of duodenal wall and distended stomach **(Figure 1A,B)**. Gallbladder was abutting the duodenum but features of cholecysto-duodenal fistula (pneumobilia and delineation of gallbladder by oral contrast) were absent.

We speculate that evolvingcholecysto-duodenal fistula might have resulted in these atypical findings like the absence of pneumobilia or no noticeable fistulous communication on imaging. This correlated intra-operatively, where the gallbladder was adherent to duodenum, but there was no fistulous connection. The patient was operated with a high index of suspicion of duodenal malignancy Even postoperative cut-section of duodenum revealed mass with ulcerated mucosa at the D1-D2 junction. The correct diagnosis could only be made at final histopathological examination.

To conlude, Duodenal intramural gallstone causing GOO is the rarest spectrum of complicated gallstone disease. The condition can masquerade as malignancy and may result in unwarranted major surgery.

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Strongyloidiasis: An Unusual Cause of Gastric Outlet Obstruction

Strongyloides stercoralis (S. stercoralis) is a helminthic infection with diverse clinical manifestations varying from an asymptomatic infection to a potentially fatal hyper-infection syndrome (HIS) and disseminated Strongyloidiasis (DS).[1] The uncommon gastrointestinal manifestations of *S. stercoralis* infestation are gastrointestinal bleed, duodenal obstruction, small bowel obstruction; perforation, pancreatitis and rarely gastric outlet obstruction.[2-3] Unfortunately, even in tropical countries where *S. stercoralis* infestation is common, there is a delay in the diagnosis due to the low index of suspicion. We report a patient who presented with gastric outlet obstruction (GOO) due to *S. stercoralis* infestation and responded to anti-helminthic therapy.

Case Report

A 23-year-old female presented to our hospital with recurrent non-bilious vomiting of 4 weeks. The frequency of vomitus gradually increased from 2-3 times per day to 6-8 times per day over the same period. There was no history of abdominal pain, hematemesis, melena or fever. In the past, she had been diagnosed with idiopathic thrombocytopenic purpura for which she had been treated with oral prednisolone at a daily dosage of 40 mg for four weeks, which was gradually tapered and finally discontinued one month prior to the onset of the vomiting. General physical examination was unremarkable except for mild pallor and pedal edema. Routine investigations were as follows:

Hemoglobin was 8.6 gm/dL, total leukocyte count 7,600 cells/mm^3, absolute eosinophil count 450 cells/mm^3, platelet count 30,000 cells/mm^3, total bilirubin 1.6 mg/dL, AST 82 IU/L, ALT 38IU/L, ALP 186 IU/L, total protein 5.2 gm/dL, Serum albumin 3.1 gm/dL, Blood urea 22 mg/dL and serum creatinine was 0.6 mg/dl. Stool examination did not show ova or cysts; ELISA for HIV I-II was negative. CECT abdomen showed gastric antral wall

thickening with normal small intestine **(Figure 1D, 1E)**. Upper gastrointestinal endoscopy (UGIE) showed edema and nodularity in antrum and in the first part of duodenum (D1) along with mild narrowing at the D1-D2 junction **(Figure 1A)**. Multiple biopsies were taken from the antrum and duodenal bulb. The histopathology showed multiple larvae of S. stercoralis invading into the lamina propria of the duodenum and antrum **(Figure 1B, 1C)**. She was started on tablet ivermectin 6 mg once a day for 6 weeks. Her symptoms started to improve 1 week after starting ivermectin and she became asymptomatic 6 weeks later. No recurrence was noted on follow up after 6 months. Repeat UGIE 6 weeks after ivermectin treatment showed normal antrum and duodenal mucosa **(Figure 1F)**.

Discussion

The commonest causes of benign GOO in India are peptic ulcer disease, corrosive induced injury and gastro-duodenal tuberculosis.[4] A recent study from India has suggested that there is a paradigm shift in the etiology of benign GOO with these three conditions accounting for nearly 75% of all patients diagnosed to have GOO due to nonmalignant disease.[5] Endoscopic biopsies from the antrum or duodenum are an essential part of the work up in a patient with GOO. Upper gastrointestinal endoscopy findings in Strongyloidiasis are non-specific and include gastric fold enlargement, mucosal edema and erythema, sub-epithelial hemorrhages, ulceration and a deformed duodenal bulb. In the present case, it was the histology which helped in the diagnosis. In previous reports, endoscopic biopsy helped in establishing the correct diagnosis in almost 90% of the cases.Thus, in all suspected cases of *S. stercoralis* infection endoscopic biopsies from the abnormal appearing mucosa of the stomach or duodenum are a must.

To conclude, Strongyloidiasis is an uncommon cause of benign GOO which should be considered as a differential diagnosis in immunosuppressed patients. Endoscopic biopsy is helpful in making the correct diagnosis. Oral ivermectin is the drug of choice in both immune competent and immunosuppressed patients.

