

LETTER TO THE EDITOR

Primary hyperparathyroidism and chronic pancreatitis

To the Editor,

We read with interest the article by Bhadada *et al.*¹ from Chandigarh published OnlineEarly in your journal. The article rekindles interest in the association between primary hyperparathyroidism (PHPT) and pancreatitis. Although recent publications on primary hyperparathyroidism tend to downplay this association, Pyrah and Hodgkinson² attempted to classify the circumstances in which pancreatitis is found in association with PHPT as early as 1966. We recently attempted to modify the classification to include four major classes,³ which we cited and discussed in our article:

- 1 PHPT presenting as acute pancreatitis.
- 2 PHPT presenting as acute recurrent pancreatitis with no evidence of chronic pancreatitis.
- 3 PHPT presenting as chronic pancreatitis with or without pancreatic calcification.
- 4 PHPT complicated by acute pancreatitis in the postoperative period.

Class 3 PHPT with chronic pancreatitis, which is the focus of the study by Bhadada *et al.*, has accounted for 54% of pancreatitis cases seen with PHPT in our center, and 37% of presentations seen in over 87 patients with pancreatitis and PHPT from four additional large series in literature. Although the authors have not commented on any additional patients with acute pancreatitis seen among the 59 patients with PHPT, the presence of pancreatic disease in over 18% of patients with PHPT is similar to the experiences at our center (12%).³

Unlike in the study reported, we did see significantly higher values of calcium among patients with pancreatitis and PHPT compared to patients with PHPT without pancreatic involvement.³ Even other etiologies of pancreatitis including alcohol, ductal hypertension, ischemia, and viral infections may trigger pancreatitis via elevation of intracytoplasmic calcium.⁴ This leads us to believe that hypercalcemia, in addition to being a direct risk for pancreatic injury, may also augment pancreatic disease in patients with other causes of ongoing pancreatic injury. The authors of the present study excluded two patients with a history of alcohol

intake and PHPT. However, it would be interesting to note the clinical course of pancreatic disease in these two patients after parathyroid surgery. In our group of patients with PHPT and an additional etiology for pancreatitis, there were no further episodes of pain after parathyroid surgery.

We propose that the reason for larger numbers of patients with PHPT presenting with pancreatitis in India may be twofold. First, PHPT in India is symptomatic to a large extent. Second, unusual pancreatic diseases like tropical chronic pancreatitis is a common cause of chronic pancreatitis in India and its etiopathogenesis is still elusive.⁵ Sustained hypercalcemia may unmask subclinical or preclinical tropical chronic pancreatitis in patients predisposed to the disease.

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