Pancreatography After Recovery from Massive Pancreatic Necrosis

JOHN M. HOWARD, M.D. and STEVEN M. WAGNER, M.D.

From the Departments of Surgery and Medicine, Medical College of Ohio, Toledo, Ohio

Massive retroperitoneal necrosis may follow life-threatening acute pancreatitis. At delayed operation, the surgeon may not be able to delineate dead pancreas from dead adipose tissue. The question arises: has "gloved hand" debridement resulted in pancreatectomy? The histologists report only "necrotic debris, of uncertain origin." To obtain objective data, pancreatography was performed in 13 patients, 10 weeks to 23 months after onset of massive pancreatic necrosis. Each patient had required delayed laparotomy for debridement and external drainage at some earlier stage of their illness. Pancreatography was correlated with the clinical assessment of diabetes and steatorrhea. Except in specific cases involving internal fistulae, pancreatography has not been previously reported in such patients. The results demonstrate that the main pancreatic duct usually maintained its normal length and configuration. Necrosis or stricture of the main duct, if it occurred, was more likely to be followed by diabetes. Steatorrhea was clinically detected in a single patient only. The necrotic tissue, up to several kilograms in wet weight, is largely dead adipose tissue. The pancreas, especially its head, is resistant to necrosis, much more resistant than is the retroperitoneal fat.

AS CONSERVATIVE TREATMENT of acute pancreatitis improves, increasing numbers of patients are surviving attacks that are associated with massive retroperitoneal necrosis. Serial computed tomography (CT) scanning increasingly permits delineation of the natural history of such attacks. Delayed necrosectomy and external drainage, usually occurring 2 months to 1 year after onset of such attacks, led us to believe that necrosectomy sometimes had accomplished a total or near-total pancreatectomy—that is, that the entire pancreas had undergone gangrene and partial liquifaction, allowing "pancreatectomy without resection." Anatomic delineation of the pancreas at laparotomy, under such circumstances, was usually impractical.

Invariably, histologic examination of the debrided tissue revealed only amorphous necrotic tissue, without delineation of its former structure.

Reprint requests and correspondence: Dr. John M. Howard, Medical College of Ohio, C.S. 10008, Toledo, OH 43699.

Submitted for publication: April 6, 1988.

The question arose—had the pancreas sloughed or remained viable in such patients? To a major degree, the answer can be obtained after recovery by anatomic delineation of the pancreatic duct by ERCP. Clinical assessment of pancreatic function, although helpful, has been less specific.

Clinical Observations

Thirteen patients were studied after severe acute pancreatitis (Table 1). Twelve patients were studied by pancreatography after recovery, one at autopsy after massive necrosis and multiple debridements.

Each of the patients had experienced life-threatening acute pancreatitis. Each had been hemodynamically unstable, requiring prolonged stay in the intensive care unit (mean of 13.5 days), respiratory support for a minimum of 3 days (twelve patients), and intravenous (I.V.) hyperalimentation. The Ranson index of severity[1] was quite high, as indicated in Table 1. Each patient had been initially treated without surgery, usually for a minimum of 2 months, until sepsis or other complications necessitated necrosectomy and external drainage. The initial surgical exploration was usually achieved transabdominally; subsequent explorations retroperitoneally. The lapse of several months after onset resulted in rather clear delineation of the necrotic retroperitoneal tissue. Necrosectomy could usually be achieved by simply lifting the dead tissue out without the use of cutting instruments. The dead tissue, 35–4000 mg (mean of 640 mg per operation) in wet weight, was bathed in encapsulated fluid in amounts varying between 250 ml to 6 l. The surgeon could often identify extension of the necrotic tissue into the mesocolon or into the mesentery of the small intestine. To reiterate,

