Gastrocutaneous Fistulas Associated with Pancreatic Abscesses

An Aggressive Entity

ANDREW L. WARSHAW, M.D., ASHBY C. MONCURE, M.D., and DAVID W. RATTNER, M.D.

Previous reports of gastrocutaneous fistulas emphasize their benign nature and the probability of spontaneous healing without the need for surgical closure. In distinct contrast we report our experiences with six patients whose gastrocutaneous fistulas were caused by fulminant pancreatitis and pancreatic abscesses. High-output acid fistulas appeared days to weeks after drainage of left upper quadrant-infected collections. None occurred at the time of initial abscess drainage. Most originated high on the greater curvature of the stomach and traversed the abscess cavity. Three of six patients had previous splenectomies, but these operations were remote in time from the appearance of the fistula. Nonoperative management was successful in only two of six patients. Massive hemorrhage from the fistula tract contributed to the deaths of three patients. The pathogenesis of these fistulas appears to be consequent to gastric injury resulting from adjacent pancreatic inflammation. We conclude that (1) gastrocutaneous fistulas associated with pancreatitis are unlikely to heal even with drainage of the abscesses and are often complicated by hemorrhage, (2) surgical closure of the fistula will often be necessary and should not be unreasonably delayed, and (3) when performed in a semi-elective setting, resection of the damaged gastric segment, perhaps with an omental or serosal patch to buttress the gastric suture line, has a good chance of success.

THE DEVELOPMENT OF EXTERNAL fistulas after acute pancreatitis is well recognized. Most fistulas arise from the pancreatic ductal system, often after debridement of pancreatic necrosis or external drainage of pancreatic pseudocysts. Colocutaneous fistulas are the second most frequently encountered and are thought to be due to ischemic necrosis of the bowel.[1,2] In contrast gastrocutaneous fistulas arising as a consequence of severe pancreatitis have not been widely appreciated or considered as an entity.

Most gastrocutaneous fistulas occur after iatrogenic gastric injury (particularly after splenectomy), breakdown of a gastroenteric anastomosis, or failure of healing of a gastrostomy tube tract.[3,4] Previous reports emphasize the benign nature of gastrocutaneous fistulas and the probability of spontaneous healing without surgical intervention.[3-6] We encountered six cases of gastrocutaneous fistulas arising as a consequence of pancreatic abscesses over a 12-year period. These fistulas responded poorly to nonoperative management and were associated with a 50% incidence of life-threatening hemorrhage from the fistula tract. To our knowledge, this is the first report specifically addressing the association of gastrocutaneous fistulas with pancreatic abscesses.

From the Surgical Services, Massachusetts General Hospital, and the Department of Surgery, Harvard Medical School, Boston, Massachusetts

Correspondence and reprint requests: Andrew L. Warshaw, M.D., Massachusetts General Hospital, ACC 336, Boston, MA 02114.

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Materials and Methods

We identified six cases of gastrocutaneous fistulas associated with pancreatic abscesses in our practice at the Massachusetts General Hospital. Gastrocutaneous fistulas originating at the site of a previous gastrostomy, developing as a complication of a primary gastric or splenic procedure, or as a complication of radiation therapy were excluded. Both the office and hospital records of all patients were available for review. Follow-up until death or fistula closure was complete in all patients.

Results

Incidence

Six patients were identified with gastrocutaneous fistulas complicating pancreatic abscesses in 12 years. During the period of this study, approximately 130 cases of pancreatitis and four to five pancreatic abscesses were treated each year at the Massachusetts General Hospital. Five of the patients were men. The patients ranged in age from 24 to 78 years, with a mean age of 49 years.

Presentation

All patients had severe acute pancreatitis. Pancreatitis was caused by gallstones in 3 cases, trauma in 2 cases (1 motor vehicle accident with blunt abdominal trauma, and 1 operative injury to the pancreas), and alcohol in 1 case. The appearance of a gastrocutaneous fistula was noted between 6 and 9 weeks (average, 7.5 weeks) from the onset of pancreatitis in all patients. Before the recognition of a gastrocutaneous fistula, all patients had surgical drainage of a pancreatic abscess. Five of the six abscesses were localized in the retroperitoneal left upper quadrant and the other was a large lesser sac collection. No gastric injury or gastric fistula was apparent at the time of the initial abscess drainage. The diagnosis was generally made days to weeks later by a sudden increase in the amount of drainage appearing *via* the drain tract or by the development of a recurrent abscess in the left upper quadrant. The drainage fluid in all cases was acid (pH < 2). The clinical diagnosis was confirmed by upper GI series in four patients and by fistulogram in two patients (Figs. 1 and 2).

