Arabian Journal of Science

ISSN:2308-5703

Impact factor:13.5 *International Multidisciplinary Online Journal* July, 2026

DIAGNOSIS INDICATORS OF DAMAGE TO THE PANCREATIC DUCT IN ACUTE PANCREATITIS

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Abstract. Contrast-enhanced CT was performed in 227 patients, in 67 patients – on the 2nd and 3rd days from the disease onset. Damage of the pancreas was estimated according to the configuration of necrosis: the depth of necrosis in the sagittal plane (transverse necrosis) and the mutual location of necrosis and healthy parenchyma. At the 1st type of configuration it was more distal than necrosis, at the 2nd type – absent. Peripancreatitis was assessed according to Ishikawa criteria, we studied alpha-amylase activity in fluid collections. The effect of the depth of necrosis and its volume (the Balthazar criterion) on the severity of peripancreatitis was compared. Necrotizing pancreatitis was detected in 200 patients: in 132 patients – type 1 (necrosis of the head, isthmus, body, initial part of the tail), in 30 – type 2 (necrosis of the tail), in 38 – necrotizing pancreatitis with localization of acute necrotic collections only in the retroperitoneal tissue. Advanced peripancreatitis was revealed in 80% of patients at early term and was present in 19 of 26 patients with early organ failure. It was more severe in type 1 in comparison with type 2 by Ishikawa criteria (5.28 ± 0.25 vs 4.27 ± 0.43, $p < 0.05$), by accumulation volume (278 and 166 cc, $p < 0.05$), by presence of high (over 1000 units/L) alpha-amylase activity 1in fluid. The depth of necrosis greater than the volume determined the degree of peripancreatitis in the first and second CT studies (data from multivariate variance and regression analyses). Deep transverse necrosis in patients with type 1 configuration and high alpha-amylase activity in fluid collections are early signs of disconnected pancreatic duct syndrome. They determine the severity of peripancreatitis and systemic complications. Computed tomography allows you to diagnose this in the first 2–3 days from the onset of the disease.

Keywords: pancreas, necrotizing pancreatitis, the depth of necrosis, configuration of necrosis, peripancreatitis, disconnected pancreatic duct syndrome, computed tomography

Introduction The problem of damage to the pancreatic duct (PD), known in the literature as disconnected pancreatic duct syndrome (DPDS), has been considered for many years as a problem of chronic pancreatitis. Insufficient attention is paid to the issues of depressurization of the ductal system of the pancreas (pancreas) in the early stages of necrotizing pancreatitis.

DPDS diagnostics are not standardized. The first systematic review on the diagnosis of DPDS in severe and moderate OP, presented by an authoritative pancreatitis study group, reported 100% sensitivity of the diagnosis using ERCPG and alpha-amylase activity in the fluid secreted through drainage. The sensitivity of MR pancreaticography, including using secretin, was 83%, and the specificity was 100%. The sensitivity of CT scans of the abdominal cavity ranged from 0 to 80%.

Future tasks include studying the level of duct damage and the degree of damage (partial or complete rupture). These data indicate the urgency of the problem and the great interest of researchers.

The purpose of the work. To study the signs of damage to the pancreas in the early phase of necrotizing pancreatitis.

Materials and methods. A retrospective study (2018-2023) was conducted, which included 227 OP patients, 163 (72%) of them were admitted on the first day of the disease. The patients underwent CT with intravenous bolus administration of a contrast agent. The images obtained in the native, arterial and venous phases of the study were analyzed. Pancreatic necrotic changes were assessed in

areas where there was no accumulation of contrast agent according to generally accepted density units. The localization of necrosis in various parts of the pancreas (head, isthmus, body, tail), its volume (according to Balthazar), depth in the sagittal plane (transverse necrosis), and the presence of viable pancreatic parenchyma located distally to necrosis were studied. The depth of necrosis during CT was studied by the depth of necrotic lesion of the parenchyma of the gland perpendicular to its central axis. Images in the axial, coronal, and sagittal planes were studied. Conventionally, 3 degrees of transverse necrosis were identified: 1st degree – <50% of the lesion in the sagittal plane, when necrosis affected less than 1/2 of the cross-section; 2nd degree – >50% of the lesion, that is, more than 1/2 of the cross-section; 3rd degree – 100%, if preserved lobules were not detected in a perpendicular plane relative to the central axis of the pancreas glands. Necrosis <50% of the parenchymal cross-section was considered shallow, suggesting that the probability of damage to the pancreas would be lower than with deep necrosis (>50% and 100%). Special attention was paid to the syntopy of necrosis and viable pancreatic parenchyma (necrosis configuration). With type 1 configuration, a viable pancreatic parenchyma remained distal to the transverse necrosis zone; with type 2 configuration, it was not present.

