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Pancreatic Abscess and other Pus-Harboring Collections Related to Pancreatitis: A Review of 108 Cases

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This is a report on 108 cases collected from 1970 to 1987, in the same department, of surgically-detected pancreatic abscesses or pus-harboring collections. The purulent areas were either of a spreading pattern or represented a clearly localized mass. To the spreading pattern belong 47 cases of necrotizing pancreatitis, without discontinuity in the clinical course from the early toxic to the late septic phase, 4 cases of acute pancreatitis, initially in remission and later complicated by septic collections, and 4 cases which developed after an acute attack of chronic pancreatitis. The abscess pattern was made up of 19 each of pseudocysts and predisposing pancreatitis, 10 cases of chronic pancreatitis, and only 5 necrotizing "nonstop" pancreatitis.

The surgical treatment in all cases consisted of multiple drainages and postoperative irrigation. We exclude 3 cases of associated open packing. The etiological, clinical, and biochemical features of each group of patients are reported and discussed. Computed tomography availability seems to be the most important improvement reported as regards diagnosis and surgical tactics. The overall mortality rate was 15.7% with a significant difference between the 2 patterns (23.6% for the spreading pattern versus 7.5% for the abscess pattern). On the basis of this experience, it is possible to establish a relationship between the gross appearance of the collection and the underlying pancreatic disease with differences in terms of prognosis, morbidity, and mortality. Finally, a simple nomenclature can be chosen which is capable of distinguishing between the diverse pancreatic purulent collections. While the presence of pus may characterize the course of severe acute pancreatitis in many cases, the low incidence of "true" pancreatic abscess is emphasized.

Sepsis is a recognized major threat to patients with acute pancreatitis. Despite the fact that the issue has received increasing attention, there is still much confusion about terms such as "pancreatic abscess" [1–9], "infected pancreatic necrosis" [2], "phlegmon" [3, 10, 11], "peripancreatic infection and sepsis" [12], "infected pancreatic abscess" [13], "major pancreatic infections" [14], "pancreatic sepsis" [15], "pseudocyst abscess" [16] and "purulent pancreatitis" [17]. This lack of uniformity reflects both our poor understanding of the pathophysiology of pancreatic infections and the need for more accurate clinical-pathological correlations [18].

Faced with this confusion when trying to develop a computer database for all of our clinical records on patients with acute and chronic pancreatitis, we decided to conduct a separate review of our data concerning pancreatitis-related sepsis in order to establish standard criteria of classification. The work soon proved harder than we expected, but the end result is, in our opinion, rewarding.

In this article, we propose to sum up the results of our review with the aim of demonstrating that, in the vast majority of cases, there is a clear relationship between the gross appearance of the purulent collection and the underlying pancreatic disease. A simple and realistic nomenclature is possible and the differences existing among the various described collections have clear prognostic implications.

Methods

From 1970 to 1987, a total of 1,090 patients underwent surgery in our department for chronic and acute pancreatitis. The present retrospective review includes only those patients whose operative records clearly report the presence of detectable amounts of purulence. The surgeons (all belonging to the same "pancreatic team") found purulence in 108 cases (9.9%), detailing the site and the extension of the collections and describing the relative proportions of pus and necrotic tissue. They also cultured the material and performed biopsies in cases presenting with "walled" collections. Patients with detectable pus ranged in age from 19 to 76 years with an average age of 43 years; the male:female ratio was 2.3:1.1.

Fifty-two cases (48.1%) belonged to a group of 120 patients suffering from necrotizing pancreatitis surgically treated after at least the fourth day from the onset of the disease [19–21]. Twenty-three patients (21.3%) underwent surgery because of a local and or systemic sepsis developing after remission of a predisposing episode of acute pancreatitis occurring 8–42 days previously (average, 17 days). Nineteen patients (17.6%) were operated on for pseudocysts suspected as being infected (15 associated with chronic pancreatitis and 4 associated with acute pancreatitis). There were 14 cases (13%) of chronic pancreatitis requiring surgery because of a severe acute attack associated with a septic clinical course (Table 1). The main etiological,

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Table 1. Patterns of purulent collections related to pancreatic diseases and mortality rate.

