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Association Between Ascites and Clinical Findings in Patients with Acute Pancreatitis: A Retrospective Study

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Study Design A
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Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Background: Complications are the most important outcome determinants for acute pancreatitis (AP). We designed this single-center retrospective study to evaluate the clinical findings (complications, disease severity, and outcomes) of 218 patients with AP and to identify variables associated with ascites.

Material/Methods: We extracted clinical data from consecutive patients with AP and divided them into 2 groups based on presence or absence of ascites. We compared disease severity, complications, and outcomes between groups.

Results: We analyzed data from 218 patients with AP (43 with ascites and 175 without it). The patients with ascites had a more severe disease (higher incidence of pancreatic inflammation [90.70% vs 68.57%; P=0.003], higher modified computed tomography severity index score [2.00 (0.00-2.00) vs 4.00 (4.00-6.00); *P*<0.001], higher incidence of moderate/severe AP [53.49% vs 13.14%; *P*<0.001]) and poorer outcomes (higher incidence of ventilation [6.98% vs 0.57%; *P*=0.025] and vasopressor use [4.65% vs 0%; *P*=0.038], and longer hospital stays [10.00 (7.00-13.00) vs 8.00 (5.00-10.00); *P*=0.007]) than those without ascites. Moreover, patients with ascites also displayed a higher risk for pancreatic fluid collection (odds ratio [OR]=9.206; 95% confidence interval [CI], 2.613-32.447; *P*<0.001), renal failure (OR=5.732; 95% CI, 1.025-32.041; *P*=0.024), respiratory failure (OR=6.242; 95% CI, 1.034-37.654; *P*=0.029), and pleural effusion (OR=5.186; 95% CI, 1.381-19.483; *P*<0.001) than those without ascites.

Conclusions: The findings from the experience of a single center of patients with AP showed that pancreatic fluid collections, renal failure, respiratory failure, and pleural effusion were associated with the development of ascites.

Keywords: Ascites • Pancreatitis • Risk Factors • Treatment Outcome

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2374 6 — 32

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Background

Acute pancreatitis (AP) is the second most common cause of prolonged total hospital stay and the fifth leading cause of in-hospital deaths [1]; and, its incidence has increased [1,2]. Local and systemic complications (including pancreatic fluid collection, pancreatic necrosis, renal failure, respiratory failure, and cardiovascular failure) occur frequently in patients with AP [3]. These complications – especially organ failure – are the most important determinants of patient outcomes [4]; AP mortality is approximately 3% in patients without complications, 15% in those with pancreatic necrosis, and up to 35% in those with persistent organ failure [5]. In addition, a population-based cohort on patients with AP showed that the median time from the onset of systemic complications to death was as short as 3 days [6]. Moreover, the mortality rate due to complications is high even after the first 2 weeks [4]. Because complications are not always obvious in the early stage of the disease, understanding the risk factors of AP complications would be of great value in helping clinicians to prevent, identify, and carry out timely management of these complications [7].

Ascites is caused by leakage of pancreatic secretions into the peritoneum [8] and damage of capillary walls and plasma extravasation [9], and it occurs in up to 38.5% of patients with AP [10]. Ascitic fluid contains an abundance of pancreatic proteases, pancreatic lipases, and inflammatory cytokines that are highly toxic and lethal [11] and also contribute to the development of intra-abdominal hypertension [10]; thus, ascites is an independent predictor of the severity and poor prognosis of AP [12]. Although complications govern the outcome and mortality in patients with AP, limited data on the association between pancreatic ascites and AP complications exists [13]. Ascites can be easily detected in the diagnostic images of early-stage AP. Discerning whether ascites is associated with AP complications may improve prompt detection and management of those and thereby reduce complication-related mortalities. This retrospective study identified 218 patients diagnosed with AP at a single center and evaluated their clinical manifestations (including complications, disease severity, and outcomes), comparing those in patients with and without ascites.

Material and Methods

Ethics Statement

The Institutional Review Board of Maoming People's Hospital, Maoming, China approved this study (No. 2020MI-141-01). The requirement for informed consent was waived by the Institutional Review Board on account of the retrospective study design.

Participants

Data from all adult (≥18 years) inpatients with AP at Maoming People's Hospital from January, 2015 to December, 2019 were eligible for inclusion in the study. We excluded data from patients with a history of pancreatitis, those with ascites caused by liver or kidney disease, those with carcinoma, pregnant patients, and those with incomplete medical information.