Local complications (parapancreatitis) were assessed according to the classification of K. Ishikawa et al. Widespread parapancreatitis was considered to be changes corresponding to grade III–V, local – grade I–II according to this classification. About the type of peripancreatic accumulations (acute peripancreatic fluid accumulation (APFA) or acute necrotic accumulation (ANA)) They were judged according to criteria proposed by the international consensus Classification of Acute Pancreatitis 2012: revision of the Atlanta classification and definitions by international consensus.

A comparative analysis of various characteristics of pancreatic necrosis and the severity of retroperitoneal tissue damage was performed. In 62 patients with necrosis localized in the head and isthmus of the pancreas, the incidence of RP was studied depending on the volume of viable parenchyma located distal to the necrosis, which was calculated using the formula for objects of irregular configuration: V = A H In H With H 0.5, where A is length, B is width, C is height, 0.5 is the coefficient. The severity of systemic complications in the early phase of the disease was assessed using the APACHE II, MODS II (Marshall) scales. The presence of an internal pancreatic fistula was judged by the high activity of α-amylase in the fluid (>1000 u/l) obtained by puncture of the clusters.

Statistical analysis was performed using IBM SPSS Statistics 19 and MS Excel 2007 programs. The groups were checked for the normality of the distribution using the Kolmogorov–Smirnov method, and the variance for uniformity was carried out using the Livign criterion. Quantitative indicators of different groups were compared using the Student's criterion (for 2 comparison groups), the Bonferroni-adjusted Student's criterion (for comparison >2 groups), analysis of variance (for comparison >2 groups), the paired Student's criterion for comparing the indicator before and after treatment, and the Dunnet criterion for comparing several groups with the control group. The qualitative indicators of the various groups were compared using the criterion $\chi 2$. The linear relationship between the two quantitative features was assessed using the Pearson correlation coefficient. To determine the degree of influence of each of the independent variables (depth and volume of pancreatic necrosis) on the distribution of the dependent variable (prevalence of parapancreatitis), a multifactorial analysis Of Variance (ANOVA) was used. To identify a correlation between a dependent variable and a set of several independent variables within each group, multiple regression analysis was used to determine the regression coefficient β for each independent predictor and its statistical significance (p). The significance level (p-level) was determined as two-way. The differences in the indicators were considered significant at a significance level of less than 5% ($p < 0.05$).

Results and discussion

1. Pancreatic necrosis and parapancreatitis. The severity of pancreatic damage is usually assessed by the Balthazar CT severity index - the sum of the scores of pancreatic necrosis (the criterion is the volume of necrosis) and inflammatory changes, the most significant of which are determined by the number of fluid accumulations. The author showed that as the index values increase, the number of complications of the disease and mortality increase, which makes it possible to consider it as a factor in predicting the course of the disease. However, recent international consensus studies have not noted the advantages of this indicator over clinical data determining the severity of AP. It was concluded that early CT does not improve treatment outcomes. This criterion does not consider the possibility of damage to the pancreas.



Fig. 1. Fistulogram. Acute necrotizing pancreatitis. The contrast agent is introduced through the nasopancreatic drainage (black arrows), conducted into the pancreatic duct (PD) through the zone of deep necrosis in the isthmus. The duct, pancreas and fluid collection (FC) located outside the pancreas are visible.

In the presented study, it was not the volume of necrosis, but its depth, that was taken as a sign of the severity of necrotic damage to the pancreas and damage to the pancreas. The volume of viable pancreatic parenchyma located distally to deep necrosis was taken as a sign determining the severity of parapancreatitis. Both of these signs are components of the term “pancreatic necrosis configuration" proposed by us. The depth of transverse necrosis >50% increases the probability of inclusion in the necrosis zone of the pancreas, leading to a violation of its patency. Due to the development of hypertension in the ductal system of the pancreas, there is a rupture of the ducts of the 3rd-4th order along its periphery (Fig. 1). Over time, the separation of the pancreatic site occurs, therefore, part of the pancreatic juice will deviate outside the pancreas (into the omentum, retroperitoneal) and determine the conditions for the formation of internal pancreatic fistula and parapancreatitis.

