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Pleural Effusion Is Associated with Severe Renal Dysfunction in Patients with Acute Pancreatitis

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Background: Renal dysfunction is a leading cause of death in patients with acute pancreatitis (AP) and often occurs later than respiratory complications. Whether respiratory complications can predict renal impairment remains unclear. The aim of this study was to investigate the association between pleural effusion and renal dysfunction in AP.

Material/Methods: Medical records were reviewed from individuals who were hospitalized with AP from January 1, 2015 to December 31, 2019. The patients were divided into 2 groups, based on the presence or absence of pleural effusion on admission. Disease severity, renal function parameters, and outcomes were compared between the 2 groups.

Results: A total of 222 patients were enrolled, 25 of whom had pleural effusion on admission and 197 who did not. Patients with AP who had pleural effusion had more serious illness (higher incidences of pancreatic inflammation, pancreatic fluid collection, and moderate-to-severe AP; worse Bedside Index for Severity in Acute Pancreatitis score; and a higher modified computed tomography severity index [all $P<0.05$]) plus worse outcomes (higher incidences of ventilation and vasopressor use [both $P<0.05$]). Moreover, patients with pleural effusion had a higher level of blood urea nitrogen and lower estimated glomerular filtration rate (both $P<0.05$). After adjustment for potential confounders, pleural effusion was a risk factor for renal failure in patients with AP (odds ratio 6.32, 95% confidence interval 1.08-36.78, $P=0.040$).

Conclusions: Pleural effusion is associated with severe renal dysfunction in AP. Therefore, efforts should be made to improve early recognition and timely treatment of renal failure by closely monitoring renal function in patients with AP and pleural effusion on admission.

Keywords: Pancreatitis • Pleural Effusion • Renal Insufficiency

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Background

Acute pancreatitis (AP) is the most common gastrointestinal cause of hospital admission and the fifth leading cause of in-hospital deaths; its incidence is continuously increasing [1,2]. AP-related organ failure (respiratory, cardiovascular, and renal) is a core indicator of disease severity and accounts for almost all mortality in patients with the condition [3,4]. Renal dysfunction is frequently associated with AP [5] and is a common complication of it, resulting from increased vascular permeability and hypovolemia, intravascular coagulation, and direct nephrotoxic effects, caused by the release of activated enzymes and proteases during AP [6]. The incidence of renal failure (RF) in patients with AP ranges from 14% to 44% [7,8], and although acute RF usually develops after the failure of other organs, it is associated with prolonged hospital stays and a 10-fold higher mortality rate [6,9-11]. Because renal dysfunction is not always obvious in the early stages of AP but leads to a significantly higher risk of death, it is very important to identify predictive factors for it to ensure that supportive therapies are initiated early in the disease course.

Imaging is a requirement for AP diagnosis, and with it, chest lesions including pleural effusion, pneumonia, and atelectasis can be detected in the early stages of the illness. Pleural effusion is found in up to 50% patients with AP [12,13] and is an independent predictor of AP severity [14]. In addition, pleural effusion can reduce cardiac output and produce hypoxemia, which in turn can lead to renal injury, manifested as a decreased glomerular filtration rate (GFR) and renal tubular epithelial cell damage [7]. Moreover, respiratory complications occur at an early stage of AP and are prolonged, while renal complications occur later, implying a potential role for respiratory complications in predicting subsequent renal injury [8]. Although there have been some studies of the association between pleural effusion and AP severity [14,15], whether pleural effusion can predict renal function dysfunction remains unclear.

The aim of this study was to investigate the association between pleural effusion and renal dysfunction in AP.

Material and Methods

Ethics Approval

Ethics approval was obtained from the Institutional Review Board (IRB) of Maoming People's Hospital, Maoming, China (No. 2020MI-141-01). Because of the retrospective design of the study, the IRB waived the need for informed consent.

Participants

In this study, all adult inpatients with AP who were treated at Maoming People's Hospital from January 1, 2015 to December 31, 2019 were eligible for enrollment. The exclusion criteria were a history of pancreatitis, pregnancy, or lack of important medical data.

Data Collection

The following data from enrolled patients were obtained from medical records in the hospital database: demographic information (age and sex); alcohol and smoking status; history of comorbidities; AP etiology (gallstones, alcohol, post-endoscopic retrograde cholangiopancreatography, medications, and idiopathic); physiological variables (heart rate, blood pressure, respiratory rate, temperature, and mental status); laboratory parameters (serum amylase, alanine aminotransferase, aspartate transaminase, total bilirubin, triglycerides, blood glucose, white blood cell [WBC] count, hematocrit, platelet count, blood urea nitrogen [BUN], serum creatinine, arterial pH, partial pressure of oxygen, partial pressure of carbon dioxide, serum potassium, serum sodium, and serum calcium); radiologic findings; and clinical outcomes (use of a ventilator, use of a vasopressor, Intensive Care Unit [ICU] admission, hospital stay, and mortality).

