Original Article

Magnetic resonance imaging (MRI) and clinical features of different parts of the pancreas involved in acute pancreatitis: a cross-sectional study

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Background: Patients with acute pancreatitis (AP) have different sites of pancreatic involvement. The aim of this study was to investigate the differences in magnetic resonance imaging (MRI) findings and clinical features of different sites of involvement (subtypes) in AP, with a view to complement and complete the classification of AP based on anatomical imaging features.

Methods: We consecutively collected data from inpatients with AP from January 2018 to October 2022 at a tertiary care hospital. The patients with AP were classified into three subtypes by MRI: type I mainly involved the head of the pancreas; type II mainly involved the body and tail of the pancreas; and type III involved the entire pancreas (head, body, and tail simultaneously). We examined the MRI findings and clinical features of the three subtypes, including their prevalence, gender, etiology, age, assessment of severity, prevalence of hypertension, diabetes mellitus, coronary artery disease, laboratory markers, prognosis, necrosis, and the incidence of complications. The three subgroups were analyzed using one-way analysis of variance (ANOVA), Kruskal-Wallis H-test, Chi-squared test or Fisher's exact probability method depending on the data distribution, and logistic regression and linear regression were used to determine the risk factors for poor short-term prognosis of AP and the number of days in hospital. Results were considered statistically significant at $P<0.05$.

Results: Among the 240 patients recruited, the mean age was 51±15 years (range, 12–89 years); 146 (60.83%) were male and 94 (39.17%) were female. Biliary pancreatitis accounted for 45.00% (108/240), hyperlipidemic pancreatitis for 33.75% (81/240), alcoholic pancreatitis for 8.75% (21/240), and unknown etiology for 12.5% (30/240). Some 81.25% (195/240) of the cases were edematous pancreatitis, whereas 18.75% (45/240) were necrotizing pancreatitis. Overall, 75 patients (31.25%) had type I AP, 108 patients (45.00%) had type II AP, and 57 patients (23.75%) had type III AP. These three subtypes were significantly different in terms of etiology, incidence of diabetes, C-reactive protein (CRP), severity, incidence of necrosis, local complications, clinical and imaging severity scores, and prognosis ($P<0.05$). Total pancreatic involvement (Type III) was the most severe subtype, with hyperlipidemia as the main cause. Regression analysis revealed that subtype classification is an important risk factor for prognosis.

Conclusions: We classified AP into three subtypes based on different sites of involvement and revealed

the MRI features and clinical characteristics of each subtype of AP. The subtype classification helps to characterize AP from the imaging dimension and predict the prognosis. The results of this study could be a target for future studies to adopt new classification methods.

Keywords: Acute pancreatitis (AP); subtype; magnetic resonance imaging (MRI); severity; clinical features

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Introduction

Acute pancreatitis (AP) is a disease in which pancreatic enzyme activation caused by a variety of etiological factors leads to auto-digestion, edema, hemorrhage, and even necrosis of pancreatic tissue, followed by a local inflammatory reaction in the pancreas, with or without functional changes in other organs (1). The incidence of AP has risen steadily over the past decade (2). AP involves dynamic processes and heterogeneity in the pancreatic parenchyma following the initiation of inflammation. Most of these patients present with mild and self-limiting disease, but necrotizing pancreatitis (NP) occurs in 33.2% (3) or more of patients with AP, whose mortality may reach 15% (4). Necrotizing AP tends to evolve into severe AP and is characterized by improper trypsinogen activation and the death of secretory cells, followed by the systemic release of cytokines and inflammatory mediators, which results in inflammatory cell activation, fever, and multiple organ failure (MOF) (5).

Magnetic resonance imaging (MRI) and computed tomography (CT) are imaging methods employed in the diagnosis and differential diagnosis of AP and in the early detection of severe AP. When evaluating AP, MRI is on par with or even outperforms CT (6). The CT severity index (CTSI) or modified CTSI scoring system is extensively used in clinical settings to evaluate the features of AP, including peripancreatic inflammation, pancreatic parenchymal necrosis, and extrapancreatic consequences (7,8). The optimal time for CT scanning in AP is 72–96 hours after the onset of symptoms (1). However, for pancreatitis, it is important to assess the development and severity of the disease as early as possible for clinicians to intervene and treat the disease in a timely manner. In recent years, research has also shown that for patients undergoing CT within 24 hours of symptom onset, the extrapancreatic inflammation on CT (EPIC) score can be used to predict the onset of early organ failure in AP with similar accuracy to conventional scoring systems (9). Similarly, the magnetic resonance severity index (MRSI) (10,11) and extrapancreatic inflammation on MRI (EPIM) scoring system have been used. EPIM is more useful than EPIC in assessing the severity of AP and provides an earlier indication of the onset of severe AP and organ failure (12).

Most published studies have been based on the morphology and local changes of AP on CT/MRI. The morphology of AP on CT/MRI is related to the severity and prognosis of AP. The pancreas is anatomically divided into three parts: the head, body, and tail. According to earlier studies, AP is more likely to occur in the body and tail of the pancreas (10,13). In clinical practice, the presentation of AP in CT/MRI scans may vary among patients, with some demonstrating involvement primarily in the head of the pancreas, others in the body and tail, and still others exhibiting involvement of the entire pancreas. However, their imaging and clinical features have not been reported.