In two patients with severe pancreatitis, the pancreatic abscess was also associated with colonic infarction or perforation. A transverse colostomy was performed at the time of drainage of the abscess in these patients, and subsequent radiologic studies demonstrated gastrocolocutaneous fistulas (Fig. 3).

Three patients had splenectomies before the development of gastrocutaneous fistulas. The indications for splenectomy were splenic rupture due to blunt trauma, operative injury to the spleen during colonic resection, and splenic infarction due to severe necrotizing pancreatitis. Splenectomies were performed in these patients 5 to 9 weeks before the development of gastrocutaneous fistulas.

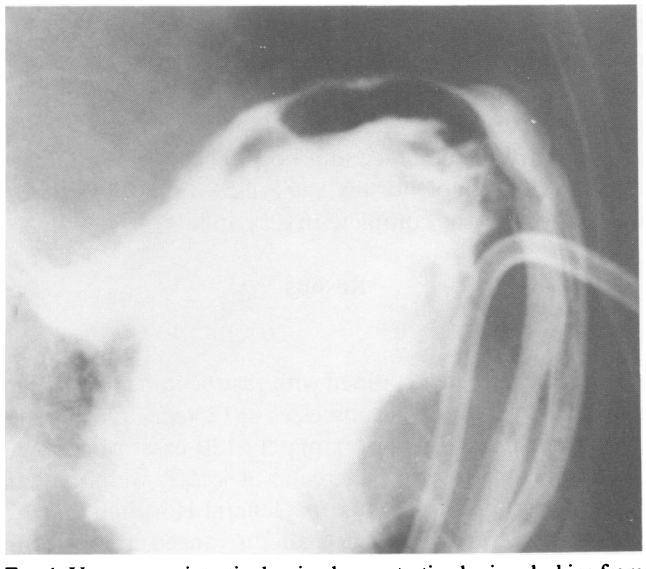
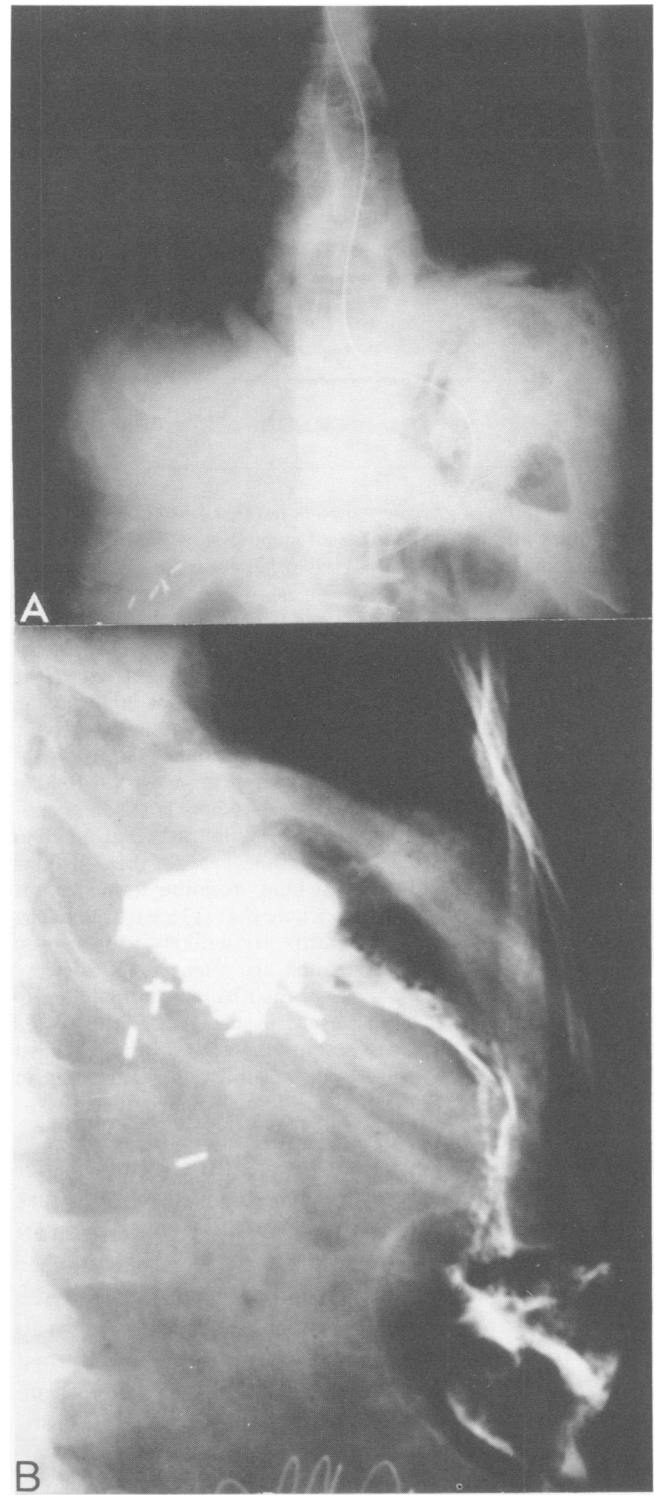


