Acta med. Nagasaki. 21 : 54–64

Mobilization and Drainage of the Pancreatic Bed as a Treatment for Severe Acute Pancreatitis

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Received for publication, November 29, 1976

A new surgical procedure, mobilization and drainage of the pancreatic bed, was performed on two cases of severe acute pancreatitis in shock state, one edematous type and another necrotic type, and prompt improvement of shock state and excellent healing of pancreatitis were observed clinically. The authors have experimentally proved that shock in acute pancreatitis developed as the retroperitoneal tissues directly absorbed the pancreatic exudate. The advantage of this surgical procedure was also proved on experimental dogs whose severe hypotension was successfully treated. The authors believe that this procedure is a simple and reasonable surgical method for acute pancreatitis.

INTRODUCTION

It has been generally believed that surgical intervention for severe acute pancreatitis is contraindicated in shock state, because conservative medical treatment has resulted in lower mortality than surgical treatment of which ideal operative approach has not yet been agreed. Some cases, however, failed to respond to conservative medical treatment and took fatal courses despite the great efforts.

The authors have performed laparotomy on those patients with severe acute pancreatitis, mobilizing the pancreas from the retroperitoneal tissues, and leaving an external drainage in the retroperitoneal space. As the result, dramatic improvement of shock state and excellent cure of acute pancreatitis ensued. The advantage of this operative procedure has been confirmed on experimental animals with acute pancreatitis.

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CASE REPORT

There were two cases of acute pancreatitis, an edematous type and another necrotic type.

Case 1 A 32-year-old house-wife was hospitalized in shock state following the chief complaints of dyspnea, cyanosis, and epigastric pain. Pulse rate was 120, and blood pressure was 60/45 mm.Hg. No anemia or jaundice was observed. Local tenderness and rigidity were marked over the epigastric region. Bowel sounds were diminished. Clinical data on admission were : leukocytosis, albuminuria, and elevation of urine amylase, serum amylase, serum GOT, serum GPT, fasting blood sugar and urea nitrogen (Table 1).

Table 1. Laboratory Data on Admission

	Case 1	Case 2
Leukocyte Count	14000	14400
Hemoglobin (mg/dl)	11.2	15.6
Hematocrit (g/dl)	32	48
Urine Protein (mg/dl)	222	38
Urine Glucose	0	0
Urine Amylase (Wohlgemuth unit)	8192	128
Serum Amylase	1024	32
Liver Function Test		
Total Bilirubin (mg/dl)	0.2	3.2
Kunkel	8	3.4
Total Cholesterol (mg/dl)	123	131
Cholin Esterase (△pH)	0.6	0.7
Alkaline Phosphatase (K.A. unit)	6.6	11.7
Serum GOT (R. F. unit)	944	77
Serum GPT	1043	123
Serum Electrolytes (Na, K, Cl, Ca)	Normal	Normal
Urea N (mg/dl)	50	50
Blood Glucose (mg/dl)	150	130

First, the shock state was treated conservatively with cardiac and circulatory drugs, blood transfusion, electrolyte correction and adrenal corticosteroid hormone, but poorly responded to these conservative treatment. Therefore, laparotomy was performed 24 hours after admission, revealing accumulation of 70 ml. of yellowish cloudy ascites. The pancreas was edematously enlarged. Edema was also found in the retroperitoneal tissues around the pancreas. No hemorrhage or necrosis was evident in the pancreas or in the surrounding

visera. Pathological examination of the biopsy piece of the pancreas showed pancreatic interstitial edema and pancreatic cell degeneration.

Case 2 A 37-year-old fisherman was refered from a general practitionar because of vomiting and lower abdominal pain under the diagnosis of intestinal obstruction. Past history revealed cholelithiasis followed by cholecystectomy four years prior to admission. No history of habitual alcoholic drinking or trauma was recorded. His face was agonal. No anemia was evident, although mild dyspnea and jaundice were noted. Pulse rate was 132, and blood pressure was 112/96mm.Hg. The abdomen showed marked tension, epigastric tenderness and rigidity. Laboratory findings were more or less the same with the first case, except amylase in urine and serum were slightly increased (Table 1).

Laparotomy revealed approximately 500 ml. of hemorrhagic cloudy ascites and numerous lesions of rice-sized fat necrosis around the upper abdominal viscera. The head of the pancreas was edematous, and the body and the tail showed hemorrhage, necrosis and abscess formation. These changes extended to the retroperitoneal tissues around the pancreas. A biopsy specimen of the pancreas provided histopathological evidences of marked edema, hemorrhage and fat necrosis.