TABLE 1. *Clinical Correlation: Subsequent Findings*

Patient	Age (years)/ Sex	Etiology Pancreatitis	Index of Severity[1]	Lapse of Time Before Pancreatogram	Findings of Main Pancreatic Duct	Diabetes	Steatorrhea
1	61/F	Gallstones		11 mos. after last operation 21 mos. after onset	Normal ERCP	No	No
2	54/M	Alcohol (?)	7	15 mos.	Slight dilatation, foreshortened? (ERCP)	60 units insulin/day	Yes
3	36/F	Common duct stones	6	19 mos.	ERCP, pancreatic duct obstructed 3 cm from ampulla	32 units insulin/day	No
4	66/F	Prior cholecystectomy for gallstones	5	5 mos.	Normal ERCP	No	Unknown
5	42/F	Indeterminate	5	10 mos.	Normal ERCP	No	No
6	70/M	Indeterminate	6	7 mos.	Normal ERCP	±	No
7	41/F	Hyperlipidemia Gallstones	7	4 mos.	Normal ERCP	60 units insulin/day	No
8	38/F	Gallstones	5	5 mos.	Stenosis of duct in tail of pancreas with small cystic cavity	No	No
9	38/M	Gallstones		10 mos.	External fistula at body of pancreas Obstruction of duct toward ampulla	Insulin dependence developed several years later	Mild
10	36/M	Alcohol	6	1 year	Normal ERCP	No	No
11	80/F	Indeterminate	5	23 mos.	Normal ERCP	No	No
12	68/F	Postoperative	5	10 weeks	Normal ERCP	No	No
13	77/M	Common duct stone	6	Pancreatic duct inspected at autopsy, 5 months after onset	Pancreatic head viable with patent and intact ducts. Slough of distal pancreas		

within the limited exposure of the operation and the restrictions imposed by inflammation and the necessity of avoiding hemorrhage, anatomic identification of the pancreas, *per se*, was usually not achieved.

Each patient required necrosectomy, with ten of the 13 patients requiring necrosectomy more than once.

Diabetes was determined by repetitive measurement of fasting and postprandial blood glucose levels after recovery. Steatorrhea was determined by clinical assessment only, but when suspected, was confirmed by measurement of 72-hour fecal fat without special dietary restrictions.

Results

The magnitude of necrosis is shown in Figure 1. In this patient, necrosectomy was performed three times, and each operation seemed compatible with total necrosis and total removal of the pancreas. ERCP, 11 months after recovery (Fig. 2), revealed the outline of an apparently normal pancreatic duct. Figure 3 reflects a similar sequence of events. Thus, continuity of the main pancreatic duct was found in all but three, or possibly four, of the patients (Table 1).

