ACUTE PANCREATITIS, DIAGNOSIS AND TREATMENT*

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ACUTE PANCREATITIS remains a disease of serious implications whose diagnosis is frequently missed and whose proper treatment is a subject of discussion and controversy.

Mortality rates in acute pancreatitis vary because of differences in the proportions of severe cases in the various series reported. Thus statistical data on the incidence, morbidity, and mortality of the disease has not been established at a reasonably accurate level. In the series of cases that have been reported the mortality rate has varied between 20 and 50 per cent.[1] However, these reports refer commonly to series with many severe cases in which the diagnosis is based upon demonstration of the lesion at operation or at autopsy. Elman[2, 3] made classical contributions to the making of a preoperative or antemortem diagnosis of milder and of severe cases. With a larger number of mild cases being discovered, much lower mortality rates have been reported.

Difficulties in diagnosis arise because pancreatitis can produce a variety of clinical pictures which closely imitate other intra-abdominal diseases. Variations in the clinical picture may or may not correspond to the different degrees of pathologic change in the pancreas, which vary from mild edema to extensive hemorrhage and necrosis. Once the condition is initiated, it may progress from the mildest to the most destructive changes, with the possibility of cessation of progress at any stage of pancreatic involvement.

The place of surgery in treatment is still not agreed upon. For some years there has been a trend away from operative treatment during the acute phase of the disease, but recently there have been advocates for operative treatment.[4, 5]

We wish to present data on 50 patients in whom a diagnosis of acute pancreatitis was made at Cook County Hospital over a period of four years. We have been impressed with: (1) the difficulties in the clinical diagnosis encountered by the house staff; (2) the inadequacy of surgical therapy in altering the progress of the condition in most instances, and (3) the failure to institute promptly enough a regimen of therapy designed to relieve the patient of the progressive pathologic changes which, when uncorrected may prove fatal.

DIAGNOSIS

Table I shows the distribution of the 50 patients according to age, sex, race and the mortality in each group. The largest number were in the age range between 30 and 50 years. In one reported series,[6] however, the patients were in an older age group, while in another series[7] a nine-year-old

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patient was explored and acute pancreatitis was found.

TABLE I.—*Relation of Age, Sex, and Race to Mortality of Acute Pancreatitis.*

Age	Number	Deaths	Sex		Deaths
20–29	6	1	Male	26	9
30–39	15	6	Female	24	8
40–49	13	5		—	—
50–59	8	3		50	17
60–69	5	0			
70–79	2	1	Race		Deaths
80–over	1	1	White	34	13
	—	—	Colored	16	4
	50	17		—	—
				50	17

Table II indicates the duration of the illness before admission to the hospital. The mortality rate rises with delay in treatment. Delay in seeking treatment is frequently the cause for failure to recognize the condition, for delay diminishes the value of the amylase test in establishing a correct diagnosis.

TABLE II.—*Duration of Illness before Admission to Hospital.*

	Number	Deaths	
Under 24 hours	16	3	18.7%
24–38 hrs.	9	2	22.0%
48–72 hrs.	1	0	0
Over 3 days	15	9	60. %
Unspecified	9	3	33.3%
	—	—	
	50	17	

Table III indicates that abdominal pain and vomiting are a basis for suspecting acute pancreatitis. In one-third of the patients the symptoms arose following a heavy meal or the use of alcohol, while in one-half the patients there was a history of a previous similar episode. Physical findings require careful evaluation, since they also commonly fit to a lesser or greater degree several of the acute abdominal conditions more commonly encountered. Rigidity and rebound tenderness are most marked in the epigastrium, but the board-like rigidity of the perforated ulcer is usually not present.

The absence of bowel sounds and presence of distention were noted in 19 and 17 patients respectively. These signs simulate

