Hyperparathyroidism

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Oral Pathology 2018

Overview

The parathyroid glands can be found in the neck, near the posterior surface of the thyroid gland. There are typically four parathyroid glands in humans. The chief cells, the main functional cells of the parathyroid glands, are primarily responsible for secreting parathyroid hormone (PTH) when there's low calcium level in the blood. According to Alhefdhi, parathyroid hormone are "inversely proportional to serum calcium levels, as high serum calcium levels decrease PTH release and low serum calcium levels increase PTH secretion (305). Hyperparathyroidism occurs when there is an overproduction of PTH from the parathyroid glands. This excess levels of PTH results in higher than normal levels of calcium in the blood; a condition called hypercalcemia (Alhefdhi 305).

Etiology

There are three types of hyperparathyroidism: primary hyperparathyroidism (pHPT), secondary hyperparathyroidism (sHPT), and tertiary hyperparathyroidism (tHPT). In primary hyperparathyroidism, an unfavorable secretion of PTH occurs because of one or more overly active parathyroid glands. Primary hyperparathyroidism (pHPT) is mostly caused by an adenoma or a benign tumor in one of the parathyroid glands, and accounts for approximately 75-80% of pHPT cases (Alhefdhi 305). Occasionally, it can be caused by multiple tumors or hyperplasia of the parathyroid glands, which accounts for 10-15% of pHPT cases (Alhefdhi 305). Less than 1% of pHPT cases are caused by cancer of the parathyroid gland (Alhefdhi 305). In some cases, "the use of thiazides may lead to mild hypercalcemia by reducing urinary calcium excretion, and the

use of lithium may lead to increase serum calcium and PTH by decreasing the sensitivity of the calcium sensing receptors to calcium" (Alhefdhi 306).

In secondary hyperparathyroidism (sHPT), the parathyroid glands reacts to an external ailment like vitamin D deficiency or renal failure by overproducing PTH. Secondary hyperparathyroidism occurs most frequently in cases related to chronic kidney disease because of the kidney's inability to produce sufficient levels of vitamin D (Komaba S37). The insufficient levels of vitamin D results in low calcium levels which influence the parathyroid glands to secrete calcium in the blood. When this continues, the parathyroid glands eventually becomes enlarged and overly active, causing high level of calcium in the body. Malnutrition and malabsorption can also make the parathyroid overactive and lead to sHPT (Komaba S41).

Tertiary hyperparathyroidism (tPHT) occurs due to prolong and/or untreated secondary hyperparathyroidism. Tertiary hyperparathyroidism (tHPT) is caused by an "as excessive persistent secretion of PTH […] in patient with secondary hyperparathyroidism" (Gawrychowski 423). In this case, parathyroid gland becomes overactive, resulting in a permanent elevated level of PTH in the body. This disorder is often occurs in patients kidney transplant or in dialysis patients (Gawrychowski 423).

Clinical Presentation

Approximately 80% of patients with primary hyperparathyroidism are asymptomatic; they do not show any signs or have any symptoms of hypercalcemia (Alhefdhi 306). However, some general signs and symptoms that may be present but not linked directly to pHPT are hypertension, accumulation of calcium salts in cardiac and coronary, kidney stones, enlargement of left ventricular, bone pain, weakness, lack of appetite, and sleep problems (Alhefdhi 306). Symptomatic pHPT include kidney stones, apparent bone loss, "gastrointestinal symptoms, such

as nausea, constipation, peptic ulcer, and pancreatitis, or neuropsychiatric symptoms, such as depression, dementia, confusion, lethargy, social function impairment, psychosis, and coma." (Alhefdhi 306).

Demographic

Primary hyperparathyroidism affects approximately 1 in 500 females and 1 in 2,000 males aged older than 40 years (Alhefdhi 305). In addition, there is a "significantly higher incidence of pHTP among black individuals (92 per 100,000 in woman and 46 per 100,000 in men) than in white individuals (81 per 100,000 in women and 29 per 100,000 in men)" (Bilezikian 2).

Biopsy / Histology / Radiographs

Primary hyperparathyroidism cannot be diagnosed by biopsy, histologic or radiologic testing. Primary hyperparathyroidism can only be identified by blood test. The blood test will reveal if there are high levels of PTH when high levels of calcium exists in the body. Once diagnosed with pHPT, however, ultrasound can be performed to determine specific location of malformed parathyroid gland(s), and CT scan to show presence of kidney (Bilezikian 26). Use of a dual-energy x-ray absorptiometry can be used to measure bone mineral density in hip, lumbar spine, and forearm to determine the level of bone loss when evaluating patient with hyperparathyroidism (Bilezikian 26).

Differential Diagnosis

According to Alhefdhi, "elevated levels of serum calcium and PTH are associated with 95% risk of a diagnosis of classic pHPT" (306). Since there are no signs and symptoms of pHPT, it is difficult to make a diagnosis of pHPT without performing blood test. According to Alhefdhi, "CT scans can identify parathyroid adenomas in 90% of the case. While false-positive findings

can be seen for thyroid nodule, tortuous vessel, or laterally displaced esophagus, false-negative results can be seen in cases of small or ectopic adenomas or distorted neck anatomy due to previous surgery " (307). Therefore, blood test is the best method to determine if there's elevated calcium in blood. Once confirmed, additional tests like bone densitometry, urine test and imaging test of kidneys can be performed to further evaluate patient's condition.

Treatment

Treatment of primary hyperparathyroidism varies based on severity of the disease. If there are no symptoms in pHPT patients, then treatment options includes carefully monitoring the level of calcium, bone density, and kidney function. Medications are also used to help decrease bone loss and to stabilize levels of calcium in the blood. Patients with symptomatic pHPT, which includes elevated calcium levels, kidney or bone disease, or parathyroid cancer, should consider parathyroidectomy; surgery to remove the parathyroid glad (Alhefdhi 308).

Prognosis

Parathyroidectomy is the only curative therapy for this disorder. It has a success rate of approximately 95-98% (Alhefdhi 308). In order to prevent development of "systemic complications" of pHPT, it is recommended for patient to undergo parathyroidectomy as quickly as possible after being diagnosed (Alhefdhi 308). After surgery, patients' calcium and parathyroid levels are monitored periodically. If calcium levels remain normal 6 months after the surgery, then the surgery is considered successful (Alhefdhi 308). Depending on severity of this disorder, if left untreated, the high levels of calcium in blood can cause vomiting, bone tenderness, irregular heartbeat, high blood pressure, confusion (Burgess). Severe cases of hyperparathyroidism can lead to loss of consciousness and coma if left untreated (Burgess).

Professional Relevance

Parathyroid hormone plays a critical role in calcium and phosphorus metabolism and as a result, has a significant influence on bone and teeth mineralization (Khalekar 581). According to Khalekar, “intraoral manifestations" in patients with hyperparathyroidism varies from “obliteration of pulp chamber by pulp stone, alterations in dental eruption, loosening and drifting of teeth, malocclusions, spacing of teeth, partial loss of lamina dura, periodontal ligament widening, teeth become sensitive to percussion and mastication, floating teeth delay or cessation of dental development, brown tumor, generalized bone ratification of jaw, soft tissue calcifications, caries, hypercalcemia may result in sialolithiasis mandibular tori, patient may complain of vague jaw bone pain.” (581). Therefore, dental hygienist should have a good understanding of this disorder and the effects it has on patients’ oral health. This will help the dental hygienist develop an accurate oral treatment plan for the patient.

Citations

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