Pancreatic Head Mass: How Can We Treat It? Acute Pancreatitis: Surgical Treatment

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Acute pancreatitis is an inflammatory process of variable severity, ranging from a mild, selflimited form with interstitial edema of the pancreas to a severe form with extensive pancreatic necrosis and hemorrhage [1-3]. Pancreatic necrosis combined with septic conditions is the leading cause of mortality in acute pancreatitis. Although aggressive organsystem support has resulted in an improved survival rate in the early stage of the disease, patients continue to die at a later stage from complications, septic culminating in multiorgan failure [4]. Overall infection rates in acute pancreatitis do not exceed 10%, but infected necrosis occurs in up to 70% of patients with necrotizing pancreatitis [5-7]. reported mortality rate of these The complications ranges from 15 to 80% [5,6,8, 9]. It is generally accepted that the initial treatment of acute pancreatitis should be conservative unless there is a specific indication for surgical intervention. There are, however a number of clear-cut indications for which surgery should be performed: 1) patients with infected necrosis should undergo urgent surgical debridement, after bacterial infection has been diagnosed; 2) ongoing multiorgan failure despite maximum intensive medical therapy over a period of 48-72 h is accepted as an indication for surgery; 3) abdominal complications (persistent ileus, intra-abdominal bleeding, suspected gastrointestinal perforation) tract pose additional indications for surgical treatment [8, 10, 11].

It has recently become apparent that there are three recognizable forms of pancreatic infection according to the morphological, clinical and laboratory criteria: infected pancreatic necrosis, a pancreatic abscess and an infected pseudocyst [6, 7, 10-15]. The incidence of pancreatic infection, including infected necrosis, pancreatic abscesses and infected pseudocysts, complicating acute pancreatitis ranges from 8% to 12% [4, 5, 8]. However infected necrosis is reported to develop in 40% to 70% of all patients with necrotizing pancreatitis [6, 7, 16] depending on the extent of the necrosis and the duration of the pancreatitis.

Diagnosis

The basis of appropriate treatment of infected pancreatic necrosis is an early, accurate diagnosis. Delayed diagnosis has been identified as a major factor resulting in poor outcome and mortality [10]. Criteria for the diagnosis of infected pancreatic necrosis include confirmation of previous or present pancreatitis, clinical evidence of sepsis, determination of the APACHE II score and the retrieval of microorganisms before and at the time of the operation. Extensive clinical experience has shown that contrast-enhanced computer tomography (CT) is the gold standard for differentiating the morphological features of the pancreas at an early or later stage of pancreatitis [17-19]. The introduction of helical CT scanners has allowed threedimensional reconstruction of the intraabdominal viscera. and volumetric assessment of the extent of intra- and

extrapancreatic necrosis, and peripancreatic and retroperitoneal exudation. However, CT diagnosis of infection is rarely possible. A characteristic appearance, the "air bubble" phenomenon produced by gas-forming bacteria, is seen in only 20% to 55% of all patients with infected necrosis [7]. The most appropriate diagnostic method for the early detection of infected pancreatic necrosis is guided percutaneous fine-needle aspiration with Gram staining and culturing of the aspirate [19-20].

Surgical Treatment

There is general agreement that infected pancreatic necrosis is an absolute indication for surgery; non-operative or percutaneous management is usually associated with a fatal outcome. A variety of approaches have been advocated for the surgical management of infected pancreatic necrosis. They include different techniques, ranging from tissuesparing methods to aggressive, extensive resection. During the 1980s, three main patterns of management could be identified in the surgical management of necrotic and infective complications of acute pancreatitis: "conventional" treatment. including 1) resection of the involved pancreatic tissue or necrosectomy, followed by simple drainage of the peripancreatic bed; 2) "lavage" treatment, in which necrosectomy is followed by continuous closed local irrigation or lavage of the involved pancreatic and retroperitoneal and 3) "open abdominal area: an involving management" (laparostomy), followed necrosectomy by various combinations of planned and staged reoperations. The mortality rate of the three patterns was 42% (range 24-84%) [21-23], 12.5% (range 6.3-23%) [8, 12, 24, 25] and 21% (range 11-55%) [26-28] when "conventional" treatment, "lavage" treatment and "open abdominal management" were applied, respectively.

