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NECROTIZING COLITIS COMPLICATING NECROTIZED PANCREATITIS: LOOK OUT FOR INTESTINAL PNEUMATOSIS

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Acute pancreatitis is a common cause of acute abdominal pain and is associated with a wide variety of complications. Pancreatic necrosis is one of the most important complications and is considered to be the most important indicator of disease severity as the increased frequency of death in acute pancreatitis is directly correlated with the development and extent of pancreatic necrosis. In addition to pancreatic necrosis, wide spectrums of colonic complications have been described, including functional and mechanical ileus, ischemic necrosis and fistula formation. In acute pancreatitis bowel ischemia usually involves the transverse colon or the hepatic and splenic flexures and may range in severity from mild superficial mural involvement totransmural colonic necrosis. This article reports a case of large bowel infarction as a complication of severe necrotizing pancreatitis in a 35-year-old male patient.

Key-word: Pancreatitis.

Bowel ischemia is a devastating disease process encompassing a wide spectrum of clinical and radiological findings, ranging from a mild self-limiting form to bowel infarction and perforation. The conditions most frequently leading to bowel ischemia include vascular occlusion due to arterial or venous disease and hypoperfusion associated with non-occlusive vascular causes. Many other diseases may also cause such vascular changes, including abdominal inflammatory conditions such as pancreatitis, appendicitis, diverticulitis, and peritonitis. Pancreatitis is associated with wide spectrum of colon lesions, including ileus, ischemic necrosis and fistula formation, the reported incidence being 1-15% among adult cases (1). In one series, 27% of patients with acute pancreatitis were complicated by ischemic enterocolitis (2). Early recognition of this particular complication of acute pancreatitis is important, as it is associated with very high morbidity and mortality. Here we report a patient who developed colonic infarction as a complication of acute pancreatitis.

Case report

A 35-year-old man presented to the emergency department with 1-day history of sudden onset of severe epigastric pain and profuse vomiting. On admission his vitals were stable. He had no previous hospital admissions and no previous similar episodes, but confessed hav-

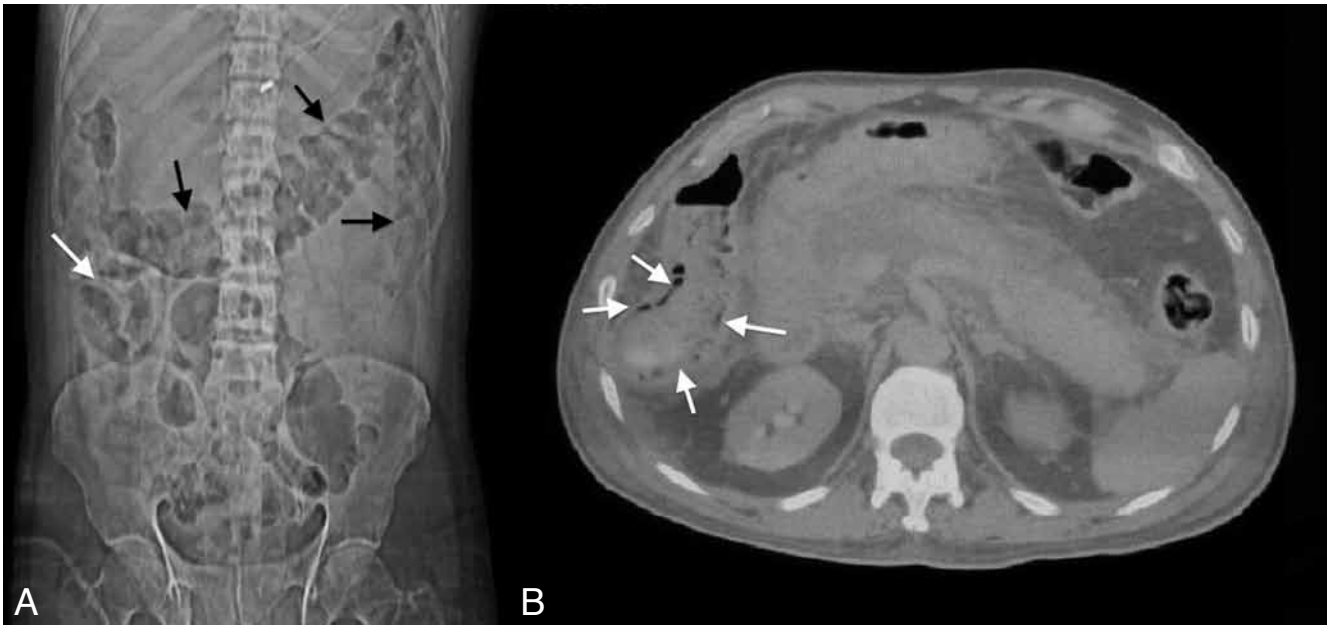

Fig. 1. — A. Frontal CT scout view shows focal dilatation of transverse colon with paucity of proximal and distal gas shadows consistent with colon cut-off sign (black arrows). An ill define speckled gas shadow was also present in right hypochondrium (white arrow). B. CT scan with lung window confirmed these speckled gas shadow to be mural air within wall of ascending colon consistent with pneumatosis intestinalis.