Total		Deaths		Group A[a]		Deaths: A		Group B[b]		Deaths: B	
No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
108		17	(15.7)	55	(50.9)	13	(23.6)	53	(49.1)	4	(7.5)
52	(48.1)	12	(23.1)	47	(90.4)	12	(25.5)	5	(9.6)[c]		
23	(21.3)	4	(17.4)	4	(17.4)			19	(82.6)	4	(21.0)[d]
19	(17.6)							19	(100.0)		[e]
14	(13.0)	1	(7.1)	4	(28.6)	1	(25.0)	10	(71.4)		[f]

[a]Purulence described as mixed to necrosis.
[b]Purulence described as isolated.
[c]First episode of acute pancreatitis without discontinuity in the clinical course.
[d]Predisposing pancreatitis in remission with period of latency between acute episode and development of purulence.
[e]Pseudocyst.
[f]Acute attack (or post-endoscopic retrograde cholangiopancreatography study) in chronic pancreatitis.

Table 2. Etiological factors and related mortality rate.

	Total		Deaths		Group A		Deaths		Group B		Deaths	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Biliary	53	(49.1)	8	(15.1)	25	(45.4)	5	(20.0)	28	(52.8)	3	(10.7)
Alcohol	37	(34.2)	5	(13.5)	16	(29.1)	4	(25.0)	21	(39.6)	1	(4.7)
Postoperative	5	(4.6)	2	(40.0)	5	(9.1)	2	(40.0)				
ERCP	5	(4.6)	1	(20.0)	1	(1.8)	1	(100.0)	4	(7.5)		
Trauma	5	(4.6)	1	(20.0)	5	(9.1)	1	(20.0)				
Other	3	(2.8)			3	(5.4)						
Total	108		17	(15.7)	55	(50.9)	13	(23.6)	53	(49.1)	4	(7.5)

Table 3. Main clinical observed features.

	Total		Group A		Group B		Statistical significance
	No.	(%)	No.	(%)	No.	(%)	
Systemic sepsis[a]	36	(33.4)	28	(50.1)	8	(15.1)	$p = 3 \times 10^{-5}$
Fever[b]	92	(85.2)	52	(94.5)	40	(75.5)	$p = 5 \times 10^{-3}$
Pain	99	(91.7)	44	(80.0)	44	(83.0)	p = not significant
Renal failure[c]	25	(23.1)	19	(34.5)	6	(11.3)	$p = 3 \times 10^{-3}$
Peritonitis	26	(24.1)	19	(34.5)	7	(13.2)	$p = 8 \times 10^{-3}$
Ileus	80	(74.1)	53	(96.4)	27	(50.9)	$p = 2 \times 10^{-5}$
Mass	36	(33.4)	8	(14.5)	28	(52.8)	$p = 2 \times 10^{-8}$
Respiratory failure[d]	37	(34.2)	30	(54.5)	7	(13.2)	$p = 3 \times 10^{-5}$

[a]°C: > 38.5, erythrocyte sedimentation rate: >40, white cell: <3,000, >13,000, with blood pressure: >80 mmHg × 10 min (19 of 36 patients had blood-borne germs).
[b]°C: > 38.5.
[c]Creatinine: >2 mg/dl.
[d]PO_2: <60 mmHg.

clinical, and biochemical features of the 108 patients studied are reported in Tables 2, 3, and 4. The patients [except for 15 belonging to the pre-CT (computed tomography) period] were studied with preoperative CT and/or ultrasound (US) and serial postoperative controls at different times. The surgical approach was transperitoneal in 85% of cases and extraperitoneal in 15%. After removing both pus and necrotic tissue and breaking all septa within the margins of the collections, we applied silastic tube drains to the upper and lower poles of the formations. In the postoperative period, we started irrigation treatment with 2–10 liters/day (on the basis of the type and extent of the disease) of normal saline solution plus antiproteases (1,000,000 U.I.K./500 ml) when an active process was suspected and antibiotics (administered at maximal doses); antibiotics were also administered intravenously. A broad-spectrum antibiotic was administered initially and, subsequently, specific antibiotics were given when culture identified specific bacteria.