FIG. 1. Upper gastrointestinal series demonstrating barium leaking from the greater curvature of the gastric fundus.



FIGS. 2A and B. (A) Abdominal roentgenogram demonstrating a mottled density behind the stomach, consistent with pancreatic necrosis and abscess. (B) Fistulogram from the same patient 2 weeks after drainage of the subphrenic abscess. The fistula originates high on the greater curvature of the stomach.

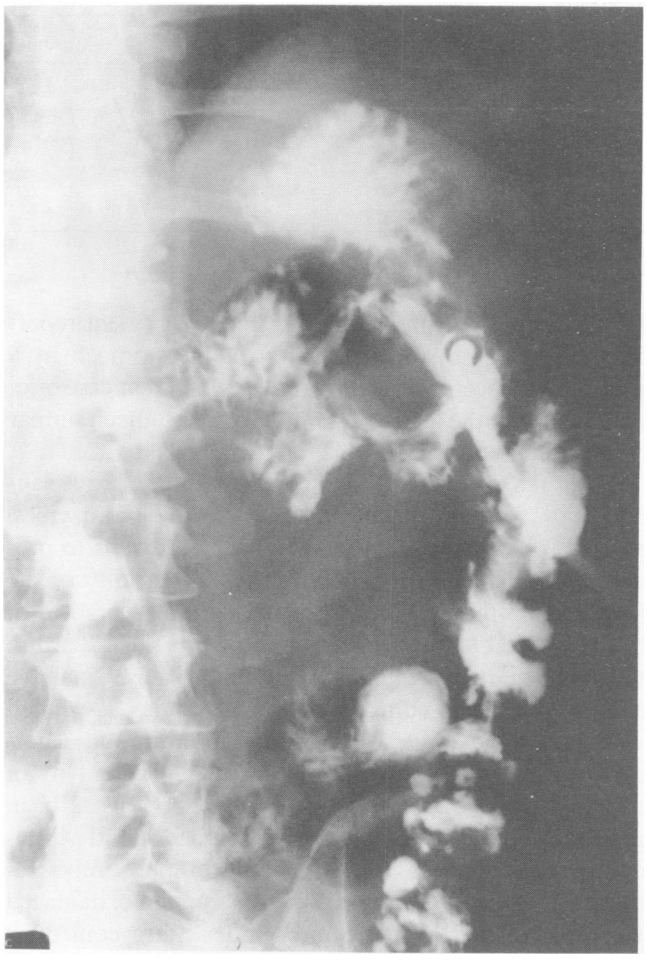


FIG. 3. Fistulogram demonstrating a gastrocolocutaneous fistula after drainage of a pancreatic abscess. The drain sits in the abscess cavity between the splenic flexure and the stomach. Fecal drainage had been noted, but the communication with the stomach was not appreciated before this study.

Soft rubber drains were in the bed of abscess cavities near the stomach in four of six cases at the time the fistula was diagnosed.

