical situation, the degree of therapy depends on the urgency of the clinical situation the degree of hypercalcemia and the underlying causes of the disorder.

Surgical therapy:

- Chronically high calcium levels, hypercalciuria, its reduce bone density, it leads to partial/complete removal of parathyroid gland.

Nonsurgical therapy

- To assess the metabolic bone loss, measure the urinary calcium excretion. Continued ambulation and avoid immobility .
- Dietary measures also include maintenance of a high fluid intake and a moderate calcium intake.
- Tablet.bisphosphonates- inhibit osteoclastic bone reabsorption .
- Estrogen and progestron therapy-reduce the serum and calcium level, oral phosphate-inhibit the calcium absorption and administered the diuretics, calcium agents.

Complication

- Hemorrhage and fluid, electrolyte disturbances,tetany, unpleasant tingling of the hands and around the mouth.

Nursing management

- Administered the intravenous infusion-calcium gluconate.
- Monitored the intaken and out put chart.
- Promote the bone calcification.
- Encourage the patient to do the exercise daily.
- Promote rest and sleep.
- Reduce the anxiety and frustration
- Participate the patient in self care activities of daily living with minimal discomfort and fatigue
- To correct the hormonal deficiency
- Provide a warm environment, prevent skin breakdown,monitor the mental status,gradually increased dietary habits.

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