Identification of pancreas necrosis in severe acute pancreatitis: imaging procedures *versus* clinical staging

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SUMMARY One hundred and five of 395 patients with acute pancreatitis were surgically treated in our clinic from 1981 to 1984. Ninety three of these patients were examined with contrast enhanced computed tomography and/or ultrasound and were clinically assessed according to Ranson's objective criteria before operation. At operation, 77 patients showed necrotising pancreatitis and 16 showed biliary acute interstitial pancreatitis. Ninety per cent of the cases with extensive and 79% of those with minor necroses of the pancreas had been demonstrated with contrast enhanced computed tomography. Ultrasound failed to be diagnostic in 24% of the patients due to meteorism; the sensitivity of the diagnostic studies for pancreatic necrosis was 73% regardless of the extent of the process. Using the early objective signs, seven patients with acute interstitial pancreatitis were classified as having a severe attack, whereas 30 patients with necrotising pancreatitis were categorised as mild attacks. We conclude that the contrast enhanced computed tomography is an aid in deciding on conservative or surgical treatment in a case of acute pancreatitis. Ultrasound does not appear to be an adequate method for determining pancreatic necrosis. The early objective signs fail to sufficiently identify the necrotising form of acute pancreatitis.

Even severe cases of acute pancreatitis can be cured by medical therapy if they are restricted to interstitial inflammation. Necrotising pancreatitis, however, holds a high risk of local infection and generalised sepsis and can, if it involves larger areas of the pancreas, lead to retroperitoneal tissue invasion and progressive toxic organ complications.¹⁻³ These complications could be avoided by the surgical removal of necrotic tissue and exudate.²⁻⁶ This in turn poses the problem of identifying and quantifying the necrosis of the pancreas in time for a successful operation. We therefore conducted a prospective clinical study in which the capacity of contrast enhanced computed tomography, ultrasound, and early objective signs⁷ to assess necrotising pancreatitis was determined by comparing the

preoperative evaluations furthered by these methods with the intraoperative findings.

Methods

PATIENTS

Three hundred and ninety five patients were treated for acute pancreatitis from 1981 to 1984; 163 of these were admitted to the surgical ward for a severe or complicated course of the disease, 105 were surgically treated. Half the patients were referred from outlying clinics. Surgery was indicated when necrotising pancreatitis was suggested by organ failures resistant to therapy and/or laboratory findings of severe acute pancreatitis combined with increasing or persistent abdominal symptoms and when acute cholecystitis caused by gall stone disease was simultaneously present. Ninety three patients were included in a prospective study of diagnostic methods. Twelve patients were excluded because of equipment failure or emergency laparotomy. The com-

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puted tomography and ultrasound studies were evaluated on a routine basis.

BIOLOGICAL AND AETIOLOGICAL DATA

The average age of the patients was 46 years (17–83). Sixty one patients (65.6%) were men, 32 patients (34.4%) women. The aetiologically relevant factors were alcohol abuse in 41 (44.1%), gall stones in 27 (29.0%) and trauma in four (4.3%) patients; the aetiology was unclear in 21 (22.6%) patients. Alcohol abuse was noted as an additional aetiological factor in four of the patients with gall stones (14.8%).

IMAGING PROCEDURES

Seventy seven patients were examined with computed tomography, 75 with ultrasound, and 59 patients with both. The computed tomography used was either Somatom 2 (Siemens) or GE CT/T8800. Each patient received 200 ml of water soluble contrast medium by mouth, scans were then done before and after the i v administration of contrast medium. The slice thickness was 5 mm, exposure time 12 seconds. The systems sector scan Siemens, the linear array scan Imager 3001 ATL or the MK 100 ATL were used for the ultrasound examinations.

