Celiac crisis is a rare life-threatening presentation of celiac disease, observed mainly in children less than 2 years of age. It presents as an acute severe exacerbation of underlying disease, causing multiple metabolic derangements and shock.

A 30-year-old lady was admitted with diarrhea of one-month duration. Stools were large volume, watery, without blood or mucus, with a frequency of 15-20 per day. There was no history of fever. There was no response to an antibiotic course received outside. She had had similar diarrheal episodes since 3 years and had lost 15 Kg during this period despite normal appetite.

On admission she was dehydrated and had features of malnutrition, in the form of pallor, paper-money skin, stomatitis and cheilosis. Initial investigations showed microcytic hypochromic anemia, normal-anion-gap metabolic acidosis and severe hypokalemia (2.1 mEq/L). Liver function tests were abnormal (AST 174 IU/L, ALT 78 IU/L, albumin 2.4 g/dL) but viral markers (anti-HAV, HBsAg, anti-HCV, and anti HEV) were negative. Stool examination was normal.

She was hydrated and potassium was supplemented orally and parenterally. Despite aggressive potassium correction, hypokalemia persisted, and on day 4 of admission she developed acute flaccid pure motor quadriplegia. She had a low single-breath count and was transferred to the ICU. Her potassium requirement remained high (350 mEq/d) despite control of diarrhea. On evaluation of hypokalemia, the patient had kaliuresis (26.9 mEq/L). Her trans-tubular potassium gradient was high (>4), but arterial blood gas analysis revealed normal-anion-gap metabolic acidosis. Urinary chloride was also elevated (220 mEq/L). Urinary anion gap was negative (-170 mEq/L) indicating high urinary ammonium excretion. This suggested an extra-renal loss of potassium. Her neurological status improved gradually, and she was transferred out of the ICU on day 7.

Barium meal follow-through revealed mildly dilated jejunal loops with flocculation of barium. Anti-gliadin antibodies were strongly positive (IgA 63.4 U/mL, IgG 111.1 U/mL: positive >17). UGI endoscopy revealed pancytopenia and scalloping of duodenal folds. Biopsy from the second part of duodenum showed subtotal villous atrophy. She was put on gluten-free diet, to which she had good response. Potassium normalized over the next few days.

Celiac crisis presents as severe acute diarrhea with life-threatening metabolic derangement in a patient with celiac disease. We report a 30-year-old lady who was admitted with one-month history of worsening small bowel-type diarrhea. She developed acute quadriplegia due to refractory hypokalemia. Celiac disease was diagnosed on the basis of positive serology and histological features. She improved with aggressive correction of hypokalemia and gluten-free diet. Celiac crisis is a rare presentation of this heterogeneous disease in adulthood. [Indian J Gastroenterol 2006;25:259-260]
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 age1 and occasionally in adults.2

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-contrasted state, resulting in kaliuresis, thus worsening hypokalemia. This explains the high potassium requirement in our patient. Despite urinary potassium and trans-tubular potassium gradient 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.3 Quadriparesis 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.3

Steroids are indicated in the treatment of celiac crisis.4 They are known to reduce mucosal inflammation, restore brush-border enzymes and epithelial cell height within 2 days.5 We withheld steroids in our patient for the fear of worsening hypokalemic paralysis.

Adult-onset celiac disease is being increasingly reported in this country.6 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.

References

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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.1,2

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.