Operative Procedure Both cases underwent mobilization and drainage of the pancreatic bed. This procedure will be called "MD-procedure" in this article. As shown in Fig.1, two incisions were made on the posterior peritoneum, one along the antimesentric border of the duodenum and another along the caudal border of the pancreas. The posterior surface of the pancreas has been free from the retroperitoneal tissues by blunt

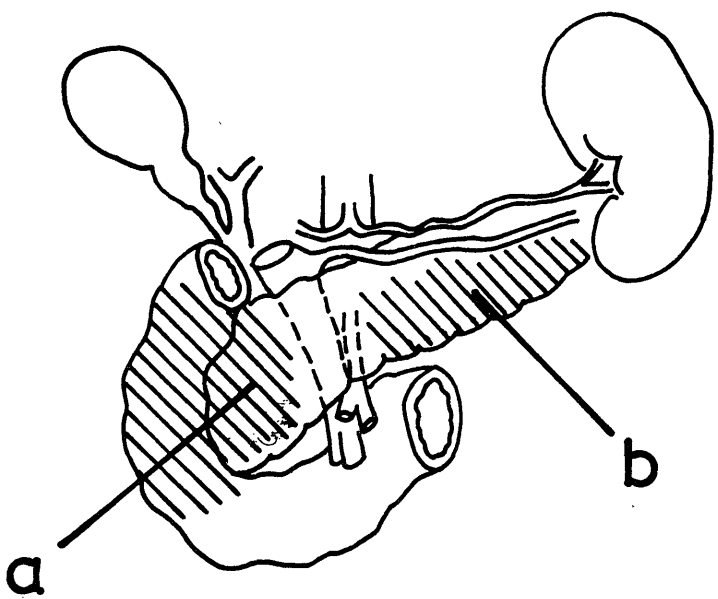


Fig. 1. Mobilization and drainage of the pancreatic bed (MD-procedure) show the area of mobilized pancreatic bed. a and b represent the position of external drainage used catheter.

dissection, avoiding trauma to the superior mesentric vein and splenic vein. During this maneuver the pancreatic exudate filled the area. The soft catheters for external drainage were placed in both right and left sides of the pancreatic bed and the peritoneal cavity.

Postoperative Course Considerable amount of exudate was drained through the drainage catheters from the head and tail of the pancreas in the pancreatic bed as illustrated in Fig. 2. The amount of drainage from the pancreatic head in the first case was 95 ml. on the day of operation, 185 ml. on the first postoperative day, and 435 ml. on the second day. Then, it decreased gradually until it finally disappeared on the 7th day. The exudate from the tail of pancreas reached the maximum of 190 ml./day. The peritoneal fluid was minimum and disappeared on the third day. In the second case, the amount of exudate was greater from the tail than the head of pancreas, 70 ml. from the latter and 180 ml. from the former on the day of operation. The enterobacteria in the exudate were identified only in the second case.

As shown in Table 2, the exudate from the pancreatic bed was more alkaline, pH 7.8, than the exudate from the peritoneal cavity, pH 7.2-7.4. The protein content, electrolytes of the pancreatic exudate resembled to the normal human lymph and differed from the nature of the serum and the ascites.

