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Acute Pancreatitis in a Patient with Malnutrition Due to Major Depressive Disorder

To the Editor:

Alcohol abuse and bile duct stones account for about 80% of acute pancreatitis. Hypertriglyceridemia, drugs and hypercalcemia are other rare causes of pancreatitis. The etiology of about 10% of pancreatitis remains unknown despite extensive investigations.[1] We report a case of "idiopathic" pancreatitis in a patient with protein-energy malnutrition due to major depressive disorder.

CASE REPORT

A 50-year old black male presented to the emergency department with severe epigastric pain, nausea and vomiting. He was emaciated with a body mass index of 16 kg/m^2. The physical examination revealed epigastric tenderness and sluggish bowel sounds. Admission laboratory values revealed an elevated serum lipase at 669 U/L, total bilirubin $<$0.2 mg/dL, AST 257U/L, ALT 212U/L, alkaline phosphatase 400 U/L, serum albumin 2.8 g/dL, serum triglycerides 55 mg/dL, and calcium 9.1 mg/dL. Urine drug screen was negative. The patient was on no medications at the time of admission. Computed tomography (CT) scan of the abdomen revealed pancreatitis. The common bile duct was normal and no gallstones were identified. His prior medical history included protein-energy malnutrition secondary to anorexia from major depressive disorder, and was negative for cholelithiasis, alcohol abuse and pancreatitis. Before the episode of abdominal pain, the patient was reported to be anorectic for about 3 months after he stopped taking psychotropic medications for major depressive disorder. He had lost about 9 lbs during this period. This episode occurred when he started feeding again after a period of prolonged starvation. His symptoms improved with conservative management and he was subsequently discharged home on medications for depression. The serum lipase was 56 U/L at discharge.

DISCUSSION

Pancreatitis secondary to protein-energy malnutrition from feeding disorders such as anorexia nervosa or, rarely, bulimia has been described in the literature.[2-7] Pancreatitis in protein-energy malnutrition due to major depressive disorder has not been reported. The exact pathophysiology of pancreatitis in protein-energy malnutrition remains unknown. Protein-energy malnutrition causes acinar cell atrophy, epithelial metaplasia and cystic dilatation of the ducts in the pancreas. These changes have been demonstrated in both humans[8-13] and monkeys.[14] There is also increased zymogen granule release and high trypsinogen levels.[11-13] In thin malnourished patients the third part of the duodenum is compressed between the aorta and the vertebral column posteriorly and the superior mesenteric neurovascular pedicle anteriorly due to loss of cushion effect of a fat pad around the superior mesenteric neurovascular bundle.[3,15-18] This phenomenon has been termed the superior mesenteric artery syndrome and may cause duodenal ileus and perhaps high pancreatic duct pressures. This effect is accentuated by refeeding after a period of prolonged starvation.[3,7] Protein-energy malnutrition is also associated with an increase in inflammatory mediators such as tumor necrosis factor-alpha (TNF-alpha), IL-1 and IL-6,[19-21] which have been proposed to play a role in pancreatitis. Levels of TNF, IL-1 and IL-6 are elevated at the onset and may also cause progression of acute pancreatitis by their leukocyte activating properties after the initial pancreatic acinar cell injury.[22-25] Altered pancreatic architecture, high trypsinogen levels, and increased pancreatic duct pressures might ultimately lead to activation of trypsinogen to trypsin within the pancreas and subsequent activation of other proteases, triggering inflammatory processes leading to pancreatitis.[22-26] Pancreatitis occurring in protein-energy malnutrition and anorexia and refeeding has been termed the "dietary chaos syndrome."[2] Protein-energy malnutrition should be suspected as a cause of pancreatitis in the appropriate clinical context, particularly during the period of refeeding after prolonged starvation.

Savio Reddymasu, MD
Daniel E. Banks, MD
Department of Medicine
Louisiana State University Health Sciences Center
Shreveport, La.

Paul A. Jordan, MD
Section of Gastroenterology
Department of Medicine
Louisiana State University Health Sciences Center
Shreveport, La.

Requests for reprints should be addressed to Savio Reddymasu, MD, LSU Health Sciences Center, 1501 Kings Hwy, P.O. Box # 205, Shreveport, LA, 71115.

E-mail: saviocharan@gmail.com

doi:10.1016/j.amjmed.2005.09.023

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Virchow's Node and Horner's Syndrome

To the Editor:

A 50-year-old man with medical history of adenocarcinoma of the stomach was admitted to our department with complaints of severe, prolonged pain in the left shoulder region, and weight loss. Physical examination revealed left supraclavicular mass, ptosis, miosis, enophthalmos of the left eye, and dryness of the left side of the face. The plane chest x-ray showed left apical opacity invading to the first rib consistent with Pancoast's tumor.

Pancoast's tumors involve the apex of the lungs; invade the first and second ribs posteriorly; involve the lower brachial plexus nerve roots (T1 and C8), which produces pain that radiates down the inner aspect of the arm and forearm; and often involve the stellate ganglion, causing Horner's syndrome.

Horner's syndrome (Figure) is the loss of sympathetic innervation to the dilator muscle. The denervated pupil dilates more slowly than the normal one, leading to ipsilateral miosis. Müller's muscle is sympathetically innervated and provides several millimeters of eyelid elevation. Sympathetic denervation results in moderate ptosis with preserved levator function. Weakness of the inferior tarsal muscle elevates the lower eyelid, contributing to a narrowed palpebral fissure. Most patients also experience hemifacial anhidrosis.

Virchow's node (Figure) is an enlarged left supraclavic-

Requests for reprints should be addressed to Igor Maslovsky, MD, Barzilai Medical Center, Ashkelon 78278, Israel.

E-mail address: igorgn@012.net.il.

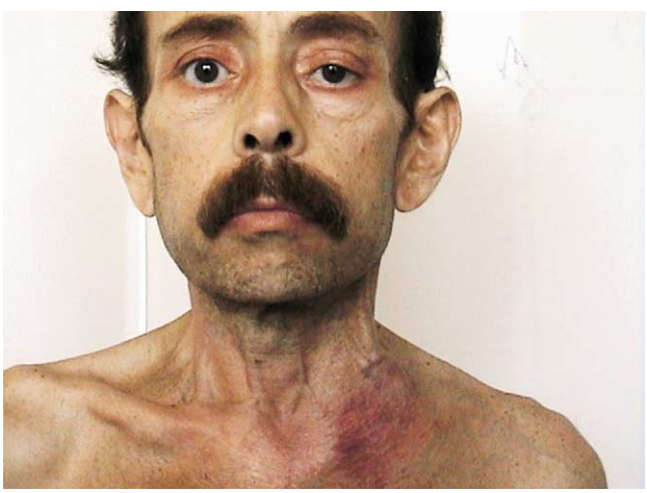

Figure Supraclavicular mass and signs of sympathetic eye denervation are seen in this patient.