Surgical Intervention Strategies of Necrotizing Pancreatitis With Abdominal Compartment Syndrome

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Objective: Acute pancreatitis can usually recover after conservative treatment. Five to 10 percent of acute pancreatitis may proceed into peripancreatic fluid collection and necrosis development, called necrotizing pancreatitis (NP), which has a high mortality rate. If it is accompanied by the occurrence of abdominal compartment syndrome (ACS) and does not respond to medical therapy, surgical intervention is indicated.

Methods: We analyzed our experience of surgical intervention strategies for NP patients with medically irreversible ACS from January 1, 2004, to December 31, 2018.

Results: Of the 47 NP patients with ACS, mean Ranson score was 6.5, mean Acute Physiology and Chronic Health Evaluation II score was 22.2, and Modified computed tomography severity index score was all 8 or greater. The mean total postoperative hospital length of stay was 80.2 days, of which the mean intensive care unit length of stay was 16.6 days. The overall complication rate was 31.9%. The mortality rate was 8.5%. Among the 47 patients, only fungemia was significantly associated with mortality incidence.

Conclusions: The combination of multiple drainage tube placement, feeding jejunostomy, and ileostomy at the same time were effective surgical intervention strategies for NP patients with ACS, which brought a lower mortality rate.

Key Words: surgical intervention, necrotizing pancreatitis, abdominal compartment syndrome, multiple drainage, feeding jejunostomy, ileostomy

The Atlanta classification of acute pancreatitis (AP) was proposed in 1992 and has been widely adopted.[1] Through this classification, clinicians are allowed to have a unified standard for clinical research and communication. Because of the increased awareness of the pathophysiology and prognosis of necrotizing pancreatitis (NP) as well as the advancement of diagnostic imaging, corrections of the Atlanta classification have been made to further accurately assess the severity and objectively describe the manifestations of local pancreatic complications. The revised Atlanta classification of AP was pronounced on August 29, 2012. Accordingly, AP can be classified into 3 categories by disease severity: mild, moderate, and severe (Fig. 1). Generally, it is a medical disease and patients can usually recover after conservative treatment. If it proceeds into severe AP, there may be some peripancreatic fluid collection and necrosis development. Severe AP is commonly defined by progression of pancreatic symptoms/signs and accompanied by organ failure lasting more than 48 hours, which needs intensive care for organ support.[2,3] When more than 30% of the pancreas is affected by necrosis, we call it NP, which accounts for 5% to 10% of severe AP cases and is associated with a mortality rate as high as 25% in the intensive care unit (ICU).[2]

There are 2 different manifestations of NP. Some cases have pure fluid collections without any solid or nonliquified components around the pancreas arising within 4 weeks of the diagnosis of AP, which are referred to as acute peripancreatic fluid collections. If acute peripancreatic fluid collection is not eliminated after treatment, it may develop into a cyst with a well-defined enhanced wall after 4 weeks, which is known as pancreatic pseudocyst. Other cases that have fluid collections containing necrotic materials are considered acute necrotic collection (ANC). Similarly, if acute necrotic collection does not improve after 4 weeks, it will develop into a well-circumscribed area of mixed fluid-solid collection, called walled-off necrosis.