According to CT data, necrotizing pancreatitis was detected in 200 (88%) of 227 patients: 162 (71%) had pancreatic necrosis, 38 (17%) had parapancreatic necrosis (ANA in retroperitoneal tissue) without pancreatic necrosis. In 27 (12%) patients, pancreatic necrosis and necrosis in retroperitoneal tissue were not detected, which indicated the presence of interstitial pancreatitis. The nature of peripancreatic accumulations in necrotizing pancreatitis corresponded morphologically to ANA, and in interstitial pancreatitis to APFA.

1.1. Necrosis configuration and parapancreatitis. The first type of necrosis configuration was detected in 132 (81%) of 162 patients with pancreatic necrosis, type 2 in 30 (19%;). In type 1

configuration, transverse necrosis was located in the head of the pancreas in 28 (21%) patients, in the isthmus – in 49 (37%), in the body – in 17 (13%), in several departments – in 38 (29%) patients, with the isthmus of the pancreas involved. Deep transverse necrosis of the pancreas was detected in 90 (68%) of 132 patients with type 1 configuration of necrosis, most often noted with localization in the isthmus – in 39 (80%) of 49 patients – and multiple necrosis – in 36 (95%) of 38. For the head, this indicator was 39% (11 out of 28 patients), for the body – 24% (4 out of 17 patients). Shallow necrosis prevailed in the pancreatic head and body, respectively: 17 (61%) and 13 (76%; $p < 0.001$) compared with patients with isthmus lesions. With type 2 configuration, deep transverse necrosis was diagnosed in 22 (73%) of 30 patients, which was comparable to type 1 configuration.

Configuration type	**Characteristics of parapancreatitis according to Ishikawa**				***p***
	shallow necrosis (<50%)		**shallow necrosis (>50% и 100%)**		
	the number of patients abs.	**significance**	**the number of patients abs.**	**significance**	
1	42	3,9 ± 0,3	90	5,6 ± 0,25	<0,001
2	8	2,6 ± 0,57	22	4,27 ± 0,37	<0,05
p	-	<0,05	-	<0,01	-

Table 1. Severity of peripancreatitis (by 2 flanks) depending on the depth of transverse necrosis

Despite the same number of patients with deep pancreatic necrosis in types 1 and 2 of the configuration, the prevalence of parapancreatitis in type 1 was significantly higher than in type 2, totaling 5.28 ± 0.25 and 4.27 ± 0.43 on the right and left flanks according to Ischikawa ($p < 0.05$). In type 1 configuration, right-sided and left-sided lesions of retroperitoneal tissue were equally noted (2.5 ± 0.15 and 2.78 ± 0.14), in type 2 configuration of necrosis, parapancreatitis developed mainly in the left type (2.92 ± 0.22), on the right the indicators corresponded to 1.35 ± 0.3 ($p < 0.01$). In the table. 1 presents data on the severity of parapancreatitis in 162 patients with deep and shallow transverse pancreatic necrosis. With an increase in the depth of necrosis, the degree of parapancreatitis increased, while significantly higher values were noted in patients with type 1 configuration of necrosis. The differences related not only to the spreading factor, but also to the nature of parapancreatic accumulations: in type 1, the structure of inflammatory changes was dominated by a liquid component with high alpha-amylase activity (>1000 u/l), which confirms the presence of an internal pancreatic fistula, in type 2 – infiltrative, and in the presence of liquid, alpha-amylase activity was low. At the same time, the volume of accumulations before any intervention in type 1 of the necrosis configuration was significantly higher than in type 2: 278 ± 34 and 166 ± 45 cm3 ($p < 0.05$).

1.2. Viable parenchyma located distal to necrosis and parapancreatitis. The frequency of widespread parapancreatitis was calculated for different volumes of viable pancreatic parenchyma located behind transverse necrosis. The analysis of the variation series (n = 62) allowed us to form 3 groups, the values of which in terms of the volume of viable parenchyma of the gland significantly ($p < 0.01$) differed from each other: 13.86 ± 0.8 cm3 (n = 15), 28.4 ± 0.98 cm3 (n = 34) and 53.1 ± 2.9

cm3 (n = 13). As the volume of viable parenchyma behind necrosis increased, the frequency of RP increased, which was detected respectively in 5 (33%), 21 (62%) and 12 (92%) patients. The significance of the differences between groups 1 and 2 was p = 0.07, between groups 1 and 3 - p < 0.01, between groups 2 and 3 - p < 0.05. In the same patients, RP with shallow necrosis was detected in 6 (24%) of 25 patients, with deep (>50%) necrosis – in 13 (54%) of 24 patients (p = 0.03), with complete transverse necrosis – in 9 (69%) of 13 patients (p < 0.01 compared to shallow necrosis). There were no significant differences between the number of patients with RP with deep transverse necrosis (>50% and 100%).