The average in vivo perfusion time was 7 days (range, 3–89). The drains were removed, on average, after 24 days (range, 6–93) on the basis of clinical-biochemical evaluation, particularly, the features of drained material and the findings on repeated CT and US studies performed approximately every 10 days. Three patients (2 after wound leakage) were treated with both the described technique and open wound packing because of the terrible local and systemic situations.

From 1978 on, all patients were fed by total parenteral nutrition. The statistical analysis was done using Fisher's exact test, establishing the level of significance at $p < 0.05$.

Table 4. Laboratory data observed (at onset of the complication).

	Total		Group A		Group B		Statisical significance
	No.	(%)	No.	(%)	No.	(%)	
Leukocytes[a]	92	(85.2)	52	(94.5)	40	(75.5)	$p = 5 \times 10^{-3}$
Anemia[b]	86	(79.6)	47	(85.4)	39	(73.6)	p = not significant
Hyperazotemia[c]	48	(44.5)	39	(70.9)	9	(17.0)	$p = 1 \times 10^{-8}$
Hyperglycemia[d]	50	(46.3)	31	(56.4)	19	(35.8)	$p = 2 \times 10^{-2}$
Hyperamylasemia[e]	82	(75.9)	49	(89.1)	39	(73.6)	$p = 3 \times 10^{-2}$

[a] = <3,000, >13,000.
[b] = Hematocrit decreased 10 points within 48 hours.
[c] = >30 mg.
[d] = >200 mg.
[e] = >200 I.U. (International Units).

Results

On the basis of the 108 operative reports, we were able to subdivide the patients into 2 groups: Group A (spreading pattern)—In this group, the pus was mixed with the retroperitoneal "slough"; the collection had neither clear boundaries nor walls; Group B (localized pattern)—In this group, the pus was well localized and the collection had a more or less pronounced wall; furthermore, pus was prevalent sometimes with small amounts of floating necrotic tissue.

Moreover, it was possible to assign each patient to 1 of the 2 groups without borderline cases. Fifty-five patients were assigned to the spreading type, and 53 patients were assigned to the localized group. Forty-seven of 52 cases of necrotizing pancreatitis without discontinuity in the clinical course from the toxic to the septic phase belonged to group A and the other 5 to group B ($p = 10^{-8}$).

Of the cases clinically characterized by a predisposing pancreatitis, initially on remission, 82.6% belong to the localized pattern ($p = 0.002$), as did all cases of pseudocyst ($p = 2 \times 10^{-7}$) and 71.4% (p = not significant) of the chronic pancreatitis cases observed (Table 1).

Multiple pus-containing collections were detected in about 80% of cases (95% in group A and 60% in group B; p = not significant). The most frequent localizations were on the left side (50% of cases) followed by the right side (25% of cases), both right and left (20%), and intrapancreatic only (5%).

The findings relating to etiology are summarized in Table 2. If we exclude postoperative pancreatitis, which was significantly more frequent in the spreading pattern ($p = 0.03$), the other etiological factors showed no differences in incidence between the 2 groups. From a clinical point of view, all the main parameters considered except the presence of a palpable mass had a higher frequency in group A than in group B (Table 3). A more significant frequency of biochemical positive criteria was reported in group A than in group B (Table 4). The overall mortality rate reported was 17 patients (15.7%), 13 (23.6%) in group A and 4 (7.5%) in group B ($p = 0.02$). No significant relationship was found between mortality rate and the etiology of pancreatitis, excluding the postoperative forms.

The deaths in the spreading pattern consisted of 12 patients of 47 suffering from necrotizing pancreatitis suddenly complicated by a stormy septic clinical course and 1 of 4 chronic patients. In the localized pattern, all the deaths occurred in the 19 patients with predisposing pancreatitis.

Table 5. Observed complications.