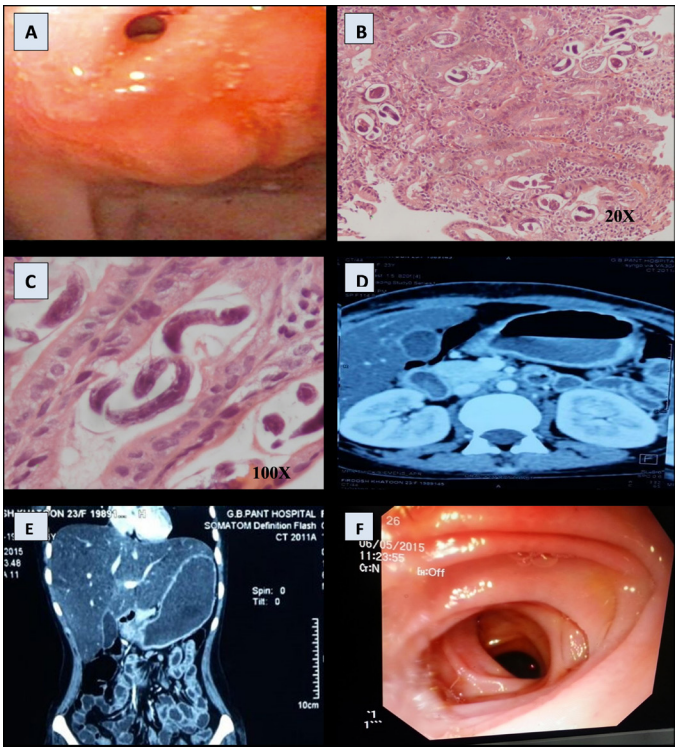

Figure 1 (A): Endoscopic image of the stomach showing antral hypertrophy with concentric narrowing of pyloric opening, (B,C): Duodenal biopsy showing rhabditidiform larvae in lamina propria. (H & E X 20,100) (D): CT scan (axial image) showing concentric transmural thickening of antrum. (E): CT scan (coronal section) showing transmural thickening of antro-pyloric region (F): Endoscopic image of duodenum post Ivermectin therapy showing complete mucosal healing.

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Asymptomatic Expulsion of Self-Expanding Metal Stent Inserted for Drainage of Walled-Off Pancreatic Necrosis

The advantages of endoscopic drainage of peripancreatic fluid or necrotic collection include lower rates of morbidity, lack of nidus for infection, and a lower incidence of percutaneous fistula formation.[1,2] Conventional plastic stents placed for drainage may need frequent revisions, and these have a higher chance of obstruction and dislodgement. Hence, fully covered metal stents are now preferred, especially for the management of walled-off necrosis (WON) by cystogastrostomy or cystoenterostomy. Stent migration has very rarely been reported with covered lumen-opposing self-expandable metal stents (CSEMS).[3,4]

We report the spontaneous, symptom-free expulsion of such a stent from a patient in whom cystogastrostomy was done for WON.

Case Report

A 24-year-old man presented with acute abdominal pain and was diagnosed to have moderately severe acute pancreatitis (revised Atlanta classification).[5] An initial CT of the abdomen showed features of acute necrotizing pancreatitis (CT severity index 9). In the fourth week, the patient had persistent fever and leucocytosis. Repeat CT showed a 10.9 cm x 6.6 cm x 7.8 cm WON. ERCP revealed a pancreatic duct leak, which was managed by duct stenting. The WON was treated by endoscopic cystogastrostomy using a 20 mm x 16 mm CSEMS (Nagi Stent, Taewoong Medical Co. Ltd., Gyeonggi-do, Korea) **(Figure 1)**. Following the procedure, the patient was relieved of fever and pain. One week later, he complained of acute-onset severe abdominal pain. Free air was seen in the peritoneum on a plain radiograph but no leak of oral contrast was found on CT scan. He was managed conservatively.

Two months later, the patient developed a second episode of pancreatitis. Repeat CT imaging revealed no residual collection and the PD stent was in situ; however, the Nagi stent was missing **(Figure 2)**. This was confirmed by endoscopy and fluoroscopy **(Figure 3)**. The patient is doing well one year later.

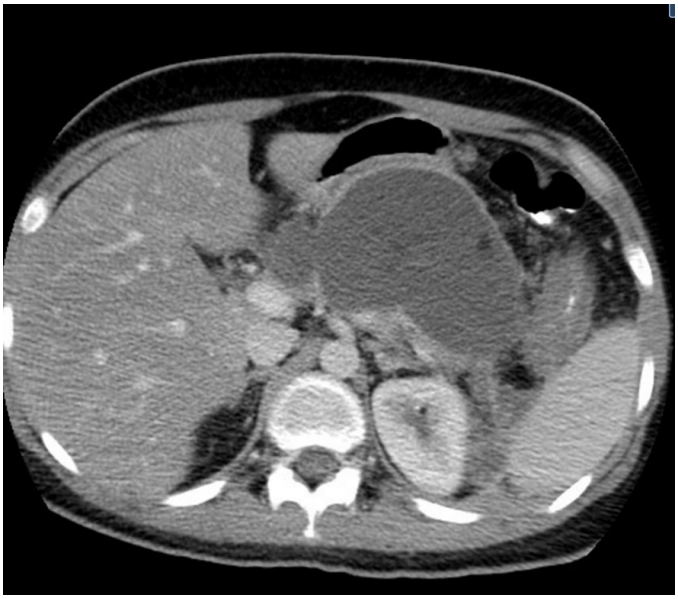

Figure 1: CT showing walled-off necrosis.