These collections may be presented within the pancreatic parenchyma, adjacent to it, or both, and then extended to the retroperitoneal cavity. They can be sterile or infected.[4] One week after AP onset, the necrotic tissue may be infected, but the degree of necrosis is not
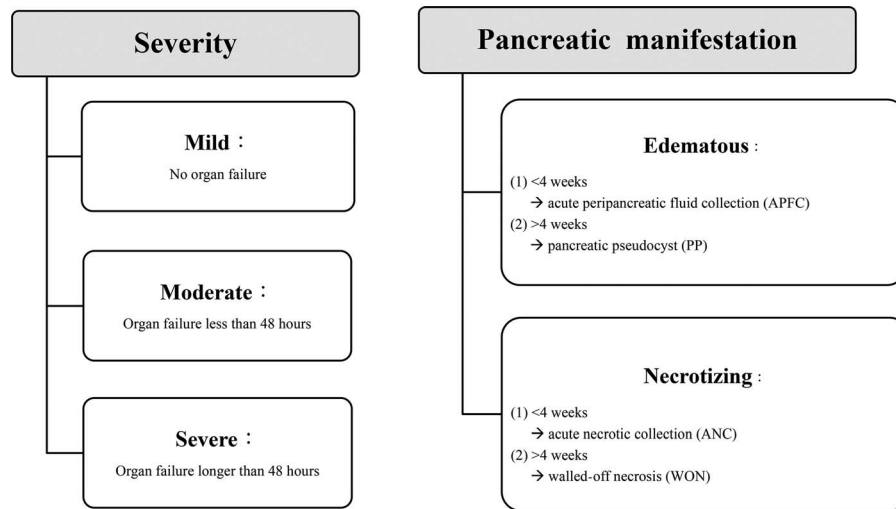
FIGURE 1. The classification of AP.

positively related to the chance of infection. The identification of infection is clinically important because it determines the timing of antibiotic administration. Air bubbles found in the pancreas or the peripancreatic surrounding tissues in computed tomography (CT) and positive bacterial culture report from fine-needle aspiration or radiological drainage or surgical removal from pancreatic tissue may be strongly considered as infection status. While sterile necrosis is associated with a 5% to 10% mortality rate, the mortality rate increases to 20% to 30% when necrosis becomes infected.[5–7] Thus, early recognition and institution of appropriate therapy are necessary.

The treatment of AP in the early phase is to control systemic inflammatory response syndrome and prevent organ failure and most of these are medical treatments. After the second week, we have to pay attention to local complications of infection. If there is an infection, intervention therapy is currently recommended 4 weeks later, when the necrotic area becomes more obvious. An incremental approach (step-up approach) should be adopted. Image-guided percutaneous drainage technique is considered first, and surgical debridement is the last rescue procedure. However, if there was severe retroperitoneal edema, retroperitoneal bleeding, retroperitoneal fluid accumulation, ascites, ileus, spastic intestinal obstruction, or gastrointestinal (GI) tract bleeding present which resulted in intra-abdominal hypertension and the abdominal compartment syndrome (ACS) and that had no response to medical therapy, we had to deal with them immediately by surgical laparotomy.[8]

In this study, we reported and analyzed our experience with surgical intervention strategies for the NP patients with ACS.

MATERIALS AND METHODS

Clinical Patient Data

This study was a retrospective cohort study. The study was conducted in accordance with the Declaration of Helsinki, and the study protocol was approved by the institutional review board of the Taipei Veterans General Hospital (approval number: 2019-10-002CC). Written informed consent from the participants for this retrospective study was waived by the institutional review board.

Data from patients who had NP with medically irreversible ACS and who needed surgical intervention in the Taipei Veterans General Hospital (VGH-TPE) from January 1, 2004, to December 31, 2018, were collected. All the clinical data were recorded from the date of surgical intervention until December 31, 2020. The follow-up period of all the survival cases ranged from 2 to 16 years. Necrotizing pancreatitis was diagnosed by CT images. Medically irreversible ACS was diagnosed by repeat measurement of sustained high intra-abdominal pressure (IAP) more than 20 mm Hg and accompanied by organ failure without improvement after medical and conservative management options. Clinically, bladder pressure was measured and recorded every 4 hours for these patients, which ultimately represented IAP. All the patients with NP and medically irreversible ACS underwent emergent laparotomy for debridement of the pancreatic necrosis, multiple drainages over retroperitoneal spaces, as well as the intra-abdominal cavity, feeding jejunostomy, and ileostomy during the first surgical intervention.

Preoperative clinical characteristics and postoperative complications were recorded. The postoperative complications were divided into early complications, which happened during the same admission period as the first laparotomy and late complications occurred during different admission periods. The outcome recorded not only the complications but also the mortality rate and hospital length of stay (LOS). We further analyzed the risk factors associated with mortality and complication.