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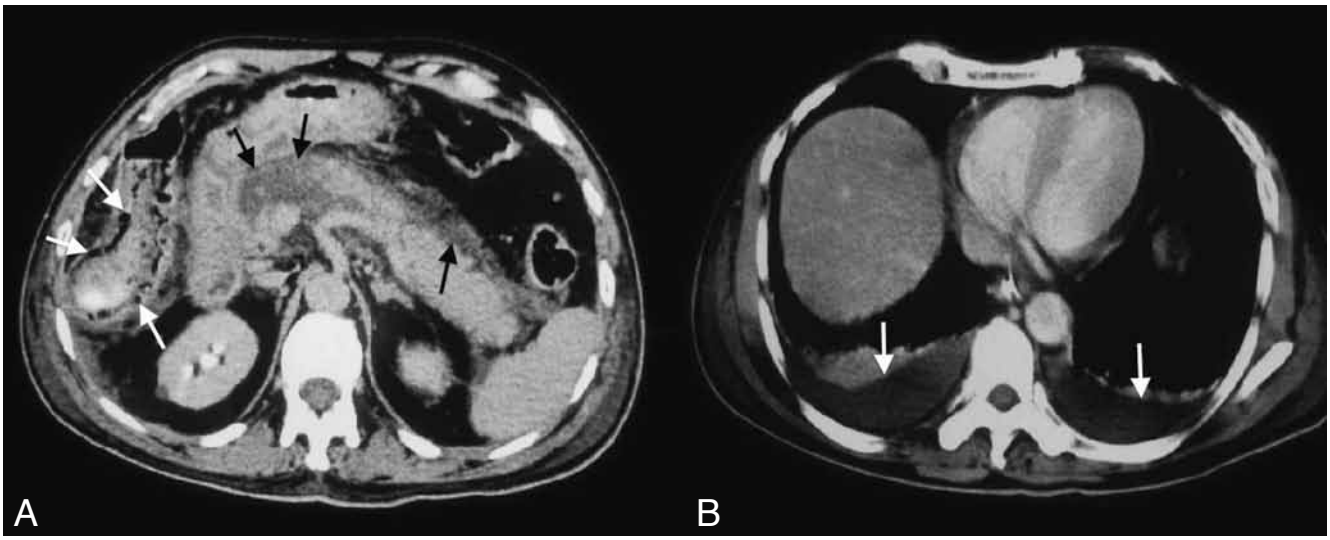

Fig. 2. — A. CT scan at the level of pancreas showed necrosis in head and neck region along with peripancreatic fluid (black arrows). Bowel wall thickening along with mural air can be seen within ascending colon and hepatic flexure (white arrows). B. CT scan at the level of lung base revealed bilateral pleural effusion with basal atelectasis (white arrows).

ing binged on alcohol 3 days back. Physical examination revealed abdominal distention with mild right-sided tenderness but no evidence of peritonism. However patient appeared dehydrated. Laboratory examinations including urine analysis, complete blood count, liver and renal functions were within normal range except for mild leucocytosis (WBC count-28,300/mm3). Serum amylase [1614 U/L (normal value < 220 U/L)] and serum lipase [815 U/L (normal value < 30 U/L)] were elevated. C reactive protein was also elevated at 314 mg/L.

On erect chest radiograph, there was no free gas under diaphragm. However small bilateral pleural effusion was present. Supine Abdominal radiograph revealed focal dilatation of transverse colon with paucity of proximal and distal gas shadows (Fig. 1A). An ill define speckled gas shadow was present in right hypochondrium just below liver in the region of hepatic flexure. Ultrasonography of the abdomen demonstrated absence of gallstones and any dilatation of intrahepatic bile ducts or common duct. Pancreas and adjacent region was not visualized due to excessive bowel gas.

Computed tomography (CT) of the abdomen showed heterogeneous diffusely enlarged pancreas with surrounding fat streakiness and free fluid (Fig. 2A). Non-enhancing area consistent with necrosis was seen in the head and neck region of pancreas. Circumferential thickening of ascending colon extending up to the level of hepatic flexure was also present. There was evidence of water halo sign along with air within bowel wall suggestive of pneumatosis intestinalis (Fig. 1B). Bilateral small pleural effusion was also present (Fig. 2B). These findings were consistent with diagnosis of severe acute pancreatitis with necrotizing colitis.

Patient's condition was relatively stable considering the severity of radiologic findings and conservative management was advised. Patient was shifted to intensive care unit and was monitored closely. Prophylactic intravenous antibiotics and fluids were started. Three days after admission, the patient's condition remained stable. A feeding tube was placed and enteral feeding started. Physiologic and biochemical parameters continued to improve and white cell count returned normal. A repeat CT seven days after admission showed resdiual pancreatic necrosis without significant peripancreatic fluid collections. The pneumatosisintestinalis had completely resolved notwithstanding some residual thickening of the wall of the ascending colon and hepatic flexure. On day 10 oral feeding was started and patient was discharged on day 18. Patient remained asymptomatic at follow-up 2 months later.

