The parathyroids and the gut.

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BACKGROUND: This review discusses gastrointestinal manifestations of parathyroid diseases. Parathyroid hormone is the primary regulator of calcium physiology. Hypoparathyroidism can be idiopathic, hereditary, or secondary to surgery in the neck. Hyperparathyroidism is usually from adenomas or hyperplasia. Hypoparathyroidism is associated with steatorrhea that may improve with medium-chain triglycerides, correction of the hypoparathyroidism, or administration of vitamin D. Hyperparathyroidism results in constipation because of reduction in neuromuscular excitability by high calcium levels. According to old literature, the incidence of peptic ulcer disease (PUD) in patients with hyperparathyroidism is 9% compared with autopsy rates of 4% to 5%.

DISCUSSION: Any association is difficult to prove today, as hyperparathyroidism is usually mild due to early detection of cases through routine automated measurements of calcium. In addition, PUD is less prevalent now than before the advent of proton pump inhibitors. The presence of ulcers or ulcer symptoms may correct in some patients after parathyroidectomy, suggesting an association. The incidence of pancreatitis in patients with primary hyperparathyroidism ranges from 1.5% to 12% and may be because of the hypercalcemia. Complicating the issue is secondary hyperparathyroidism in response to hypocalcemia from pancreatitis. Pancreatitis may improve in some individuals after parathyroidectomy.

CONCLUSIONS: Pancreatitis may follow parathyroid surgery because of an acute rise in calcium levels with manipulation of the parathyroid glands or to a blunted response of calcitonin-producing cells from fatigue. Parathyroid diseases have a few distinct effects on the gut: steatorrhea in hypoparathyroidism, and constipation, PUD, and pancreatitis in hyperparathyroidism.

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