1.3. Does the depth of transverse necrosis or its volume determine widespread parapancreatitis? To comparatively assess the effect of criteria for the volume and depth of pancreatic necrosis on the prevalence of parapancreatitis, multifactorial variance and regression analysis was used in patients with types 1 and 2 of necrosis configuration before any interventions during the first and second CT scans. A multifactorial ANOVA analysis of the effect of independent variables of necrosis depth and volume on the prevalence of parapancreatitis in patients with type 1 configuration at the first CT scan (on 5.8 days, n = 90) showed a significant effect of necrosis depth on the overall prevalence of parapancreatitis (p = 0.009) and its prevalence on the right flank (p = 0.013). A significant effect of necrosis volume was found only on the right flank (p = 0.016). On the second CT scan (day 14, n = 29), there was a significant effect of the depth of necrosis on the overall prevalence of parapancreatitis (p = 0.053) and its spread along the right flank (p = 0.042). There was no reliable effect of necrosis volume on the severity of parapancreatitis. In patients with type 2 necrosis, the first CT scan (6.5 days, n = 20) revealed a significant (p = 0.01) effect of the depth of pancreatic lesion on the overall prevalence of parapancreatitis. There was no significant effect of necrosis volume. During the second CT scan (16.7 days, n = 11), there was no significant effect of the depth and volume of pancreatic necrosis on the prevalence of parapancreatitis.

Regression analysis showed a significant (p < 0.05) direct relationship between the depth of pancreatic lesion and the prevalence of parapancreatitis in both the first and second CT scans in patients with type 1 necrosis. There was no significant relationship between the volume of necrosis and the prevalence of parapancreatitis in this group of patients. Regression analysis in patients with type 2 pancreatic necrosis demonstrated a significant direct relationship between the depth of pancreatic lesion and the prevalence of parapancreatitis only in the first study, with the second such relationship was not noted.

Thus, multifactorial variance and regression analyses also showed that the prevalence of parapancreatic changes in retroperitoneal tissue is primarily related to the depth of transverse pancreatic necrosis. This dependence was pronounced and could be traced in dynamics to a large extent in patients with type 1 configuration of necrosis. The data obtained allow us to draw an important conclusion that the depth of necrosis, in contrast to its volume, in patients with type 1 configuration of necrosis is an important criterion for the progression of parapancreatitis in dynamics, whereas in type 2 configuration of necrosis this indicator is not decisive.

2. Early organ failure, configuration of pancreatic necrosis and parapancreatitis. According to the modern concept of AP, systemic complications caused by organ dysfunction determine the severity of patients' condition in the early phase of the disease (the first week). Early IT was noted in 28 (14%) of 200 patients with necrotizing pancreatitis. The configuration of necrosis was studied in 26 patients with early OF. Two patients underwent only native CT, which allowed them to assess the severity of parapancreatitis, but not the necrotic lesion of the pancreas. In 19 (73%) of 26 patients, type 1 configuration of pancreatic necrosis was observed (in 15 patients, the necrosis zone affected the isthmus of the pancreas), type 2 necrosis was detected only in 4 (15%; p < 0.01). In another 3 (12%) patients, in the absence of necrosis in the pancreas, acute necrotic accumulations in retroperitoneal

tissue were noted. In the configuration of type 1 necrosis, the structure of organ dysfunction was dominated by multiple organ failure (12 (63%) patients), represented by acute respiratory failure (ARF) and acute cardiovascular insufficiency (ACI; n = 3), ARF, ACI and acute kidney injury (ACI; n = 9), which had the character of a constant. In 7 patients, only one was noted, in 3 it was transient. With type 2 configuration, 3 patients had organ failure (ORF or ACI), and 1 had multiple organ failure (ORF, ACI, and ACI).

The analysis indicates a significantly higher severity of parapancreatitis in patients with early OH compared with patients who did not have IT in the early stages ($p < 0.01$). At the same time, patients with three organ insufficiency showed significantly ($p < 0.05$) more pronounced changes in retroperitoneal tissue in both left-sided and right-sided lesions, which confirms the role of parapancreatitis in the development of organ dysfunctions. Almost all patients with pancreatic necrosis (20 out of 23) had deep transverse necrosis (n = 9, >50%, 11 had complete transverse necrosis), while in 14 patients the volume of necrosis did not exceed 30% (2 points according to Balthazar). 17 (74%) of the 23 patients died: 16 had type 1, and 1 had type 2 pancreatic necrosis configuration. Death occurred during the first 11 days (5-11) in 7 patients (6 had insufficiency of three organ systems), in 10 – within 15 to 48 days (insufficiency of three systems was noted in 5). In 6 surviving patients, early organ dysfunction was represented by ORF (n = 3), ACI (n = 1), ORF and ACI (n = 1), ORF and ACI (n = 1).