CLINICAL CLASSIFICATION AND DATA

All 93 patients were clinically assessed and shown to have signs and symptoms of complicated acute pancreatitis after their admission to the surgical ward. Sixty five patients (69.9%) had systemic complications. Pulmonary insufficiency ($pO_2 < 60$ mmHg) occurred in 39 patients, acute renal insufficiency (serum creatinine > 1.4 mg/dl) in 27, shock (systolic blood pressure < 80 mm Hg) in 13 and sepsis (body temperature $> 38.5^\circ\text{C}$, chills, metabolic acidosis, platelet reduction, raised WBC in 18 patients. A paralytic ileus was found in 13 patients, eight patients had gastrointestinal bleeding. Eighty seven patients presented with leukocytosis, 60 with raised blood sugar concentrations LDH was raised in 35 of the 68 patients in whom it was determined and 51 of 78 patients had hypoalbuminaemia of less than 3.5 g/dl. These parameters were not determined in 25 and 15 patients, respectively, because the routine laboratory protocol was not followed on admission. Serum amylase was raised in 75 patients.

The clinical data were then reviewed for the criteria of Ranson's objective signs⁷ and the patients classified accordingly as having 'mild' or 'severe' acute pancreatitis. Ten of the 11 objective criteria were evaluated; fluid sequestration ($> 6 \frac{1}{24}$ h) was omitted because of the difficulty involved in assessing it accurately. Except for the LDH mentioned above and the serum calcium, which was not

determined in two patients, all 10 criteria were completed in each patient. The Ranson criteria were calculated in 45 secondary referral patients (48.4%).

We do not consider these criteria as prognostic signs, but as an abbreviated formula for clinical severity which, in conjunction with the imaged morphological findings, provides an impression of the urgency of surgical intervention.

MORPHOLOGICAL CLASSIFICATION AND DATA

The pancreas was exposed ventrally and the adjacent structures and retroperitoneal spaces were inspected. The necrotic tissue was either manually or instrumentally removed and the extent of the pancreatic necrosis determined by the weight and dimensions of the extracted pancreatic tissue: tissue of up to 3×5 cm and 50 g was classified as a 30% necrosis, tissue of up to 5×8 cm and 120 g as a 50% necrosis, necrosis involving the entire pancreas and weighing more than 120 g was graded as subtotal, a weight of over 190 g was considered a total pancreatic necrosis. Only two categories were used for the purpose of this study – that is, minor necrosis and extensive necrosis. The latter refers to pancreatic necrosis of 50% to 100%, which was found to be associated with a significantly higher morbidity.⁴ Specimens of the removed necrotic tissue were submitted for histological evaluation. The samples revealed both necrotic pancreas as well as fatty tissues. Although the categories mentioned above are thus based on approximate values only, they are, as demonstrated by Beger *et al.*,⁴ nevertheless of clinical relevance.

Minor necrosis of the pancreas was found in 35 (45.4%), and extensive necrosis in 26 patients (33.8%). Sixteen of the latter presented with subtotal or total necrosis. Bacteriological samples were submitted in 58 cases, of which 23 (39.7%) showed bacterial contamination of the necrotic tissue at the time of operation. Additional findings and their relationship to the morphological categories are listed in Table 1.

SURGICAL PROCEDURES AND RESULTS

Debridement and postoperative continuous local lavage with additional peritoneal lavage in cases of peritonitis were carried out in most of the patients. In patients with gall stone disease, cholecystectomy and/or choledochotomy with T-drainage was done simultaneously. Biliary surgery was combined with pancreatic drainage in patients with biliary acute interstitial pancreatitis. The postoperative lavage of the necrotised area in the retroperitoneal space was continued until the drainage fluid no longer showed tissue debris and microorganisms. The lavage period averaged 24 (10–115) days.

Table 1 Intraoperative pathomorphological findings

Morphological category	Extrapaneatic necrosis		Pancreatitis associated ascites	
	Patients (n)	%	Patients (n)	%
Acute interstitial pancreatitis* 16 pts	—		6	37.5
Necrotising pancreatitis† 77 patients	40	52.0	44	57.1
Minor necrosis (30% of pancreas) 35 patients	11	31.4	14	40.0
Extended necrosis (50–100% of pancreas) 42 patients	32	76.2	30	71.4

*Acute interstitial pancreatitis

†Necrotising pancreatitis.

