

Spontaneous Bowel Perforation during the Course of Acute Pancreatitis

— A case report —

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We recently experienced a case of spontaneous perforation of infected necrosis into the colon and duodenum during the course of acute pancreatitis in a 63 year-old male patient. Enteric perforations or fistulas in the setting of acute pancreatitis implicate severe underlying pathology and have substantial morbidity and mortality. In the meantime it has generally been accepted that infected pancreatic or peripancreatic necrosis should be managed surgically as soon as possible. Enteric perforations in the present case contributed to transient improvement of the patient's infection sign and condition, and thus an early operation was able to be avoided. Delayed surgical management resulted in complete recovery of the patient without postoperative morbidity. Herein we report an unusual complication of acute pancreatitis.

Key Words: Acute pancreatitis, spontaneous enteric perforation

INTRODUCTION

Colonic complications as a consequence of acute pancreatitis are reported to occur in 1% of patients (Lukasch, 1967; Abcarian et al. 1979). These include obstruction, necrosis, perforation, and fistula formation. The terms, perforation and fistula formation, have been used interchangeably and both are late complications of the disease, associated with a

protracted course and probably a consequence of pancreatic or peripancreatic suppuration, or pseudocysts (Adams et al. 1994). Perforation or fistula formation may involve all levels of the alimentary tract, but the transverse colon is reported to be the most frequently involved segment (Negro et al. 1991; Adams et al. 1994). Multiple anatomic sites involvement has rarely been reported in the literature (Jones, 1944; Morton, 1945; Casio and Onstad, 1972; Shatney and Sosin, 1973; Mason et al. 1975; Negro et al. 1991; Kim et al. 1992; Adams et al. 1994). These complications in acute pancreatitis implicate severe underlying pathology and have substantial morbidity and mortality (Bouillot et al. 1989; Farthmann et al. 1993; Adams et al. 1994).

We have recently experienced a case of spontaneous perforation of infected necrosis into the colon and duodenum during the course of acute pancreatitis in a 63 year-old male patient. These perforations contributed to transient improvement of the patient's in-

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fection sign and condition, and thus an early operation was able to be avoided. Delayed surgical management resulted in complete recovery of the patient without postoperative morbidity. Herein we report an unusual complication of acute pancreatitis and discuss the therapeutic strategies that were successfully used.

CASE REPORT

A 63-year-old man was admitted to the Young Dong Severance Hospital because of a 10-hour duration of the acute onset of epigas-

tric and periumbilical pain. He had been relatively well except for a perianal fistula which required surgery 8 years earlier. He had consumed a small amount of distilled liquor daily. On admission, he complained of nausea, vomiting, and back pain, but denied fever, dyspnea, diarrhea, and constipation. Upon examination, the body temperature was 36.5°C; the blood pressure 110/80 mmHg; and the pulse rate 80/min. He appeared acutely ill and the examination of the abdomen revealed marked epigastric and periumbilical tenderness.

His laboratory findings were as follows: white blood cell count 11,500/mm³, hemoglobin 17.5 g/dl, platelets 127,000/mm³, serum amylase 1186 U/L, serum lipase 4760 U/L, AST 68 U/L,