Thus, the aim of this study was to classify AP according to the different parts of the pancreas involved in AP on MRI, to study the differences in the MRI manifestations and clinical features of each subtype of AP, including the prevalence, age, etiology, and severity, and scores, laboratory indices, prognosis, and the type of AP among the three subtypes, as well as to explore the prognostic risk factors of patients with AP. We present this article in accordance with the STROBE reporting checklist (available at https://qims.amegroups.com/article/view/10.21037/qims-24-693/rc).

Methods

Study design

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and was approved by the Ethics Committee of the Affiliated Hospital of North Sichuan Medical College (approval

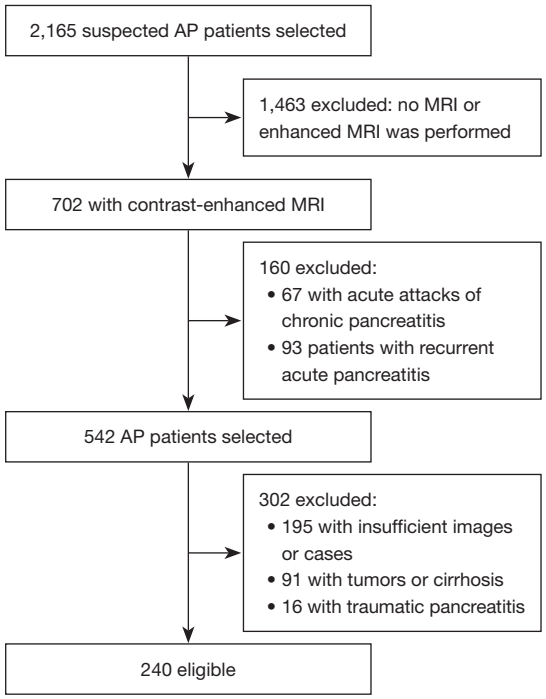

Figure 1 Patient flow chart. AP, acute pancreatitis; MRI, magnetic resonance imaging.

number 2024ER168-1) and the requirement for individual consent for this retrospective analysis was waived. We conducted a cross-sectional study. AP was classified into three subtypes based on the different regions of pancreatic involvement, as revealed by MRI.

We compared the differences between the three subtypes in terms of imaging manifestations and clinical features. Univariate and multivariate regression analyses were then used to identify risk factors associated with short-term prognosis and length of hospital stay in AP patients.

Participants

According to the 2012 revised Atlanta diagnostic criteria for AP (1): two of the following three criteria must be met for a diagnosis of AP: (I) persistent abdominal pain; (II) serum lipase (or amylase) levels more than three times above the normal limit; and (III) typical AP imaging manifestations. Patients who may have had AP were extracted from our hospital's hospital information system (HIS) between January 2018 and October 2022. The medical records of all patients were examined to collect their clinical information. The medical records of all enrolled patients were followed up two to five weeks after the first MRI examination.

The inclusion criteria for this study were as follows: (I) hospitalized patients with a first diagnosis of AP; (II) magnetic resonance (MR) examination within one week of AP onset; and (III) corresponding clinical and laboratory data within three days before and after the MR examination. The exclusion criteria were as follows: (I) no MRI or enhanced MR images; (II) acute attacks of chronic pancreatitis or recurrent AP; (III) incomplete images or incomplete medical records; (IV) tumors or cirrhosis; and (V) traumatic pancreatitis. A flowchart of patient enrollment is provided in *Figure 1*.

We checked the medical records of all included patients on the hospital medical record system and collected their clinical information, such as prevalence of the three subtypes, gender, etiology, age, severity, prevalence of hypertension, diabetes mellitus, coronary artery disease, sodium, potassium, calcium, chloride levels, C-reactive protein (CRP) levels, length of hospital stay and short-term prognosis, incidence of necrosis and complications, as well as Acute Physiology and Chronic Health Evaluation II (APACHE II) (14) and Bedside Index of Severity in Acute Pancreatitis (BISAP) (15). The clinical severity of AP was determined within three days of the MRI scan using the 2012 Revised Atlanta Classification (2012RAC) (1).

MRI

MRI for all patients was performed on two 3.0-T systems [MR750, GE Medical Systems, Waukesha, WI, USA (patients n=143); and uMR790, United Imaging, Shanghai, China (patients n=97)]. The MR750 sequences included the following: coronal and axial single-shot fast spin-echo T2-weighted imaging (SSFSE T2WI), axial fast recovery fast spin-echo T2-weighted imaging (FRFSE T2WI) with fat saturation, T1-weighted in-phase and out-of-phase imaging obtained from three-dimensional liver acquisitions with volume acceleration flex (3D LAVA-flex), and dynamic contrast-enhanced 3D LAVA-flex with fat saturation imaging. uMR790 scanning parameters: T2WI with fast spin echo sequence. T1WI was performed with a three-dimensional (3D) volume interpolated fast scrambled gradient echo sequence and enhancement; coronal magnetic resonance cholangiopancreatography (MRCP) was performed with a single excitation fast spin echo sequence; and 3D LAVA dynamic enhancement was performed with 20 mL of gadolinium (Magnevist; Bayer Schering, Guangzhou, China) administered intravenously at

2–3 mL/s, which was followed by a 20-mL saline solution flush. Dynamic enhancement was performed at 16 seconds (early hepatic arterial phase), 30 seconds (hepatic arterial phase), 60 seconds (venous phase), and 120 seconds (delayed phase) after the injection. Table S1 and Table S2 list the sequences and parameters of these two machines.