Statistical Analysis

Nominal data were compared between groups by the χ^2 test and Fisher exact test. Continuous data were reported as mean (standard deviation [SD]) and compared using the independent t test (parametric data) or Mann-Whitney U test (nonparametric data). A P value of less than 0.05 was considered statistically significant. All analyses were done using SAS version 6.0 (SAS, Inc, Cary, NC).

RESULTS

Clinical Characteristics of the NP Patients With Compartment Syndrome

There were 47 patients who had NP with ACS (39 male and 8 female). The mean Ranson score was 6.5 (SD, 1.3) and the mean APACHE II score (Acute Physiology and Chronic Health Evaluation II score) was 22.2 (SD, 3.4). The modified CT severity index score of all the patients was 8 or greater. The proportion of NP caused by alcoholism was 74.5% (35 patients), whereas cholelithiasis was

TABLE 1. Clinical Characteristics of the NP Patients With Abdominal Compartment Syndrome (N = 47)

Characteristics	
Age, mean (SD, range), y	58.4 (11.8, 26–82)
Sex, n (%)	
Male	39 (83.0)
Female	8 (17.0)
Body mass index, mean (SD, range), kg/m^2	23.7 (3.4, 18.7–34.0)
Etiology, n (%)	
Alcoholism	35 (74.5)
Cholelithiasis	8 (17.0)
Other	4 (8.5)
Ranson score, mean (SD)	6.5 (1.3)
Modified CT severity index score ≥8, n (%)	47 (100)
APACHE II score, mean (SD)	22.2 (3.4)
Sepsis, n (%)	47 (100)
Septic shock, n (%)	34 (72.3)
Respiratory failure, n (%)	40 (85.1)
Coma, n (%)	0 (0)
Acute renal failure, n (%)	36 (76.6)
Hepatic impairment, n (%)	30 (63.8)

17.0% (8 patients). The mean age of the patients was 58.4 years (SD, 11.8 years; range, 26–82 years). Before the emergent laparotomy, 47 patients (100%) experienced sepsis, 34 patients (72.3%) faced septic shock that needed inotropes to maintain the mean blood pressure higher than 65 mm Hg, 40 patients (85.1%) had respiratory failure and needed ventilator support, no patient was in coma (Glasgow Coma Scale ≤8), 36 patients (76.6%) had renal insufficiency and needed renal replacement therapy, and 30 patients (63.8%) possessed hepatic impairment. All of the clinical outcomes for NP patients with compartment syndrome (N = 47) are summarized in Table 1.

Outcome of the NP Patients With Compartment Syndrome

The mean total hospital LOS was 105.5 days (SD, 23.0 days). The mean interval from medical admission to laparotomy was 25.3 days (SD, 7.0 days). The mean postoperative hospital LOS was 80.2 days (SD, 7.8 days). The mean ICU LOS was 16.6 days (SD, 8.1 days) and the mean ventilation day was 14.3 days (SD, 5.6 days). There were 8 patients (17%) who needed a tracheostomy

TABLE 2. Outcomes of the NP Patients With Abdominal Compartment Syndrome (N = 47)

Outcomes	
Total hospital LOS, mean (SD, range), d	105.5 (23.0, 52–207)
Preoperative hospital LOS, mean (SD, range), d	25.3 (7.0, 7–42)
Postoperative hospital LOS, mean (SD, range), d	80.2 (7.8, 44–200)
ICU LOS, mean (SD, range), d	16.6 (8.1, 0–67)
Ventilation day, mean (SD, range), d	14.3 (5.6, 0–67)
Tracheostomy, n (%)	8 (17)
TPN, mean (SD, range), d	9.1 (2.7, 0–22)
Mortality, n (%)	4 (8.5)
Overall complication, n (%)	15 (31.9)

TABLE 3. Early Complications in the NP Patients With Abdominal Compartment Syndrome (N = 47)