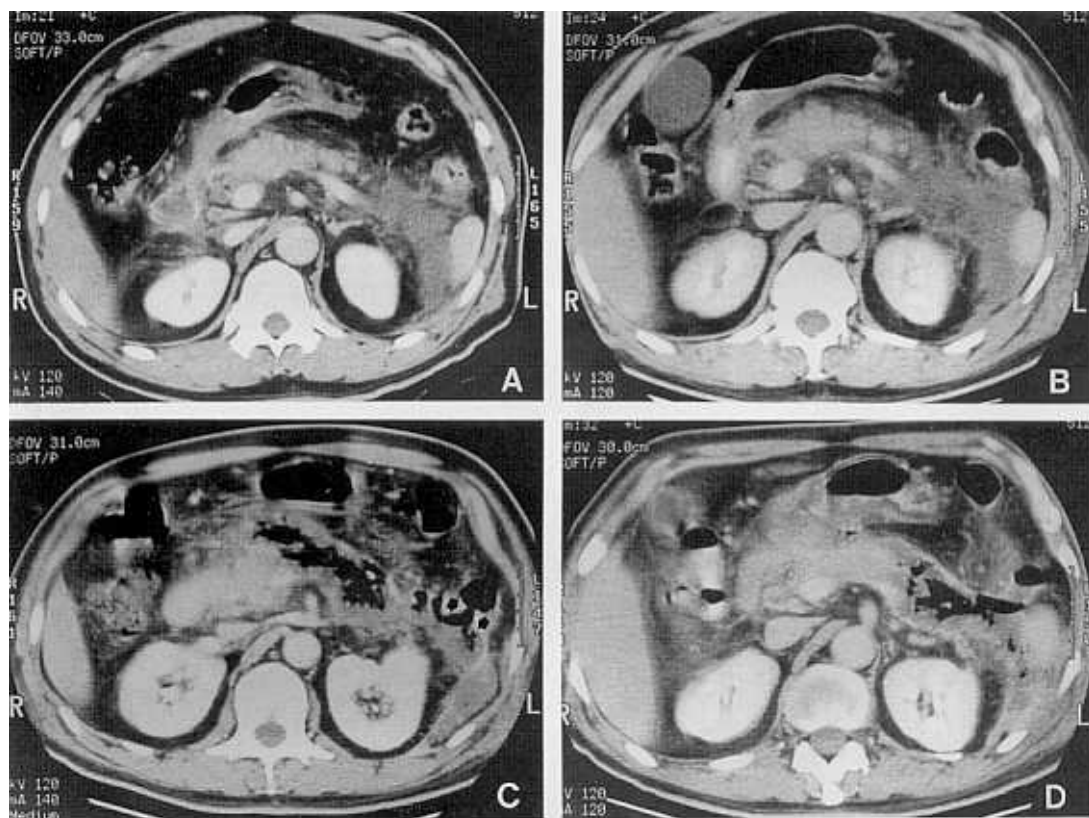


Fig. 1. An abdominal C-T scan performed on admission shows an enlarged pancreas with fluid collection in the left anterior pararenal space and infiltration of peripancreatic fat(A). An abdominal CT scan performed on the 4th hospital day shows progression of acute pancreatitis(B). A follow-up CT performed on the 29th hospital day shows air density in the peripancreatic space and it seems to communicate with the splenic flexure(arrows)(C). A follow-up CT performed on the 53th hospital day shows decrease of peripancreatic fluid collection and air accumulation but persistence of pancreatic swelling(D).

ALT 57 U/L, and total bilirubin 2.3 mg/dl.

An abdominal CT scan performed on admission showed an enlarged pancreas with fluid collection in the left anterior pararenal space and peripancreatic fat (Fig. 1A). High spiking fever ($>39^{\circ}\text{C}$) developed several hours after the admission. Because of the persistence of abdominal pain and fever, a follow-up abdominal CT scan was performed on the 4th

hospital day (HD), which showed progression of acute pancreatitis (Fig. 1B). Despite the treatment with antibiotics, a low-grade fever persisted and fine needle aspiration performed on the 26th HD revealed *Staphylococcus aureus* and *Klebsiella pneumoniae*. On the 27th HD, he had an episode of mild rectal bleeding and watery diarrhea. A follow-up CT performed on the 29th HD showed air density in the