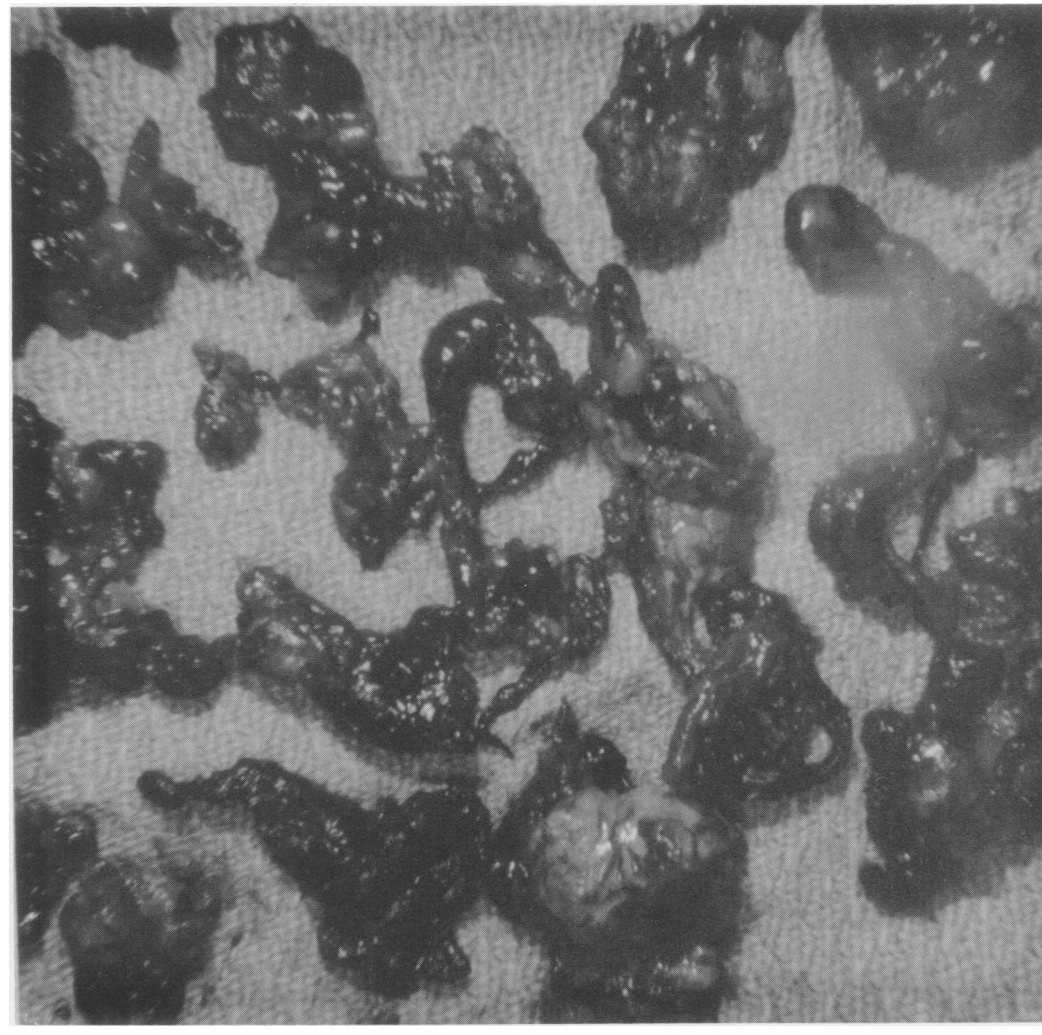


FIG. 1. Photograph of tissue removed by "pancreatic necrosectomy" after severe acute pancreatitis. The surgeon believed that a total or near-total "gloved-hand" pancreatectomy had been performed. Three comparable necrosectomies were performed.

In another patient in whom the CT scan had shown evidence of massive necrosis (Figs. 4A and B), the pancreatic duct appeared to be fibrotic, mildly dilated and, perhaps, foreshortened (Fig. 5). Two patients developed transient external pancreatic fistulae after necrosectomy and external drainage.

There appeared to be a rough tendency for impairment of pancreatic function to parallel the degree of destruction of the ductal system. Thus, of the four patients currently requiring insulin, two and possibly three had abnormal

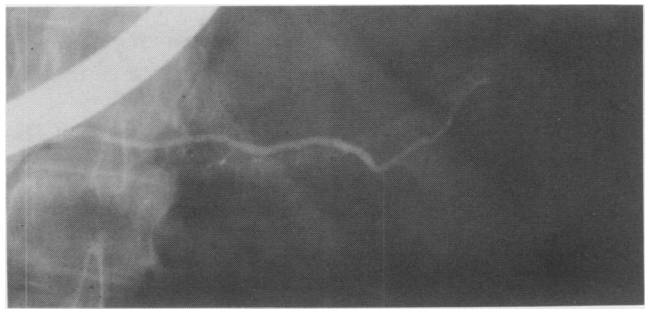


FIG. 2. Normal pancreatogram (ERCP) 11 months after necrosectomy and external drainage (necrotic tissues shown in Figure 1).