Since 1986 we have adopted the "lavage" treatment in our practice, consisting of wide-ranging necrosectomy and other surgical

interventions, combined with continuous widespread lavage [24]. In all patients (175 patients), the operative management consisted of wide-ranging necrosectomy throughout the entire area affected, using bilateral subcostal laparotomy. The abdomen was explored for classification of the extent of pancreatic and extrapancreatic necrosis. Through division of gastrocolic duodenocolic the and the ligaments, the extent of necrosis in the head, body and tail of the gland could readily be assessed and measured. For accurate exploration of the retroperitoneum, Kocher's mobilization of the duodenum and mobilization of the right and left colon were performed. In our cases, the infected necrotizing process was situated in the right and left retrocolic area in 70%, in the left subphrenic area in 20%, and in the retroduodenal and subhepatic area in 10%. Debridement or necrosectomy, either digitally or by the careful use of an instrument combined with permanent normal saline lavage, permitted the exclusive removal of all demarcated devitalized tissue, preserving the vital pancreatic tissue [25] and removing the infected necrotic tissue from the whole affected retroperitoneal area. After surgical debridement, meticulous hemostatis and extensive intraoperative lavage with 8 to 10 liters of normal saline were applied, and for postoperative closed continuous local lavage 4 to 11 large (Charr. 28-34) silicone rubber tubes were inserted into the entire area affected. They were inserted only into the pancreatic region and the retroperitoneal spaces, without any connection with the intraabdominal region.

In 79 of the 175 cases (45%) some other surgical intervention distal pancreatic resection and splenectomy (n=30), splenectomy (n=4), subtotal pancreatectomy (n=6), colon resection (n=3), cholecystectomy (n=35), cholecystectomy and bile duct drainage (n=6), partial hepatic resection (n=1) or appendectomy (n=2) - was also performed. Continuous washing and suction drainage was applied for an average of 42.5 days (range 21-90), with a median of 9.5 liters (range 5-20) of normal saline per 24 h. In the first few

postoperative days, the amount of lavage fluid was generally 15-20 liters, which was later reduced, depending on the clinical course and on the appearance and quality of the outflowing liquid. Reoperation was necessary in 22% (38 patients) of all patients. 32 of them had developed a secondary abscess in the area of the original necrosis cavity and 2 had developed a colonic fistula, which was cured by large bowel resection, while massive diffuse local bleeding was responsible in 4 patients. Only one of the reoperated cases died following reoperation as a result of local bleeding. Pancreatic fistulas were observed in patients; in 8 cases they closed 16 spontaneously, but in the remaining 8 cases they became long-standing, high-output ones with a high amylase concentration (mean 435 500 U/L). In all 8 of these patients, octreotide therapy (3x0.1 mg/day) was combined with total parenteral nutrition, and 13 days (range 7-19) of this treatment led to complete closure of the fistulae [29]. Systemic complications occurred mainly in connection with local complications and reoperation. In 32 patients respiratory failure required mechanical ventilation for over 24 h. Renal and circulatory insufficiency developed in 10 and 5 patients, respectively.

Eleven patients died following surgery and therefore the overall mortality rate was 6.3% (11 of 175 patients). The cause of death was bacterial sepsis in 7 patients, bacterial and fungal sepsis in one patient, fungal sepsis in one patient, myocardial infarction in one, and stroke in one patient. The hospital stay of the surviving patients amounted to a median of 45 days (range 24-95).