MR image interpretation

After the MRI examination was completed, the raw MR data and images were transferred to the picture archiving and communication system (PACS). Two observers (with 6 and 9 years of respective experience in abdominal MRI interpretation) independently examined the AP findings on MRI within a blinded zone of laboratory information and clinical findings. For disputed evaluation results, the results were recorded after consultation and agreement between the two observers. This included the extent of pancreatic parenchymal and peripheral inflammatory involvement, assessment of pancreatic parenchymal and peripancreatic necrosis, and the occurrence of complications. On imaging images, pancreatitis is divided into two types, namely interstitial edematous pancreatitis (IP) and NP. IP is a milder form of pancreatitis without parenchymal or peripancreatic necrosis. It usually presents with localized or diffuse enlargement of the pancreas; there may be peripancreatic effusion and inflammatory changes in the peritoneum and mesentery. We determine pancreatic necrosis by demonstrating on enhanced MRI as speckled, patchy, or large patchy areas of non-enhancement in and around the pancreatic parenchyma. In NP, the area of necrosis includes the pancreas itself and its surrounding adjacent tissues, and is therefore subdivided into three subclasses: pure parenchymal necrosis (PN), extrapancreatic necrosis (EXPN), and peripancreatic and parenchymal necrosis (PPN). The degree of pancreatic necrosis was classified into three grades: less than 30% of the necrotic area, 30–50%, and more than 50% (1). Acute peripancreatic fluid collection (APFC), pseudocysts, acute necrotizing effusion (ANC), and wall of necrosis (WON) are considered complications of AP (1). The MRSI (16) and EPIM scoring systems (17) were used to grade the severity of AP. Mild, moderate, and severe AP were scored by the MRSI (MRSI scores of 0–3, 4–6, and 7–10, respectively) (16,18).

AP was classified into three subtypes based on the different regions of pancreatic involvement, as revealed by MRI (*Figure 2*).

Type I AP is bounded by the abdominal aorta, with inflammation involving only the right pancreatic head and neck, the right paracolic groove, the right peritoneum, the right perirenal fascia, and the duodenum. The body and tail of the pancreas, located to the left of the abdominal aorta, are not affected.

Type II AP is defined as inflammation involving the body and tail of the pancreas on the left side of the abdominal aorta, part of the peritoneum and mesentery on the left side, the left perirenal fascia, the retroperitoneum, and the perisplenic area. The head of the pancreas on the right side of the abdominal aorta and its surroundings are not involved.

Type III AP is defined as involvement of the entire pancreas and may have features of both type I and type II AP as described above, such as inflammation involving the head, neck, body and tail of the pancreas, extensive swelling of the mesentery to the left and right of the abdominal aorta, bilateral thickening of the perirenal fascia, gastrointestinal involvement, and extensive exudation from the pancreas, peri-splenic area, and paracolic grooves on both sides of the colon.

Clinical outcomes of AP patients

It has been shown that two to five weeks after an episode of AP, there is a high incidence of abdominal complications, which can lead to exacerbation and prolonged hospitalization (19). The medical records of all enrolled patients were followed up two to five weeks after the first MRI examination. In the follow-up group, short-term adverse prognostic conditions were defined as a hospital stay of more than five weeks or readmission for worsening AP within two to five weeks after discharge, and within two to five weeks of hospitalization, sudden onset of elevated white blood cell count, organ failure, systemic inflammatory response syndrome (SIRS), worsening of infection, recurrent abdominal pain requiring resuscitation during the hospital stay, or imaging of pancreatic swelling, parenchymal neovascularization, or peripancreatic necrosis, increased ascites, worsening bowel inflammation, or death. Conversely, the absence or reduction of these conditions was defined as improvement.

Statistical analysis

The software SPSS 26.0 (IBM Corp., Armonk, NY, USA) was used to analyze the data. Continuous variables such