Five patients with necrotising pancreatitis (6.5%) and one patient with acute interstitial pancreatitis (6.3%) died during the postoperative period. The median of the postoperative hospital stay was 26 (minimum:1–maximum:116) days (survivals: 26, deaths: 25 days), that of the intensive care period was three (0–42) days (survivals: three deaths: 11 days).

STATISTICAL EVALUATION

Sensitivity (correct positives: correct positives + false negatives) was the category used to evaluate the results of the computed tomography and ultrasound examinations.

Results

CONTRAST-ENHANCED COMPUTED TOMOGRAPHY

Of the 77 patients examined with computed tomography, 10 suffered from acute interstitial pancreatitis, 67 from necrotising pancreatitis. Twenty nine patients showed minor necrosis of the pancreas, 38 showed extensive necrosis (Tables 2 and 3).

Necrotising pancreatitis was determined in 85.1% of the patients. The extent of the pancreatic necrosis was correctly assessed in 72.4% of the minor and in 65.8% of the extensive necrotising processes. Minor necroses were overestimated in 6.9%, a false negative interpretation as acute interstitial pancreatitis was made in 20.7% of the patients. In 23.7% of the patients, extensive necroses were interpreted as minor processes, whereas 10.5% of the diagnoses were false negative (Table 3). As a whole, the necrotising process as such was identified more often in patients with an extensive necrosis (89.5%) than in patients with minor necrosis of the pancreas (79.3%).

Table 2 Assessment of pancreas necrosis by contrast-enhanced computed tomography

Intraoperative findings	Computed tomography diagnosis			
	Necrotising pancreatitis		Other findings in the pancreas	
	Patients (n)	%	Patients (n)	%
Necrotising pancreatitis 67 patients	57*	85.1	10†	14.9
Acute interstitial pancreatitis 10 patients	5‡	50.0	5§	50.0

*Right positives.

†False negatives: 8 patients acute interstitial pancreatitis, 1 patient acute interstitial pancreatitis in calcifying chronic pancreatitis, 1 patient tumour of the pancreas.

‡False positives: 3 patients minimal necrosis, 2 patients necrosis of approx. 30% of the pancreas.

§Right negatives.

Table 3 Assessment of pancreas necrosis of different extent by contrast enhanced computed tomography

Intra-operative findings	Computed tomography diagnosis					
	Minor necrosis		Extended necrosis		Other findings in the pancreas	
	Patients (n)	%	Patients (n)	%	Patients (n)	%
Minor necrosis* 29 patients	21	72.4	2	6.9	6	20.7
Extended necrosis† 38 patients	9	23.7	25	65.8	4	10.5

*approx. 30% of the pancreas.

†50% to 100% pancreas.

Necrotising pancreatitis was also suspected in five of the 10 patients with acute interstitial pancreatitis – that is, as minimal necrosis in three, as a 30% necrosis in two cases, but no pancreatic necrosis was found intraoperatively. It is noteworthy, however, that there was no false positive diagnosis of an extensive pancreatic necrosis.

ULTRASOUND

Seventy five patients were examined with ultrasound. Sixty two patients suffered from necrotising pancreatitis, 13 from severe acute interstitial pancreatitis. The necrotising process was minor in 26, extensive in 36 patients (Tables 4 and 5).

The examinations turned out to be non-diagnostic in 18 patients (24.0%) because of bowel gas collections; this was the case in one-third of the patients with a vast necrotic area. As a whole, only 56.4% of

Table 4 *Assessment of pancreas necrosis by ultrasound*

Intraoperative findings	Ultrasound diagnosis				Non-diagnostic due to meteorism Patients (n) %	
	Necrotising pancreatitis		Other findings in the pancreas			
	Patients (n)	%	Patients (n)	%	Patients (n)	%
Necrotising pancreatitis 62 patients	35	56.4 (72.9)*	13†	21.0 (27.1)*	14	22.6
Acute interstitial pancreatitis 13 patients	2	15.4 (22.2)*	7‡	53.8 (77.7)*	4	30.8

*Percentage of the diagnostic studies

†11 patients acute interstitial pancreatitis, 1 patient calcifying chronic pancreatitis, 1 patient normal pancreas.

