Chronic diarrhea and diffuse gastric wall thickening: What is the common link?

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The gastric wall thickening is a diagnostic challenge for gastroenterologists and can be caused by a wide variety of benign and malignant disorders including lymphoma, adenocarcinoma, Menetriers' disease, Crohn's disease, peptic ulcer disease, sarcoidosis and tuberculosis. We present a case of diffuse gastric wall thickening that presented with long-standing chronic diarrhea and on evaluation was found to be having pancreatic gastrinoma.

A 50-year-old male was referred to us for evaluation of complaints of intermittent large volume diarrhea and nonbilious vomiting of 4 years duration. During the episodes of diarrhea he used to pass large volume, watery stools but no blood or mucus. He had lost around 8 kg of weight over last 2 years. There was associated mild epigastric discomfort and occasional vomiting. Physical examination was unremarkable. He had been evaluated elsewhere and had normal complete blood counts, biochemistry, immunoglobulin A tissue transglutaminase serology and stool examination. Ultrasound of the abdomen was normal. An esophagogastroduodenoscopy showed thickened gastric folds with an inconclusive biopsy. However, the contrast enhanced computed tomography (CT) scan of the abdomen showed a hypodense mass lesion in the region of the uncinate process of the pancreas with thickened gastric walls [Figure 1]. Positron emission tomography-CT scan showed increased metabolic activity in the thickened gastric wall with active tracer uptake in the lesion in the uncinate process (SUVmax 10.6) [Figure 2] and no detectable metastases. Endoscopic ultrasound (EUS) revealed a hypoechoic 2.6 cm × 1.8 cm well-circumscribed lesion in the uncinate part of the pancreas. Fat planes with surrounding vessels were normal and preserved [Figure 3]. Rest of the pancreas and pancreatic duct were normal. Serum gastrin levels were markedly elevated (2664 pg/mL; *N* <100 pg/ml). Considering the results of above-mentioned investigations, a diagnosis of gastrinoma was kept. Review of the patient's history indicated that the intake of proton pump inhibitors had alleviated his symptoms and resulting in the intermittent diarrhea whenever he stopped the drug. A pylorus-preserving pancreatico-duodenectomy was done. There was a well-encapsulated lesion in the uncinate region of the pancreas. Histopathology showed tumor cells arranged in cords

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separated by fine fibrovascular septa. These cells had finely stippled monomorphic nuclei (hematoxylin and eosin stain, × 450) [Figure 4]. Immunostain done for gastrin showed cytoplasmic positivity (PAP, × 450) [Figure 4 inset]. Ki-67 was negative. Patient is asymptomatic on follow-up.

Zollinger–Ellison Syndrome (ZES) is a neuroendocrine tumor characterized by hypergastrinemia, which most commonly occur in the duodenum. Pancreas is the second commonest location.[1] The diarrhea in ZES is mediated by the excessive acid production due to an increased parietal cell mass and the resulting reduced duodenal pH. This compromises the activity of pancreatic enzymes that need alkaline pH. Traditionally the patients presented with complications in the form of bleeding, penetration, esophageal stricture and perforation.[2] However, the presentation now is characterized by typical duodenal ulcer contrasting with multiple ulcers and atypical locations noted earlier.[3] Also, there is an increased recognition of gastroesophageal reflux disease like symptoms as a presenting feature. Diarrhea in absence of a peptic ulcer has been noted as a presentation in <10% of cases but is more commonly recognized now.[3] Interestingly use of proton pump inhibitors blunted the tumor and modified the presentation to intermittent episodes of diarrhea. The review of patient's medication history may provide a clue to the underlying disease and may help diagnosing the disease before an adverse oncologic outcome.[4,5] Some reports have indicated that a quarter of patients with gastrinoma may have underlying MEN 1 and therefore the diagnosis of MEN 1 must be sought in all patients diagnosed as the gastrinoma.[6]

The localization of gastrinomas on EUS depends primarily on the location of the lesion. Pancreatic

Figure 1. Contrast enhanced computed tomography scan of abdomen showing thickened gastric wall