TABLE III.—*Symptoms and Signs in Acute Pancreatitis.*

A. Symptoms	Number
1. Abdominal pain	50 (100%)
2. Vomiting	39
3. Back pain	16
4. Onset after heavy meal	12
5. Obstipation over 24 hrs.	11
6. Fainting or collapse	7
7. Onset following alcohol	4
8. Diarrhea	3
9. Melena	1
10. Onset after trauma	1
11. Previous attacks	26
B. Signs	**Number**
1. Rigidity	27 (54%)
2. Rebound tenderness	26
3. Temp. above 100 on adm.	23
4. Absent bowel sounds	19
5. Distention	17
6. Jaundice	7
7. Blood pressure below 90	3
8. Grey-Turner sign	1

peritonitis or late intestinal obstruction. Both of these conditions actually occur as a result of pancreatitis. Fat necrosis in the mesentery and in the subperitoneal fat about the pancreas with the accumulation of hemorrhagic peritoneal fluid are responsible for the picture of generalized peritonitis. The enlarged head of the pancreas may mechanically obstruct the duodenum, but more commonly the local peritonitis about the pancreas, with bathing of the upper loops of the jejunum in the peritoneal fluid, results in local ileus and accounts for the picture of small bowel obstruction. The absence of an obvious cause for obstruction with tenderness in the flanks should make one suspicious of acute pancreatitis.

Jaundice was noted in seven patients and accounts for the incrimination of the disease of the biliary passages as the primary condition. Since the common duct is intimately related to the head of the pancreas and may be occluded by swelling of the latter, jaundice in the presence of acute abdominal symptoms warrants a suspicion of pancreatitis as a cause.

TABLE IV.—*Laboratory Findings in Acute Pancreatitis.*

1. Amylase Determinations (19 patients)		
Amylase	Under 48 Hrs.	Over 48 Hrs.
8 units	1	0
16	3	1
24	0	1
32	4	5
48	0	2
64	1	3
96	3	0
128	4	0
200	1	0
256	1	1
512	1	0
32 and below	8	7
Above 32	11	6
58% elevated in 1st 48 hrs. (11 in 19)		
46% elevated after 48 hours (6 in 13)		
2. Icterus Index (15 patients)		
7 or below		0
7–15		5
15–20		2
20–40		4
40–60		2
60–100		1
Over 100		1
Elevated in all cases examined (30% of the total group)		
3. White Blood Count (50 patients)		
		Deaths
Below 10,000	5	2
10,000 to 20,000	30	6
Over 20,000	9	5
Highest 45,000	1	1
4. Urine Examination (50 patients)		
Glycosuria	5 (3 of these died)	
Albuminuria	11 (4 of these died)	
5. X-Ray—Flat Plate Abdomen		33
Distended loops of bowel		11
Small bowel		6
Large bowel		2
Large and small		3
Evidence of fluid (haziness)		2

A clinical picture of shock with low blood pressure is noted only in the severe cases with extensive pancreatic destruction. These usually run a rapidly fatal course. While the severe cases occur least commonly, they have been accorded the greatest concern in diagnosis and frequently constitute the only classical description of this disease. Local discoloration of the abdominal wall, as described by Grey-Turner[8] is usually a late finding in severe cases. This sign was present in only one case in our series.

Table IV shows the laboratory studies made in this group of patients. The concentration of serum amylase is usually increased, but extensive destruction of the pancreas may prevent the formation of amylase or lipase. Usually in one to four days, normal values return following subsidence of the acute process. Serum lipase may remain increased for a longer period of time than does the serum amylase. Hence, in patients admitted late in the course of the acute disease, evidence of the nature of the condition may be determined by an elevated serum lipase even though the amylase level has returned to normal. In this series the lipase determinations were not done, and the infrequent use of the amylase determination played a role in the failure to diagnose the condition more frequently.

The icteric index was elevated in all of the 15 patients in whom this examination was made. While obstruction to the common duct due to edema of the head of the pancreas is the most likely cause, an associated hepatitis must also be considered.

Albuminuria was found in 11 patients. Four of these died. Glycosuria was found in five patients, and three of these died. Thus albuminuria and glycosuria must be regarded as evidence of severe disease.

The calcium level in the serum has been studied by Edmonson and Berne[9] and by others in acute pancreatitis. The normal level of serum calcium is about 10 mg. per 100 cc. A drop below this level has been noted in acute pancreatitis. As the level approaches 7 mg. per 100 cc. the condition is said to be extensive and may be fatal. Gambill *et al.*[10] found serum calcium levels of 5.1 to 5.9 in a fatal case. It is not known what changes occur in the bound and ionizable calcium, but the loss of calcium in the formation of the areas of fat necrosis is obvious.[11]

Roentgen ray studies of the abdomen in acute pancreatitis are of value in ruling out other conditions. The findings of calcification in the region of the pancreas may be a