‡ four patients acute interstitial pancreatitis, 3 patients normal pancreas.

Table 5 *Assessment of pancreas necrosis of different extent by ultrasound*

Intraoperative findings	Ultrasound diagnosis				Non-diagnostic due to meteorism	
	Necrotising pancreatitis		Other findings in the pancreas			
	Patients (n)	%	Patients (n)	%	Patients (n)	%
Minor necrosis* 26 patients	18	69.2 (75.0)*	6	23.1 (25.0)*	2	7.7
Extended necrosis† 36 patients	17	47.2 (70.8)*	7	19.4 (29.2)*	12	33.3

*Percentage of the diagnostic studies

†The rate of non-diagnostic studies correlated significantly with the extent of the pancreatic necrosis according to the increasingly frequent paralysis of the intestines causing bowel gas collections. Minor and extended necroses were usually not differentiated in the study reports.

Table 6 *Clinical staging in 93 patients with acute interstitial pancreatitis or necrotising pancreatitis*

	Objective signs (n)				
Morphological category	0-2 Patients (n)	%	3-10 Patients (n)	%	Mean value
Acute interstitial pancreatitis 16 patients	9	56.2	7	43.8	2.8
Necrotising pancreatitis 77 patients	30	39.0	47	61.0	4.0
Minor necrosis (30% of pancreas) 35 patients	23	65.7	12	34.3	3.1
Extended necrosis (50-100% of pancreas) 42 patients	7	16.7	35	83.3	4.9

the cases with necrotising pancreatitis were identified by ultrasound. Based on the number of correct examinations, the sensitivity was 72.9%; it was not affected by the extent of the pancreatic necrosis. Extensive pancreatic destruction was imaged in only 70.8% of the cases. A false positive diagnosis of necrotising pancreatitis was arrived at in two of the nine patients with severe acute interstitial pancreatitis.

CLINICAL GRADING

The mean numbers of objective signs listed in Table

6 indicate that necrotising pancreatitis ran a more severe course than acute interstitial pancreatitis and that the clinical severity increased proportionally to the extent of pancreatic necrosis. The clinical variability, however, is considerable. Of the patients with acute interstitial pancreatitis, 43.8% were classified as having severe pancreatitis (three and more signs). On the other hand, 39% of the patients with necrotising pancreatitis and 16.7% of the patients with an extensive necrosis were classified as mild pancreatitis according to the criteria. No deaths occurred among patients with nil to two

signs. Mortality was 11.3% in patients with three and more signs.

Discussion

The routine use of imaging procedures in the diagnosis of acute pancreatitis has substantially contributed to the efficacy of pancreatitis therapy. These procedures permit the visualisation of the pancreas and adjacent tissues and spaces and – by means of follow up examinations – yield an estimate of the dynamics of the inflammatory process. This in turn permits the timely operation of patients with progressive organ destruction.

In our hospital, patients with complicated and severe acute pancreatitis are usually examined with contrast enhanced computed tomography and/or ultrasound in order to identify necrotising pancreatitis. This study reports on the results of these examinations in 93 patients who were investigated prospectively and who were operated because they failed to respond to maximum conservative therapy in the course of pancreatic necrosis or acute biliary disease. The imaging procedures were carried out upon admission and the patients were clinically assessed and classified according to Ranson's objective criteria.⁷ Of the patients so classified, 48% were secondary referrals from outlying clinics. They were referred to our Pancreas Center because their condition deteriorated to a point which exceeded the therapeutic capacity of the primary admission clinic and a secondary assessment was deemed necessary.

Ten of the criteria were used, fluid sequestration was omitted. Sixty five patients had systemic organ complications, 60 patients had diabetes; hypoalbuminaemia was found in 51 of the 78 patients in whom this parameter was determined. Seventy seven patients had necrotising pancreatitis; 16 patients with severe biliary acute pancreatitis serve as a control group.