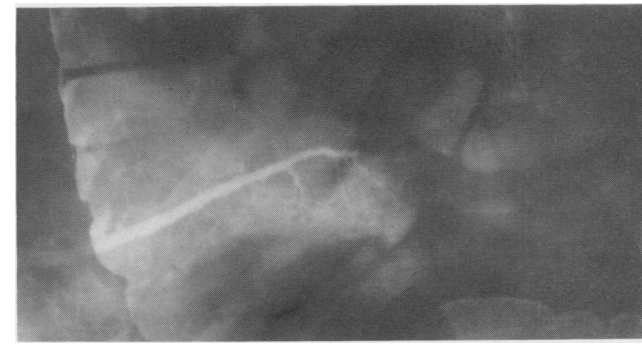
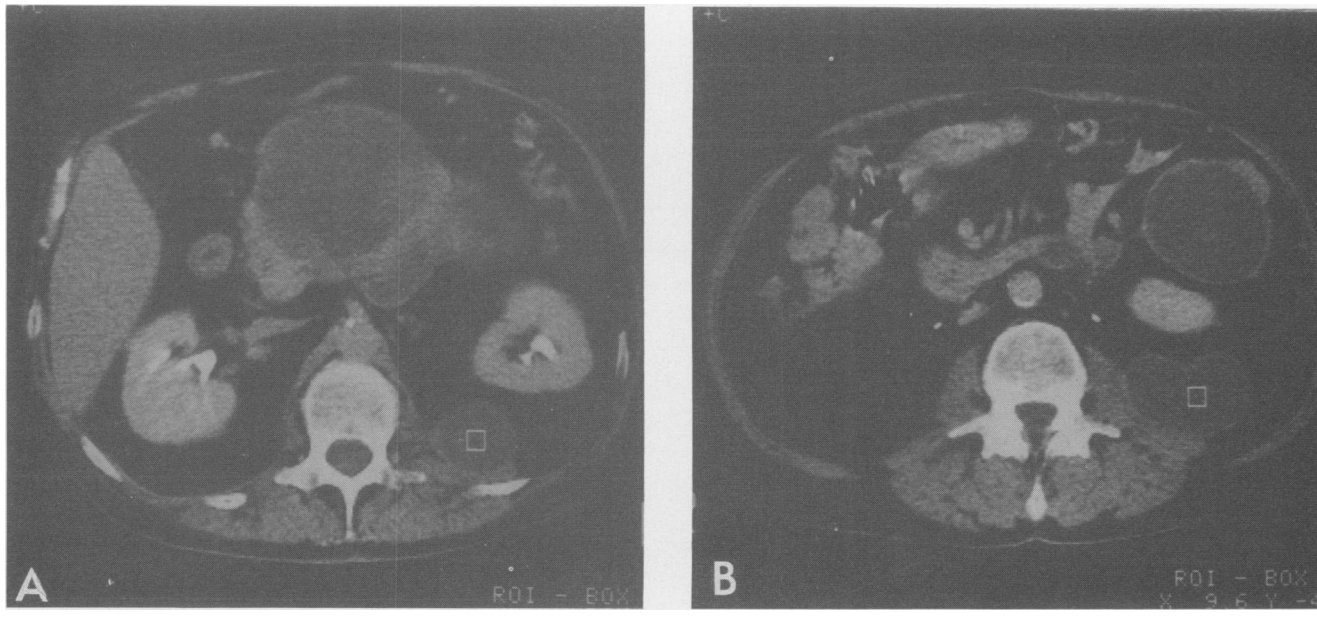


FIG. 3. Normal retrograde pancreatogram, 10 months after onset of massive pancreatic and retropancreatic necrosis. Two operations were required for adequate necrosectomy and external drainage.



FIGS. 4A and B. CT scans of a patient showing large collections of fluid and necrotic tissue in the areas of the body and tail of the pancreas, as well as adjacent to the left kidney.

pancreatograms. Only one patient requires exocrine pancreatic supplementation, and this patient had an abnormal pancreatogram.

Discussion

Correlation of the operative findings with those of delayed pancreatography clearly reveals that, under the conditions described, the surgeon may not be able to delineate necrotic pancreas from retroperitoneal necrotic adipose tissue. In several instances, follow-up pancreatography revealed a normal or near normal anatomic outline of the pancreas after the surgeon's operative note had recorded the sloughing of the entire gland. This observation is in keeping with the observations of Leger[2] and Testas,[3] who independently observed that the necrosis of such an acutely inflamed pancreas, when resected early in the course of the attack, was often superficial, effecting only the surface of the gland to a depth of a few millimeters. This limitation in the depth of pancreatic necrosis may explain the relative adequacy of the endocrine and exocrine function of the gland after recovery.

