One year later, when he came for a routine check up (still well), he brought his younger brother (aged 36 years) who lived with him and worked with him in the fields. The brother had complaints of upper abdominal pain of 6 months' duration. He was also not a consumer of alcohol and there was no significant past medical or surgical history. General examination was unremarkable and there was no palpable mass in the abdomen. Ultrasonopraphy revealed a 4 cm x 3 cm cystic mass in the lesser sac with internal echoes suggestive of pancreatic pseudocyst. The pancreas appeared normal. This patient also underwent laparoscopic cystogastrostomy. A year after this both brothers are well and symptom free.

Both our patients had no past history of pancreatitis, were not consumers of alcohol or known to have gallstone disease. Additionally they had adequate nutritional intake and there was no history of trauma. The similar presentations and identical pathologies suggest a common etiology. They were farmers who lived and worked together, and although an environmental (exposure) factor cannot be ruled out, we could not identify any. Occurring in brothers, there is a possibility of this having a familial or hereditary etiology.[1] Pancreatic pseudocysts without antecedent symptomatic pancreatitis, to our knowledge, have not been described to occur in siblings.

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Small bowel volvulus around feeding jejunostomy tube

Placement of tube or needle catheter jejunostomy as an adjunct to major upper gastrointestinal surgery and following upper gastrointestinal corrosive acid injury is now widely accepted.[1] Minor functional disturbances associated with feeding jejunostomy are well understood and are acknowledged in most studies.[2] However, major complications requiring emergency re-laparotomy related to jejunostomy feeding tube are rare.

A 19-year-old girl had a surgically placed feeding jejunostomy tube for corrosive stricture of esophagus and stomach antrum following acid ingestion. She underwent antrectomy with Billroth I gastro-duodenal anastamosis three months following corrosive injury, and was on regular antegrade endoscopic esophageal dilatation using Savary-Gilliard dilators.

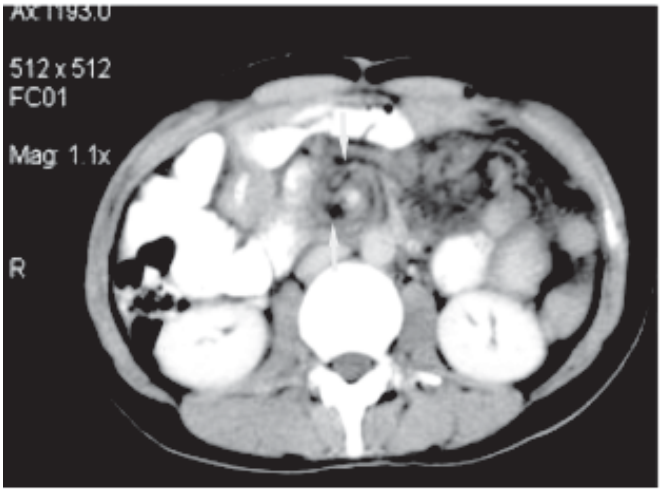

Fig. : Contrast - enhanced CT scan showing "whirlpool sign"

After 9 months, she was admitted with acute severe abdominal pain. On examination, abdomen was not distended. There were no clinical signs of peritonitis. X-ray abdomen was normal. Contrast-enhancement CT scan (Fig) showed jejunostomy feeding tube *in situ*, with a "whirlpool sign",[3] i.e., convergence of mesenteric vessels toward the twisted site in the small bowel mesentery, with mesenteric edema and no free fluid in the abdomen, suggesting small bowel volvulus with impending vascular compromise with no intraperitoneal leak.

At emergency re-laparotomy the jejunal loops were twisted around the jejunostomy tube fixation site on the abdominal wall, resulting in small bowel volvulus with mesenteric edema and bowel ischemia with no peritoneal contamination. Untwisting of the small bowel was done, which resulted in a pink viable small bowel with good mesenteric pulsation. The previous jejunostomy site was dismantled and closed with 3/0 vicryl-interrupted sutures. The patient had an uneventful recovery.