Data Collection

We extracted the following data from electronic medical records of the eligible patients: demographic characteristics, smoking status, alcoholism status, medical history, etiology, complications, treatment, outcomes, imaging findings, and laboratory parameters (including white blood cell and platelet counts, serum levels of amylase, calcium, alanine aminotransferase, aspartate transaminase, total bilirubin, triglycerides, blood glucose, hematocrit, creatinine, and blood urea nitrogen).

Definitions

Alcoholism was defined as a daily alcohol consumption >30 g/day in men and >20 g/day in women. Hypocalcemia was defined as serum calcium levels <2.12 mmol/L [14]. Ascites was diagnosed in patients with peritoneal fluid around the sublienal space and among intestinal loops by ultrasonography or computed tomography (CT) [15]. The diagnosis of acute pancreatitis required the presence of 2 of the 3 following criteria: (1) characteristic epigastric abdominal pain; (2) elevation of serum amylase or lipase levels to more than 3 times the upper normal limit; and, (3) abdominal imaging findings consistent with the diagnosis [16]. The severity of AP was classified based on the revised Atlanta criteria: mild (no organ failure or local or systemic complications), moderately severe (organ failure resolving within 48 h and/or local or systemic complications without persistent organ failure [<48 h]) and severe (persistent organ failure [>48 h]) [16]. Local (pancreatic fluid collections and pancreatic necrosis) and systemic complications (renal failure, circulatory failure, and respiratory failure) of AP were diagnosed as follow: (1) Pancreatic fluid collection was diagnosed based on the presence of a homogeneous collection of dense fluid adjacent to the pancreas without evidence of pancreatic necrosis and confirmed by CT [17]. (2) Pancreatic necrosis was diagnosed based on the presence of a heterogeneous and non-liquid density of varying degrees in different pancreas locations confirmed by CT [17]. (3) Renal failure was defined as a serum creatinine >177 μmol/L [18]. (4) Circulatory failure was defined as systolic blood pressure < 90 mmHg [18]. (5) Respiratory failure was defined as PaO_2 <60 mmHg [18]. Pleural effusion was diagnosed as blunting of the costophrenic or cardiophrenic angle confirmed by CT [19]. Pancreatic inflammation was defined as focal or diffuse enlargement of

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Table 1. Patient characteristics.

	No-ascites (n=175)		Ascites (n=43)		P value
Age (years)	54.70±18.05		52.65±19.37		0.656
Male sex	103	(58.86)	28	(65.12)	0.453
Alcoholism	13	(7.43)	7	(16.28)	0.132
Smoking status					0.919
Never	158	(90.29)	38	(88.37)	
Former	4	(2.29)	1	(2.33)	
Current	13	(7.43)	4	(9.30)	
Hypertension	21	(12.00)	7	(16.28)	0.452
Diabetes	15	(8.57)	3	(6.98)	0.975
CCI	2.00	(1.00-3.00)	1.00	(0.00-3.00)	0.289
Etiology					0.330
Gallstones	76	(43.43)	15	(34.88)	
Hypertriglyceridemia	35	(20.00)	10	(23.26)	
Alcohol	5	(2.86)	1	(2.33)	
Post-ERCP	0	(0)	1	(2.33)	
Medications	13	(7.43)	2	(4.65)	
Idiopathic	46	(26.29)	14	(32.56)	

Data are presented as means±standard deviations, medians (interquartile ranges) or n (%). CCI – Charlson Comorbidity Index; ERCP – endoscopic retrograde cholangio-pancreatography.

the pancreas with or without intrinsic pancreatic inflammatory changes [20]. We calculated the Charlson Comorbidity Index [21] and modified CT severity index (CTSI) as published [20]. Briefly, we assigned a score of 1, 2, 3, or 6 to 19 different medical condition categories (such as myocardial infarct, lymphoma, and metastatic solid tumor) to add them all up and obtain an overall Charlson Comorbidity Index score [21]. We assessed the modified CTSI based on the severity of 3 indicators (pancreatic inflammation, pancreatic necrosis, and extrapancreatic complications). Using this modified index, we categorized the AP severity as mild (0-2 points), moderated (4-6 points), or severe (8-10 points) [20].

Statistical analysis

We used SPSS (IBM Corp., Armonk, NY, USA) to analyze the statistical data. Continuous data are presented as means±standard deviations for normally distributed variables, and medians and interquartile ranges for non-normally distributed variables. We compared continuous variables using the *t* test for normally distributed variables or Mann-Whitney U test for non-normally distributed variables between patients with and without ascites. Categorical data are presented as frequencies and proportions, and we compared them using the chi-squared test between 2 groups. To assess the associations between ascites and severity, complications, and outcomes of AP, we performed a multivariate logistic regression analysis with a backward conditional method, and a multivariable linear regression (with patients without ascites as the reference group) to adjust for potential confounders (ie, age, sex, hypocalcemia, blood glucose level, and white blood cell count) [22,23]. We expressed results as odds ratios (ORs) with 95% confidence intervals (CIs) and beta with standard errors (SE). A two-sided *P* value <0.05 was considered to indicate statistical significance.