	Group A		Group B	
	No.	(%)	No.	(%)
Pancreatic fistulas	19	(34.5)	16	(30.2)
Enteric fistulas	18	(32.7)	13	(24.5)
Wound infection	14	(25.4)	7	(13.2)
Pus collections	16	(29.1)	2	(3.8)[a]
Bleeding	6	(10.1)	3	(5.7)
Stress ulcer	3	(5.4)		
Colonic obstruction			1	(1.9)
Total	76	(64.4)	42	(35.6)
		118[b]		

[a] $p = 0.01$.
[b] $p = 7 \times 10^{-6}$.

None of the patients with pseudocysts died.

The causes of death in group A were generalized sepsis (8), bleeding from drainage (3), adult respiratory distress syndrome (1), and heart failure (1). In group B, 3 were due to sepsis and 1 to bleeding.

The 108 patients experienced 118 surgical complications: 35 (29.7%) external pancreatic fistulas, 31 (26.2%) enteric fistulas, 21 (17.8%) wound infections, 18 (15.2%) new or incompletely drained pus collections, 9 (7.6%) bleeding from drainage, 3 (2.5%) bleeding from stress ulcer, and 1 (0.8%) colonic obstruction.

The number of complications observed was significantly higher in the spreading pattern than in the localized pattern ($p = 7 \times 10^{-6}$) (Table 5). Nine cases (25.7%) of 35 pancreatic fistulas required surgery, while the others were resolved by conservative treatment [22]. The 31 enteric fistulas required surgery in 3 cases of colonic fistulization and in 1 case of duodenal perforation. Reexploration was necessary in the 18 cases of new pus collections either because of inadequate initial drainage (10 cases) or because of further spread of the previous pus (8 cases); 3 patients required more than 1 operation, and 2 of them died as a result of systemic sepsis versus 5 deaths of the 15 reoperated patients. Those with bleeding from drainage required surgery in 7 of 9 cases; 4 surgical patients died.

The pus was collected intraoperatively for microbiological study; the culture findings are shown in Table 6.

In the pre-CT period, our mortality rate in pus-harboring pancreatitis was 40% (6 deaths of 15 patients observed) [1].

Table 6. Microbiological findings in 108 pancreatic collections with pus.

	No.	(%)	Deaths	(%)
Positive	83	(76.8)	17	(20.48)
Polymicrobial	68	(81.9)	16	(23.50)
Monomicrobial	15	(18.1)	1	(6.70)
Negative	25	(23.2)	–	–
Strains				
Escherichia coli	28	(17.40)		
Pseudomonas ael.	26	(16.14)		
Anaerobes	23	(14.30)		
Staphylococcus aureus	19	(11.80)		
Proteus	16	(9.93)		
Streptococcus faecalis	14	(8.70)		
Mycetes	13	(8.07)		
Klebsiella sporigena	8	(4.96)		
Enterococcus aeroguinosa	5	(3.10)		
Other	9	(5.60)		
Total	161	(100.00)		

Today, thanks also to the spatial information supplied by scan studies, the mortality rate is 11.8% (11 deaths of 93 patients). Moreover, in our previous report [1], 7 cases (46.7%) of 15 without CT had to undergo 1 or more reoperations to drain an inadequately evacuated collection; such a situation occurred 3 times (3.2%) in the CT group (93 patients). Seventy-four patients required intensive care (44 in group A and 30 in group B) for more than 24 hours. The mean hospitalization period in the survivors was 31 days (range, 14–116) with a difference between group A and B (A: mean, 53 days; range, 16–116; B: mean, 23 days; range, 14–78).

Discussion

By analogy with the axiom "no acid, no ulcer," we can state "no pus, no abscess." The different features displayed by infection in pancreatic diseases have made it difficult to establish criteria capable of clearly distinguishing between the various pus-characterized pancreatitis states.

Trusting in the homogeneous judgment of our surgical team (who have been working together since 1970), we reviewed the hospital records of patients in which both the terms *pancreatitis* and *pus* appeared in the operative report. Essentially, in the 108 cases admitted to this study, we noticed 2 kinds of descriptions: necrotic tissue with mixed pus, and pus as "pure" material in a well-filled mass. Retrospectively, these 2 pus patterns were well correlated with the various diseases involved: more than 90% of the cases of necrotizing pancreatitis without interruption between the toxic and the septic phases belonged to the first pus pattern (i.e., mixed with a large amount of necrotic tissue).