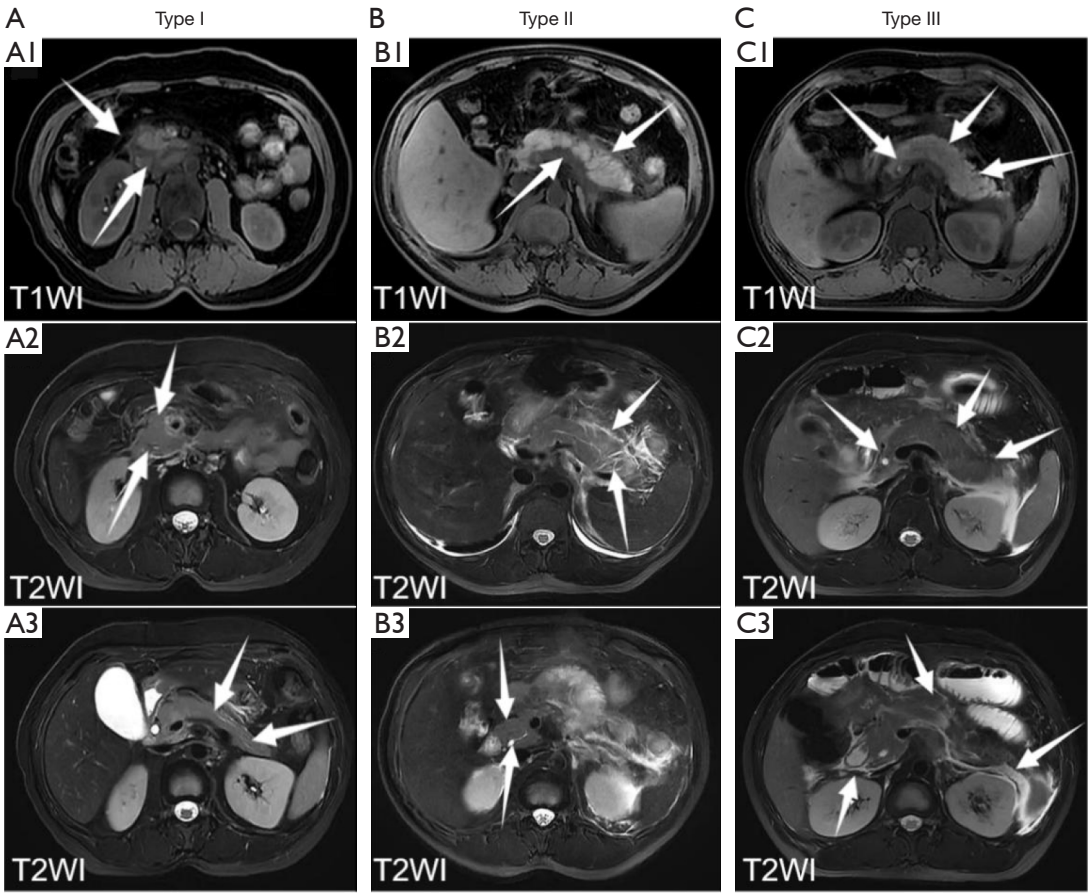

Figure 2 The three subtypes of AP on MR images. (A) A 51-year-old female AP patient with inflammation involving only the head and neck of the right pancreas (A1/A2 white arrows); the body and tail of the pancreas, located to the left of the abdominal aorta, are unaffected (A3 white arrows) (type I). (B) A 61-year-old male AP patient with inflammation involving the body and tail of the pancreas to the left of the abdominal aorta, with blurred peripancreatic fat gaps, thickening of the left perinephric fascia, and part of the mesentery (B1/B2 white arrows); the head of the pancreas and its periphery were not involved (B3 white arrows) (type II). (C) A 57-year-old male AP patient with diffuse pancreatic enlargement, blurring of the peripancreatic fat space, extensive swelling of the adjacent mesentery, bilateral thickening of the perirenal fascia, and extensive peripancreatic oozing (C1/C2/C3 white arrows) (type III). T1WI, T1-weighted imaging; T2WI, T2-weighted imaging; AP, acute pancreatitis; MR, magnetic resonance.

as age, days of hospitalization, biochemical indices such as CRP, APACHE II score, BISAP score, MRSI score, and EPIM score for the three subtypes were expressed as medians (range) or means ± standard deviations, and categorical variables such as sex, etiology, necrosis, and local complications were expressed as n (%). Normally distributed data were analyzed by one-way analysis of variance (ANOVA) and further compared two-by-two using the least significant difference (LSD) test; nonnormally distributed data were compared two-by-two using the Kruskal-Wallis H test and further compared two-by-two using the Bonferroni method. Unordered categorical variables were analyzed using the chi-square test or Fisher's exact probability method. Based on qualitative and quantitative data, variables were first screened using univariate logistic regression and linear regression, respectively, and statistically significant variables screened were analyzed by collinearity analysis using the variance inflation factor (VIF) method, and then variables with no significant collinearity were included in stepwise regression analyses to determine the risk factors for poor short-term prognosis of AP and the number of days of hospitalization. Final results were considered significant at $P<0.05$.

Table 1 Baseline demographic characteristics, clinical and MRI characteristics of the different sites of involvement in acute pancreatitis (N=240)

Characteristics	All (n=240)	Type I (n=75)	Type II (n=108)	Type III (n=57)	P (H;χ^2) value (I *vs.* II *vs.* III)	P value (I *vs.* II)	P value (I *vs.* III)	P value (II *vs.* III)
Age (years)	51±15	51±16	49±15	53±14	0.396	0.459	0.540	0.181
Male gender	146 (60.83)	43 (57.33)	67 (62.04)	36 (63.16)	0.751	0.524	0.500	0.889
Causes					0.012	0.588	0.005	0.012
Bile duct	108 (45.00)	43 (57.33)	55 (50.93)	10 (17.54)				
Hypertriglyceridemia	81 (33.75)	18 (24.00)	32 (29.63)	31 (54.39)				
Alcoholism	21 (8.75)	6 (8.00)	7 (6.48)	8 (14.04)				
Idiopathic	30 (12.50)	8 (10.67)	14 (12.96)	8 (14.04)				
Hypertensive	64 (26.67)	23 (30.67)	26 (24.07)	15 (26.32)	0.613	0.324	0.578	0.758
Coronary heart disease	41 (17.08)	11 (14.67)	18 (16.67)	12 (21.05)	0.034	0.725	0.337	0.479
Diabetes	44 (18.33)	10 (13.33)	17 (15.74)	17 (29.82)	0.034	0.677	0.015	0.026
Na^+ (mmol/L)	137.93±2.657	137.72±2.87	138.07±2.54	138.01±2.62	0.715	0.435	0.545	0.947
K^+ (mmol/L)	3.655±0.273	3.65±0.28	3.67±0.27	3.63±0.28	0.598	0.586	0.644	0.320
Cl^+ (mmol/L)	103.32±2.858	103.27±2.88	103.54±2.96	102.97±2.63	0.470	0.539	0.543	0.224
Ca^+ (mmol/L)	2.235±0.139	2.23±0.14	2.25±0.12	2.21±0.17	0.282	0.437	0.420	0.115
hs-CRP (mg/L)	32.607±21.862	24.16±13.38	30.09±16.63	48.49±30.17	<0.001	0.048	<0.001	<0.001
Prognosis of AP					<0.001	0.012	<0.001	<0.001
Poor	56 (23.33)	4 (5.33)	23 (21.30)	29 (50.88)				
Good	184 (76.67)	71 (94.67)	85 (78.70)	28 (49.12)				
Hospitalization (days)	13.76±7.03	10.91±5.05	12.91±6.179	19.14±7.92	<0.001	0.036	<0.001	<0.001
Local complication	43 (17.92)	2 (2.67)	21 (19.44)	20 (35.09)	0.027	0.054	<0.001	<0.001
Necrotizing AP	45 (18.75)	6 (8.00)	18 (16.76)	21 (36.84)	0.034	0.067	<0.001	<0.001