Results

Patient Characteristics

Based on the inclusion and exclusion criteria, we analyzed data from 218 patients with AP. During the course of the disease, 43 patients (19.72%) developed ascites (41 diagnosed by CT and 2 by ultrasonography). **Table 1** summarizes the findings of a comparison of the characteristics between the patients with and without ascites. We found no significant intergroup differences with respect to demographics, comorbidities, or AP etiology. In addition, laboratory results were similar between the 2 groups of patients, except for hypocalcemia (**Table 2**).

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Table 2. Laboratory variables in patients with or without ascites.

	No-ascites (n=175)	Ascites (n=43)	P value
Serum amylase (U/L)	809.00 (246.06-2029.86)	403.00 (147.00-1600.00)	0.103
Hypocalcemia	34 (19.54) (n=174)	15 (34.88) (n=43)	**0.031**
WBC (10^9/L)	11.44 (8.87-15.53)	12.75 (7.64-17.05)	0.785
Platelets (10^9/L)	225.00 (180.00-269.00)	235.00 (174.00-266.00)	0.794
ALT (U/L)	52.90 (17.80-225.70) (n=163)	52.60 (24.60-134.20) (n=43)	0.962
AST (U/L)	42.70 (22.28-135.43) (n=164)	52.90 (25.60-117.40) (n=43)	0.515
Total bilirubin (μmol/L)	27.30 (17.80-54.30) (n=163)	22.80 (16.40-57.00) (n=43)	0.449
Triglycerides (mmol/L)	1.20 (0.79-2.65) (n=152)	1.23 (0.76-6.38) (n=38)	0.623
Blood glucose (mmol/L)	7.14 (5.34-10.09) (n=174)	6.69 (4.89-8.93) (n=43)	0.242
Hematocrit (%)	41.30 (37.50-45.30)	40.70 (36.80-44.20)	0.379
Serum creatinine (μmol/L)	86.50 (74.21-99.40) (n=161)	81.70 (68.15-101.77) (n=42)	0.335
BUN (mmol/L)	4.11 (3.22-5.36) (n=171)	3.98 (3.00-6.53) (n=43)	0.780

Data are presented as medians (interquartile ranges) or n (%). ALT – alanine aminotransferase; AST – aspartate transaminase; BUN – blood urea nitrogen; WBC – white blood cells.

Table 3. Disease severity and complication-related variables in patients with or without ascites.

	No-ascites (n=175)	Ascites (n=43)	P value
Disease severity			
Pancreatic inflammation	120 (68.57)	39 (90.70)	**0.003**
Modified CTSI	2.00 (0.00-2.00)	4.00 (4.00-6.00)	**<0.001**
Severity			**<0.001**
Mild	152 (86.86)	20 (46.51)	
Moderate/severe	23 (13.14)	23 (53.49)	
SIRS	42 (37.71)	12 (27.91)	0.595
Complications			
Pancreatic fluid collection	15 (8.57)	20 (46.51)	**<0.001**
Pancreatic necrosis	1 (0.57)	1 (2.33)	0.356
Renal failure	3 (1.86) (n=161)	5 (11.90) (n=42)	**0.011**
Respiratory failure	3 (1.71)	4 (9.30)	**0.041**
Circulatory failure	4 (2.29)	2 (4.65)	0.742
Pleural effusion	12 (6.86)	13 (30.23)	**<0.001**

Data are presented as medians (interquartile ranges) or n (%). CTSI – CT severity index; SIRS – systemic inflammatory response syndrome.

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Table 4. Outcomes of patients with or without ascites.

	No-ascites (n=175)	Ascites (n=43)	P value
Use of ventilation	1 (0.57)	3 (6.98)	**0.025**
Use of vasopressor	0 (0)	2 (4.65)	**0.038**
ICU admission	2 (1.14)	3 (6.98)	0.054
Mortality	0	1 (2.33)	0.196
Hospital stay (days)	8.00 (5.00-10.00)	10.00 (7.00-13.00)	**0.007**

Data are presented as medians (interquartile ranges) or n (%). ICU – Intensive Care Unit.

