Development of Colonic Necrosis Following Severe Acute Pancreatitis

Yoshikazu Umeno, Junji Otsuka, Eizaburo Sasatomi* and Koji Irie*

Abstract

We herein describe a 70-year-old male patient who developed colonic necrosis following severe acute pancreatitis. He was referred to our hospital with a diagnosis of acute pancreatitis. In the course of the disease, he developed sudden and massive hematochezia and died. The autopsy findings revealed large bowel ischemia with transmural infarction. The possible pathogenic mechanisms of colonic ischemia are also discussed.

(Key words: pancreatitis, colonic complication, pathogenesis

Introduction

Colonic involvement is an uncommon but potentially lethal complication of acute pancreatitis. It has been estimated that 1% of the patients with acute pancreatitis develop colonic complications (1-4). Such accompanying lesions show a wide spectrum, including functional ileus, mechanical ileus due to surrounding compression, ischemic necrosis, and fistula formation. The most common manifestation is colonic ileus, which can be seen as the "colon cut-off" sign on plain abdominal film. Necrosis of the colon rarely complicates acute pancreatitis (4, 5). In this article, we report a patient with acute pancreatitis who developed serious colonic necrosis.

Case Report

A 70-year-old man suffered from sudden epigastralgia after eating breakfast on April 3, 1998. His past history was unremarkable except for a two-year history of atrial fibrillation with no medication. He had no history of alcohol abuse. The pain gradually worsened with accompanying symptoms such as nausea and vomiting, and he was admitted to a local hospital. The laboratory findings at admission were normal except for an elevation of pancreatic enzymes. At two days after the onset of symptoms, however, the white blood cell count was 9,200/µl, red blood cell count 470x10^4/µl, hemoglobin 15.1 g/dl, platelet count 8.7x10^4/µl, amylase 187 IU/l, blood urea nitrogen (BUN) 35.7 mg/dl, creatinine 1.34 mg/dl, Ca 7.2 mg/dl, lactate dehydrogenase (LDH) 715 IU/l, total protein 5.7 g/dl, C-reactive protein (CRP) 29.4 mg/dl, and PaO_2 53.6 mmHg under room air. Abdominal computed tomography (CT) revealed a swollen pancreas with edema in the surrounding areas, cholelithiasis and the common duct stones, and ascites, and the patient was thus classified to be Grade IV (Fig. 1). From these findings, he was diagnosed as having severe acute pancreatitis according to the 1990 revised criteria for grading the severity of acute pancreatitis established by the Research Committee for Intractable Diseases of the Pancreas, Japanese Ministry of Health and Welfare. In spite of performing total parenteral nutrition and administering 500 mg/day of gabexate mesilate, his condition deteriorated. The hypoxemia worsened and the platelet count continued to decrease. He was then transferred to our hospital for further treatment on April 7, 1998.

On admission, his consciousness was disturbed and he suffered from delirium. His blood pressure was 146/76 mmHg, and his pulse was arrhythmic with a rate of 112/min. His abdomen was markedly distended with diffuse tenderness and mild guarding, tympanic sounds on percussion, and decreased sounds on auscultation. The laboratory findings were as follows: white blood cell count 5,100/µl, red blood cell count 423x10^4/µl, hemoglobin 12.9 g/dl, platelet count 5.3x10^4/µl, amylase 69 IU/l, BUN 15.4 mg/dl, creatinine 0.7 mg/dl, Ca 6.8 mg/dl, Na 127 mEq/l, K 3.7 mEq/l, Cl 93 mEq/l, aspartate aminotransferase 37 mU/ml, alanine aminotransferase 21 mU/ml, LDH 654 mU/ml, total protein 5.5 g/dl, CRP 22.3 mg/dl, and PaO_2 90.6 mmHg under a reservoir mask at 6 l/min. A chest radiographic examination revealed left pleural effusion and atelectasis in the left lower lung field.

In addition to 500 mg/day of gabexate mesilate, the administration of 1,000 mg/day of citocline, 10,000 units/day of heparin sodium, 150 units/day of human anti-thrombin III, and...
Figure 1. Abdominal computed tomography shows a diffusely enlarged pancreas and inflammatory changes in the surrounding tissue. Gallstones and ascites are also noted.

