Laboratory screening for hyperparathyroidism.

Younes NA1, Shafagoj Y, Khatib F, Ababneh M.

Abstract

INTRODUCTION:
The clinical syndrome produced by excess parathyroid hormone (PTH) is referred to as hyperparathyroidism (HPT). Autonomous growth of PTH producing cells is defined as primary hyperparathyroidism (pHPT). In its classic form pHPT is characterized by painful bones, kidney stones, abdominal groans, psychic moans, fatigue overtones and hypercalcemia. Chronic stimulation of the parathyroid glands secondary to low circulating calcium level results in secondary hyperparathyroidism (sHPT). Tertiary hyperparathyroidism (tHPT) results from prolonged secondary hyperparathyroidism when the glands take on an autonomous function manifested by hypercalcemia and high PTH levels despite resolution of the original stimulus.

REVIEW:
The paper reviews the physiologic regulation of PTH secretion and types and forms of HPT. Calcium homeostasis is discussed, emphasizing interactions of PTH, PO4 and vitamin D that can lead to HPT. In addition, the paper reviews the contribution of serum calcium, chloride, phosphorus and PTH levels to the diagnosis of HPT, the role of urinary calcium in the diagnosis of familial benign hypocalciuric hypercalcemia (FBHH), and the role of alkaline phosphatase and bone mass measurements as markers of severity of hyperparathyroid bone disease.

CONCLUSIONS:
It is concluded that the diagnosis of hyperparathyroidism can be made with a very high confidence rate by documenting an increased serum PTH level with an increased ionized or total calcium level in pHPT, increased serum PTH level with low or normal calcium level and an underlying renal failure or vitamin D deficiency in sHPT. Early management of HPT is important because many of the nonspecific complains, or classic symptoms, or metabolic conditions often improve after proper control of hyperparathyroidism.