Despite the advantages of feeding jejunostomy, serious complications do occur and can be life-threatening. In a large study, intestinal occlusion and volvulus occurred in 0.14% of all needle catheter jejunostomy applications.[4] Zapas *et al*[5] reported that routine insertion of feeding jejunostomy as an adjunct to major upper abdominal procedures may not be justified, as its benefit-risk ratio was low.

Small bowel volvulus at the anchored site of jejunostomy tube can be prevented by broad-based fixation (6-10 cm) of the jejunal loop to the parietal peritoneum of the anterior abdominal wall using three or four 3/0 silk sutures.

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Olanzapine-induced diabetes due to pancreatitis

Although the new antipsychotic drug olanzapine shares many pharmacological and clinical properties with clozapine, it is believed to have lesser side effects than the latter. Recently, new-onset diabetes, ketoacidosis and some cases of pancreatitis have been linked to olanzapine treatment.[1]

A 25-year-old man with paranoid schizophrenia had been treated with haloperidol for 4 years. When this resulted in marked extrapyramidal symptoms, haloperidol was slowly reduced and olanzapine treatment was started at 10 mg per day. After 22 months of olanzapine monotherapy he was admitted to hospital in poor physical condition with acute abdominal pain. He suffered from nausea, vomiting, polydipsia and polyuria. The abdomen was firm, distended and board-like with extensive guarding. Bowel sounds were not heard during auscultation.

Blood tests prior to beginning olanzapine therapy were normal. However, on admission to hospital, ALT was 130 IU/L (reference range: 10-41), random blood glucose was 283 mg/dL (75-110), total cholesterol 202 (0-200), triglyceride 301 mg/dL (0-200), calcium 5.5 mg/dL (8.6-10.6), glycosylated hemoglobin A1c 7.1% (4.8-6.0), C-reactive protein 390 mg/dL (0-10), amylase 130 IU/L (0-115), lactate dehydrogenase 450 U/L (80-240), leukocytes $15.6x10^9$/L (3.5-11.0). He did not use alcohol, narcotics or other medication. CT scan showed swelling of the pancreas with enlarged, poorly defined borders.

The patient was transferred to the intensive care unit for management of acute pancreatitis, and olanzapine was discontinued. Although treatment resulted in clinical and laboratory amelioration of pancreatitis, the patient remained diabetic and is continuing insulin therapy.

Psychiatric patients may have substance abuse and alcohol consumption as well as trauma, cholelithiasis, hypercalcemia and other drugs as cause of pancreatitis. In our case we ruled out all other possible causes of acute pancreatitis. A few cases of olanzapine-induced pancreatitis have been reported earlier.[1,2,3]

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Cherry pip bezoars causing acute small intestinal obstruction presenting as diabetic ketoacidosis

Most bezoars occur in patients who are mentally challenged. We report a patient who swallowed approximately fifty cherries with the pips under the mistaken impression that it would cure her diabetes.

A 35-year-old lady, a known diabetic on treatment, was brought to our hospital ketotic, with blood sugar level of 547 mg/dL. She was previously controlled on oral hypoglycemics. She had complained of abdominal pain and her relatives had mentioned that she had eaten several cherries that day. On presentation she was tachypneic and comatose but hemodynamically stable. She was intubated, resuscitated, and started on insulin-glucose infusion. Her blood sugar levels decreased over the next 8 hours. She was extremely tender in the abdomen. Bowel sounds were initially sluggish but later became exaggerated. Abdominal X-ray did not reveal any abnormality.

She was taken up for laparotomy twelve hours after presentation. On laparotomy about fifty cherry pips were found causing luminal obstruction at the terminal ileum. They were milked into the colon through the ileocecal valve, upon which the obstruction was relieved. She made an uneventful recovery. She admitted later to having eaten the cherries on the assumption that this would cure her diabetes.

Acute intraluminal occlusion causing small bowel obstruction is uncommon. Among the causes bezoars are the most common, usually in patients with depressive illness and psychiatric illness.[1] Bezoars have been described with undigested food and vegetable matter serving as nuclei.[2] Bezoars of vegetable matter causing obstruction have been