Such a situation can be considered as a macroscopic expression of infection present in 52 (43.4%) of our 120 patients surgically treated after the fourth day from onset of necrotizing pancreatitis [19–21]. In these patients, we noticed positive cultural findings of necrotic tissue in 85 (70.8%) samples: pus was detected in 52 (61.2%), while 33 (38.8%) were infected without pus [23].

Such a condition is called "infected phlegmon" by some authors [3, 11]; others [3, 10, 15, 24, 25] prefer to use the term "abscess." In agreement with the Ulm team [2], the term *infected necrosis* (with or without pus), in our opinion, avoids confusion and should be used. In these cases, the coexistence of multiple organ failure may be misleading and sepsis overlooked. It may be deduced that this did not happen in patients with predisposing pancreatitis occurring, on average, 17 days before, and initially completely cured as regards the "enzymatic toxic phase." In fact, the majority of these cases (i.e., patients with predisposing pancreatitis) were also well identified by the patterns utilized: more than 80% belong to group B (i.e., presence of an obvious mass of pus).

All the observed pseudocysts occurred in group B. Their pseudowalls were clearly distinguished by surgeons from the above described pus mass after a predisposing episode. This was possible in all cases of pseudocysts in chronic pancreatitis, while the differential diagnosis was more difficult in 2 of 5 cases of pseudocyst after acute pancreatitis because of the incomplete formation of the walls.

The pus collection after predisposing pancreatitis does not have a true pyogenic wall [1, 26] and, on the other hand, "young" pseudocysts infected with pus may mimic the above-mentioned mass of pus. Possibly, the latter may represent the first step in the development of a pus-filled pseudocyst: we think the 2 different situations described are due both to the different observation times (average of 21 days in patients with pseudocysts and 14 days in pus collection in our experience) and to different local reaction capability. In the light of these considerations, the distinct differentiation between primary and secondary pancreatic abscess appears well founded [5, 27]. On the basis of the above described experience, we suggest the use of the following terminology:

1. Infected pancreatic necrosis (with or without pus); certainly, infected necrosis may present little purulence in the retroperitoneal slough, but this finding in our opinion cannot characterize an abscess.
2. Infected pancreatic pseudocyst (with or without pus).
3. Pancreatic abscess: A pus-harboring mass following (after as much as 2 weeks) a predisposing acute pancreatitis, initially in remission. These masses develop either earlier or, probably, with a different mechanism from that of infected pseudocysts; moreover, unlike pseudocysts, these "true" abscesses present a poorly-defined wall.

The different pathological features of the pus in abscess, infected necrosis, and pseudocyst have always been carefully described in our surgical reports, although in the early 1970's the problem of nomenclature was not regarded as being as important as it is today. We have eliminated terms such as peripancreatic infection and sepsis [12], infected pancreatic abscess [13], major pancreatic infection [14], pancreatic sepsis [15], pseudocyst abscess [16], and primary or secondary abscess [5, 27] because of their confused meaning, and we now avoid using the term "phlegmon" because of the different meanings it has in the Latin and Anglo-American schools [3, 25, 26]. In the Latin meaning, a "phlegmon" is always an infected collection, while it is used nowadays to identify noninfected edematous pancreatitis collections.

In conclusion, the presence of pus may characterize the course of acute pancreatitis in many severe cases, but the pancreatic abscess, meaning a well-filled mass of pus after predisposing pancreatitis, is a rare late complication. As re-

gards the relationship between the gross appearance of the pus collections and the underlying pancreatic disease, we note that the simple nomenclature proposed above caters to the 2 pus patterns considered.