3. The timing of the formation of parapancreatitis in pancreatic necrosis. According to the international consensus, local complications (parapancreatitis) develop at the end of the first week (first phase) of the disease, and may progress further. The study showed that parapancreatitis does not form at the end of the first week, but already in the first days of the disease. CT on day 2-3 was performed in 67 (41%) of 162 patients with pancreatic necrosis, 54 (80%) had widespread parapancreatitis. This indicates that not only pancreatic necrosis, but also parapancreatitis is a morphological substrate against which systemic complications manifest themselves in the first phase of the disease.

The data obtained allow us to discuss the lack of evidence that the clinical picture is a priority, and CT results only complement the picture, and that early CT does not lead to improved treatment outcomes. Based on the proposed criterion for the configuration of pancreatic necrosis, we can confidently conclude that early CT allows us to identify risk factors for pancreatic damage and widespread parapancreatitis associated with the formation of an internal pancreatic fistula (deep transverse necrosis with distally located viable pancreatic parenchyma). It also makes it possible to identify the “threatened” group of patients early, which accounts for the majority of systemic complications in the first week of the disease and complications of the second phase of AP.

Conclusion. Deep transverse necrosis in patients with type 1 configuration and high activity of α-amylase in fluid accumulations are early signs of damage to the pancreas. They determine the severity of parapancreatitis and systemic complications. CT allows you to get information in the first 2-3 days after the onset of the disease.

References

1. Balthazar E.J., Ranson J.H., Naidich D.P., Megibow A.J., Caccavale R., Cooper M.M. Acute pancreatitis: prognostic value of CT. Radiology. 1985; 156 (3): 767–772. https://doi.org/10.1148/radiology.156.3.4023241.
2. Ishikawa K., Idoguchi K., Tanaka H., Tohma Y., Ukai I., Watanabe H., Matsuoka T., Yokota J., Sugimoto T. Classification of acute pancreatitis based on retroperitoneal extension: application of the concept of interfascial planes. Eur. J. Radiol. 2006; 60 (3): 445–452. https://doi.org/10.1016/j.ejrad.2006.06.014.

3. Banks P.A., Bollen T.L., Dervenis C., Gooszen H.G., Johnson C.D., Sarr M.G., Tsiotos G.G., Vege S.S. Classification of acute pancreatitis – 2012: revision of Atlanta classification and definitions by international consensus. Gut. 2013; 62 (1): 102–111. https://doi.org/10.1136/gutjnl-2012-302779.
4. Kuo D.C., Rider A.C., Estrada P., Kim D., Pillow M.T. Acute pancreatitis: what's the score? J. Emerg. Med. 2015; 48 (6): 762–770. https://doi.org/10.1016/j.jemermed.2015.02.018.
5. Balthazar E.J., Robinson D.L., Megibow A.J., Ranson J.H. Acute pancreatitis: value of CT in establishing prognosis. Radiology. 1990; 174 (2): 331–338. https://doi.org/10.1148/radiology.174.2.2296641.
6. Working Group IAP/APA Acute Pancreatitis Guidelines. IAP/APA evidence-based guidelines for the management of acute pancreatitis. Pancreatology. 2013; 13 (4 Suppl. 2): e1–15. https://doi.org/10.1016/j.pan.2013.07.063.
7. Dyuzheva T.G., Dzhus E.V., Ramishvili V.Sh., Shefer A.V., Platonova L.V., Galperin E.I. Early CT predictors of the different form peripancreonecrosis development. Annaly khirurgicheskoy gepatologii = Annals of HPB Surgery. 2009; 14 (4): 54–63. (In Russian)
8. Dyuzheva T. Pancreatic necrosis configuration determines local complications development and the choice of treatment approach in acute pancreatitis. HPB. 2014; 16 (suppl.): 657.
9. Verma S., Rana S.S. Disconnected pancreatic duct syndrome: updated review on clinical implications and management. Pancreatology. 2020; 20 (6): 1035–1044. https://doi.org/10.1016/j.pan.2020.07.402.
10. Garg P.K., Singh V.P. Organ failure due to systemic injury in acute pancreatitis. Gastroenterology. 2019; 156 (7): 2008–2023. https://doi.org/10.1053/j.gastro.2018.12.041.