The operation was done an average of eight days after admission to the hospital, regardless of whether the referral was primary or secondary. The postoperative mortality was 6.5%. Considering the high risk of most of these patients, this rate is relatively low and reflects the benefits of surgical therapy in necrotising pancreatitis. The rationale for the method used is based on the fact that necrotic tissue releases toxic substances and invites bacterial contamination and intraabdominal sepsis. Removal of the tissue debris can prevent this. That is achieved by thorough intraoperative debridement and continuous postoperative lavage of the retroperitoneal space involved in the inflammatory process for as long as there is evidence of necrotic tissue and/or microorganisms in the drainage.

Computed tomography measures the density of structures. Acute pancreatitis is characterised by loss of density, increase in volume and alterations of the contours of the pancreas and – if the process spreads beyond the pancreatic capsule – of retroperitoneal structures.^{8,9} Necrotising pancreatitis presents as a non-homogeneous density of the pancreas.^{8,10} Chronic pancreatitis, neoplasia, and fatty degenerative changes can, however, look quite similar.¹¹ It is therefore recommended to apply a rapid iv bolus of contrast medium in order to differentiate necrotic portions of the pancreas.^{10,12–14} After the injection, a phasic change of density can be observed, delimiting necrotic tissue and exudative masses as non-perfused areas which retain their original density.¹³

By using this procedure, we detected 85.1% of the cases of necrotising pancreatitis. Extensive necrosis (89.5%) was assessed with greater accuracy than minor necrosis (79.3%), and there was a tendency to underestimate the extent of pancreatic necrosis. On the other hand, small, non-perfused areas were also seen in some of the patients with acute interstitial pancreatitis. Thus a circumscribed pancreatic necrosis diagnosed by computed tomography involves the risk that the necrotic area is in fact larger or else that there is no necrosis in the pancreas at all. The imaging of minor perfusion loss therefore necessitates a follow up of examination in order to exclude false positive diagnoses of necrotising pancreatitis. A follow up is advisable at any rate in patients with severe symptoms in cases like this, because pancreatic perfusion in necrotising pancreatitis may be normal initially and fail completely a few hours later. According to our data, the diagnosis of an extensive necrotising process offers a reliability of 90% and can be accepted as an indication for surgery.

There are only few studies available on the presentation of necrotising pancreatitis in computed tomography investigations. The rates of sensitivity range from 89–100%.^{8,15,16} Intravenous contrast medium was applied in only one study. In this study, perfusion deficits were noted in eight of nine patients whose necrotising pancreatitis was verified at operation.¹⁶ Another study carried out without iv contrast medium reported that exudate or haemorrhage were seen in eight of nine patients,¹⁵ a similar investigation found necrotising pancreatitis presenting as volume increase and in homogeneous structure of the pancreas with exudative masses.⁸ Non-enhanced computed tomography was not able to supply a definite identification and quantification of the necrotising process. Figure 1 shows a picture of exudative pancreatitis as imaged by the non-enhanced computed tomography. The pancreas is

not seen. Figures 2 and 3 show the demarcation of necrotic pancreatic regions as imaged by the contrast enhanced computed tomography.

Sonography uses the reflection of sound waves at the contact surfaces of structures with different acoustic impedance.¹⁷ The diagnosis of acute pancreatitis is based on the attributes, size, form, contour, and echogenicity of the pancreas,^{9 18} a decrease of echogenicity being the primary characteristic.¹⁹

Necrosis presents a non-homogeneous echo which can also be seen in fibrosis and tumours. Obese abdominal walls (which reduce the penetration of sound waves) or meteorism (which absorbs sound waves) render the imaging of the pancreas inaccurate or impossible.¹⁷ Twenty to sixty five per cent of the studies in patients with acute pancreatitis could not be evaluated because of meteorism.^{9 11 14}

Ultrasound examinations were non-diagnostic be-

Fig. 1 Acute interstitial pancreatitis with peripancreatic exudate. Computed tomography (CT) before application of contrast medium. The pancreas and exudative masses cannot be differentiated. P=pancreas, E=exudate, A=aorta, D=duodenum, C=V cava inf, K=kidney, S=spleen.