The surgical management of our patients with infected pancreatic necrosis was directed towards removal of the devitalized intra- and extra-pancreatic tissue in all affected areas. It seems very important to explore every possibly infected site since ineffective debridement can endanger the recovery of the patients and increase the likelihood of reoperation. In agreement with several authors, it is not necessary to remove every small bit of the devitalized tissue, because any necrotic or necrotizing tissue is washed out by the lavage fluid later in the postoperative period [13, 14, 24, 25]. The success of the postoperative closed continuous lavage depends on the number and the size of the drainage tubes [30]. Generally, we applied 4 to 11 large silicone tubes inserted in all the sites affected. As infected necrotic processes can extend into intra- and extrapancreatic areas, other surgical interventions are also advisable. This fact explains our surgical strategy, namely in 79 of 175 patients (45%) the necrosectomy and continuous lavage were combined with several surgical interventions (distal pancreatic resection, splenectomy, cholecystectomy, colon resection, etc.). We attribute the improved survival rate to adequate surgical debridement and additional surgical intervention combined with continuous widespread washing and suction drainage in all affected areas. A large volume of saline solution for continuous lavage through multiple drainage tubes is a safe and non-traumatic procedure which can eliminate the infected, necrotic tissue. Effective surgical treatment together with adequate supportive therapy can give good results in patients with infected pancreatic necrosis.

In conclusion, improved results can be achieved by aggressive surgical treatment, continuous. long-standing washing and suction drainage together with supportive including immunonutrition. therapy, modification of the cytokine production [31, 32] combined with adequate antibiotic and antifungal medication [33, 34]. This surgical strategy provides the possibility of recovery in cases of necrotizing pancreatitis combined with septic complications.

Key words Candida; Glutamine; Infectious Pancreatic Necrosis Virus; Pancreatitis, Acute Necrotizing; Pentoxifylline

Abbreviations CT: computed tomography

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References

1. Corfield AP, Cooper MJ, Williamson RCN. Acute pancreatitis: a lethal disease of increasing incidence. Gut 1985; 26:724-9.

2. Gendell JH, Egan J. Acute pancreatitis. Western J Med 1987; 146:598-602.

3. Balthazar EJ. CT diagnosis and staging of acute pancreatitis. Radiol Clin N Am 1989; 27:19-37.

4. Frey CF, Bradley EL III, Beger HG. Progress in acute pancreatitis. Surg Gynecol Obstet 1988; 167:282-6.

5. Allardyce DB. Incidence of necrotizing pancreatitis and factors related to mortality. Am J Surg 1987; 154:295-300.

6. Bittner R, Block S, Büchler M, Beger HG. Pancreatic abscess and infected pancreatic necrosis. Dig Dis Sci 1987; 32:1082-7.

7. Schoenenberg MH, Rau B, Beger HG. Diagnose und therapie des primaren pankreasabscesses. Chirurg 1995; 66:588-96.

8. Beger HG, Büchler M, Bittner R, Block S, Nevalainen T, Roscher R. Necrosectomy and postoperative local lavage in necrotizing pancreatitis. Br J Surg 1988; 75:207-12. [88164274]

9. Wilson C, McArdle CS, Carter DC, Imrie CW. Surgical treatment of acute necrotizing pancreatitis. Br J Surg: 1988; 75:1119-23.

10. D'Egidio A, Schein M. Surgical strategies in the treatment of pancreatic necrosis and infection. Br J Surg 1991; 78:133-7.

11. Bradley EL III. A clinically based classification system for acute pancreatitis.

Summary of the International Symposium on Acute Pancreatitis, Atlanta, Ga, September 11 through 13, 1992. Arch Surg 1993; 128:586-90. [93256758]

12. Bassi C, Vesentini S, Nifosi F, Girelli R, Falconi M, Elio A, Pederzoli P. Pancreatic abscess and other pus-harboring collections related to pancreatitis: a review of 108 cases. World J Surg 1990; 14:505-12. [90342342]

13. Imrie CW. Indications for surgery : the surgeon's view. In: Beger HG, Büchler M, Malfertheiner P, eds. Standards in Pancreatic Surgery. Berlin: Springer-Verlag, 1993: 148-56.

14. Büchler M, Uhl W, Beger HG. Surgical strategies in acute pancreatitis. Hepatogastroenterol 1993; 40:563-8.

15. Bradley EL III. Operative management of acute pancreatitis: ventral open packing. Hepatogastroenterol 1991; 38:134-8.

16. Fedorak IJ, Ko TC, Djuricin G, McMahon M, Thomson K, Prinz A. Secondary pancreatic infections: are they distinct clinical entities?. Surgery 1992; 112:824-31.