Data are presented as mean ± standard deviation or number (%). $^+$, indicates elemental ions. MRI, magnetic resonance imaging; Na, sodium; K, potassium; Ca, calcium; Cl, chloride; hs-CRP, high-sensitivity C-reactive protein; AP, acute pancreatitis.

Results

Patient clinical characteristics

This study included 240 AP patients, 146 males and 94 females, with an average age of 51±15 years. All patients completed follow-up. Among the 240 AP patients recruited, 75 (31.25%), 108 (45.00%), and 57 (23.75%) had type I, type II, and type III AP, respectively. The etiology of AP can be categorized into hyperlipidemic, cholestatic, and other causes. Among these patients, 45.00% (108/240) had cholestatic pancreatitis, 33.75% (81/240) had hyperlipidemic pancreatitis, 8.75% (21/240) had alcoholic pancreatitis, and 12.5% (30/240) had other etiologies (*Table 1*). The main etiology of type III AP was hyperlipidemia, whereas the etiologies of type I and type II AP were mainly biliary tract disease. The differences in etiology between type III AP and type I AP and type II AP were statistically significant (P<0.05).

Patients with type III AP had the highest mean CRP levels, whereas those with type I AP had the lowest. The CRP levels were significantly different (P<0.05) among the three subtypes according to post hoc comparisons. However, the differences between the three subtypes were not statistically significant (P>0.05) in terms of age, sex, Na^+, K^+, Ca^+, or Cl^+ (*Table 1*).

The prevalence of diabetes mellitus in patients with type III AP was significantly greater than that in patients with type II and type I AP (P<0.05), whereas the difference in the prevalence of diabetes mellitus in patients with type I and type II AP was not statistically significant, and the

Table 2 Assessment of severity among these three AP subtypes (N=240)

Characteristics	All (n=240)	Type I (n=75)	Type II (n=108)	Type III (n=57)	P (H;χ^2) value (I *vs.*II *vs.* III)	P value (I *vs.* II)	P value (I *vs.* III)	P value (II *vs.* III)
Severity of AP (2012RAC)					<0.001	0.096	<0.001	<0.001
Mild	174 (72.5)	69 (92.00)	79 (73.15)	26 (45.61)				
Moderate	38 (15.83)	2 (2.67)	21 (19.44)	15 (26.32)				
Severe	28 (11.67)	4 (5.33)	8 (7.41)	16 (28.07)				
APACHE II	3 (2, 5)	3 (1, 5)	3 (2, 5)	4 (3, 7)	0.006	0.363	0.026	0.001
BISAP	1 (1, 2)	1 (1, 2)	1 (1, 2)	2 (1, 2)	0.003	0.179	0.037	0.001
MRSI score	3 (2, 5)	3 (1, 5)	3 (2, 5)	4 (3, 6)	<0.001	0.270	<0.001	<0.001
EPIM score	4 (3, 5)	4 (3, 5)	4 (3, 5)	5 (4, 7)	<0.001	0.959	<0.001	<0.001

Data are presented as medians (interquartile spacing) deviation or number (%). AP, acute pancreatitis; 2012RAC, 2012 Revised Atlanta Classification; APACHE II, Acute Physiology and Chronic Health Evaluation II; BISAP, Bedside Index of Severity in Acute Pancreatitis; MRSI, magnetic resonance severity index; EPIM, extrapancreatic inflammation on magnetic resonance imaging.

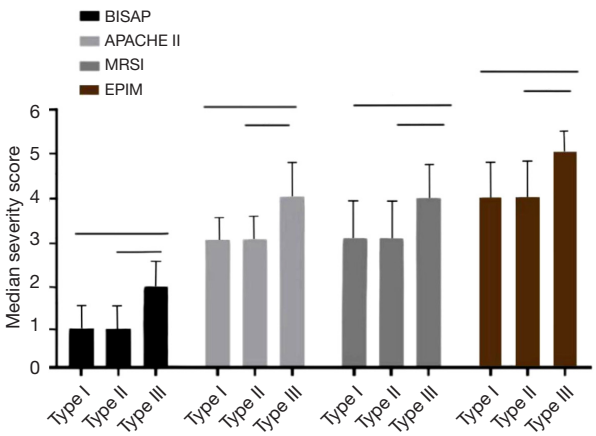

Figure 3 Characteristics of three subtypes of clinical and imaging AP severity scores. BISAP, Bedside Index of Severity in Acute Pancreatitis; APACHE II, Acute Physiology and Chronic Health Evaluation II; MRSI, magnetic resonance severity index; EPIM, extrapancreatic inflammation on magnetic resonance imaging; AP, acute pancreatitis.

difference in the prevalence of hypertension and coronary artery disease was not statistically significant among the three subtypes of AP (P>0.05) (*Table 1*).