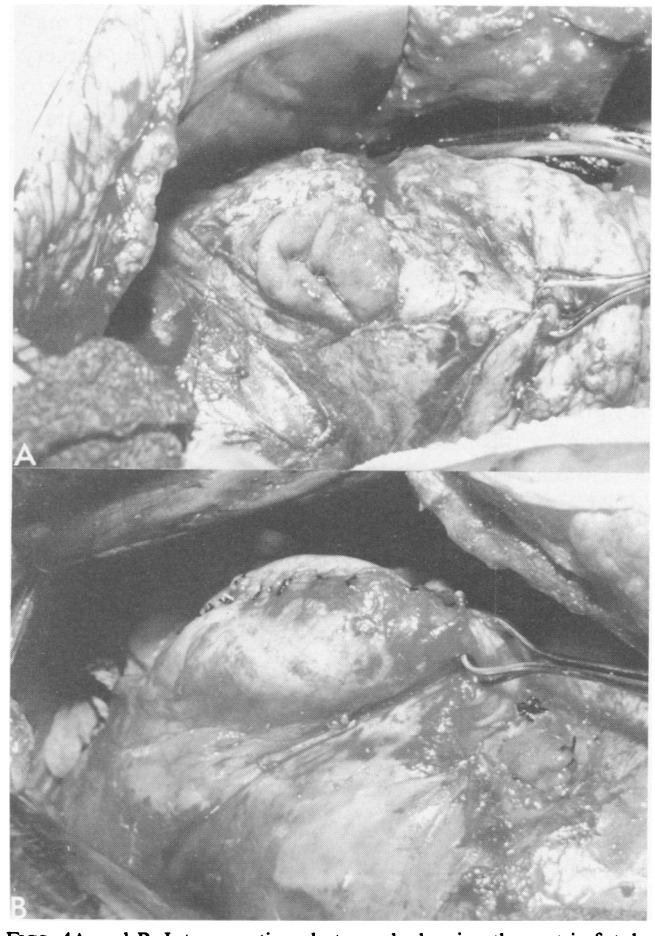
Complications

All patients had a complex clinical course with signs of undrained sepsis at the time the gastrocutaneous fistula was recognized. The most striking complication was massive hemorrhage from the fistula tract in three of six patients. All of these patients required transfusion of more than 10 units of blood in a 48-hour period and all manifested hemodynamic evidence of hypovolemia. Two other patients had bloody drainage from the fistula at some point in their illness, but no hemodynamically significant bleeding.

Outcome

The three patients with massive hemorrhage from the fistula required urgent angiographic and surgical intervention. All of these three patients ultimately died. One of these patients sustained an iatrogenic dissection of the celiac axis during attempted angiographic embolization of the bleeding site. This led to infarction of the upper abdominal viscera and death. In another patient who was 78 years old, hypotension caused by hemorrhage led to a perioperative myocardial infarction and death on the third postoperative day. The third patient with massive hemorrhage had successful surgical closure of the gastrocutaneous fistula and control of bleeding but died of persistent sepsis 2 weeks after operation. Surgical closure of the gastrocutaneous fistula was also successfully performed in a semi-elective setting in one other patient (Figs. 4A and B).

Both patients with gastrocolocutaneous fistulas were successfully treated by drainage of the abscess cavity and proximal colostomy for fecal diversion, but without direct surgical closure of the gastric defect. The gastric fistulas subsequently closed in 3 and 4 months, respectively. One patient whose barium enema showed closure of the colonic fistula had his transverse colostomy closed, and the



FIGS. 4A and B. Intraoperative photograph showing the gastric fistula after mobilization of the greater curvature. (B) Completed three-layer closure of the gastric perforation.

TABLE 1. *Characteristics of Patients with Gastrocutaneous Fistulas*

Patient	Age	Cause of Pancreatitis	Splenectomy	Colonic Fistula	Hemorrhage	Mortality
1	24	Blunt trauma	+	+		
2	78	Biliary			+	+
3	68	Operative trauma	+		+	+
4	47	Alcohol	+			
5	61	Biliary		+		
6	62	Biliary			+	+

other patient underwent an elective left colectomy 3 months after recovering from his acute illness (Table 1).