Disease Severity and Complications in Patients with and without Ascites

In terms of disease severity, patients with AP and ascites showed a higher incidence of pancreatic inflammation detected by CT scan (90.70% vs 68.57%; *P*=0.003), higher modified CTSI scores (2.00 [0.00-2.00] vs 4.00 [4.00-6.00]; *P*<0.001) and a higher incidence of moderate/severe AP (53.49% vs 13.14%; *P*<0.001) than the patients without ascites. However, the incidence of SIRS was similar in the 2 groups (**Table 3**).

In terms of local complications, patients with ascites showed a higher incidence of pancreatic fluid collection than those without ascites (46.51% vs 8.57%; *P*<0.001). Pancreatic necrosis was almost absent in both groups (**Table 3**).

In terms of systemic complications, renal failure (11.90% vs 1.86%; *P*=0.011), respiratory failure (9.30% vs 1.71%; *P*=0.041), and pleural effusion (30.23% vs 6.86%; *P*<0.001) were significantly higher in the ascites group than in the other group, while the incidence of circulatory failure was similar in the 2 groups (4.65% vs 2.29%; *P*=0.742; **Table 3**).

Outcomes in Patients with and without Ascites

Outcomes such as the incidences of use of ventilation (6.98% vs 0.57%; *P*=0.025) and vasopressors (4.65% vs 0%; *P*=0.038) were significantly higher and the hospital stays (10.00 [7.00-13.00] vs 8.00 [5.00-10.00]; *P*=0.007) were significantly longer in the ascites group than in the no-ascites group. In addition, the patients with AP and ascites had a higher incidence of intensive care unit (ICU) admission (6.98% vs 1.14%), although the difference was not significant (*P*=0.054). Mortality was rare in both groups (**Table 4**).

Multivariate Logistic and Multivariable Linear Regression Analyses

We applied a multivariate logistic regression analysis to assess the associations between ascites and disease severity, complications, and outcomes of AP. Patients with ascites presented

Table 5. Logistic regression analyses of ascites as a risk factor for severe acute pancreatitis and its complications.

	OR (95% CI)	P value
Disease severity		
Pancreatic inflammation	5.332 (1.207-23.576)	**0.004**
Moderate/severe AP	60.531 (13.798-265.461)	**<0.001**
SIRS	–	–
Complications		
Pancreatic fluid collection	9.206 (2.613-32.447)	**<0.001**
Pancreatic necrosis	–	–
Renal failure	5.732 (1.025-32.041)	**0.024**
Respiratory failure	6.242 (1.034-37.654)	**0.029**
Cardiovascular failure	–	–
Pleural effusion	5.186 (1.381-19.483)	**<0.001**
Outcomes		
Use of ventilation	12.900 (1.551-107.260)	**0.029**
Use of vasopressor	–	–
ICU admission	8.108 (1.168-56.279)	**0.032**
Mortality	–	–

Data were adjusted for age, sex, hypocalcemia, blood glucose, and white blood cell counts. Data from patients without ascites served as the reference group. AP – acute pancreatitis; CI – confidence interval; ICU – Intensive Care Unit; OR – odds ratio; SIRS – systemic inflammatory response syndrome.

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Table 6. Multivariate linear regression analysis for ascites as a predictor of severe acute pancreatitis.

	Beta	Standard error	*P* value
Modified CTSI	3.072	0.225	**<0.001**
Hospital stay (days)	–	–	–

Data were adjusted for age, sex, hypocalcemia, blood glucose level, and white blood cell counts. Data from patients without ascites served as the reference group. CTSI – CT severity index.

higher risks of pancreatic inflammation (OR=5.332; 95% CI, 1.207-23.576; *P*=0.004), moderate/severe AP (OR=60.531; 95% CI, 13.798-265.461; *P*<0.001), pancreatic fluid collection (OR=9.206; 95% CI, 2.613-32.447; *P*<0.001), renal failure (OR=5.732; 95% CI, 1.025-32.041; *P*=0.024), respiratory failure (OR=6.242; 95% CI, 1.034-37.654; *P*=0.029), pleural effusion (OR=5.186; 95% CI, 1.381-19.483; *P*<0.001), use of ventilation (OR=12.900; 95% CI, 1.551-107.260; *P*=0.029), and ICU admission (OR=8.108; 95% CI, 1.168-56.279; *P*=0.032; **Table 5**) than the patients without ascites.

Our multivariable linear regression analysis showed that patients with AP and ascites also had a higher modified CTSI than the patients without ascites (beta=3.072, SE=0.225; *P*<0.001; **Table 6**).