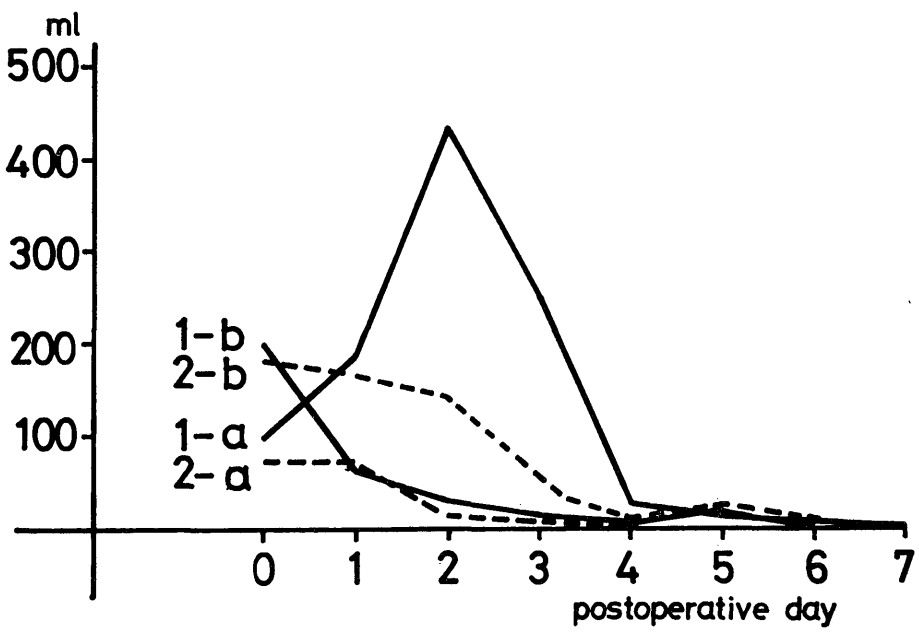


Fig. 2. Flow volumes of pancreatic exudate obtained from pancreatic bed. 1 and 2 represent patient number. a and b represent drainage from the pancreatic head and tail.

In both cases, blood pressure and pulse rate quickly returned to the normal level after the operation without requiring special medication, and it was observed the dramatic relief of abdominal pain and other complaints. Abnormal laboratory findings at admission disappeared within one month after operation. The patients were discharged in good health.

Table 2. Laboratory Data in Serum, Ascitic Fluid and Pancreatic Exudate (lst day after operation)

	Case 1			Case 2		
	Serum	Ascitic Fluid	Pancreatic Exudate	Serum	Ascitic Fluid	Pancreatic Exudate
pH	7.4	7.4	7.8		7.2	7.8
SG	1023	1020	1023		1010	1022
Total Protein	7.0	5.0	7.6	6.2	0.6	5.3
Albumin	51.0	62.8	58.7	54.6	64.8	65.0
α_1-Globulin	5.6	1.3	3.3	10.6	7.0	5.0
α_2-Globulin	11.1	7.9	7.6	18.2	9.3	6.7
β-Globulin	9.3	7.9	8.7	6.1	10.9	11.7
γ-Globulin	23.0	20.1	21.7	10.5	8.0	11.7
Amylase	1024	64	512	8	32	512
Na (mEq/L)	135	54	139	149	50	140
K (mEq/L)	4.1	2.1	5.6	4	1.5	5.2
Cl (mEq/L)	95	37	87	110	35	100
Ca (mEq/L)	5.9	2.7	2.9	5.3	1.2	4.9

EXPERIMENTAL STUDIES

Materials and Methods The adult mongrel dogs weighing approximately 10 kg. were subjected for the experiment. The pancreas of the dog is not enclosed in the retroperitoneal space like the human pancreas, but is free to move in the abdominal cavity. Therefore, it was necessary to fix the tail of the pancreas with suture into the retroperitoneal space after dissection of the capsule of the posterior surface of the pancreatic tail and the posterior peritoneum over the ganglion solare, carefully avoiding injury. The experiment was started more than three weeks after this operation.

Under GOF anesthesia the thoracic duct was exposed in the left supraclavicular fossa, and a polyvinyl catheter was passed into the duct to form an external drainage. Femoral arterial and portal pressure was directly and continually recorded on a polygraphic recorder.

Acute pancreatitis was produced by injecting the autogenous bile, 0.5 ml./kg. body weight, containing 10% duodenal juice, into the pancreatic duct through the orfice of the main pancreatic duct in the speed of one ml. per minute after ligation of accessory pancreatic duct under laparotomy. It was necessary to confirm the good filling of the bile in the pancreatic tail. In all animals arterial blood pressure dropped below 50 per cent of the preinjection level within one to two hours and then MD-procedure was performed.

The pancreatic exudate during acute pancreatitis was collected in the following technique. The covering peritoneal membrane of the pancreas was carefully stripped avoiding injury to the pancreas, which was covered with a rubber condome and the exudate was allowed to flow into this condome when acute experimental pancreatitis was induced by injection of the bile into the pancreatic duct as mentioned above. The mean value and standard deviation of the collected exudate in 5 dogs were pH 8.0±0.2 and Sp. Gr. 1,023±18, and amylase concentration was 11,048±4,032 Smith Roe units/100 ml.