Complications	n (%)
Overall early complication	12 (25.5)
Internal bleeding	9 (19.1)
(1) Retroperitoneal venous bleeding	7 (14.9)
(2) Splenic artery bleeding	1 (2.1)
(3) Left gastric artery	1 (2.1)
Gastrointestinal tract perforation	5 (10.6)
Fungemia	12 (25.5)

and prolonged ventilation support. The mean total days with parenteral nutrition supplement was 9.1 days (SD, 2.7 days). The overall complication rate was 31.9% (15 patients). The mortality rate was 8.5% (4 patients). The outcome of all patients is summarized in Table 2.

Complications of the NP Patients With Compartment Syndrome

Complications were divided into 2 categories: early complications are complications that happened during the same admission period as the laparotomy, whereas late complications occurred during different admission periods. The early complications (Table 3) included internal bleeding (9 patients, 19.1%), GI tract perforation (5 patients, 10.6%), and fungemia (12 patients, 25.5%). The late complications (Table 4) included intestinal obstruction (2 patients, 4.3%), pancreatic pseudocyst (1 patient, 2.1%), common bile duct stricture (2 patients, 4.3%), and colon stricture (4 patients, 8.5%).

Analysis of Risk Factors Associated With Mortality in NP Patients With Compartment Syndrome

Table 5 and Table 6 show the analysis of the risk factors associated with mortality and overall complications in NP patients with ACS, respectively. Among the 47 patients, only fungemia was significantly associated with mortality incidence.

DISCUSSION

According to the guidelines, indications for open surgical intervention for NP included infected necrosis with uncontrolled organ failure, symptomatic extensive and multifocal collections, persistent collections after percutaneous or endoscopic intervention, and the presence of ACS.[2,9] Our cases were NP patients with ACS resulting from massive abdominal abscess or inflammation accompanied by bowel obstruction or ileus.

Traditionally, because of its accuracy and relative simplicity, intrabladder pressure monitoring was usually regarded as the

TABLE 4. Late Complications in the NP Patients With Abdominal Compartment Syndrome (N = 47)

Complications	n (%)
Overall late complication	8 (17.0)
Intestinal obstruction	2 (4.3)
Pancreatic pseudocyst	1 (2.1)
Common bile duct stricture	2 (4.3)
Colon stricture	4 (8.5)

TABLE 5. Analysis of Risk Factors Associated With Mortality in the NP Patients With Abdominal Compartment Syndrome (N = 47)

	Survivor (n = 43)	Nonsurvivor (n = 4)	*P*
Age, mean (SD), y	57.6 (11.8)	66.8 (9.7)	0.126
Sex, male/female, n	36 /7	3/1	0.539
Body mass index, mean (SD), kg/m^2	23.7 (3.3)	23.2 (5.3)	0.753
Etiology, alcoholism/cholelithiasis/other, n	33/7/3	2/1/1	0.384
Ranson score, mean (SD)	6.5 (1.3)	6.5 (1.9)	0.869
Modified CT severity index score ≥8, n (%)	43 (100)	4 (100)	
APACHE II score, mean (SD)	21.9 (3.0)	26.3 (5.1)	0.098
Sepsis, n (%)	43 (100)	4 (100)	
Septic shock, n (%)	31 (72.1)	3 (75)	1.000
Respiratory failure, n (%)	37 (86.0)	3 (75)	0.488
Coma, n (%)	0	0	
Acute renal failure, n (%)	34 (79.1)	2 (50)	0.229
Hepatic impairment, n (%)	28 (65.1)	2 (50)	0.613
Preoperative hospital LOS, mean (SD), d	25.5 (6.8)	23.6 (10.2)	0.898
TPN, mean (SD), d	6.8 (4.8)	9.3 (2.4)	0.064
Internal bleeding, n (%)	7 (16.3)	2 (50)	0.160
Gastrointestinal tract perforation, n (%)	3 (7.0)	2 (50)	0.051
Fungemia, n (%)	9 (20.9)	3 (75)	0.046*