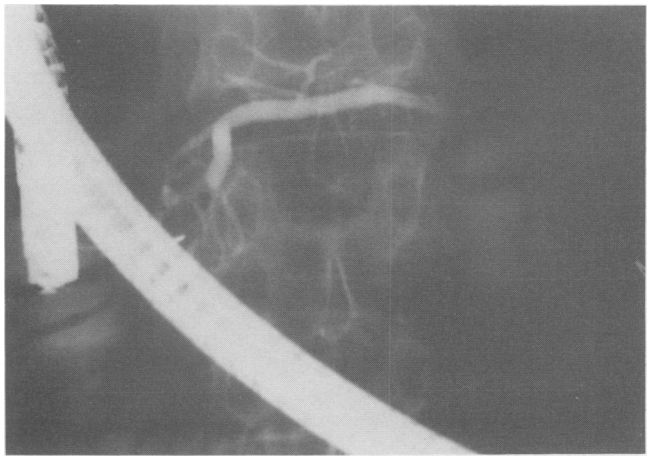


FIG. 5. Retrograde pancreatogram 18 months after massive pancreatic necrosis shown in Figure 4. The duct may have been foreshortened by necrosis or fibrosis. No external fistula followed necrosectomy and external drainage.

In a recent report of 31 patients with massive necrosis of the pancreatic and/or peripancreatic tissues and in whom delayed necrosectomy and external drainage were required, an external pancreatic fistula was found to have transiently developed in eight patients.[4] Even though the retroperitoneal fluid bathing the necrotic tissue characteristically had a high amylase content, communication of the peripancreatic cavity with the pancreatic ductal system, over the course of the natural history of the disease, was seldom demonstrable.

In a 4-year follow-up of patients with necrohemorrhagic pancreatitis treated conservatively, Angelini and associate[5] rarely found overt diabetes—an indirect indication that the anatomic and functional integrity of the gland had not been entirely destroyed.

McLatchie,[6] Darras,[7] and Satake[8] noted a useful role of ERCP in outlining internal pancreatic fistulae resulting

from acute pancreatitis, yet a systemic study of pancreatography in patients surviving massive necrosis apparently has not been previously performed.

Conclusions

The studies indicate that, in patients with life-threatening acute pancreatitis, the massive amount of necrotic tissue found retroperitoneally consists mainly of necrotic adipose tissue. The pancreas usually remained viable. If the pancreas undergoes full-thickness necrosis and slough, the body or the tail of the gland, not the head, appears to be the most vulnerable.

References

1. Ranson JHC, Rifkind KM, Turner JW. Prognostic signs and nonoperative peritoneal lavage in acute pancreatitis. Surg Gynecol Obstet 1976; 143:209–219,.
2. Leger L, Chicke B, Louvel A. Necrosis in acute pancreatitis: surgical and anatomopathological comparisons. Seven cases. Nouv Press Med 1977; 6:337–340.
3. Testas P. Presented at the meeting of the Royal College of Surgeons of Edinburgh, Edinburgh, Scotland, May 28, 1987.
4. Howard John M. Delayed debridement (necrosectomy) and external drainage of the massive pancreatic or peripancreatic necrosis. Surg Gynecol Obstet (in press).
5. Angelini G, Pederzoli P, Caliari S, et al. Long-term outcome of acute necrohemorrhagic pancreatitis: a four-year follow-up. Digestion 1984: 30:131–137.
6. McLatchie GR, Meek D, Imrie CW. The use of endoscopic retrograde choledochopancreatography (ERCP) in the diagnosis of internal distulae complicating severe acute pancreatitis. Br J Radiol 1985; 58:395–397.
7. Darras T, Gulbis A, Khazzaka E, et al. Endoscopic retrograde pancreatography in the diagnosis of pancreatico-pleural fistulae. J Belge de Radiologie 1977; 60:423–426.
8. Satake K, Cho K, Sowa M. Demonstration of a pancreatic fistula by ERCP in a patient with chronic pleural effusion. Am J Surg 1978; 136:390–392.