days. One month later she was passing 1-2 well-formed stools per day. Her weight had increased by 2 Kg and serum potassium was normal. One year after discharge, she is doing well on gluten-free diet.

This lady with celiac disease presented with diarrhea and developed life-threatening hypokalemia and acidosis. This rare presentation of celiac disease (celiac crisis) has been described in children less than 2 years of age and occasionally in adults.

An acute severe exacerbation of underlying mucosal inflammation leads to uncontrolled diarrhea, which in turn results in dehydration and loss of ions and bicarbonate in stool. This leads to metabolic acidosis and shock. Underlying malabsorptive state would predispose to hypoalbuminemia and hence a decreased effective arterial volume. These patients do not tolerate volume losses, and acid-base and electrolyte imbalance is more severe in them. There is also a compensatory rise in renin and aldosterone levels in response to a volume-contracted state, resulting in kaliuresis, thus worsening hypokalemia. This explains the high potassium requirement in our patient. Despite urinary potassium and trans-tubular potential difference of potassium being high, urinary chloride was increased in her, leading to negative urinary anion gap, indicating a primary extra-renal source of potassium loss.

Celiac disease has been associated with neurologic and psychiatric disorders. Quadriplegia in our patient improved with potassium correction. This may not be a direct neurologic manifestation of celiac disease, but our patient did have abnormal conduction latencies in her nerve conduction study. She may have an underlying subclinical neuropathy, which could be linked to celiac disease and would require follow up. Evidence for peripheral neuropathy has been found in up to 49% of celiac disease patients.

Steroids are indicated in the treatment of celiac crisis. They are known to reduce mucosal inflammation, restore brush-border enzymes and epithelial cell height within 2 days. We withheld steroids in our patient for the fear of worsening hypokalemic paralysis.

Adult-onset celiac disease is being increasingly reported in this country. Diarrhea may not be the presenting symptom in these patients. Extra-intestinal features of this disease are protean and need to be recognized, so that patients can be diagnosed early. Celiac crisis, described mainly in children younger than two years of age, is rare these days due to earlier diagnosis and effective therapy. This case describes another clinical course of celiac disease and emphasizes the need to consider it even in adults suffering from acute diarrhea with severe electrolyte and acid-base imbalance.

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Received December 21, 2005. Accepted February 5, 2006

Infected pseudocyst in tropical pancreatitis presenting as psoas abscess
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Infected pseudocyst as a consequence of tropical pancreatitis presenting as psoas abscess is unusual. We report a 40-year-old man who presented with pain in the right lumbar region. CT and MRI of the abdomen revealed pancreatic pseudocysts with abscess formation in the psoas muscle, and evidence of chronic calcific pancreatitis. He was managed by percutaneous drainage of the abscess along with antibiotics and other supportive measures. [Indian J Gastroenterol 2006;25:260-261]

Psoas abscess as an unusual presentation of pancreatic pseudocyst has been reported in literature, the majority being in alcoholic pancreatitis.

A 40-year-old man was admitted with complaints of severe pain in the right lumbar region with radiation to the thigh since two days. He had intermittent low-grade fever for the previous one week. He had history of recurrent episodes of mild abdominal pain in the past with a recent episode about two months back. There was no history of consumption of alcohol or drugs or any other systemic illness. On admission, he was febrile but general examination was otherwise normal. Abdominal examination revealed tenderness in the right lumbar region.
There was severe pain on extension of hip and psoas test was positive. There was no palpable abdominal mass or ascites and rest of the systemic examination including spine was unremarkable.

Investigations: white cell count 19,000/cm³ (80% polymorphs) and ESR 80 mm in first hour. Serum amylase and lipase levels were 644 IU/L and 964 IU/L, respectively. Liver and renal biochemistry, lipid profile, serum calcium and blood sugar were normal. X-ray showed multiple dense calcifications close to L1 vertebra. Ultrasonography revealed a large focal collection in the region of right psoas muscle below the level of right kidney, with evidence of pancreatic calcification. CT scan showed bulky right psoas muscle with a hypodense lesion with enhancing walls of size 7 cm x 5 cm x 7 cm. The pancreas was atrophic, with multiple large, dense calcifications in the head and dilated pancreatic duct with intraductal calculi. MRI of the thoraco-lumbar region showed hyperintense signal in the right psoas muscle suggestive of fluid collection (Fig). The vertebral bodies and disk spaces were normal.

Aspiration showed purulent foul-smelling fluid and biochemical analysis showed amylase value of 10,256 IU/dL, fluid protein 4.5 g/dL, WBC count 12,750/mm³ (75% polymorphs). Gram stain showed gram-negative bacilli and culture revealed growth of E. coli. AFB stain and culture were negative. ERCP revealed dilated main pancreatic duct with calculi, but no ductal leak could be demonstrated. Ultrasound-guided percutaneous drainage of the abscess was done. He was managed with parenteral antibiotics and other supportive treatment for two weeks. CT scan three months later showed complete resolution of the abscess.

Pseudocysts occur in about 25% of patients with chronic pancreatitis but are relatively uncommon in tropical pancreatitis, occurring in 5%-6% of cases. Infected pseudocyst due to chronic pancreatitis presenting as psoas abscess is infrequent and occurring as the initial manifestation or complication of tropical chronic pancreatitis has not been reported. The mechanism is possibly tracking of the fluid along the psoas muscle. Adequate treatment combines antibiotic therapy with prompt surgical drainage. Percutaneous catheter drainage has also been shown to be effective in selected patients with loculated infected fluid collections in the absence of significant pancreatic or peripancreatic necrosis or severe disease.

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Received December 26, 2005. Received in final revised form February 14, 2006. Accepted March 29, 2006

Metachronous gastric diffuse large B-cell lymphoma and adenocarcinoma

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The development of gastric carcinoma in a patient with gastric lymphoma is rare. Helicobacter pylori is a common etiologic agent for both these conditions. We report a 38-year-old lady who was initially diagnosed to have gastric lymphoma and developed early gastric carcinoma on follow up. She was operated on for the carcinoma and is in complete remission since.

Synchronous gastric adenocarcinoma and mucosa-associated lymphoid tissue (MALT) lymphoma in association with Helicobacter pylori infection, although rare, has been well documented. Metachronous gastric adenocarcinoma following gastric MALT has been reported in 7 cases; H. pylori infection is the common etiologic factor. However, gastric adenocarcinoma following gastric diffuse large B-cell lymphoma (DLBL) has not been reported in literature so far.

A 38-year-old lady was investigated elsewhere for dyspeptic symptoms and intermittent vomiting. Upper gastrointestinal endoscopy (UGIE) revealed extensive infiltrative growth in the body and antrum of the stom-