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Hyperpara Thyroidism and its Management

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ABSTRACT

Hyperparathyroidism is an increase in parathyroid hormone (PTH) levels in the blood. This occurs from a disorder either within the parathyroid glands (primary hyperparathyroidism) or as response to external stimuli (secondary hyperparathyroidism). Symptoms of hyperparathyroidism are caused by inappropriately normal or elevated blood calcium leaving the bones and flowing into the blood stream in response to increased production of parathyroid hormone. In healthy people, when blood calcium levels are high, parathyroid hormone levels should be low. With long-standing hyperparathyroidism, the most common symptom is kidney stones. Other symptoms may include bone pain, weakness, depression, confusion, and increased urination. Both primary and secondary may result in osteoporosis (weakening of the bones). In 80% of cases, primary hyperparathyroidism is due to a single benign tumor known as a parathyroid adenoma. Most of the remainder are due to several of these adenomas. Very rarely it may be due to parathyroid cancer. Secondary hyperparathyroidism typically occurs due to vitamin D deficiency, chronic kidney disease, or other causes of low blood calcium. The diagnosis of primary hyperparathyroidism is made by finding elevated calcium and PTH in the blood.

Keywords: Vitamin D; Osteoporosis; Parathoromone; Renal Rickets.

INTRODUCTION

It is caused by overproduction of parathoromone by the parathyroid glands and is characterized by bone decalcification and development of renal calculi containing calcium. Primary Hyperparathyroidism occurs two or 4 times more often in women than in men. Rare in children younger than 15 years of age, but it increases 10 fold between the ages of 15 and 65 years. It is caused by over production of parathoromone by the parathyroid glands and is characterized by bone decalcification and development of renal calculi containing calcium.

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TYPES OF HYPERPARATHYRIODISM

Primary Hyperparathyroidism occurs two or 4 times more often in women than in men. Rare in children younger than 15 years of age, but it increases 10 fold between the ages of 15 and 65 years. Half of the people diagnosed with hyperparathyroidism do not have symptoms. Secondary hyperparathyroidism, with manifestation similar to those of primary hyperparathyroidism occurs in patients who have

chronic renal failure and so called ***renal rickets*** as a result of phosphorous retention, increased stimulation of the parathyroid glands and increased parathyroid hormone secretion.

Clinical Manifestations

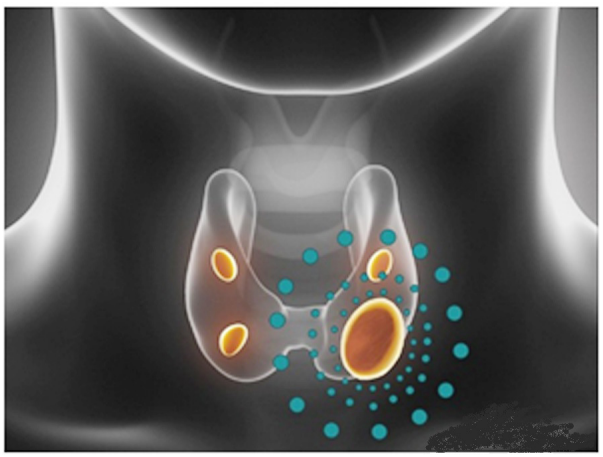

Fig. 1: Showing enlarged parathyroid gland (***Source:*** https://www.hyperparathyroidmd.com/hyperparathyroidism/

- Patient may have no symptoms or may experience signs and symptoms resulting from involvement of several body systems.
- Apathy, fatigue, muscle weakness, nausea, vomiting, constipation, hypertension and cardiac dysrhythmias may occur.
- All these signs are attributable to the increased concentration of calcium in the blood.
- Psychological effects may vary from irritability and neurosis to psychoses caused by the direct action of calcium on the brain and nervous system.
- An increase in calcium produces a decrease in the excitation potential of nerve and muscle tissue.
- The formation of stones in one or both kidneys related to the increased urinary excretion of calcium and phosphorous is one of the important complications of hyperparathyroidism and occurs in 55% of patients with primary hyper parathyroidism.
- Renal damage results from the precipitation of calcium phosphate in the renal pelvis and parenchyma which causes renal calculi, obstruction, pyelonephritis and renal failure.
- Musculoskeletal symptoms accompanying hyperparathyroidism may be caused by demineralization of the bones or by bone tumors composed of benign giant cells resulting from over growth of osteoclasts.
- The patient may develop skeletal pain and tenderness especially of the back and joints, pain on weight bearing, pathological fracture, deformities and shortening of body stature.
- Bone loss attributable to hyperparathyroidism increase the risk of fracture.
- The incidence of peptic ulcer and pancreatitis is increased with hyperparathyroidism and may be responsible for many of the GI symptoms that occur.
- Due to increased serum calcium levels which leads to increased serum levels of gastrin and acetylcholine. These changes result in stimulation of gastric acid secretion and peptic ulcer disease.

ASSESSMENT & DIAGNOSTIC FINDINGS

Primary hyperparathyroidism is diagnosed by persistent elevation of serum calcium levels and an elevated concentration of parathoromone. An elevated serum calcium level alone is a non-specific finding because serum levels may be altered by diet, medications and renal and bone scans in advanced disease. The double antibody parathyroid hormone test is used to distinguish between primary hyperparathyroidism and malignancy as a cause of hypercalcemia. Ultrasound, MRI, thallium scan and fine needle biopsy have been used to evaluate the function of the parathyroid and to localize parathyroid cysts, adenomas or hyperplasia.

