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. Ultrasonography 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. There 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 anastomosis three months following corrosive injury, and was on regular antegrade endoscopic esophageal dilatation using Savary-Gilliard dilators.

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”, 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 al5 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 suf-
fered from nausea, vomiting, polydipsia and polyuria. The ab-
domen 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 bor-
ders.

The patient was transferred to the intensive care unit for
management of acute pancreatitis, and olanzapine was discon-
tinued. 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, cholelithi-

asis, 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 re-
ported 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 pre-
sentation she was tachypneic and comatose but hemodynamici-
ably 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 exagger-
ated. Abdominal X-ray did not reveal any abnormality.

She was taken up for laparotomy twelve hours after pre-
sentation. 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 recov-
er. 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