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Primary Hyperparathyroidism and Hypoparathyroidism

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Primary Hyperparathyroidism and Hypoparathyroidism

INTRODUCTION

CALCIUM HOMEOSTASIS

Bone contains 99% of the human body's calcium. The remaining 1% is contained in the blood and extracellular fluid and within cells. The calcium in blood circulates in 3 forms: protein bound (50%), complexed to ions (10%), or as free ions (40%). It is the free calcium ions (ionized calcium) that are biologically relevant. Because of calcium's importance in enzymatic reactions, nerve and muscle function, and many cellular functions, the serum calcium concentration in humans is tightly regulated by parathyroid hormone (PTH) and vitamin D. Although calcitonin is an important calcium-regulating hormone in fish and some rodents, a role for it in regulating human calcium homeostasis is uncertain.

Blood calcium concentrations are acutely regulated by PTH. PTH secretion is regulated by the calcium-sensing receptor on parathyroid cells, which when activated by circulating calcium decreases PTH secretion. PTH acts to increase resorption of bone by stimulating osteoclast activity. PTH also causes increased synthesis of 1,25-dihydroxyvitamin D (the active vitamin D metabolite) by the kidney and increased phosphate excretion in the urine. Vitamin D regulates calcium on a more chronic basis. Vitamin D is a steroid synthesized in the skin in response to ultraviolet light. It is metabolized sequentially by the liver and then the kidney to result in the biologically active 1,25-dihydroxyvitamin D metabolite. The principal action of 1,25-dihydroxyvitamin D is to increase absorption of calcium from the gastrointestinal tract. Hypercalcemia and hypocalcemia frequently result from derangements in these normal calcium regulatory mechanisms.

PRIMARY HYPERPARATHYROIDISM

Primary hyperparathyroidism is an abnormality of the parathyroid glands that causes inappropriate PTH secretion, with resultant hypercalcemia; primary hyperparathyroidism is the most common cause of hypercalcemia in the outpatient population. The increase in identified cases of primary hyperparathyroidism during the 1970s is largely attributed to the use of automated

blood testing, which detected hypercalcemia in asymptomatic patients. Secondary hyperparathyroidism is an appropriate increase in PTH secretion in response to hypocalcemia.

Today, only a minority of cases of primary hyperparathyroidism are symptomatic, and the severe complications of nephrocalcinosis and osteitis fibrosis cystica are rarely encountered. Management of the symptomatic patient with primary hyperparathyroidism is relatively straightforward, as the symptoms dictate that surgery usually is necessary once the patient is medically stable. Management of the asymptomatic patient is more challenging. No prospective trials have compared outcomes of patients randomized to surgery or medical management. Likewise, no studies have directly compared options for medical management. Because evidence suggests that mild primary hyperparathyroidism can remain stable for years without causing complications, there is debate over the best approach to managing the asymptomatic patient with mild primary hyperparathyroidism.¹

HYPOPARATHYROIDISM

Hypoparathyroidism is an important endocrine cause of hypocalcemia. Hypocalcemia is a frequently encountered laboratory abnormality among inpatients and outpatients. The presentation can range from life-threatening tetany to an asymptomatic laboratory finding.

ASYMPTOMATIC PRIMARY HYPERPARATHYROIDISM

CASE PRESENTATION

Initial Presentation

A 60-year-old woman presents for evaluation of hypercalcemia. The abnormality was detected on routine laboratory testing during a preoperative evaluation for upcoming dental implant surgery.

History

The patient feels well. She denies any abdominal pain, constipation, bone pain, or neurologic or psychiatric symptoms. She has no history of kidney stones or