IDIOPATHIC HYPOPARATHYROIDISM

Effects of Parathyroid Hormone Deficiency

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This article describes the diagnosis, symptoms, manifestations, and treatment of autoimmune hypoparathyroidism.

Parathyroid Hormone

The four (or sometimes more) parathyroid glands, situated on the outer tips of the thyroid lobes, produce and secrete parathyroid hormone (PTH). PTH, along with the hormone vitamin D, regulates the body's calcium and phosphate levels and activates the conversion of 25-hydroxyvitamin D to 1, 25-dihydroxyvitamin D, the active form of vitamin D that stimulates calcium and phosphate absorption from the gastrointestinal tract. When the parathyroid glands fail to secrete sufficient amounts of thyroid hormone, a condition of hypoparathyroidism develops. Low calcium and high phosphorus levels and their associated symptoms are the most significant effects of hypoparathyroidism.

Types of Hypoparathyroidism

Hypoparathyroidism can occur as a primary disorder caused by a genetic mutation or it can be caused by autoimmune destruction of the parathyroid glands. Alternately, hypoparathyroidism can occur as an acquired disorder caused by surgical removal of the parathyroid or thyroid glands, by magnesium deficiency or excess or by infiltrative disease caused by malignancy, chemical and drug toxicity, Wilson disease, sarcoidosis, radiation injury, mechanical injury, or infection. This article discusses the condition of autoimmune hypoparathyroidism.

Symptoms

Idiopathic or autoimmune hypoparathyroidism is usually diagnosed within the first decade of life although on occasion it is first diagnosed at a later age. Autoimmune hypoparathyroidism may occur alone, and it may occur in patients with one or more other autoimmune disorders, which may develop years later, such as Graves' disease, pernicious anemia, hypogonadism, chronic active hepatitis and alopecia. In addition, hypoparathyroidism may occur in association with certain disorders such as candidiasis, autoimmune thyroid disease, and adrenal insufficiency in one of the autoimmune polyendocrine syndromes.

Hypocalcemia is the most serious consequence of hypoparathyroidism. Approximately one-half of the body's calcium is in an ionized or active form, and the other half is bound or linked to plasma proteins. Hypoparathyroidism causes a significant reduction in the ionized or active form of calcium.
Symptoms occur when ionized calcium levels drop to less than 2.5 mg/dl. The symptoms of low calcium (hypocalcemia) are primarily neurologic and are related to the hyperexcitability of neuronal membranes caused by inadequate calcium levels. Cardiac effects of hypocalcemia occur in severe hypocalcemia although most patients receive treatment before heart involvement occurs.

Neurologic symptoms of hypocalcemia related to hypoparathyroidism in adults include altered mental status, confusion, depression, psychosis, gait disturbances, muscle cramping, muscle spasms, tetany, seizures, extremity (hands and feet) and periorbital (area surrounding the eye) paresthesias (numbness or burning, tingling/tickling). In infants, hypocalcemia in hypoparathyroidism causes hyperirritability, vomiting, abdominal distention, diminished respiration, twitching, tremors, intermittent cyanosis, muscle rigidity with normal mental status, and seizures.

**Diagnosis**

About 60 percent of patients with autoimmune hypoparathyroidism demonstrate antibodies to parathyroid tissue or to the calcium-sensing receptor within the parathyroid gland. The criteria for a diagnosis of autoimmune hypoparathyroidism include: hypoparathyroidism based on low calcium, elevated phosphorus and low PTH levels without surgical or mechanical causes in the presence of PTH or calcium-sensing parathyroid gland receptor antibodies, or who have an association with another autoimmune disease.

Clinical signs include Trousseau sign in which a hand spasm occurs when an inflated blood pressure cuff is left on the arm for several minutes, and Chvostek sign in which tapping on the facial nerve in front of the ear causes contraction of the muscles of the eye, mouth, and nose. Electrocardiographic studies may show a prolonged QT interval.

**Treatment**

To restore calcium levels in patients with hypocalcemia, patients are treated with calcium gluconate administered intravenously. For maintenance and prevention of hypocalcemia, patients with autoimmune hypoparathyroidism are treated lifelong with calcium supplements and ergocalciferol, vitamin D-2 (Calciferol) to stimulate absorption of calcium and phosphate from small intestine. A diet high in calcium and low in phosphorus is recommended.

**Resources:**


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