TABLE V.—*Clinical Diagnosis, Type of Treatment and Mortality*

Clinical Diagnosis	Total	Surg.	Died	Med.	Died
1. Acute pancreatitis	20	1	1	19	3
2. Perforated peptic ulcer	7	6	2	1	0
3. Bowel obstruction	5	4	2	1	1
4. Acute appendicitis	4	3	0	1	1
5. Acute cholecystitis	4	3	1	1*	0
6. Acute abdomen	3	2	2	1†	1
7. Obstructive jaundice	2	2	1	0	0
8. Peritonitis	1	0	0	1†	1
9. Abdominal abscess (Case 3)	1	1	1	0	0
10. Ruptured spleen	1	1	0	0	0
11. Ca of stomach (original diag. intest. obst.) (Case 1)	1	0	0	1*	0
12. Penetrating peptic ulcer (Case 4)	1	1	0	0	0
	50	24	10	26	7

* Late operation.
† Terminal on admission.

TABLE VI.—*Errors in Diagnosis in Acute Pancreatitis.*

Group	Total	Surg.	Died	Med.	Died
1. Severe "acute abdomen"					
Table V-6	3	2	2	1	1
-8	1	0	0	1	1
-4	1	0	0	1	1
All 5 in this group died					
2. Disease of the biliary tract					
Table V-5	4	3	1	1	0
-7	2	2	1	0	0
Five of the six subjected to surgery. Two died					
3. Intestinal obstruction					
Table V-3	5	4	2	1	1
Three of the five died					
4. Acute peritonitis					
Table V-4 (acute appendicitis)	3	3	0	0	0
-2 (Perf. pep. ulc.)	6	6	2	0	0
-2 (Forme fruste)	1	0	0	1	0
5. Abdominal mass					
Table V- 9 (Abdom. abscess)	1	1	1	0	0
-10 (Rup. spleen)	1	1	0	0	0
-11 (Ca of stomach)	1	0	0	1	0

clue to the diagnosis in the recurrent case.[12] A flat plate of the abdomen may show a bowel pattern suggestive of intestinal obstruction (Table IV). It must be appreciated that bowel obstruction or ileus may be an accompaniment of acute pancreatitis, and subsidence of this phenomenon usually occurs with subsidence of the acute process.

The clinical diagnoses are indicated in Table V. From the variety of incorrect diagnoses several groups are discernible (Table VI). It is noteworthy that the clinical diagnosis of acute pancreatitis was not made in 30 of the 50 patients. Either surgery or autopsy disclosed the correct diagnosis in this group. This should call attention to the importance of the direct diagnosis of the conditions which simulate acute pancreatitis sufficiently to make the clinical error possible. Table VI closely parallels the classification of Paxton and Payne,[13] who have made extensive studies of the material at the Los Angeles County Hospital.

As our data indicate, the four acute abdominal conditions most frequently imitated by acute pancreatitis are mechanical bowel obstruction, biliary tract disease, peritonitis, and penetrating or perforated peptic ulcer. It is of utmost importance for

the physician to maintain a high index of suspicion for pancreatitis and to obtain a serum amylase determination as early as possible in the course of any acute abdominal disease.

Differentiation of acute pancreatitis from bowel obstruction can be difficult because of the severe ileus which often accompanies pancreatitis with abdominal distention, vomiting, obstipation, and distended loops of bowel shown by roentgen ray. An onset with cramping pain and the presence of obstructive bowel sounds are indicative of bowel obstruction rather than pancreatitis. A history of previous attacks is more suggestive of pancreatitis than of bowel obstruction, such a history having been elicited in over 50 per cent of our cases (Table III). Abdominal tenderness is usually more pronounced in acute pancreatitis than in a non-strangulated bowel obstruction. In a strangulated bowel, obstruction tenderness is more localized and often lower in the abdomen than in acute pancreatitis. The following history illustrates a case of acute pancreatitis in which a diagnosis of mechanical small bowel obstruction was made. Some of the points which should have suggested the correct diagnosis are placed in italics.

Case 1.—B. M., a white female, age 68, was admitted January 15, 1948, because of *steady pain* in the upper abdomen and repeated vomiting for 15 hours. Peri-umbilical epigastric pain began about one hour after supper. There had been increasing abdominal distention and no passage of gas or feces by rectum during this time. *There had been an attack of epigastric pain 2 weeks previously, coming on after a heavy meal* and lasting only a few hours.