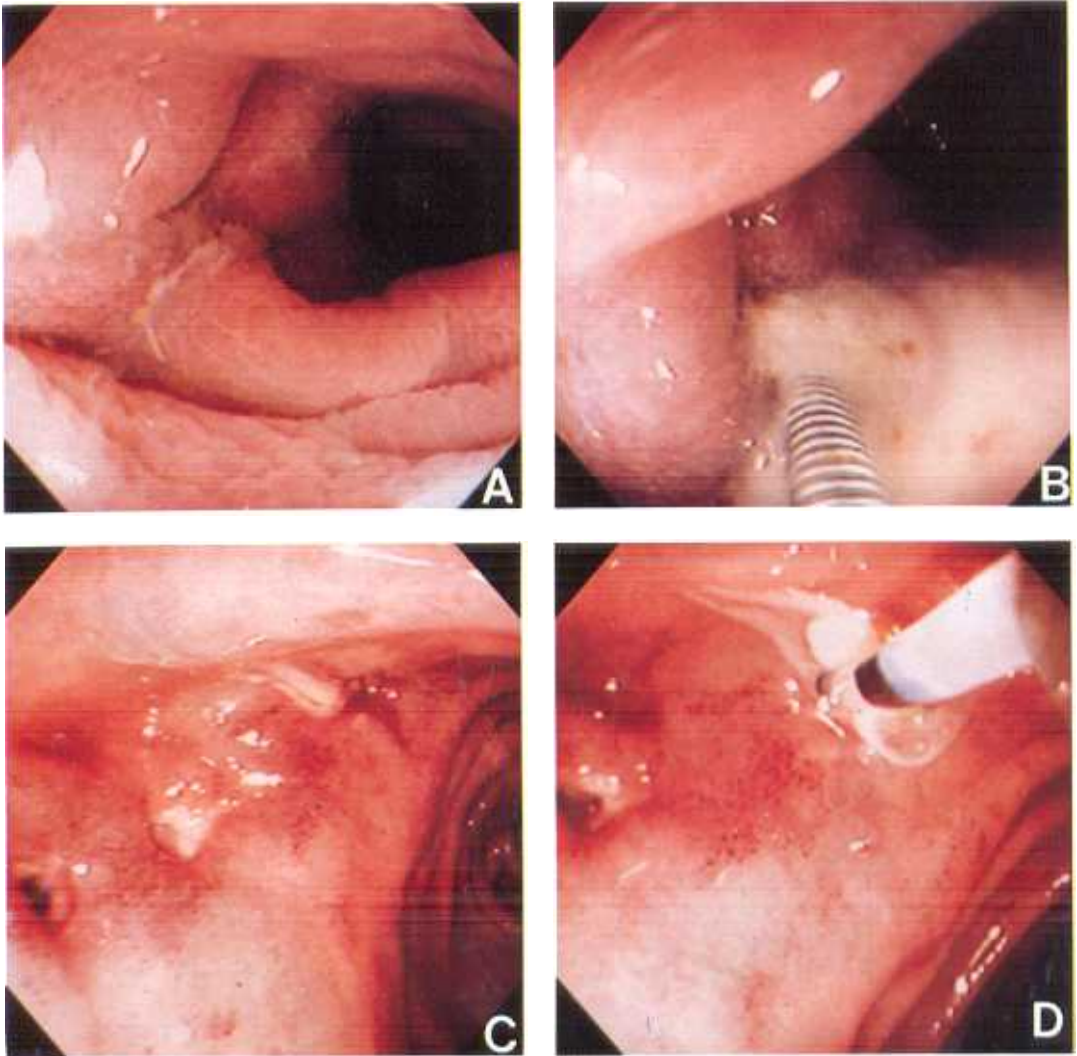


Fig. 2. Colonoscopic findings show hyperemic mucosa coated with exudate on 40 cm from the anal verge (A) and pus gushed out when the biopsy forcep was inserted through the lesion (B). Duodenoscopic findings show 3 holes coated with exudates on the posterior wall and superior border of the duodenal bulb (C). The cannula was inserted through one of the holes (D).

peripancreatic space and it seemed to communicate with the splenic flexure(Fig. 1C).