Results 1. Changes of arterial and portal pressure in relation to the injection route of the pancreatic exudate: The pancreatic exudate during acute pancreatitis was injected 0.5 ml./kg. into the peritoneal cavity, the vein, the portal vein and the retroperitoneal space near the ganglion solare, and the change of arterial and portal pressure was measured. In the intraperitoneal group, no change was noted in three hour observation. In the intravenous and intraportal groups, mild transient decrease of arterial and portal pressure was observed immediately after injection. In the retroperitoneal group, arterial

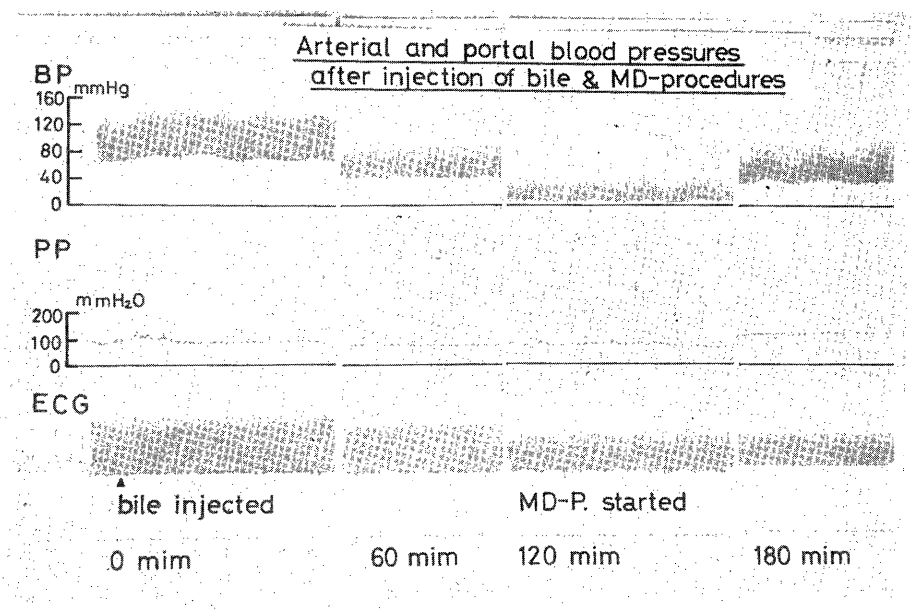


Fig. 3. Changes of arterial and portal blood pressure following bile injection and MD-procedure.

pressure fluctuated shortly after injection, and then dropped significantly. It took 110, 125, 140 and 175 minutes in 4 dogs to drop to 50 per cent of the pre-injection arterial pressure, followed by further progressive decrease. On the other hand, portal pressure increased temporarily and then gradually decreased.

2. Changes of arterial and portal pressure following bile injection and MD-procedure : The Fig. 3. illustrated arterial and portal pressure changes following bile injection and MD-procedure. Injection of the bile was followed by transient mild elevation of the arterial

Table 3. Arterial and Portal Blood Pressures after Bile Injection and MD-procedure in Dogs with Fixation of the Pancreas.

	Arterial Pressure (mm.Hg.)				Portal Pressure (mm.H_2O)			
	Time after injection			Time after MD-procedure	Time after injection			Time after MD-procedure
	Before	60 min.	120 min.	60 min.	Before	60 min.	120 min.	60 min.
No. 1	120/60	85/40	35/10	105/40	105	77	70	115
No. 2	110/52	48/26		70/22	120	79		102
No. 3	132/56	122/50	66/18	95/28	132	128	95	121
No. 4	116/62	80/44	38/8	88/24	100	81	68	92
No. 5	106/82	52/18		68/20	125	117		117