*$P < 0.05$, statistical significance.

preferred method for indirect measurement of IAP.[10–13] We measured the intrabladder pressure regularly through the NP patient's indwelling urinary Foley catheter and regarded it as IAP. When the IAP increased to more than 12 mm Hg, we needed to stop feeding the patient, decompress obstructed bowel using nasogastric tube and anal tube, drain the abscess collections percutaneously, and apply diuretics or dialysis to remove excess body fluids. If the previously mentioned conservative treatment failed to effectively improve intra-abdominal hypertension, and the IAP continued to rise to more than 20 mm Hg, emergent laparotomy had to be performed.[14]

Figure 2 shows CT images of a 52-year-old male patient with alcoholism, Ranson score of 7, APACHE II score of 23, sepsis, septic shock under inotropes, respiratory failure under mechanical ventilator, acute renal failure under renal replacement therapy, hepatic impairment, fungemia, and IAP of 21 mm Hg. The CT images showed extension of necrosis to more than 50% of the pancreas with abscess over the bilateral paracolic gutters. Figure 3 presented large amounts of both pancreatic necrosis and retroperitoneal abscess during the operation. Figure 4 demonstrated the location of the drainage tubes. Figure 5 revealed the whole disease process including multiple drainages stage (Fig. 5A), periodic irrigation of the intra-abdominal abscess via multiple drainages stage (Fig. 5B), before reversal of ileostomy stage (Fig. 5C), and after closure of ileostomy stage (Fig. 5D).

The most important work in our emergent laparotomy was not debridement of the necrosis/abscess radically but placing multiple

TABLE 6. Analysis of Risk Factors Associated With Overall Complication in the NP Patients With Abdominal Compartment Syndrome (N = 47)

|	Complications (n = 32)	No Complications (n = 15)	*P*
Age, mean (SD), y	59.3 (9.5)	56.4 (15.8)	0.601
Sex, male/female, n	25/7	14/1	0.406
Body mass index, mean (SD), kg/m^2	23.8 (3.5)	23.4 (3.2)	0.686
Etiology, alcoholism/cholelithiasis/other, n	22/7/3	13/1/1	0.384
Ranson score, mean (SD)	6.6 (1.2)	6.3 (1.4)	0.891
Modified CT severity index score ≥8, n (%)	32 (100)	15 (100)	
APACHE II score, mean (SD)	22.1 (2.7)	22.5 (4.6)	0.623
Sepsis, n (%)	32 (100)	15 (100)	
Septic shock, n (%)	23 (71.9)	11 (73.3)	1.000
Respiratory failure, n (%)	28 (87.5)	12 (80.0)	0.664
Coma, n (%)	0	0	
Acute renal failure, n (%)	25 (78.1)	11 (73.3)	0.725
Hepatic impairment, n (%)	21 (65.6)	9 (60.0)	0.753
Preoperative hospital LOS, mean (SD), d	28.5 (7.4)	23.8 (6.4)	0.069
TPN, mean (SD), d	9.1 (2.5)	9.1 (3.1)	0.103
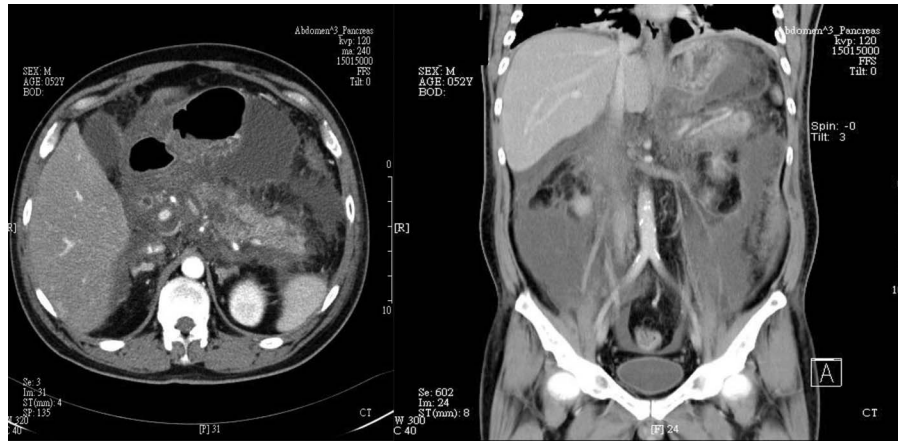
FIGURE 2. Computer tomography images of 1 of our NP patients. Left, axial plane view; right, coronal plane view.