Colonoscopy and endoscopic retrograde cho-

langiopancreatography(ERCP) were performed on the 31st HD. Colonoscopic findings showed hyperemic mucosae coated with exudates on

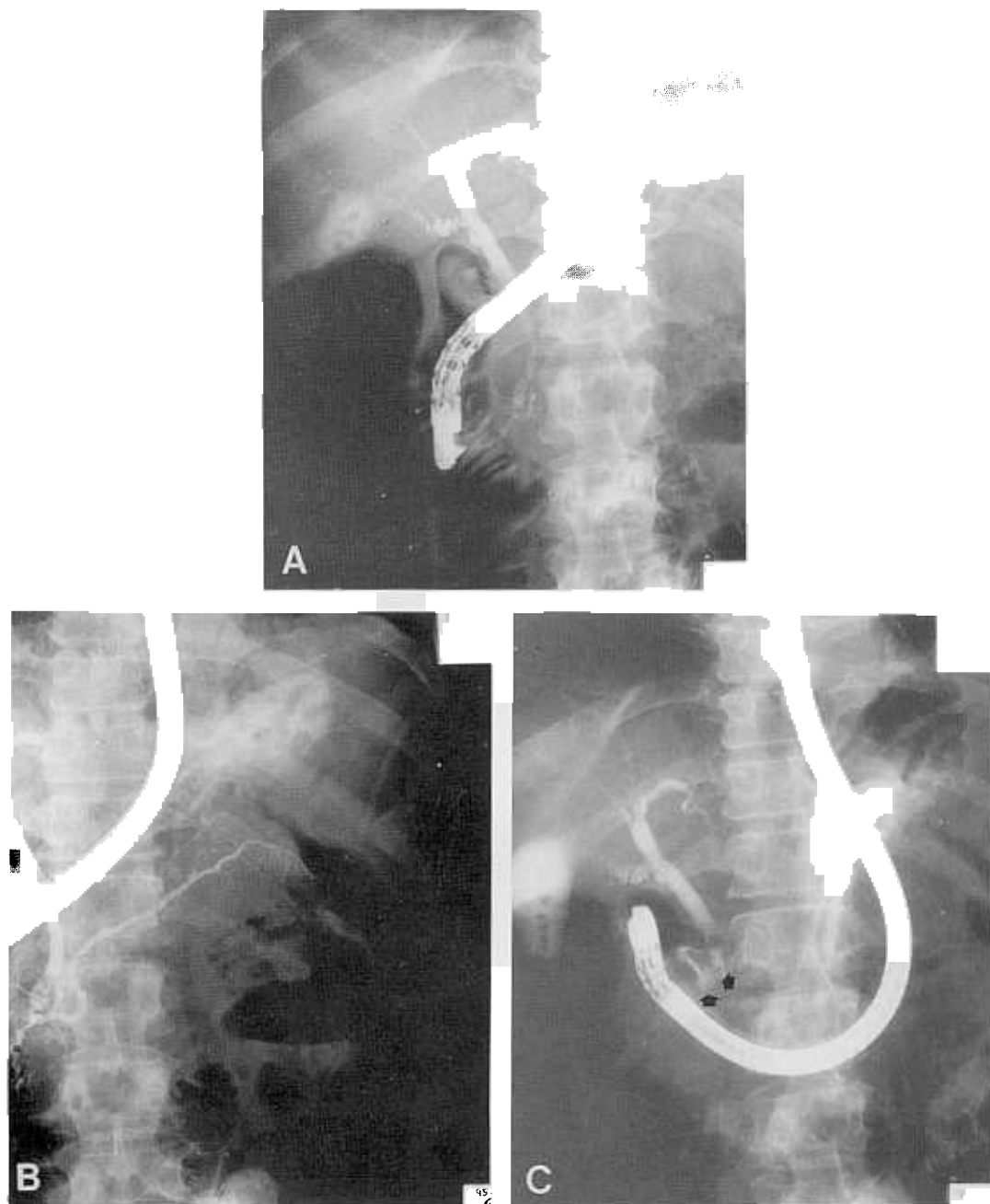


Fig. 3. ERCP shows multiple gall stones but normal bile duct(A) and normal pancreatic duct(B). Fistulogram through one of the holes reveals spread of dye into the retroperitoneal space(arrows)(C).