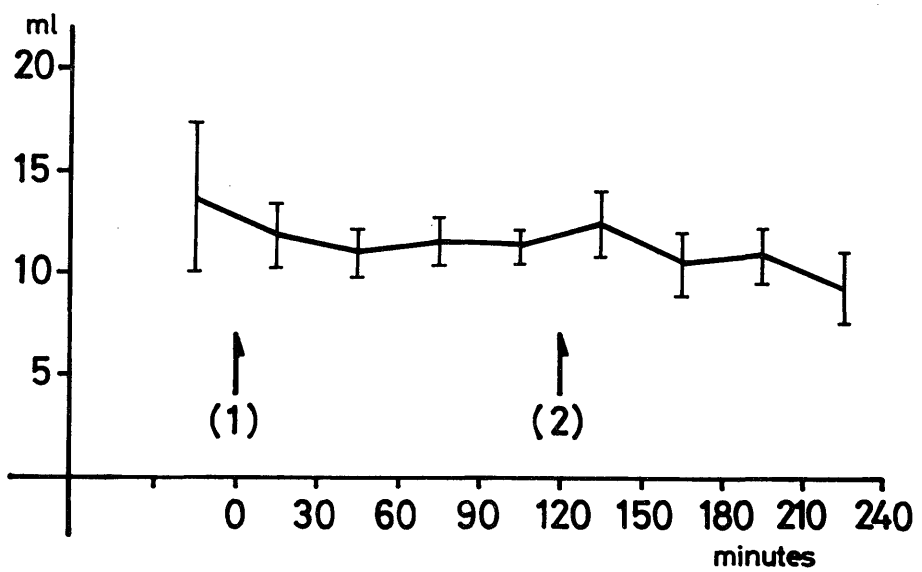


Fig. 4. Volume of lymph flow from thoracic duct. 1 or 0 time represents termination of injection of bile. 2 or 120 minutes represents termination of MD-procedure. The vertical lines for each point represent the range of standard deviation (N=5).

pressure, but then the pressure dropped markedly, from normal level of 120/60 mm.Hg. to 85/40 mm.Hg. in 60 minutes, and further down to 35/10 mm.Hg. in 120 minutes. MD-procedure was performed at this point followed by prompt recovery blood pressure, to 105/40 mm.Hg. in 60 minutes. The portal pressure changed in parallel with the arterial pressure with insignificant difference. It dropped from 105 mm.H_2O to 70mm. H_2O in 120 minutes after bile injection, and it rose back to 115 mm.H_2O in 60 minutes after MD-procedure. The Table 3 illustrates arterial and portal pressure changes on five animals.

Arterial and portal pressure did not change simultaneously with the injection of the bile in the dogs whose pancreas was not fixed surgically to the retroperitoneal space.

3. Exudate from thoracic duct drainage: The amount of exudate from thoracic duct drainage was measured every 30 minutes on the animals subjected to the above experiment. The average amount of exudate before bile injection was 13.7±3.7ml. / 30 minute /10 kg. body weight, and decreased insignificantly after bile injection and MD-procedure (Fig. 4), although the fluid turned hemorrhagic after injection of the bile. In addition, although the exudate from thoracic duct drainage after injection of the bile was injected 0.5ml./kg. into the femoral and portal vein, no change was observed in the arterial and portal pressure.

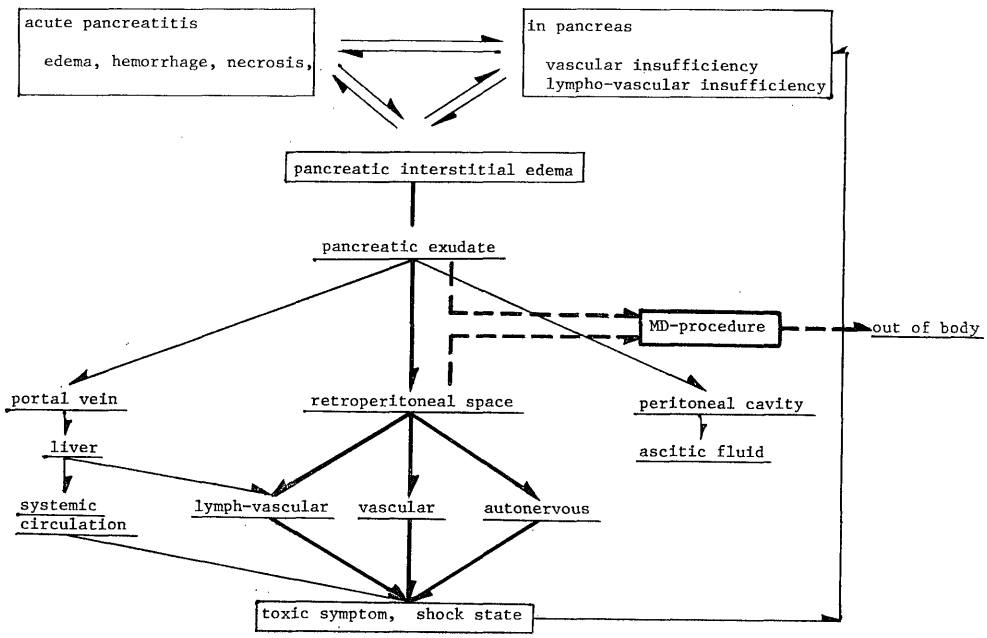
Histological findings of these pancreas resembled to the findings of clinical specimens; interlobular and intercellular edema and cell degeneration in the early stage, and hemorrhage and fat necrosis in the later stage.