MRI characteristics

Of the 240 patients diagnosed with AP, 81.25% (195/240) had edematous AP, and 18.75% (45/240) had necrotizing AP on MRI. The overall local complication rate was 17.92% (43/240). There were 6 (8.00%, 6/75) type I necrosis cases, 18 (16.67%, 18/108) type II necrosis cases, and 21 (36.84%, 21/57) type III necrosis cases. Local complications occurred in two cases (2.67%, 2/75) in type I, 21 cases (19.44%, 21/108) in type II, and 20 cases (35.09%, 20/57) in type III (*Figure 2B,2C*, *Table 1*). Among the three subtypes of AP patients, the prevalence of local complications and necrosis was significantly greater in type III AP patients than in type I and II AP patients, and the difference was statistically significant (P<0.05). However, the prevalence of local complications and necrosis was not statistically significant between type I and type II AP patients (P>0.05).

Comparison of the severity among these three subtypes

The 2012RAC, BISAP, APACHE II, MRSI, and EPIM scores were used to evaluate the severity of these three AP subtypes. According to the 2012 Revised Atlanta Classification (2012RAC), 72.5% (174/240) of the AP patients had mild AP, 15.83% (38/240) had moderately severe AP, and 11.67% (28/240) had severe AP. All of the above scoring details are presented in *Table 2* and *Figure 3*.

The 2012 RAC-based severity classification and BISAP, APACHE II, MRSI, and EPIM scores were significantly different among the three subtypes (P<0.05). Patients with type III AP were more severe, with all scores significantly higher than those with types I and II AP. However, the differences in severity and BISAP, APACHE II, MRSI, and EPIM scores between type II and type I patients were not statistically significant (P>0.05) (*Table 2*).

Table 3 Univariate and multivariate analysis of AP short-term prognosis

Variables	Univariate analysis		Stepwise regression analysis	
	OR (95% CI)	P value	RC (95% CI)	P value
Subgroups	2.284 (1.725 to 3.024)	<0.001	0.118 (0.072 to 0.164)	0.001
Sex	0.819 (0.446 to 1.503)	0.519		
Age	0.999 (0.980 to 1.019)	0.916		
Etiology	0.874 (0.643 to 1.189)	0.391		
Severity (2012RAC)	2.097 (1.573 to 2.797)	<0.001	0.108 (0.056 to 0.161)	0.001
Complications	4.418 (2.192 to 8.904)	<0.001		
Ca^+	0.815 (0.095 to 7.023)	0.852		
Cl^+	0.887 (0.794 to 0.992)	0.353		
Necrosis	3.121 (1.563 to 6.230)	0.001		
APACHE II	1.013 (0.910 to 1.127)	0.817		
BISAP	1.373 (0.975 to 1.934)	0.070		
EPIM	1.198 (0.984 to 1.458)	0.071		
MRSI	1.345 (1.157 to 1.564)	<0.001		

Dependent variable: short-term prognosis. Subgroups: different parts of the pancreas involved in acute pancreatitis. Type I, head of the pancreas; type II, body and tail of the pancreas; type III, whole pancreas (head, body and tail of the pancreas are involved). +, indicates elemental ions. AP, acute pancreatitis; OR, odds ratio; CI, confidence interval; RC, regression coefficient; 2012RAC, 2012 Revised Atlanta Classification; Ca, calcium; Cl, chloride; APACHE II, Acute Physiology and Chronic Health Evaluation II; BISAP, Bedside Index of Severity in Acute Pancreatitis; EPIM, extrapancreatic inflammation on magnetic resonance imaging; MRSI, magnetic resonance severity index.

Comparison of the prognosis among these three subtypes groups

Short-term prognosis and hospital length of stay were used to evaluate the prognosis of AP patients. Of the 240 AP patients recruited, 23.3% (56/240) had a poor short-term prognosis, and 76.6% (184/240) had a good short-term prognosis.

The number of patients with short-term poor prognosis in the three groups were 4 (5.33%, 4/75) cases of type I, 23 (21.30%, 23/108) cases of type II, and 29 (50.88%, 29/57) cases of type III. The length of hospitalization was 10.91±5.05 days for type I, 12.91±6.18 days for type II, and 19.14±7.92 days for type III. Among the three subtypes, type III patients had the longest hospitalization and the most unfavorable short-term prognosis. Subsequently, type II followed, whereas type I had the most favorable short-term prognosis and the shortest average hospitalization duration. Statistical analysis revealed significant differences in the short-term poor prognosis and length of hospitalization among the three subtypes, and post hoc comparisons confirmed that these differences were also statistically significant (P<0.05).