drainages adequately. We first separated the greater omentum from the colon and then opened the lesser omentum. Two drainage tubes were placed via the opening of the lesser omentum to the anterior side of the pancreatic tail and head (shown as D2 and D3 in Fig. 4). One drainage tube was placed via the right anterior pararenal space to the posterior aspect of the pancreatic head (shown as D10 in Fig. 4). Another drainage tube was placed via the left anterior pararenal space through the Treitz ligament to the pancreatic body (shown as D6 in Fig. 4). Because the abscess may often spread down to the bilateral paracolic gutters, another 2 drainage tubes would be placed over bilateral paracolic gutters (shown as D4 and D10 in Fig. 4). We also placed several drainage tubes over the numerous recesses of the peritoneal cavity, in which abscesses tend to loculate, such as lesser sac, Morison's pouch, splenic fossa, and pelvic cavity (shown as D2, D11, D5, and D8 in Fig. 4).

Another important item in the management of an NP patient was to establish nutrition without oral intake initially. Because the presence of food in the stomach would stimulate the pancreas to secrete digestive enzymes, the absence of food through the stomach and duodenum would allow the pancreas to rest without initiating digestive exocrine function. Oral reintake or nasogastric tube refeeding too early would lead to a high incidence of NP relapses.[15] It had been pointed out in the literature that giving enteral nutrition through postpyloric route for a certain period rather than total parenteral nutrition (TPN) before oral reintake/refeeding could reduce the incidence of NP relapse.[16] Postpyloric enteral feeding could also decrease the risk of aspiration. It could be achieved by placing a feeding tube either endoscopically, radiographically, or surgically introduced to the proximal jejunum. Thus, we routinely created a feeding jejunostomy during the laparotomy. Surgical feeding jejunostomy was better than endoscopic or radiographical nasojejunal feeding tubes because there was no risk of sinusitis and a lower risk of inserting the tube into the trachea accidentally.