40 cm from the anal verge (Fig. 2A) and pus gushed out when the biopsy forcep was inserted through the lesion (Fig. 2B). Duodenoscopic findings showed 3 holes coated with exudates on the posterior wall and superior border of the duodenal bulb (Fig. 2C). A cannula was inserted through one of the holes (Fig. 2D). An ERCP showed multiple gallbladder stones but a normal bile duct (Fig. 3A) and a normal pancreatic duct (Fig. 3B). A fistulogram through one of the holes revealed the spread of dye into the retroperitoneal space (Fig. 3C). Fever subsided from then on and he was relatively well until a high spiking fever developed again on the 48th HD. A follow-up CT performed on the 53rd HD day showed a decrease of peripancreatic fluid collection and air accumulation but persistence of pancreatic swelling (Fig. 1D).

Because of persistent fever, an exploratory laparotomy was performed on the 57th HD. The operative findings were as follows; the pancreas was edematous and its consistency was hard. There was yellowish necrotic debris on the posterior portion of the pancreas and peripancreatic area. A tract was found at the distal transverse colon (about 10 cm proximal to the splenic flexure), but no tracts could be found around the duodenal bulb. There were about 0.3 cm sized multiple small black pigment stones in the gallbladder. Cholecystectomy, segmental resection of the transverse colon, pancreatic and peripancreatic debridement, irrigation and drainage were performed. The histologic findings of the resected transverse colon were fistulous tract with chronic inflammation with fibrosis. He recovered uneventfully and was discharged on the 79th hospital day. He is now in good health 12 months after the operation.

DISCUSSION

In 1944, Jones reported a spontaneous perforation of a pancreatic pseudocyst into the colon and duodenum. Simultaneous involvement of the colon and duodenum due to acute pancreatitis was also described by Morton in

1945. However, their cases were diagnosed by postmortem examination. Since their reports, five more cases of simultaneous perforation or fistula formation of the colon and duodenum have appeared in the English literature (Casio and Onstad, 1972; Shatney and Sosin, 1973; Mason *et al.* 1975; Negro *et al.* 1991). In Korea, only one case of pancreatocolonic fistula due to acute pancreatitis has been reported. Interestingly, this case had both pancreatoduodenal fistula and pancreatocolonic fistula, as well (Kim *et al.* 1992). The underlying conditions of perforation or fistula formation of these 8 cases were necrosis in 2, abscess in 3, pseudocyst in 2, and infected pseudocyst in 1. However, the terms used in the previous literature are confusing. A new classification system for acute pancreatitis was made in Atlanta, Georgia, U.S.A. in 1992 and precise definitions of the terms were rendered (Bradley, 1993). Therefore, some of the pancreatic abscesses described might have been infected necrosis as in our case and infected pseudocysts should be termed as a pancreatic abscess. Nonetheless, all the underlying conditions causing perforation or fistula formation of the colon and duodenum fell into the category of severe acute pancreatitis.

The head and body of the pancreas are behind the transverse mesocolon, while the pancreatic tail is in close proximity to the splenic flexure. The inflammatory process, therefore, can spread to the transverse colon and splenic flexure (Chen *et al.* 1994), which in fact were by far the most common sites involved (Goldofsky *et al.* 1984; Negro *et al.* 1991; Adams *et al.* 1994). In acute pancreatitis, extensive peripancreatic exudate can dissect into the abdominal cavity to sites distant from the pancreas, but may involve contiguous organs such as the stomach, and duodenum (Chen *et al.* 1994). Therefore simultaneous involvement of the colon and stomach or small bowel other than the duodenum have, albeit rare, been reported (Fielding *et al.* 1989; Adams *et al.* 1994).

In most of the cases with colonic or multiple enteric perforations or fistula formations, diagnosis was made during surgery (Negro *et al.* 1991; Adams *et al.* 1994) or by postmortem examination (Morton, 1945; Mason *et al.* 1975).