Univariate logistic and linear regression and stepwise regression analysis models for predicting AP prognosis

To explore the risk factors for poor short-term prognosis in patients with AP, univariate logistic regression analysis was used to identify the basic demographic characteristics, clinical characteristics, laboratory indices, and imaging characteristics associated with short-term prognosis. Finally, univariate logistic regression analysis showed that short-term prognosis was significantly correlated with all seven factors in this regression model (P<0.1) (*Table 3*). Collinearity analysis using the VIF method revealed that these seven factors did not have significant collinearity. Subsequently, stepwise regression analyses were performed. The results showed that subtype and severity based on the 2012RAC criteria were risk factors for poor short-term prognosis (*Table 3*), with regression coefficient (RC) even exceeding the severity of 2012RAC.

To explore the risk factors for hospital length of stay in AP, we used univariate linear regression analysis to

Table 4 Linear regression analysis—length of hospital stay

Variables	Univariate analysis		Multivariate analysis	
	RC (95% CI)	P value	RC (95% CI)	P value
Subgroups	2.789 (2.069 to 3.508)	<0.001	1.295 (0.652 to 1.937)	<0.001
Sex	0.362 (–1.465 to 2.188)	0.698		
Age	0.025 (–0.032 to 0.083)	0.388		
Etiology	0.059 (–0.821 to 0.940)	0.895		
Severity (2012RAC)	4.465 (3.747 to 5.183)	<0.001	2.829 (2.017 to 3.641)	<0.001
Complications	9.781 (7.815 to 11.75)	<0.001	2.895 (0.499 to 5.291)	0.019
Ca^+	–2.745 (–9.160 to 3.669)	0.402		
Cl^+	–0.232 (–0.543 to 0.079)	0.146		
Necrosis	7.458 (5.379 to 9.537)	0.001	2.109 (–0.053 to 4.270)	0.057
APACHE II	0.331 (0.014 to 0.648)	0.042	0.075 (–0.167 to 0.318)	0.543
BISAP	0.644 (–0.311 to 1.600)	0.188		
EPIM	0.639 (0.096 to 1.181)	0.022	–0.299 (–0.791 to 0.194)	0.236
MRSI	1.625 (1.220 to 2.030)	<0.001	0.339 (–0.166 to 0.843)	0.189

Dependent variable: length of hospital stay. Subgroups: different parts of the pancreas involved in acute pancreatitis. Type I, head of the pancreas; type II, body and tail of the pancreas; type III, whole pancreas (head, body and tail of the pancreas are involved). +, indicates elemental ions. RC, regression coefficient; CI, confidence interval; 2012RAC, 2012 Revised Atlanta Classification; Ca, calcium; Cl, chloride; APACHE II, Acute Physiology and Chronic Health Evaluation II; BISAP, Bedside Index of Severity in Acute Pancreatitis; EPIM, extrapancreatic inflammation on magnetic resonance imaging; MRSI, magnetic resonance severity index.

identify the underlying demographic characteristics, clinical characteristics, laboratory indicators, and imaging characteristics associated with hospitalization days. The results showed that the number of hospitalization days was significantly correlated (P<0.05) with seven factors in this regression model (*Table 4*), and collinearity analysis using the VIF method revealed that these seven factors did not have significant collinearity. Subsequently, stepwise regression analyses were performed. The results showed that subtype, severity of 2012RAC regulations, local complications, and pancreatic necrosis were risk factors for days of hospitalization (*Table 4*).

Discussion

In this study, for the first time, we classified AP into three subtypes based on the different sites of pancreatic involvement on MRI images. The subtypes differed significantly in terms of etiology, severity, MRI findings, and clinical features. Notably, type III AP (complete pancreatic involvement), in which hyperlipidemia was the main etiology, was the most severe overall and had the worst prognosis. Multivariate regression analyses revealed that subtype classification was an independent risk factor for short-term prognosis, with RC even exceeding the severity of 2012RAC. Thus, subtype classification contributes to characterizing AP from the imaging dimension more comprehensively and predicting patient prognosis. This provides clinicians with new perspectives and ideas for more comprehensive prediction of disease severity as well as early management and treatment of AP.

In the last two decades, a variety of methods for assessing AP severity have been developed based on clinical and imaging criteria (20). Subsequently, various AP classifications have emerged (21). These classifications, which include changes in biochemical markers, the pancreas, peripancreatic tissues, and neighboring organs, are labor intensive and complex (22,23). The innovation of the method in this study lies in categorizing pancreatic inflammation on imaging according to the site of involvement, which has the advantage of being more intuitive and simpler.

AP involves various inflammatory dissemination pathways, mainly involving the pancreatic parenchyma,

retroperitoneal space, and intraperitoneal space. In the present study, AP was classified into three subtypes depending on the site of inflammatory involvement in the pancreatic parenchyma. Type I AP involves the head and neck of the pancreas. It extends anteriorly to the right paracolic groove and peritoneum, reaching the right to part of the duodenum, and posteriorly to the right perirenal fascia. Type II AP involves the body and tail of the pancreas, with inflammation spreading anteriorly to the left paracolic groove, the adjacent mesentery, the peritoneum, and to the left around the spleen and, in a few patients, to the stomach and part of the intestinal tract, as well as spreading posteriorly to the left perirenal fascia. Type III AP involves both type I and type II areas and is therefore more widespread. Currently, CTSI and MRSI score only the extent of inflammation in the pancreas, including pancreatic inflammation and necrosis, but do not reflect the major sites of pancreatic involvement. Our results indicate that subtype classification based on inflammatory involvement in the pancreatic parenchyma is a better indicator for assessing severity and prognosis, suggesting that this classification may complement and complete the assessment of AP imaging.