The next step is to understand if, in our experience, it is possible to establish a correlation between these patterns and prognostic criteria. At first sight, on the basis of the mortality rate findings in the different groups, there clearly appears to be a more severe prognosis in the spreading than in the localized pattern ($p = 0.02$). Biliary pancreatitis, for example, has been reported as more prone to infection than the others [24, 28]; these data could be confirmed in this study by the higher frequency of biliary pancreatitis (Table 2). As reported by Becker and associates [7], Pollock [29], and in our series, the postoperative cases had the most significant mortality rate (40%, $p < 0.03$) alongside both the post-endoscopic retrograde cholangiopancreatography and traumatic patients (20%). In actual fact, in our experience, there is no significantly different distribution of etiological factors related to the mortality rate between the 2 pus patterns. In our opinion, from a prognostic point of view, etiology is important as regards the acute pancreatitis per se, but not when pus is already harbored.

Signs of systemic sepsis (Table 3) were significantly more frequent in group A than in group B ($p < 3 \times 10^{-5}$) (because of the combined toxin activity damage with early infection of necrotic retroperitoneal tissue) [23, 28]. The higher presence of renal insufficiency and ileus in the spreading pattern supports this hypothesis, as does the greater incidence of complications and the longer period of hospitalization.

The overall analysis of the mean clinical features also shows a greater severity in group A patients. Only the presence of an abdominal mass was significantly more frequent in the localized than in the spreading pattern ($p < 2 \times 10^{-8}$), probably because of the major abdominal manageability of the latter patients. Becker and colleagues [7], Hurley and Vargish [30], and Balldin and Ohlssonn [31] studied the availability of different biochemical data to predict abscess formation and to establish possible prognostic criteria; the results are contradictory: Becker, for example, underlined the prognostic role of glycemia and amylasemia, while a significant "risk factor" was found by Hurley using lactic dehydrogenase, white cells, pO_2, metabolic acidosis, calcium, and fluid sequestration. In our experience, only an overall evaluation of clinical, biochemical, and CT findings is useful to predict the septic risk in acute pancreatitis: the difference shown in Table 4 can only be related, in our opinion, to the different natural history of the more severe cases in group A than in group B.

By contrast with the Stone report [23], we did not find a significantly higher mean age in the group with acute pancreatitis and pus collection than in acute pancreatitis alone (43 years versus 40) [19].

As in the experience of Malangoni and associates [32], CT has changed the trend of our approach, with earlier and more rational surgery. As in other experiences [7, 27], the pathognomonic "soap bubble" sign was detected only in 17 (18.3%) of 93 patients we studied, but the diagnosis can be made on the basis of the frequent clear-cut correlation between CT and clinical-biochemical data. This is especially true of the localized pattern, in which the presence of pus was always suspected preoperatively. Moreover, CT showed the exact number and spatial extent of the collection serving as a guide for correct positioning of the drains [1]. In about 30% of the cases reported by Stricker and Hunt [18], the surgeons underestimated the extent of the lesions. We experienced that, too, in the "pre-CT period," while such a situation has been avoided using CT. Finally, CT makes it easier to evaluate the pus collections at the right time. As Aranka and colleagues [33] emphasize, the improvement in the treatment reported in this field during recent years should also be related to the better nutritional support obtained using total parenteral nutrition [34]. The negative findings at cultural studies reported in 23.2% of our studied samples (Table 6) compares with those reported by others [2, 5–8, 10, 11]. This may be explained by the initially sterile nature of the pancreatic necrosis or may, perhaps, be due to the antibiotic prophylaxis, as claimed by Kaushik and associates [8], Donahue and coworkers [5], and Shi and colleagues [9].

Stone and associates [24] and Becker and coworkers [7] do not confirm this hypothesis: surprisingly, in this last experience, deaths associated with abscess are lower in patients who have not received antibiotics (the kinds used are not reported).

We use intravenous and locally administered antibiotic treatment (either for the need to prevent extrapancreatic infections or to carry the antimicrobial agent via the drains into the local site of the disease). Furthermore, some antibiotics which are suitable against the isolated flora responsible for the pus infections have been shown to penetrate pancreatic juice: in the light of this rationale, we deem it necessary to entirely reconsider the disputed antibiotic management of acute pancreatitis [2, 19, 21, 24, 28].