Giving enteral nutrition not only is more physiological but also can prevent intestinal mucosal atrophy without the adverse effects of TPN (such as fungemia).[17] However, patients with NP often faced adynamic ileus or poor digestive motility with blockage over colon,[18] which may be caused by a large amount of retroperitoneal abscess that spread down to the bilateral paracolic gutters. Patients could not be jejunal fed successfully when the above happens and needed prolonged TPN. The first 5 NP cases in our series only received feeding jejunostomy during the initial laparotomy. Because of severe abdominal fullness and the lack of smooth defecation, all 5 patients underwent a second operation for ileostomy approximately 7 to 10 days thereafter because the intolerance to jejunal feeding. All the subsequent NP patients underwent both feeding jejunostomy and ileostomy at the first laparotomy. Nutrition status could be established and maintained well soon after the laparotomy without TPN. Feeding through a proximal jejunostomy and defecation through a distal ileostomy enabled the body to smoothly carry out most of the digestive function of the intestine. This could further decrease the risk of bacteria translocation induced blood stream infection secondary to the use of TPN. Generally, bacterial translocation was defined as the travel of bacteria or their derivatives from the GI tract to extraintestinal sites (such as mesenteric lymph nodes, liver, spleen, kidney, and blood).[19] With regard to the bacterial translocation, there were 3 main mechanisms mentioned in the literature: (a) long-term use of antibiotics disrupted the ecological balance of the GI tract, giving rise to excessive growth of bad intestinal bacteria; (b) severe systemic inflammatory response
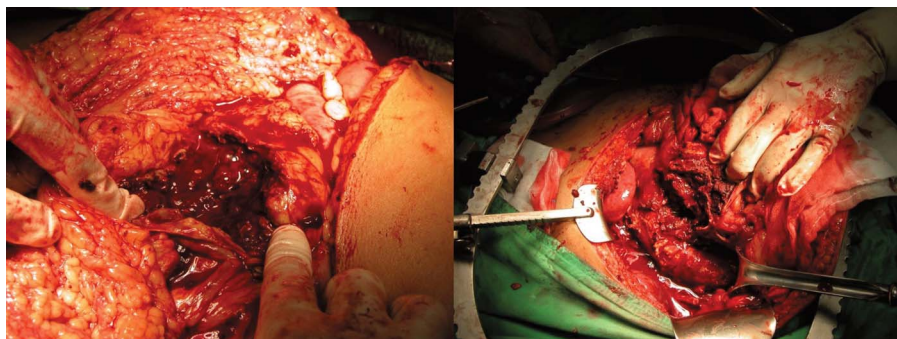
FIGURE 3. Intraoperative pictures of the pancreatic necrosis and retroperitoneal abscess.
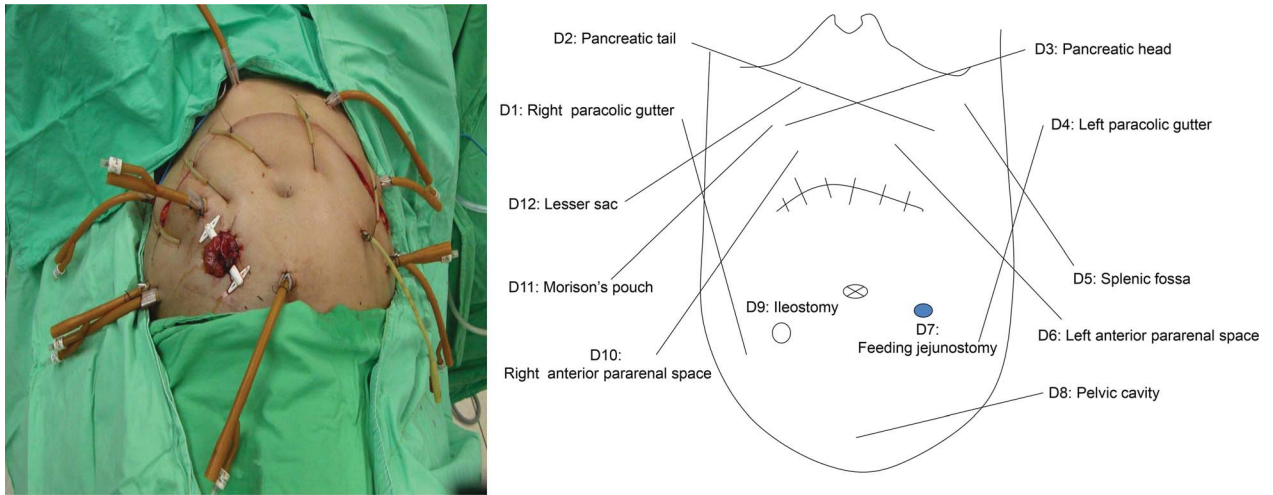
FIGURE 4. The clinical picture and cartoon picture indicating the location of the drainage tubes.

syndrome led to increased permeability of the intestinal mucosal barrier, and (c) prolonged fasting resulted in insufficient host immune defense capabilities.[20] We speculated that surgical intervention strategies with the combination of feeding jejunostomy and ileostomy at the initial laparotomy could effectively allow patients to establish proper enteral nutrition at an early stage, and then reverse the previously mentioned mechanisms to prevent local bacteria from passing through intestinal epithelial cells and vascular endothelial cells and then entering the bloodstream, leading to fatal sepsis.