DISCUSSION

Acute pancreatitis is a unique disease which differs in many aspects from inflammation of other organs. It is, therefore, important to understand pathophysiology of pancreatitis for the treatment of acute pancreatitis. The basic pathogenesis in acute pancreatitis is autodigestion (autolysis) of the pancreas by trypsin, lipase, phospholipase A and other pancreatic enzymes. This disorder of the pancreas has a few important features : (1) The essential disorder in acute pancreatitis is disturbance of lymph and blood circulation which leads to edema, hemorrhage and necrosis of the pancreas, and these changes again aggravate the original circulation disturbance[1,2,8,11,13]. (2) Toxic substance produced in acute pancreatitis gives damage to other organs and develop intoxication with shock symptoms (*Table* 4). These two developments are not always in correlation.

The authors have experimentally proved that shock in acute pancreatitis developed as the retroperitoneal tissues directly absorbed the pancreatic exudate. Arterial and portal pressure dropped significantly after injection of the pancreatic exudate into the retroperitoneal cavity as compared with intraperitoneal, intravenous and intraportal injections. WHIPLE et al[15] reported also that no toxic substance was contained in the peritoneal exudate

Table 4. The Significance of "MD-procedure" for Acute Pancreatitis.



after massive intravenous and intraperitoneal injection of the pancreatic exudate in his experimental acute hemorrhagic pancreatitis. Although mild transient decline of arterial pressure was observed after intraportal injection of the pancreatic exudate, subsequent vascular insufficiency was not apparent. According to NAKAGAWA[10] there was found a minimal change of arterial and portal pressure following injection of the autogenous bile into the pancreatic duct in the dog without fixation of the pancreas, whereas a significant drop of pressure was observed in the dog whose pancreas was fixed in the retroperitoneal space. GRAY et al[5], RYAN et al[12], and HONJO[6] also reported that shock in the acute pancreatitis was due to retroperitoneal involvement of the disease. On the other hand, POPPER et al[11], MENGUY et al[9], GIDSDORF et al [4] described aggravation of acute pancreatitis to stimulation of the mesentric nerve in the retroperitoneal space.

Surgical treatment of acute pancreatitis in the past consisted of (1) exploratory laparotomy to differentiate it from other acute abdomens and to confirm the type, progress, complication of acute pancreatitis in order to establish adequate therapeutic program, (2) peritoneal drainage to irrigate and remove the pancreatic exudate and the ascitic fluid, and also serve to manage pseudo-cyst of the pancreas, profuse hemorrhage and abscess, and (3) biliary drainage to improve bile stasis and increased biliary duct pressure, and also to reserve back-flow in the pancreatic duct. For the same purpose DOUBILET[3] performed sphincterectomy. (4) Direct approach to the pancreas itself includes longitudinal incision of the pancreas, focal excision of the lesion, and also total pancreatectomy in fulminant pancreatitis as reported by WATTS[14]. These procedure, however, have been criticized

from the viewpoint of surgical risk too high for their therapeutic effects. Laparotomy alone has a high risk in the presence of shock due to severe acute pancreatitis, which is often complicated with dysfunction of the heart, liver, kidney and endocrinal disturbance of the pancreas[7]. On the other hand, one must weigh the fact that some cases of acute pancreatitis died in spite of extensive medical treatment even though a complication is not seen developed.

Having in mind the finding that retroperitoneal involvement of acute pancreatitis was followed by shock, the authors developed the new surgical treatment, namely MD-procedure for acute pancreatitis, and proved its effectiveness clinically as well as experimentally. Acute pancreatitis was induced in experimental dogs, which demonstrated elevation of arterial and portal pressure following MD-procedure. On clinical cases, considerable amount of exudate was drained from the retroperitoneal space. Severe disturbance in the liver, kidney and heart has been prevented or recovered as shown on the improvement of blood pressure and pulse rate as well as normalization of abnormal laboratory findings. As shown in Table 4, the pancreatic exudate was drained to the outside of the body instead of filling the retroperitoneal space, resulting in reduction of the pressure of pancreatic parenchym, thus contributing to relieve lymph stasis with additional possible effect to prevent involvement of other retroperitoneal organs and influence to autonomous nerves. This procedure also allows easy discovery and management of focal hemorrhage and necrosis in the posterior portion of the pancreas. MD-procedure can be performed not only on patients who failed to improve over a 12 hours of conservative medical treatment, but also on poor-risk patients with acute fulminant pancreatitis and its complication, because the technique is simple and mechanical stimulation to the pancreas is minimum.

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