In the minority of the cases, Preoperative diagnosis was made by barium enema(Shatney and Sosin, 1973; Mason *et al.* 1975; Kim *et al.* 1992) or endoscopy(Kim *et al.* 1992; Chen *et al.* 1994). The clues for performing diagnostic work-up were hematochezia with or without diarrhea(Poole and Wallenhaupt, 1984) or air density on CT examination(Kim *et al.* 1992). Our present case showed both features, and therefore we performed colonoscopy which confirmed a fistula opening at about 40 cm proximal to the anal verge. On the same day, ERCP was performed to evaluate the state of the pancreatic duct and to rule out extra-hepatic bile duct stones. Incidentally ERCP demonstrated multiple perforations at the duodenal bulb which had communications with retroperitoneal space.

Actually, we were planning to operate on the patient because the result of fine needle aspiration revealed bacterial infection. However, as fever subsided suddenly and the patient's condition improved following enteric perforations, we decided to wait and see. It has generally been accepted that infected pancreatic or peripancreatic necrosis should be managed surgically as soon as possible. This aggressive approach has contributed to improve the morbidity and mortality of severe acute pancreatitis(Goldofsky *et al.* 1984; Farthmann *et al.* 1993). Nevertheless, early surgery still has high morbidity and mortality(Bouillot *et al.* 1989; Farthmann *et al.* 1993). Additionally, a form of pancreatic fistulas may develop after surgery and multiple operations may be required(Pemberton *et al.* 1986; Ihse *et al.* 1994). As Farthmann *et al.*(1993) pointed out, it should be borne in mind that the later the operative intervention is performed, the better the necrotic tissue will be demarcated. If we had operated on this patient earlier, perforations of the colon and duodenum would not have developed. However, as described previously, surgery during the earlier stage could not have avoided wide blunt debridement of the necrotic tissue and drainage procedure and multiple operations. Wide blunt debridement and drainage procedure themselves have been reported to be the frequent cause of formation of pancreatic fistulas(Pemberton *et al.*

1986; Ihse *et al.* 1994). Obviously, perforations of the colon and duodenum in our case seem to have helped the infected peripancreatic necrosis to drain transiently. We therefore were able to delay surgery enough to confine the necrotic process. Eight weeks later after the initial presentation, a laparotomy was performed because of a persistent low-grade fever that recurred. Debridement, drainage, cholecystectomy, and segmental resection of the transverse colon were performed and the patient recovered uneventfully. The case of Kim *et al.*(1992) might be another example of the advantages of delayed surgery.

Their patient had an attack of acute pancreatitis over 2 months prior to the operation. Although the patient suffered from the protracted course of acute pancreatitis, an one-stage operation did cure the patient uneventfully.

Enteric perforations or fistulas may obliterate spontaneously without any further intervention(Fielding *et al.* 1989; Chen *et al.* 1994). In fact, multiple duodenal perforations detected during ERCP in the present case were not found at the time of surgery.

The colonic fistula found by a barium enema in the case of Kim *et al.*(1992) was also not present at the time of surgery. Instead they found a fistula at the duodenal bulb during the operation. However, both cases required surgery and its attending risks of spreading infection. It might be expected that a patient would benefit from spontaneous decompression of an abscess through a fistulous tract. Berne and Edmondson(1966), however, emphasized that this is not usually true. Decompression is often incomplete and is frequently followed by sepsis or a severe life threatening hemorrhage(Mason *et al.* 1975). Therefore, spontaneous perforation of the infected necrosis or pancreatic abscess does not eliminate the need for adequate surgical drainage. Even in cases with pseudocysts, if spontaneous perforation occurs into the colon, it is recommended that the patient should undergo immediate surgical intervention to avert further contamination of the pseudocyst and its potentially lethal consequences(Shatney and Sosin, 1973).

In summary, although prompt surgical intervention is mandatory in infected necrosis of acute pancreatitis, spontaneous perforations into the colon and duodenum in our present case, which were confirmed by endoscopic examinations, allowed transient drainage of the infected necrosis, and thus an early operation was avoided. Delayed surgical intervention led the patient to recover without morbidity. Thus it is suggested that the timing of surgery in cases with enteric perforations or fistulas complicated by acute pancreatitis should be decided by carefully judging every individual patient.

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