2 g/day of piperacillin sodium was started. Continuous drainage was instituted for left pleural effusion. Following these treatments, the biochemical abnormalities began to improve gradually. On the 8th hospital day, PaO₂ was 88.7 mmHg under a nasal cannula at 2 l/min, the number of platelets increased to 24.7x10⁴/μL, and the CRP decreased to 5.3 mg/dl. Heparin sodium and gabexate mesilate were then discontinued on the 8th and 14th hospital day, respectively. The patient's overall condition also improved gradually, although low grade to middle grade fever persisted and he sometimes felt palpitation due to the rapid ventricular rate in atrial fibrillation. A liquid diet was started on the 37th hospital day. He was able to completely consume almost all meals and also felt a desire to defecate. However, he was not able to defecate for the first two days after starting the liquid diet. He had watery diarrhea on the 39th hospital day. Bloody stools were noted with abdominal pain on the 40th hospital day. The bloody stools persisted, and he died of hemorrhagic and hypovolemic shock on the 42nd hospital day, despite receiving such intensive care as transfusions, mechanical ventilation, and hemodialysis.

At autopsy, hemorrhagic necrosis of the colon was seen ranging from the terminal ileum to the right transverse colon and from the sigmoid to the rectum (Fig. 2). A histopathological examination revealed evidence of ischemic damage with various degrees of infarction (Fig. 3). There was no evidence of ischemic damage in the remaining segment of the colon, and congestion was seen in the small intestine. The pancreas was characterized by edema, fatty necrosis, and interstitial inflammation, but most of the pancreatic parenchyma was well preserved. An abscess measuring 3x1.5 cm in diameter was observed between the neck of the pancreas and the root of the superior mesenteric vein (SMV), and it extended to the SMV, where both vasculitis and thrombus formation were observed (Fig. 4). There was no thrombosis in either the splenic vein or the portal vein. No arterial embolus or thrombus was detected in the mesenteric arteries, and the heart was grossly normal except for the presence of ventricular hypertrophy.

Discussion

Ischemic bowel diseases are a heterogeneous group of disorders that have as their unifying feature hypoxia of the small and/or large intestine which is caused by alterations in the blood flow. Clinical manifestations vary depending on such anatomic factors as the site of ischemia, the nature of the occlusive process, collateral circulation, superimposed vascular spasm, and the degree and duration of ischemic insult (6).

Pancreatitis is one of the conditions associated with colonic
Acute Pancreatitis with Colonic Necrosis

In the present case, a complex thrombus mixed with fibrin and red cells, which is thought to be formed within a few days, was found in the SMV. Mesenteric venous thrombosis can have either a sudden acute onset, a subacute onset of weeks to months, or a chronic course, although it most often has a subacute course (9). The mortality rate in acute mesenteric venous thrombosis ranges from 20% to 50%, which is significantly lower than the mortality in other forms of acute mesenteric vascular disease (10). A number of predisposing conditions have been associated with mesenteric venous thrombosis, including pancreatitis and other intraabdominal inflammatory conditions, hypercoagulable states, congestive heart failure, portal hypertension, and nonpenetrating abdominal trauma (9-12). The pathogenesis of mesenteric venous thrombosis in inflammatory disorders is probably related to the direct perivascular extension of the local inflammatory process, or the release of thrombogenic factors secondary to inflammation (9, 11, 13, 14). As a result, we believe that the SMV thrombosis observed in the present case was caused by acute pancreatitis probably resulting from gallstones irid thereafter induced colonic necrosis.

Ischemic changes were seen in the colon from both the ascending to the right transverse colon and from the sigmoid to the rectum. The extent of the lesions conformed to the regions supplied by both the superior and inferior mesenteric artery. The extensive colonic necrosis was primarily attributable to the presence of mesenteric venous occlusion. However, such occurrence appeared to be too acute and the lesions too extensive for colonic necrosis secondary to SMV thrombosis.

The mesenteric blood flow might have decreased substantially due to an increase in the intraperitoneal pressure after the patient was put on a liquid diet. Moreover, propulsive movements occur in the colon after meals. They force a mass of feces into the rectum, and then the desire for defecation is felt. Intestinal distention and increased intraluminal pressure are thought to be responsible for colonic ischemia (15, 16). Our patient also had atrial fibrillation. Cardiac arrhythmia is one of the conditions that cause mesenteric vasoconstriction, or it is also referred to as nonocclusive mesenteric ischemia or poor mesenteric perfusion syndrome (17, 18). Based on the above described pathophysiological events, we therefore consider that acute nonocclusive ischemia may be superimposed on the SMV occlusion in the occurrence of colonic necrosis. The reason why the colon from the left transverse to the descending colon demonstrated no necrosis, is considered to be due to the fact that no thrombotic occlusion occurred in the inferior mesenteric vein.

References