PC. 17. Freeny Radiology of acute pancreatitis: diagnosis, detection of complications, and interventional therapy. In: Glazer Ranson JHC, G. eds. Acute Pancreatitis: Experimental and Clinical Aspects of Pathogenesis and Management. London: Bailliere Tindall, 1988: 275-302.

18. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JHL. Acute pancreatitis: value of CT in establishing prognosis. Radiology 1990; 174:331-6.

19. Gerzof SG, Banks PA, Robbins AH, Johnson WC, Spechler SJ, Wetzner SM, et al. Early diagnosis of pancreatic infection by computed tomography-guided aspiration. Gastroenterology 1987; 93:1315-20.

20. Banks PA, Gerzof SG, Chong FK, Worthington MG, Doos WG, Sullivan JG, Johnson WC. Bacteriologic status of necrotic tissue in necrotizing pancreatitis. Pancreas 1990; 5:330-3. 21. Allardyce DB. Incidence of necrotizing pancreatitis and factors related to mortality. Am J Surg 1987; 154:295-9.

22. Wilson C, McArdle CS, Carter DC, Imrie CW. Surgical treatment of acute necrotizing pancreatitis. Br J Surg 1988; 75:1119-23.

23. Warshow AL, Jin G. Improved survival in 45 patients with pancreatic abscess. Ann Surg 1985; 202:408-17.

24. Farkas G, Márton J, Mándi Y, Szederkényi E. Surgical strategy and management of infected pancreatic necrosis. Br J Surg 1996; 83:930-3. [96408792]

25. Büchler M, Uhl R, Isenmann R, Bittner R, Beger HG. Necrotizing pancreatitis: necrosectomy and closed continuous lavage of the lesser sac. The Ulm experience. In: Beger HG, Büchler M, Malfertheiner P, eds. Standards in Pancreatic Surgery. Berlin: Springer-Verlag, 1993: 191-202.

26. Bradley EL III. Surgical indications and techniques in necrotizing pancreatitis. In: Acute Pancreatitis: Diagnosis and Therapy. Bradley EL III, ed. New York: Raven Press, 1994: 105-17.

27. Wertheimer MD, Norris CS. Surgical management of necrotizing pancreatitis. Arch Surg 1986; 121:484-7. [86158244]

28. Schein M, Hirschberg A, Hashmonai M. Current surgical management of severe intraabdominal infection. Surgery 1992; 112:489-96. [92390834] 29. Farkas G, Leindler L, Szederkényi E. Beneficial effect of Sandostatin, a long-acting somatostatin analog, in pancreatic surgery. Hepatogastroenterol 1993; 40(Suppl. 1):182-3.

30. Pederzoli P, Bassi C, Vesentini S, Girelli R, Cavallini G, Falconi M, et al. Retroperitoneal and peritoneal drainage and lavage in the treatment of severe necrotizing pancreatitis. Surg Gynecol Obstet 1990; 170:197-202.

31. Mándi Y, Farkas G, Ocsovszky I, Nagy Z. Inhibition of tumor necrosis factor production and ICAM-1 expression by pentoxifylline: beneficial effects in sepsis syndrome. Res Exp Med 1995; 195:297-307. [96171908]

32. Mándi Y, Farkas G, Ocsovszky I, Béládi I, Balogh Á. Effects of pentoxifylline and Pentaglobin on cytokine production in septic patients. In: Faist E, Baue AE, Schildberg FW, eds. The Immune Consequences of Trauma, Shock and Sepsis-Mechanisms and Therapeutic Approaches. Berlin, Wien, Riga: Pabst Science Publishers, 1996: 420-8.

33. Farkas G, Szendrényi V, Karácsonyi S, Mezey G. Candida infection in pancreatic abscess. Dig Surg 1993; 10:254-6.

34. Farkas G, Márton J, Mándi Y, Szederkényi E, Balogh Á. Progress in the management and treatment of infected pancreatic necrosis. Scand J Gastroenterolog 1998; 228 (Suppl.):31-7. [99082999]