The samples we studied show a higher presence of gram-negative strains of intestinal origin. Polymicrobial cultures are significantly more frequent than monomicrobial cultures ($p = 10^{-8}$). Our microbiological data confirm the experience of Hurley and Vargish in a larger number of observations [30]: patients with more than 1 organism in the pus samples had a 23.5% mortality rate (16 of 68) while monomicrobial infections had a 6.7% mortality rate (1 of 15); these data are not statistically significant, probably because of the low number of monomicrobial samples. No patients with sterile pus or only a single gram-positive agent died ($p = 0.007$), suggesting a prognostic role for culture positivity.

As previously stressed, we use antiprotease substances in the irrigation fluid with the aim of preventing continuation of the necrotizing process, if it is still active. This aspect has been clearly demonstrated by Professor Frey in his discussion of Miller's report [27]: after marsupialization of the abscess on the basis of every-other-day debridements, he noticed relapsing activity of the enzymatic necrosis.

The only chance we have of combating such a condition from a conservative point of view (as an alternative to the continued open-packing debridement) is the topical use of antiprotease drugs via drains, whose usefulness has been demonstrated in experimental models [35] but, so far, not in human clinical practice.

We have no surgical experience with marsupialization, passive drainage, resection, or debridement only and have used the technique proposed by Bradley and Fulenwider in only 3 cases [36]. This is due to the tempting results obtained using lavage after a correct positioning of drains (guided by CT) in the upper

and in the more distal portions of the collection: employing this technique, our overall mortality rate (15.7%) is rewarding, especially if we consider the localized pus pattern in which the mortality rate decreases to 9.5%.

The drains—if possible, two-way which should allow a reduction of the total numbers of input lavage drains employed—should be inert and soft, in order to not damage the neighboring organs and to avoid the most important complications—fistulas and hemorrhages; the latter is the most feared complication. It is probably due both to drainage decubitus and to the inherent enzymatic activity of the pancreatic juice. Now, we currently use silastic tubes of a suitable size to drain both pus and necrotic material completely [32, 37]. The several techniques proposed yielded results similar to those reported by Becker and associates [7]. This suggests that the experience of the surgical team can improve on the basis of improvement in each technique used [38, 39]. The extraperitoneal approach can always be chosen, whenever possible, because it avoids peritoneal pus contamination. This is feasible in well-delineated collections or in reoperations, but not in the continuous necrotizing process, in forms of biliary origin or in the multiple collections, situations which represent the majority of cases reported, thereby justifying the fact that we opted for the extraperitoneal route only in 15% of cases.

In conclusion, in our opinion, the pus pattern enables us to identify the underlying pancreatitis diseases and to use a correct terminology, distinguishing infected necrosis from pancreatic abscess and infected pseudocyst; moreover, it may represent a prognostic factor. Our data confirm the experience of Becker and colleagues [7] in which the relationship between severity of predisposing pancreatitis and prognosis of ensuing pus collection is clearly demonstrated.

The highest mortality rate we reported is that in the patients suffering from severe necrotizing pancreatitis without discontinuity between the toxic and septic phases (23%), whereas our mortality rate decreases in the 23 patients with predisposing pancreatitis in remission (17.4%), and is absent in patients with pseudocysts because of their less toxic clinical behavior, as the reports by Miller and Donahue have suggested [5, 27].

Chronic patients may present pus as a consequence of severe attack (without mortality in this study) and after endoscopic retrograde cholangiopancreatography as in our deceased patients.