Discussion

Previous reports have emphasized the relatively benign nature of gastrocutaneous fistulas and the success of nonoperative management.[3,6] However no previous report has discussed the development of gastrocutaneous fistulas in the setting of pancreatic abscesses. In contrast to gastrocutaneous fistulas arising from other causes, gastrocutaneous fistulas secondary to pancreatic abscesses are life threatening, as evidenced by the 50% mortality rate in this series.

The pathogenesis of these fistulas is uncertain and may be multifactorial. Because all patients had previous surgery in the left upper quadrant, the role of iatrogenic injury to the stomach cannot be dismissed, in particular the potential for ischemic necrosis of the greater curvature after splenectomy.[3,4] Although three patients in this series had splenectomies before the development of the fistula, three others did not and thus iatrogenic gastric injury after splenectomy cannot fully account for the findings. External drainage catheters lying adjacent to the stomach for prolonged periods of time are believed to be capable of eroding the gastric wall with subsequent gastrocutaneous fistula formation.[6] In four cases in this series, erosion of the stomach by drainage catheters may have contributed to the pathogenesis of the fistula, notwithstanding that only soft rubber drains were used. Two patients no longer had indwelling drains when the fistula appeared, however. The only common denominator among all patients was ongoing active pancreatic inflammation. Vascular thrombosis is well known within areas affected by severe pancreatitis, perhaps due to the action of liberated enzymes on blood vessels, and may have caused ischemic necrosis of the gastric wall.[7] The simultaneous occurrence of colonic necrosis and fistulas in two of the six patients supports this hypothesis.[1,2]

In addition to producing vascular thrombosis, activated pancreatic enzymes may have the power to penetrate visceral walls directly. The propensity for pancreatic pseudocysts to decompress spontaneously by erosion into adjacent viscera is well known.[8] Apparently pancreatic abscesses can behave in a similar fashion.[9] A recent report documented spontaneous erosion of the stomach by a pancreatic abscess.[10] The internal fistula in that case originated high on the greater curvature of the stomach similar to the cases described in this report (Fig. 1).

Hemorrhage is a recognized complication of both pancreatic pseudocysts and abscesses.[11,12] Pancreatic enzymes, especially elastase, may digest blood vessel walls to produce pseudoaneurysms and rupture. The acidity of the fistula contents may further contribute to ulceration of the walls and blood vessels along the fistula tract. Because the stomach has a particularly rich blood supply, massive life-threatening bleeding from gastric fistulas associated with pancreatic abscesses is predictably common, occurring in one half of our patients. Although the gravity of bleeding associated with pancreatic abscesses has been noteworthy,[11-13] hemorrhage from eroded blood vessels can be arrested in most cases by angiographic embolization, and surgical resection and reconstruction of damaged viscera can then be performed under more controlled conditions.[11] Adjunctive treatment of this sort has reduced the number of deaths from pancreatic abscesses and infected necrosis to as little as 5%.[12]

During this same study period, we have treated three patients with duodenocutaneous fistulas associated with pancreatic abscesses. Somewhat to our surprise, all three of the duodenal holes healed without complication under conservative management. This apparently different behavior may indicate the deleterious role of unbuffered acid drainage from a gastric fistula, in contrast to the duodenal contents that contain alkaline bile and pancreatic secretions. The different behavior of acid and alkaline fistulas is further exemplified by a contemporaneous patient with a pancreatic abscess whose gastrocutaneous fistula resulted from a dehisced gastrostomy. That patient also experienced a high-output acid fistula, developed massive bleeding through the fistula, and ultimately was salvaged only by re-exploration and successful closure of the gastric defect.

While some gastrocutaneous fistulas associated with pancreatic abscess will heal, our experience illustrates that the risk of further complications, especially bleeding and uncontrolled sepsis, is a constant threat, greater than that from fistulas located elsewhere along the gut.[12] A trial of

nonoperative therapy consisting of H_2-blockers and nasogastric suction may be worthwhile, but specific time limits, probably no longer than a few weeks, should be set. Continuing high output from the fistula seems to indicate a low probability of healing. Limited resection of the damaged gastric segment and direct closure in two or three layers, buttressed with omentum, when possible, or with a serosal patch, has been successful.

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