Physical examination showed a moderately obese woman having severe abdominal pain. Her temperature was 99.8 orally, respirations 40, and blood pressure 130/72. The heart and lungs were normal. The abdomen was moderately distended. There was generalized abdominal tenderness, most marked in the epigastrium. Bowel sounds were absent. Pelvic examination showed moderate tenderness in the posterior cul-de-sac and in both fornices, and also on movement of the cervix.

Laboratory studies showed a hemoglobin of 100 per cent, white blood count of 16,000, and a urine negative except for 3 plus albumin. The serum amylase on the day of admission was 82 units. A flat plate of the abdomen showed a moderate amount of *gas in both the large and small bowel.*

By the second hospital day the patient's distention had become marked in spite of continued gastric suction. The temperature remained around 99. No bowel sounds were heard, and obstipation persisted. A barium enema showed the entire colon to fill well. The Medical Service felt that the patient probably had a mechanical bowel obstruction, but the surgical consultant felt that pancreatitis was the most likely diagnosis and advised continued conservative treatment. The patient's condition remained essentially unchanged until the fifth hospital day, at which time gradual improvement began. Following subsidence of the acute symptoms a gastro-intestinal roentgen ray examination was done. A filling defect was seen in the pre-pyloric region of the stomach. A gastroscopic examination was negative. Because of the barium meal findings and some continued epigastric discomfort, an exploratory operation was performed on the 37th hospital day. The stomach and duodenum were found to be normal. The gallbladder and bile ducts were likewise normal. The pancreas was firm and moderately, diffusely enlarged, and fat necrosis was noted still present in the greater omentum. The operative diagnosis was subsiding acute pancreatitis. The patient made an uneventful recovery.

Acute pancreatitis may be mistakenly diagnosed as biliary tract disease. The type and location of pain may be similar in the two conditions, and the occurrence of jaundice in acute pancreatitis is not uncommon. Jaundice was noted in seven of our 50 cases of acute pancreatitis, and an elevated icteric index was present in all of the 15 cases in which this test was made. The frequent simultaneous occurrence of pancreatitis and gallbladder disease may also lead to confusion in diagnosis at times. The following case history illustrates a marked degree of jaundice occurring in acute pancreatitis, leading to an erroneous diagnosis. Italics call attention to points which should have aroused a suspicion of the existence of acute pancreatitis.

Case 2.—E. R., a white female, age 25, was admitted on September 14, 1947, because of colicky *epigastric pain* of 5 days duration. Soon after the onset a chill with a rise in temperature to 101, accompanied by vomiting, had occurred. Three days before admission the patient noted that her urine was very dark, and one day before admission she noticed her skin becoming yellow. Fifteen months previously the patient had experienced *several recurring attacks of epigastric colic after large meals.*

Physical examination revealed a thin, acutely ill, jaundiced woman having paroxysms of abdominal pain. The temperature was 101 orally, pulse 112, respirations 22, and blood pressure 150/90. The abdomen was flat and was *diffusely tender in the upper half* with marked tenderness in the epigastrium slightly to the right of the mid-line. Bowel sounds were infrequent.

Laboratory studies showed a hemoglobin of 75 per cent and white blood count of 14,200. The urine was negative except for a 2 plus albumin and 4 plus bile. There was no uribilinogen in the urine. The stool examination was negative, including a benzidine test for blood. A flat plate of the abdomen revealed no abnormalities.

Improvement began soon after admission, and by the third day the temperature was normal, and the abdominal pain and jaundice were subsiding. However, on the sixth hospital day, after eating ice cream, the patient had a recurrence of colicky pain and on the next day had more pain and vomiting. By the tenth day the patient was having severe, *steady epigastric pain* and tenderness and *pain in the upper lumbar region of the back.* The temperature rose to 100.8 orally. The icteric index increased to 46; the white blood count was 16,400 and the serum amylase 16 units. The patient's condition remained unchanged, and on the twelfth hospital day an exploratory laparotomy was done. The pancreas was found to be diffusely enlarged and nodular. The gallbladder was thin-walled and contained no stones. The common bile duct was thin-walled and moderately distended. The liver showed excess bile pigmentation. The remainder of the abdominal exploration was negative. The abdomen was closed after exploration without any other procedure being done. The patient had a smooth postoperative course, and by the second postoperative day her condition was very good. The jaundice cleared, and the patient was discharged on the eighth postoperative day.