There were four mortality cases in our series. Two died because of uncontrolled fungemia with multiple organ failure. In our report, only fungemia was significantly associated with mortality incidence. Our analysis coincided with previous teachings that the occurrence of fungemia may increase mortality.[21,22] One case experienced stomach

perforation and massive splenic artery bleeding approximately 2 weeks after the first laparotomy. Reoperation with antrectomy and splenectomy was done. However, leakage over duodenal stump and rebleeding over splenic artery stump was encountered. Persistent NP caused the resection stump of the duodenum and splenic artery continued to be soaked in the abscess, leading to further stump erosion and leakage. Therefore, we proposed not to resect any organs during NP; otherwise, it would cause further leakage and form a vicious circle. This opinion was the same as the concept mentioned in the revised Atlanta guidelines for AP[2] to try not to perform cholecystectomy in the acute phase to prevent biliary tract leakage. The last mortality patient died because of leakage after reversal of ileostomy. Water-soluble contrast enema was arranged to check the integrity and patency of the colon before the reversal of ileostomy and showed no evidence of contraindication
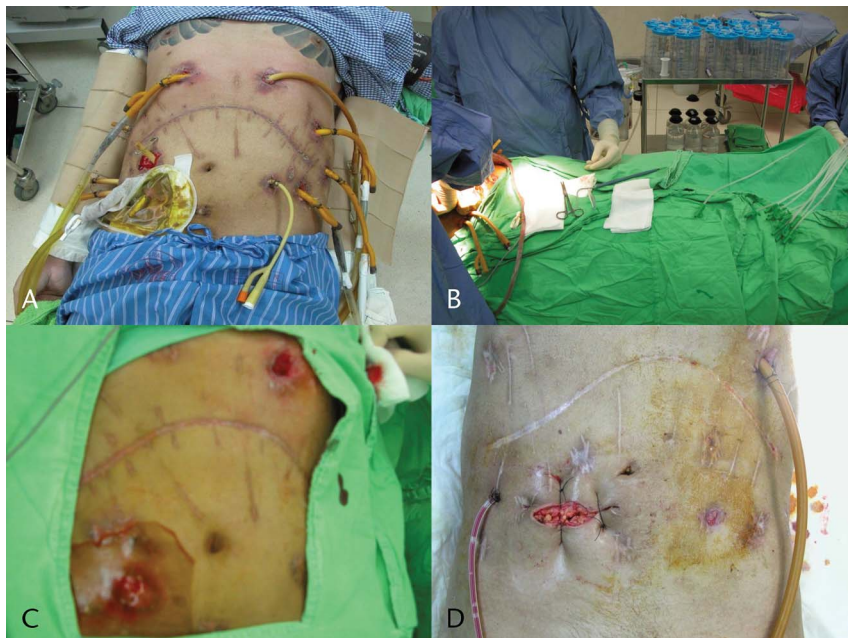
FIGURE 5. A, The picture of multiple drainages stage. B, The picture of periodical irrigation of the intra-abdominal abscess via multiple drainages stage. C, The picture of before reversal of ileostomy stage. D, The picture of after closure of ileostomy stage.

for the reversal surgery. However, a large amount of anastomosis leakage occurred approximately 7 days after the operation. Ileostomy was done again and intraoperative colonoscopy disclosed that stricture over sigmoid colon level with partial obstruction was responsible for the leakage. The patient still died because of uncontrolled severe intra-abdominal infection with sepsis. From then on, we routinely arranged both water-soluble contrast enema and complete colonoscopy before operations to close ileostomies. Once we found stricture of the colon, we would perform colectomy. There were another 3 patients who underwent partial or total colectomy at the same time when the ileostomy was reversed. They were all alive and well with good digestive condition to date.

In summary, the combination of multiple drainage tube placement and feeding jejunostomy and ileostomy at the same time were effective surgical intervention strategies for NP patients with ACS, which brought a lower mortality rate, and it took approximately 3 months of hospital LOS to recover after the operation.

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