

# Recurrent Pancreatitis, an Uncommon Presentation of Primary Hyperparathyroidism

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## Abstract

The association between pancreatitis (PTS) and primary hyperparathyroidism (PHPT) has been previously reported in a few case studies. According to consensus, PTS is a consequence rather than a cause of PHPT (1). We present a case of recurrent PTS, which helped in diagnosing an otherwise clinically silent PHPT.

## CASE

A 50-year-old female with past medical history of irritable bowel syndrome and cholecystectomy presented with acute onset of abdominal pain associated with nausea and vomiting. Laboratory studies revealed lipase 2588 U/L, amylase 284 U/L, serum calcium 11.3 mg/dL, ionized calcium 1.48 mmol/L (normal 1.12-1.32 mmol/L), phosphorus 2.8 mg/dL, cholesterol 189 mg/dL, HDL cholesterol 29 mg/dL, LDL cholesterol 119 mg/dL and triglycerides of 203 mg/dL. Computer tomography scan of abdomen was compatible with acute PTS. Intravenous fluids and pain medication were initiated, and the PTS resolved uneventfully.

Her past medical history also revealed an episode of PTS post endoscopic retrograde cholecystopancreatography (ERCP) about 15 years ago. Her recent past medical history includes three episodes of acute PTS in six months time. An ERCP done after her third episode was negative. Recurrent episodes of acute PTS without an evident cause other than the hypercalcemia resulted in further investigations. A bio-intact PTH was 118 pg/mL (normal 10-69) confirming the diagnosis of PHPT.

## DISCUSSION

In 1947, Martin and Canseco were the first to report the association of PHPT with PTS (2). Multiple case series studies and case reports have since debated the cause and effect relationship of PTS and PHPT (5,6,7,8,9,10). Kelly et al in 1968 reported the role played by calcium in the pathogenesis of PTS.

PHPT affects multiple organ systems resulting in recognized clinical syndromes. PHPT leads to nephrocalcinosis, osteoporosis, neuromuscular symptoms including fatigue, confusion, somnolence and coma; hypercalcemia crisis, pseudogout and multiple endocrine neoplasia (MEN) (2). Peptic ulcer disease and PTS are the gastrointestinal (GI) syndromes associated with PHPT. The most important GI symptoms with hypercalcemia include constipation, nausea, vomiting, anorexia, epigastric and diffuse abdominal pain (2).

Hypercalcemia leads to accelerated intrapancreatic conversion of trypsinogen to trypsin, which causes the pancreatic damage (3). PHPT is the cause for recurrent episodes of acute PTS in the current patient. In fact, recurrent PTS led to the diagnosis of an otherwise silent PHPT. Elevated serum levels of calcium, ionized calcium and bio-intact PTH needs further evaluation by an ENT surgeon for possible resection, which is currently being sought. Surgical resections of PHPT have resulted in the cure of acute PTS. However, the role of the surgical resection in preventing subacute and chronic cases of PTS is still unknown (4). This case report highlights the importance of considering PHPT in the differential diagnosis of acute PTS.

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