Acute generalized peritonitis can also easily be confused with acute pancreatitis. Again, the history is helpful, especially if previous attacks suggestive of pancreatitis have occurred. In peritonitis the history is often indicative of a preceding disease, such as acute appendicitis. The fever is usually higher in peritonitis. The following history is that of a patient with acute pancreatitis in which a diagnosis of peritonitis with subsequent abscess formation was made. Italics indicate points suggestive of the correct diagnosis.

Case 3.—M. G., a white female, age 23, was admitted on October 28, 1945, because of *pain in the epigastrium* without radiation. She had not vomited. There had been increasing abdominal distention.

Examination showed an obese woman in moderate abdominal pain with a temperature of 101 degrees and a pulse of 120. The abdomen was markedly distended, and there was *moderate epigastric tenderness without muscle spasm or rebound tenderness.* Bowel sounds were present. The liver was enlarged to 10 cm. below the costal margin.

The urine showed 2 plus sugar and 4 plus acetone, although the patient did not have a previous history of diabetes. A glucose tolerance test showed a typical diabetic response. A roentgen ray examination of the gastro-intestinal tract was negative. The serum amylase test was not done.

The patient continued to run a febrile course with elevations to 102 while under penicillin therapy. On the tenth hospital day the patient complained of pain in the left lower quadrant where tenderness and a mass were noted. A muscle splitting incision was made in the left lower quadrant and a large retroperitoneal abscess containing much foul pus and necrotic material was drained. A piece of necrotic tissue was sent to the pathologist and was reported as showing necrotic tissue suggestive of pancreatic ghost acini. The postoperative course was progressively downward, and the patient died 2 months following the operation. At autopsy there was found pancreatic necrosis, abscess formation with extension of the abscess along the descending colon, and perforation through the left side of the diaphragm.

In this case earlier drainage of the retroperitoneal accumulation of fluid would probably have been of benefit to the patient, although death might still have resulted from the extensive pancreatic necrosis.

Persisting or recurring pain in the epigastrium and back occurring in pancreatitis can lead to a mistaken diagnosis of a penetrating or intractable ulcer and may lead to the scheduling of the patient for a gastric resection, as happened in the following case. Findings which should have suggested the correct diagnosis are in italics.

Case 4.—H. K., a white male, age 44, was admitted on May 3, 1948, because of severe, steady epigastric and back pain of 4 weeks duration. The pain was almost constantly present and was frequently very severe. *It was increased by eating food or drinking alcohol.* There had been occasional vomiting after meals. *Milk and alkalis gave little or no relief from the pain.* For 2½ years the patient had been having similar attacks of pain, lasting from several days to a week or more. Following a barium meal examination he was told that he had a duodenal ulcer. He was placed on an ulcer diet and given alkalis. *One year before admission he had a severe attack of epigastric pain with distention and vomiting* for which he was hospitalized with a diagnosis of peritonitis.

Physical examination was essentially negative except for slight tenderness in the epigastrium. The temperature was normal. The hemoglobin was 78 per cent, the white blood count 9,200, and the urine negative. The stool was benzidine negative. Barium meal roentgen ray studies showed a slight duodenal deformity suggestive of peptic ulcer. The preoperative diagnosis was duodenal ulcer with intractable symptoms, and operation was performed on the twelfth hospital day. The stomach and duodenum were found to be normal, with no evidence of old or recent ulcer. The gallbladder and bile ducts appeared normal. The pancreas was enlarged, hard, and rather nodular. The appendix was removed and the abdomen closed without drainage. The operative diagnosis was recurrent acute pancreatitis.

TREATMENT

For many years it has been recognized that surgical treatment for acute pancreatitis has not always played a role in the recovery of the patient. While in some instances decompresion of a distended biliary system or drainage of a retroperitoneal collection of fluid or of an abscess or pseudocyst have aided recovery, in more instances the addition of surgical trauma with delay in the institution of effective medical therapy has made recovery more difficult and in some instances impossible.