Résumé

Voici une étude portant sur 108 cas, dans le même service, entre 1970 et 1987, de collections pancréatiques caractérisées par la présence de pus décelé à la chirurgie, soit diffus (mêlé à du tissu nécrotique) soit en collection nettement localisée. Au premier type appartiennent 47 cas de pancréatite nécrosante évoluant progressivement de la phase toxique primitive à la phase septique terminale, 4 cas de pancréatite aiguë d'abord en rémission mais compliquée par la suite de collections septiques, et 4 cas survenant au décours d'une poussée aiguë sur pancréatite chronique. Le deuxième type compte 19 cas de pancreatites avec présence de pseudokystes, 19 cas de pancréatite familiales, 10 cas au cours de pancréatites chroniques, et 5 seulement au cours de pancréatites nécrosantes progressives. Dans tous les cas le traitement a comporté drainages multiples et irrigation postopératoire, si nous exceptons 3 cas de laparostomie. Nous rapportons et discutons les caractéristiques étiologiques, cliniques et biochimiques des 2 groupes de patients. L'arrivée de la tomodensitométrie semble être le progrès le plus important réalisé en ce qui concerne diagnostic et tactiques thérapeutiques. Le taux global de mortalité rapporté est de 15.7% avec une différence significative entre les 2 groupes décrits (23.6% pour le premier et 7.5% pour le deuxième). D'après cette expérience, on peut établir un rapport entre l'aspect macroscopique de la collection et la maladie pancréatique sous-jacente avec des différences en fait de pronostic, morbidité, et mortalité. En conclusion, on peut établir une classification simple susceptible de différencier les différentes collections pancréatiques de pus. Alors que la présence de pus peut caractériser l'évolution d'une pancréatite aiguë dans bien des cas, le faible taux d'incidence d'abcès pancréatique "vraie" est souligné.

Resumen

La sepsis es una grave complicación de la pancreatitis aguda. Todavía existe considerable confusión sobre los términos relativos a abscesos y sepsis pancreática. La falta de uniformidad refleja nuestro pobre conocimiento de la patofisiología de las infecciones pancreáticas, así como la necesidad de mejores correlaciones clinicopatológicas. Nos propusimos realizar una revisión de nuestra experiencia con sepsis pancreática a fin de establecer criterios estándar de clasificación.

Entre 1970 y 1980 se operaron 1,090 pacientes por pancreatitis crónica y aguda. Se halló pus en 108 casos (9.9%); estos pacientes se pudieron dividir en 2 grupos que correspondieron a 2 patrones: el grupo A, con inflamación expansiva (mezcla de pus y tejido necrótico), sin límites definidos, y el grupo B, con una masa inflamatoria localizada.

En el grupo A se ubicaron 47 casos de pancreatitis necrotizante en que no hubo interrupción en su evolución clínica desde la fase tóxica temprana hasta la fase tóxica tardía, 4 casos de pancreatitis aguda inicialmente en remisión pero luego complicada por colecciones sépticas y 4 casos de ataque agudo en pancreatitis crónicas. El grupo B estuvo conformado por 19 casos de pseudoquiste y 19 de pancreatitis predisponente, 10 casos de pancreatitis crónica, y 5 casos de pancreatitis necrotizante progresiva.

El tratamiento quirúrgico en todos los casos consistió en drenajes múltiples e irrigación postoperatoria, excepto por 3 casos con drenaje y empaquetamiento abierto (laparostomía). Se informan y se discuten las características etiológicas, clínicas, y bioquímicas de cada grupo de pacientes. La disponibilidad de la tomografía computadorizada aparece como el avance más importante en cuanto a diagnóstico y tratamiento. La mortalidad global fue de 15.7% con una diferencia significativa entre los 2 patrones aquí descritos (23.6% para el primero versus 7.5% para el segundo). Con base en esta experiencia aparece posible establecer una relación entre la apariencia macroscópica de la colección y la enfermedad pancreática de base, con diferencias en términos de pronóstico, morbilidad, y mortalidad.

Finalmente, es posible escoger una nomenclatura capaz de diferenciar las diversas colecciones purulentas pancreáticas. En tanto que la presencia de pus puede caracterizar la evolución de

una pancreatitis severa en muchos casos, se hace énfasis en la baja incidencia de "verdaderos" abscesos pancreáticos.

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Invited Commentary

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Professor Bassi and colleagues have made an industrious contribution to the understanding of pancreatic abscess and other infective complications related to pancreatitis; however, this study was retrospective and included patients seen within a 17-year span. Even though the surgical team remained the same, many features of the diagnosis (the quality of computed tomography images and their interpretation have evolved enormously) and treatment of pancreatic abscesses have changed (even the surgeons are 17 years older!). As they have included several different categories of infective complications due to pancreatitis, and pancreatitis in their patients was either acute or chronic, the course, chemistry, microbiology, treatment, and prognosis of disease seen in this series all differed greatly, and