Discussion

Acute pancreatitis is an acute inflammatory process that is followed by complete restoration of structural and functional normalcy after the attack subsides, provided that no part of pancreas has been destroyed by necrosis. A number of conditions are responsible for this, the most common being choledocholithiasis and alcohol abuse. Most patients present with nausea, vomiting and abdominal pain. The diagnosis is usually established by the detection of elevated levels of pancreatic enzymes in the blood, urine, or both. Once the diagnosis is established, the treatment of the patients is based on the assessment of disease stage and severity. CT is used not only to confirm the initial diagnosis but also to assess the severity of attack, and detect any complications. Modified CT severity index is a very useful scoring system that grades the severity of pancreatitis into mild, moderate and severe. In this index patients are given an even number from 0-10 on the basis of presence or absence of acute fluid collections, pancreatic parenchymal necrosis and extrapancreatic findings such as pleural fluid, ascites, extrapancreatic parenchymal abnormalities, vascular complications, or involvement of the gastrointestinal tract. Patients with a score of 8-10 are labeled as having severe acute pancreatitis and have worst prognosis.

A number of systemic and locoregional complications are seen in patients of acute pancreatitis. Morbidity and mortality in acute pancreatitis is directly associated with the presence or absence of

complications. Pancreatic necrosis and colonic lesions are two such complications associated with poor clinical outcome.

Colonic complications are uncommon in the patients of acute pancreatitis. A recent analysis of pooled data reports the incidence of colonic complications from acute pancreatitis and severe acute pancreatitis as 3.3 and 15%, respectively (4). Although uncommon, colonic lesions are important as they indicate an extensive underlying inflammatory process (5). Colonic abnormalities associated with acute pancreatitis are divided into the following two groups from a pathological viewpoint: (a) Pericolitisshowing lesions mainly in the serosa and subserosa, in which pancreatic inflammation has extended directly through the mesentry; (b) Ischemic colitis, i.e., ischemic changes in mucosa, subucosa and tunica muscularis, developing into colonic necrosis and perforation in severe cases (6). Ischemic colitis is considered to result from a number of underlying patho-physiological mechanisms like congestion of blood flow due to compression by severe edema in the mesentery, thrombus formation in the mesenteric vessels, disseminated intravascular coagulopathy and, decreased blood pressure (7). Aldridge et al. reported transverse colon as the most common site of involvement (63%) followed by splenic flexure (48%), descending colon (43%), ascending colon ((23%) and sigmoid colon (13%). In our patient, the ischemic changes were most conspicuous in the ascending colon. CT revealed thickening of the colonic wall with water halo sign and pneumatosis intestinalis, findings characteristic of transmural colonic necrosis. However no evidence of hepatic portal or portomesenteric venous gas was seen.

There are many causes of pneumatosis intestinalis. It ranges from infectious, inflammatory, neoplastic, or iatrogenic mucosal injury to increased intraluminal pressure and asthma. Furthermore, it is important to differentiate it from pneumatosis cystoides coli, a benign condition of idiopathic etiology. Benign pneumatosis often appears as cystlike collections of gas in the bowel submucosa, while curvilinear, more circumferential gas collections is often considered as characteristic of bowel infarction.

The management of colonic complications of severe acute pancreatitis relies on a high index of suspicion because the clinical presentation is varied, nonspecific, and could occur quite late in the disease process (4). Because such cases mainly have been reported as case reports and series, there are no evidence-based guidelines for management (4). Although surgical intervention remains the choice of treatment when nonviability of the colon is determined, resection may be difficult and complicated. Fortunately in our case patient remained stable and responded well to conservative management probably because of good general health and young age.

References

1. Aldridge M.C., Francis N.D., Glazer G., Dudley H.A.: Colonic complications of severe acute pancreatitis. *Br J Surg*, 1989, 76: 362-367.
2. Gumaste V.V., Gupta R., Wasserman D., Dave P.B., Vieux U.: Colonic involvement in acute pancreatitis. *Am J Gastroenterol*, 1995, 90: 640-641.
3. Takahashi Y., Fukushima J., Fukusato T., Shiga J., Tanaka F., Imamura T., Fukayama M., Inoue T., Shimizu S., Mori S.: Prevalence of ischemic enterocolitis in patients with acute pancreatitis. *J Gastroenterol*, 2005, 40: 827-832.
4. Mohamed S.R., Siriwardena A.K.: Understanding the colonic complications of pancreatitis. *Pancreatology*, 2008, 8: 153-158.
5. Thompson W.M., Kelvin F.M., Rice R.P.: Inflammation and necrosis of the transverse colon secondary to pancreatitis. *AJR*, 1977, 128: 943-948.
6. Yamagiwa I., Obata K., Hatanaka Y., Saito H., Washio M.: Ischemic colitis complicating severe acute pancreatitis in a child. *J Pediatr Gastroenterol Nutr*, 1993, 16: 208-211.
7. Wiesner W., Khurana B., Ji H., Ros P.: CT of Acute Bowel Ischemia. *Radiology*, 2003, 226: 635-665.