TABLE VII.—*Fatal Cases: Time of Death in Relation to Onset of Treatment.*

Following Surgery	Deaths	No Surgery Done
2	Under 24 hrs.	1 (Autopsy)
2	Between 24 and 48 hrs.	
1	3 days	1 (Autopsy)
1	4 days	1 (Amylase 67)
1	5 days	1 (Amylase 96)
	7 days	1 (Previous surgery)
1	8 days	
1	2 weeks	
	3 weeks	1 (Autopsy)
	4 weeks	1 (Autopsy)
1	7 weeks	
10	Total	7

TABLE VIII.—*Findings at Abdominal Exploration.*

Fat necrosis	15 (62.5%)
Enlarged firm pancreas	15 (62.5%)
Hard nodules in pancreas	1
Soft boggy pancreas	1
Hemorrhage in pancreas	1
Pseudo cyst	1
Retro peritoneal abscess	1
Hemorrhage in mesentery	1
Biliary calculi	6 (25.0%)
Clear fluid	4 (16.6%)
Cloudy fluid	1
Bloody fluid	10 (41.6%)

In our series of 50 cases of acute pancreatitis, operation was performed in 24 with ten deaths, a mortality of 41.6 per cent. Non-operative treatment was used in 26 cases with seven deaths, a mortality of 26.9 per cent. Among the 19 patients in whom the correct diagnosis of pancreatitis was made on admission and nonoperative treatment employed, three died, a mortality of 15.6 per cent. Table VII indicated the duration of survival in those who died following surgery and in those who died without surgical intervention.

The findings at the time of exploration are noted in Table VIII, and the procedures carried out are listed in Table IX. In 12 of the patients operated upon the abdomen

was closed without drainage after the correct diagnosis was disclosed, and in this group there were four deaths, a mortality of 33 per cent. In the remaining 14 patients operated upon, in whom various procedures were carried out at the site of the pancreas or the biliary tract, there were six deaths, a mortality of 42.8 per cent.

TABLE IX.—*Procedures in 26* Patients Subjected to Surgery.*

	Number	Deaths
1. Closed without drainage..... Appendectomy 1	12	4
2. Drainage of peritoneal cavity	3	1
3. Cholecystostomy.....	5	2
4. Choledochostomy.....	2	1
5. Cholecystostomy plus choledochostomy.....	1	0
6. Cholecysto-jejunostomy.....	1	1
7. Abscess drained.....	1	1
8. Pseudo cyst drained.....	1	0
	26	10

* Two of these were operated upon late, one with a diagnosis of carcinoma of the stomach, the other with a diagnosis of subsiding acute cholecystitis.

Thus in our experience, abdominal exploration increased the mortality from acute pancreatitis, and the addition of other procedures in addition to exploration further increased the mortality.

There were nine patients with pancreatitis in whom decompression of the biliary passages was done, with four deaths. In six patients cholecystostomy was done; in one of these a choledochostomy was also done. In two others choledochostomy alone was done. In some of these there was distention in the bile ducts, but in only three were stones found. Three of these patients died. In one other patient a cholecysto-jejunostomy was done. This patient was jaundiced, had stones in the gallbladder, and died seven weeks after operation. In this group it is difficult to determine what course the disease might have followed had not decompression of the biliary passages been done.

Drainage of an abscess was done in one patient who did not recover. A pseudocyst was drained in another patient who recovered.

Treatment of acute pancreatitis is based on three requirements: (1) Decrease the activities of the pancreas and place the organ at rest; (2) restore to normal the altered changes in the body brought about by vomiting, inability to take food and water, and loss of fluid rich in proteins into the peritoneal cavity with a shrinkage of the blood volume, and loss of minerals; and (3) prevent or correct the severe ileus and the resulting accumulation of gas in the bowel which often occurs.

Papaverine, atropine, and sedatives are used in an attempt to reduce the secretory activities of the pancreas. Papaverine is given in doses of 1.5 gr. intramuscularly at three- to four-hour intervals to relax smooth muscle spasm in the sphincter of Oddi and permit drainage of pancreatic juice from the pancreatic duct and bile from the common duct, and thus reduce tension within the pancreas.

Atropine in doses of 0.01 gr. at three- to four-hour intervals is used to decrease vagal activity in its stimulation of pancreatic secretion. Sodium phenobarbital is given in doses of 2 gr. intramuscularly as often as is necessary to quiet the patient.

The use of whole blood and plasma is important in the treatment of severe cases to combat shrinkage of blood volume due to loss of fluids rich in proteins into the peritoneal cavity. If prolonged nasal suction is required, the use of protein hydrolysate may help to spare body proteins.

Dextrose in 5 per cent solution is used in sufficient amount to maintain a 24-hour urine output of about 1000 cc. with a specific gravity between 1.010 and 1.020. Sodium chloride is given in an amount sufficient to replace that lost through stomach suction. The use of a 0.45 per cent sodium chloride solution is convenient and restricts

the salt administration to 4.5 Gm. per 1000 cc. of fluid.