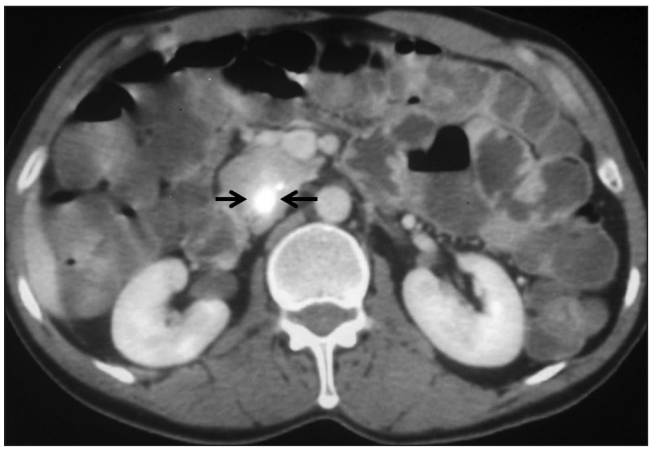

Figure 2. Positron emission tomography-computed tomography scan showing increased tracer uptake in the lesion in the uncinate process

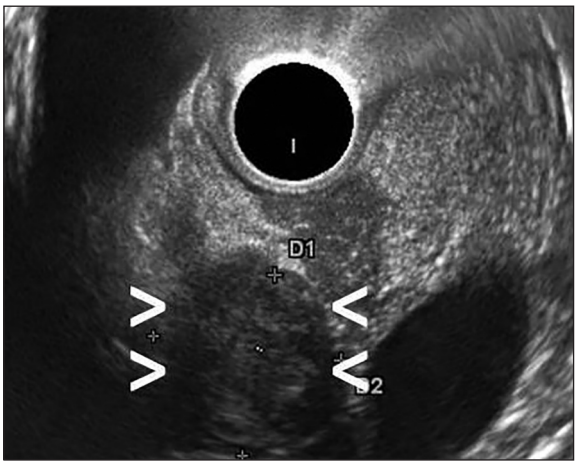

Figure 3. Endoscopic ultrasound showing a hypoechoic 2.6 cm × 1.8 cm well circumscribed lesion in the uncinate part of the pancreas

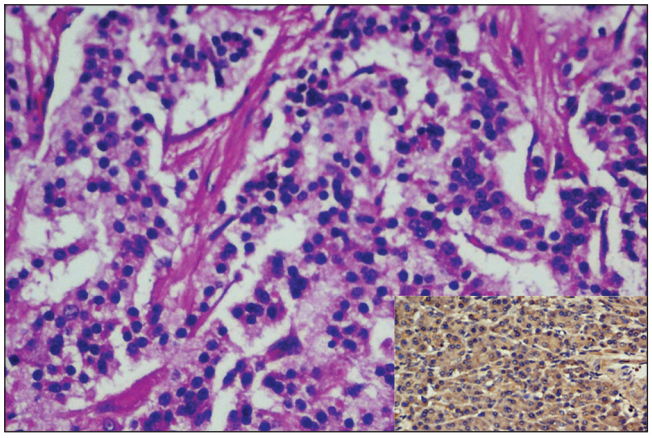

Figure 4. Histopathology showing tumor cells arranged in cords separated by fine fibrovascular septa and cells having finely stippled monomorphic nuclei (H and E, ×450). Immunostain for gastrin showing cytoplasmic positivity (PAP, ×450) (Inset)

lesions are deemed to be easier to localize when compared to the duodenal lesions. The appearance is usually in the form of hypoechoic lesions although on occasion these may be isoechoic and rarely hyerechoic. The lesions may also have a small perilesional halo. Lesions as small as 2-3 mm can be detected on EUS.[7] The diagnostic accuracy of EUS-fine needle aspiration (FNA) may not match pancreatic adenocarcinoma as these lesions may have fibrotic stroma and therefore EUS-FNA cytology may be negative. However, clinical correlation, serum gastrin and EUS visualization of pancreatic lesion suggest the diagnosis in most settings.[8,9]

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Conflicts of interest
There are no conflicts of interest.

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