Thallium Scan

- A radioactive substance, Thallium is injected at peak exercise (or when typical chest pain develops). Exercise is continued for one minute after injection to ensure maximum myocardial extraction.
- The patient is scanned within 10 minutes.
- The ECG is monitored for the 12 minutes of the procedure.

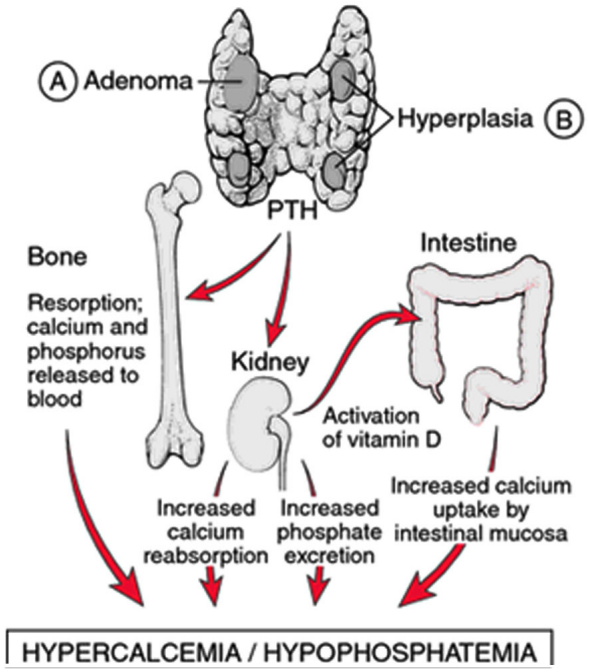

Fig. 2: Showing process of hypercalcemia and hypophosphatemia

SURGICAL MANAGEMENT

Recommended treatment is the surgical removal of abnormal parathyroid tissue. Previously, standard. Parathyroidectomy involved a bilateral neck exploration under general anesthesia. Nowadays, minimally invasive parathyroidectomy techniques allow for unilateral neck exploration using local anesthesia, these are performed on an outpatient basis. In some cases only the removal of a single diseased gland is necessary, reducing morbidity rates associated with surgery. For asymptomatic patients who have only mildly elevated serum calcium concentrations and normal renal function, surgery may be delayed and the patient monitored closely for worsening of hypercalcemia, bone deterioration, renal impairment or the development of kidney stones. Surgery is recommended for asymptomatic patients who meet the following criteria:

1. Younger than 50 years of age
2. Unable or unlikely to participate in follow up care
3. Serum calcium levels more than 1.0 mg/dl (0.25mmol/L) above normal reference range.
4. Urinary calcium levels greater than 400 mg/day
5. A 30% or greater decrease in renal function
6. With the complaints of primary hyper parathyroidism including nephrocalcinosis, osteoporosis or severe psychoneurologic disorder.

HYDRATION THERAPY

- Because kidney involvement is possible, patients with hyper parathyroidism are at risk for renal calculi.
- Therefore a daily fluid intake of 2000 mL or more is encouraged to help prevent calculus formation.
- Cranberry juice is suggested because it may lower the urinary pH.
- Cranberry extract tablets are an alternative to reduce urinary pH
- Patient is instructed to report other manifestations of renal calculi, such as abdominal pain and hematuria.
- Thiazide diuretics are avoided, because they decrease the renal excretion of calcium and further elevate serum calcium levels.
- Because of the risk of hypercalcemic crisis, the patient is instructed to avoid dehydration and to seek immediate health care if conditions that commonly produce dehydration (e.g. vomiting, diarrhea) occur.

Mobility

Mobility of the patient, with walking or use of a rocking chair for those with limited mobility, is encouraged as much as possible because bones that are subjected to normal stress give up less calcium. Bed rest increases calcium excretion and the risk for renal calculi. Oral phosphates lower the serum calcium level in some patients, long term use is not recommended because of the risk of ectopic calcium phosphate deposition in soft tissues.

Diet and Medications

Nutritional needs are met, but the patient is advised to avoid a diet with restricted or excess calcium. If the patient has a coexisting peptic ulcer, prescribed antacids are necessary. Because anorexia is common, efforts are made to improve the appetite. Stool softeners and physical activity, along with increased fluid intake, help offset constipation which is common post-operatively.

Complications: Hypercalcemic Crisis

Acute hypercalcemic crisis can occur with extreme elevation of serum calcium levels. Serum calcium levels greater than 15 mg/dl result in neurologic, cardiovascular and renal symptoms that can be life threatening. Treatment includes rehydration with large volumes of IV fluids, diuretics agents to promote renal excretion of excess calcium, and phosphate therapy to correct hypo phosphatemia and decrease serum calcium levels by promoting calcium deposition in bone and reducing gastrointestinal absorption of calcium. Cytotoxic agents (e.g. mithramycin), calcitonin and dialysis may be used in emergency situations to decrease serum calcium levels quickly. A combination of calcitonin and corticosteroids has been administered in emergencies to reduce the serum calcium level by increasing calcium deposition in bone. Other agents may be administered to decrease serum calcium levels include bisphosphonates.

CONCLUSION

Hyperparathyroidism is a condition in which one or more of the parathyroid glands is overactive and makes excess parathyroid hormone, regardless of the level of calcium in the body, which this hormone normally regulates. In other words, the parathyroid glands continue to make large amounts of parathyroid hormone even when the calcium level is normal and they should not be making hormone at all. Over production of parathyroid hormone by overactive parathyroid glands can rob you of your health. It can make you feel run down and tired and cause osteoporosis, kidney stones, and many other serious problems. Hyper parathyroidism can be fixed in most people with newer minimally invasive surgery techniques in less than 20 minutes.

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