In patients who have had excessive vomiting or diarrhea, potassium deficiency may develop, as indicated by blood potassium and electrocardiographic studies. Treatment with intravenous potassium chloride is indicated when evidence of potassium deficiency is found.

The blood calcium may reach a low level, in which case treatment with intravenous calcium gluconate may be indicated. In severe acute pancreatitis hypokalemia and hypocalcemia may co-exist. In such cases potassium administration may precipitate tetany if the hypocalcemia is not also corrected.[14] This is because of the opposing effects of potassium and calcium on muscle and nerve irritability.

Continuous naso-gastric suction with complete abstention of oral feedings is used to combat distention and vomiting and to reduce the hormonal stimulation of the pancreas by preventing acid from reaching the duodenum.

Demerol is used to control pain and is probably preferable to morphine, which has a toxic effect on smooth muscle and increases the spasm of the sphincter of Oddi. The pain in the severe cases may be very difficult to control, and in such cases the use of paravertebral sympathetic block to relieve pain, as advocated by Gage[15] and Marion,[16] may be tried. Orr and Warren,[17] however, failed to obtain consistent results with paravertebral blocks and have used continuous epidural procaine analgesia with gratifying results.

Penicillin should be given prophylactically to minimize the danger of infection and abscess formation.

It is our feeling that surgery should be reserved for the treatment of the late complications of abscess or pseudo-cyst formation. Some cases will develop a chronic pancreatitis with continued intractable pain requiring surgery. Splanchnicectomy, transcholedochal sphincterotomy, prolonged common duct drainage, partial gastrectomy, and pancreatectomy are procedures which have been used, but a discusion of this problem is not within the scope of this paper. Obstructive jaundice occurring during pancreatitis may require decompression of the biliary tract, but in most cases the jaundice will clear as the pancreatitis subsides (Cf., case No. 2). In the most severe cases with extensive necrosis of the pancreas the patient rarely survives. However, the extensive necrosis and accumulation of fluid in the retroperitoneal tissues which is often found at autopsy in the fulminating cases (Cf., case 3) suggests that retro-peritoneal drainage through a flank incision might be of value and more logical than the drainage of the lesser sac and pancreatic capsule which is sometimes done.

It should be emphasized that when a diagnosis of acute pancreatitis is made and conservative therapy instituted, great care must be taken and repeated examinations made to rule out the presence of an intra-abdominal condition requiring surgery. In some cases the presence or absence of such a condition can be determined only by abdominal exploration. The patient must also be watched closely for the development of complications of acute pancreatitis which might require surgery, such as an extensive, dissecting retroperitoneal accumulation of fluid, a pseudo-cyst, or an abscess.

SUMMARY AND CONCLUSIONS

A study of 50 cases of acute pancreatitis is presented from the standpoint of diagnosis and treatment. Methods of differentiating acute pancreatitis from the conditions with which it is often confused are discussed, and illustrative case histories are presented. The results of treatment in our series of cases are presented and discussed.

Acute pancreatitis is a disease of protean manifestations and can closely imitate such conditions as bowel obstruction, biliary tract disease, peritonitis, and penetrating or perforated peptic ulcer. In every patient

with acute abdominal pain the possibility of acute pancreatitis should be considered and a serum amylase test made as soon as possible. In addition to a careful history and physical examination, the laboratory determinations of blood sugar, blood calcium, and serum bilirubin may be useful in making the diagnosis of acute pancreatitis. Roentgen ray studies usually reveal evidence of a paralytic ileus and are chiefly useful in ruling out such conditions as bowel obstruction and perforated peptic ulcer.

It is our feeling that a plan of conservative treatment should be set in action as soon as the diagnosis of acute pancreatitis is made and that surgery should be reserved for certain complications such as extensive retroperitoneal collection of fluid, abscess, or pseudo-cyst.

It is also our experience that in some cases only surgical exploration will differentiate acute pancreatitis from conditions in the abdomen requiring surgery. However, abdominal exploration increases the mortality from acute pancreatitis and therefore should be held to a minimum by a careful diagnostic evaluation of each patient. It would also appear from our experience that if abdominal exploration discloses acute pancreatitis closure without drainage is the best procedure.

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