Fig 2 Necrotising pancreatitis. Necrosis of the pancreas tail (contrast-enhanced CT). P=pancreas, S=spleen, K=kidney, GB=gall bladder, white markings=necrotic tail of the pancreas.

Fig 3 Necrotising pancreatitis. Total involvement of the organ (contrast-enhanced CT). P=pancreas, E=exudate, A=aorta, C=V cava inf, S=spleen, K=kidney.

cause of flatulence in 24.0% of our patients; the rate was even higher in patients with extensive necrotising pancreatitis. As a consequence, necrotising pancreatitis was determined in only 56.4% of the cases. The sensitivity of the diagnostic studies was 72.9%. The extent of the necrotic areas was not distinctly visualised, and large pancreatic necroses did not improve the sensitivity.

The diagnostic potential of ultrasound in necrotising pancreatitis was thus considerably less than that of contrast enhanced computed tomography. If all types of acute pancreatitis are considered, both procedures arrive at a sensitivity of 80%.¹⁷ This does not include necrotising pancreatitis, however, because there has as yet been no systematic study of its assessment by ultrasound. Ultrasound should preferably be used to ascertain the diagnosis and follow the course of mild acute pancreatitis.

Several attempts have been made to predict the severity of acute pancreatitis.^{7 20 21} McMahon and coworkers²⁰ used diagnostic peritoneal lavage to assess 72% of the cases of severe acute pancreatitis, 71% by means of Imrie's²¹ and 82% by means of Ranson's⁷ criteria. Imrie and associates²² reported that they correctly predicted 53% of the severe attacks by means of diagnostic peritoneal lavage and 61% by using multiple criteria.²¹ Emphasis is placed on the fact that not all patients which fulfill the

criteria of severe pancreatitis actually run a severe course of disease.^{23 24} The clinical outcome was the main criterium for the predictive correctness of the studies. There has as yet been no systematic study of the relationship between morphological alterations and the clinical criteria.

Although our study does show a correlation between the number of objective signs and morphological progression, 44% of the patients with acute interstitial pancreatitis were classified as having severe acute pancreatitis, whereas 39% of the patients with necrotising pancreatitis were categorised as mild pancreatitis. Among the latter were seven patients with a 50–100% necrosis of the pancreas with the corresponding extra-pancreatic manifestations. Considering the therapeutic implications – conservative management in patients with the mild form, intensive medical care and further therapeutic measures in patients with severe pancreatitis – 8% of the patients would have been overtreated, whereas 39% would have been condemned to a prolonged course of disease and the risk of septic complications. With the exception of those cases with mild abdominal complaints lasting only a few days, the discrepancy between clinical and morphological alterations is evidently too prevalent to justify the sole reliance on clinical data in the management of acute pancreatitis.

The mortality rates in our study showed only a rough correlation with the categories of objective signs and were considerably lower than those stated in a study which emphasises the non-surgical management²² of severe acute pancreatitis.

The results of our study make it quite clear that no safe conclusion concerning the degree of morphological alteration in the pancreas can be drawn from the clinical signs and symptoms of acute pancreatitis and, in particular, from Ranson's criteria. This is also supported by the fact that only 60% of the patients with severe acute pancreatitis referred to us from other hospitals for possible surgical therapy actually required an operation. About one third of the patients recovered with medical treatment and the computed tomography imaging showed no major perfusion deficit. Ultrasound is inadequate in cases of severe acute pancreatitis, because it lacks both imaging capability if meteorism is present and sensitivity for necrotising pancreatitis in the diagnostic examinations. Contrast enhanced computed tomography aids in the decision of whether to continue medical treatment or to operate on a patient, because it images extensive necrosis of the pancreas with high sensitivity. A computed tomography examination showing a minor perfusion deficit requires a follow up to determine whether this was because of a transient disturbance of circulation or the initial stage of progressive pancreatic necrosis.

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