Regarding etiology, the three subtypes exhibit variations, with hyperlipidemia identified as the primary cause for type III AP, whereas types I and II AP are predominantly associated with biliary AP. The difference in the etiology of type III AP from that of types I and II AP was statistically significant, whereas there was no statistically significant difference in the etiology of type I and type II AP. Research has shown that hyperlipidemia is more serious than other causes of AP (24).

Pancreatic lipase breaks down triglycerides into free fatty acids (FFAs). FFAs have direct cytotoxic effects on pancreatic cells and vascular endothelial cells and increase inflammatory mediators such as tumor necrosis factor-α (TNF-α), interleukin (IL)-6, and IL-10, exacerbating the inflammatory response (25). In contrast, biliary AP is less severe than hyperlipidemic AP, possibly because biliary AP is mainly caused by biliopancreatic duct obstruction, which leads to increased pancreatic duct pressure, bile reflux, trypsin activation and pancreatic autodigestion, whereas pancreatic microcirculatory obstruction is less severe and is less likely to lead to the development of severe pancreatitis (26).

Based on the comprehensive clinical and imaging features, our findings revealed that patients with type III disease experienced the most severe form of the disease, resulting in a poorer prognosis, followed by patients with type II disease, whereas those with type I disease experienced less severe disease. The key distinctions lie in the fact that, in comparison to type I and type II AP, type III AP manifests a greater incidence of local complications and necrosis, higher clinical and imaging severity scores, a longer mean length of hospital stay, and the poorest short-term prognosis. First, the degree of dissemination or exudation of AP can reflect the severity of the disease, and the greater the extent of dissemination of retroperitoneal inflammation, the greater the severity of AP (27,28). Second, more extensive AP involvement leads to more ischemia in pancreatic tissues, more activated pancreatic enzymes in tissues, and more pancreatic necrosis and local complications, resulting in more severe conditions (29). Finally, the early local and systemic consequences of hyperlipidemic AP are more severe and fatal than those of AP caused by other etiologies (24), making type III AP, which has hyperlipidemia as its primary etiology and the most extensive involvement, more severe.

We also found no statistically significant differences in the relevant imaging evaluation scores between type I and type II patients, but only three clinical indicators (CRP, hospitalization days, and short-term prognosis) were significantly different between type I and type II AP patients. Compared with type I AP patients, type II AP patients had a greater mean CRP, longer mean hospitalization days, and worse short-term prognosis. Nevertheless, we did not find differences in imaging-related indicators between type I and type II AP patients, possibly because imaging is only used to assess lesions in the pancreas and peripancreatic tissues, whereas CRP, days of hospitalization, and short-term prognosis are also affected by other underlying diseases.

In addition, we found that subtype classification was an independent risk factor for short-term prognosis. The 2012RAC is currently the most widely used consensus for assessing the severity of AP, but our results showed that the new subtype classification system also exhibited good performance in evaluating prognosis, which underscores the importance of considering not only 2012RAC severity but also the specific subtype in predicting short-term prognosis. This may have profound implications for clinical decision-making and treatment strategies. Additionally, our study prompts a reconsideration of the existing classification systems, suggesting that a more nuanced approach may be necessary to capture the diverse manifestations of this condition. This could pave the way for the development of more tailored and effective interventions based on the

specific characteristics of each subtype. Therefore, this new subtype classification can provide a good complement for characterizing AP and predicting patient prognosis from an imaging perspective, as well as providing clinicians with new perspectives and ideas for more comprehensive prediction of disease severity as well as early management and treatment of AP.

However, our study has several limitations. First, this was a single-center study, and the inclusion of the study population may have been subject to selection bias. However, we implemented strict inclusion and exclusion criteria, with a high representation of patients and a wide range of patients, from male to female, young to old, with different etiologies, but of course, multi-center, large-sample studies are still needed in the future. Secondly, the five-week follow-up period in this study was relatively short. Our team continues to collect and conduct studies on pancreatitis and will increase the follow-up time in the future to look at the long-term clinical outcomes of the patients. Lastly, this study only dealt with the site of inflammation, and did not consider the extent of inflammation, the presence or absence of necrosis, or the occurrence of local complications. These factors could be considered in combination with these indicators in the future, or even with CTSI or MRSI, to develop a more complete imaging assessment system.

Conclusions

Different parts of the pancreas involved in patients with AP on MRI correspond to different disease severities and prognoses. Type III AP (total pancreatic involvement) is the most severe, with a greater incidence of SAP, higher clinical severity scores, MRSI and EPIM scores, a greater percentage of patients with concomitant diabetes, longer hospitalization, a greater incidence of complications and necrosis and the worst prognosis. This subtype is an independent risk factor for predicting poor short-term prognosis and days of hospitalization for AP and contributes to a comprehensive assessment of the severity of AP. The results of this study may support reconsideration of the classification of pancreatitis or an increase in anatomical distribution.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://qims.amegroups.com/article/view/10.21037/qims-24-693/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-24-693/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and was approved by the Ethics Committee of the Affiliated Hospital of North Sichuan Medical College (approval number 2024ER168-1) and the requirement for individual consent for this retrospective analysis was waived.

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