Discussion

In this study, we found that patients with AP and ascites had increased risks for more severe disease and worse prognoses than the patients without ascites. More importantly, ascites was also a risk factor for local and systemic complications of AP, including pancreatic fluid collection, renal failure, respiratory failure, and pleural effusion.

The incidence of ascites in patients with AP in our study was 19.72%, which is similar to the incidence reported by Maringhini et al (18%) [12]. Maringhini et al reported that ascites is an accurate independent predictor of AP severity [12]. Jayanta et al also revealed that patients with AP who develop ascites have significantly higher severity scores, more need for interventions, and greater mortality than patients without ascites [10]. Consistent with those results, we found that patients with ascites showed higher risks of moderate/severe AP, and need for ICU admission and ventilation, and higher modified CTSIs than patients without ascites. Ascites can occur in the early stages of AP and can be easily detected by imaging and even physical examinations; therefore, it is of great value to predict AP progression [11].

Although ascites is a sign of severity for systemic inflammatory conditions, whether it can predict AP complications is poorly understood. A previous study found an association between pancreatic ascites and the development of a pseudocyst (a delayed and local AP complication), which usually occurred more than 4 weeks after onset of the pancreatitis [1,12]. Moreover, Jayanta et al also revealed that patients with pancreatic ascites showed higher incidences of local and systemic complications (including pancreatic necrosis, acute lung injury, acute kidney injury, and shock) than patients without ascites [10]. However, that study missed many clinical data (such as alcohol and smoking status, medical history, and laboratory results) important to clarify the association between ascites and AP complications. To overcome these limitations, we compared clinical data between patients with and without ascites, adjusted the potential AP complications' confounders, and confirmed that ascites was a risk factor for local (pancreatic fluid collection) and systemic complications (renal failure, respiratory failure and pleural effusion) of AP in our cohort.

There are different possible explanations for our findings. First, ascites can be caused by the leakage of pancreatic fluid; however, pancreatic proteases and lipases, and inflammatory cytokines from ascitic fluid can also aggravate the inflammatory exudate of the pancreas, which may further contribute to pancreatic fluid collection. Second, ascites accumulation always leads to elevated intra-abdominal pressure, known as intra-abdominal hypertension (IAH) [24]. IAH impairs the venous return from the peripheral circulation to the right heart, which subsequently decreases the cardiac outflow and reduces arterial perfusion, thereby directly compressing the renal parenchyma [25]. Thus, IAH may result in renal function impairment and even renal failure. In addition, IAH also decreases the functional residual capacity of the lungs and the thoracic wall compliance by transmitting the pressure to the thorax [25]. Under these circumstances, acute respiratory distress syndrome and respiratory failure may occur, and ventilation may be needed [26]. Third, lung injury and pleurisy caused by ascites can also promote pleural effusions [27]. However, our data did not show a significant association between ascites and pancreatic necrosis and circulatory failure. This might be due to the limited sample size and the low incidence of these complications in our cohort.

Although complications account for the poor prognoses and most deaths in patients with AP, they often remain undetected, and the time from the occurrence of a systemic complication to the death of the patient can be short [4,28]. Some

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interventions against pancreatic ascites have decreased the prevalence of new complications and have improved the outcomes of patients with AP [13]. For example, an experimental study showed that abdominal paracentesis drainage may ameliorate the illness as well as complications in rats with severe AP by removing various proinflammatory mediators, such as interleukin-1β and tumor necrosis factor-α [29]. Moreover, clinical studies also have shown that removal of pancreatic ascites by peripancreatic percutaneous catheter drainage significantly decreases the incidence of complications and subsequently improves prognoses [30,31]. Therefore, clinicians should monitor the occurrence of AP complications in patients with ascites and provide early medical and surgical interventions (such as percutaneous drainage) to reduce the mortality of these patients.

Our study has some limitations. First, information about the volume and characteristics (including white blood cell counts, albumin levels, and bacterial culture) of the ascites was limited due to the retrospective nature of the study. Thus, we fell short of assessing the association between ascites characteristics (such as its volume) and AP complications. Second, although we analyzed the clinical data to our best abilities, we did not consider genetic factors, such as mutations of SPINK1, IL-1b, IL-10, which greatly influence the risk, etiology, and severity of AP [32]. Third, this was a single-center study with a small cohort. Hence, our conclusions should be interpreted with caution, and large-scale prospective clinical observations are needed to validate our findings.

Conclusions

The findings from the experience of a single center of patients with AP showed that pancreatic fluid collections, renal failure, respiratory failure, and pleural effusion were associated with the development of ascites.

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