Definitions

In accordance with the revised Atlanta classification, diagnosis of AP required at least 2 of the following 3 diagnostic features: persistent abdominal pain, serum lipase or amylase activity at least 3 times the upper limit of the normal range, and characteristic findings on abdominal imaging [3]. Pleural effusion was diagnosed as blunting of the costophrenic or cardiophrenic angle confirmed by computed tomography (CT) [12]. Alcoholism was defined as ingestion of >30 g/day of alcohol in men and >20 g/day in women. Hypocalcemia was defined as a serum calcium level <2.12 mmol/L [16]. RF was defined as creatinine ≥176.8 μmol/L (2.0 mg/dL) [17]. Severity of AP was stratified into 3 categories – mild, moderately severe, and severe – based on the revised Atlanta criteria [3]. Charlson Comorbidity Index (CCI) [18], estimated GFR (eGFR) [19], modified computed tomography severity index (CTSI) [20], Acute Physiology And Chronic Health Evaluation II (APACHE II) score [21], multiple organ dysfunction syndrome (MODS) score [22], and Bedside Index for Severity in Acute Pancreatitis (BISAP) score [23] were calculated as previously described.

Statistical Analysis

Normally distributed continuous variables were presented as mean±standard deviation and those with skewed distribution

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Table 1. Patient characteristics.

	No pleural effusion (n=197)	Pleural effusion (n=25)	P value
Age (years)	53 (38.50-66.00)	58 (41.50-74.50)	0.186
Male sex	118 (59.90)	16 (64.00)	0.693
Alcoholism	21 (10.66)	1 (4.00)	0.487
Smoking status			0.501
Never	175 (88.83)	24 (96.00)	
Former	6 (3.05)	0 (0.00)	
Current	16 (8.12)	1 (4.00)	
Hypertension	25 (12.69)	4 (16.00)	0.883
Diabetes	14 (7.11)	4 (16.00)	0.252
CCI	1.00 (1.00-3.00)	2.00 (1.00-3.00)	0.269
Etiology			0.712
Gallstones	84 (42.64)	8 (32.00)	
Hypertriglyceridemia	39 (19.80)	6 (24.00)	
Alcohol	7 (3.55)	0 (0.00)	
Post-ERCP	1 (0.51)	0 (0.00)	
Medications	13 (6.60)	3 (12.00)	
Idiopathic	53 (26.90)	8 (32.00)	

Data are presented as median (interquartile range) or n (%). CCI – Charlson Comorbidity Index; ERCP – endoscopic retrograde cholangiopancreatography.

as median and interquartile ranges. Categorical variables were presented as frequencies and percentages. Continuous data were compared using the *t* test or the Mann-Whitney U test, while categorical variables were compared using the chi-square test or Fisher exact test, as appropriate. To assess associations between pleural effusion and disease severity and RF and outcomes of AP, multiple logistic regression analysis, using the backward conditional method, was applied to adjust for potential confounders (age, sex, hematocrit, WBC count, blood glucose, serum triglycerides, and hypocalcemia) [24-27]; odds ratios (ORs) and 95% confidence interval (95%CIs) also were calculated. All data were processed using SPSS software version 22.0 (IBM Corp., Armonk, New York, United States). Results were considered significant at $P<0.05$.

Results

Patient Characteristics

A total of 356 patients with AP were diagnosed from January 2015 to December 2019. Of them, 134 were excluded, and 222 were finally enrolled in the present study. The patients were divided into 2 groups – pleural effusion (25) and non-pleural effusion (197) – based on their imaging results at admission. Information on demographic characteristics and AP etiology is presented in **Table 1**. There was a higher incidence of hypocalcemia in the pleural effusion group than in the non-pleural effusion group (44.00% vs 19.80%, $P=0.007$) (**Table 2**); however, no significant intergroup differences were observed with respect to other laboratory parameters.

Disease Severity

On comparing factors associated with disease severity between the 2 groups, we found that the incidence of pancreatic inflammation (92.00% vs 70.56%, $P=0.023$); pancreatic fluid collection (36.00% vs 13.20%, $P=0.008$); moderate-to-severe AP (52.00% vs 18.27%, $P<0.001$); BISAP score (2.00; range, 1.00-3.00 vs 1.00; range, 0.00-1.00, $P<0.001$); and modified CTSI scores (4.00; range, 4.00-6.00 vs 2.00; range, 0.00-2.00, $P<0.001$) were significantly higher in the pleural effusion than in the non-pleural effusion group (**Table 3**). Further, patients with pleural effusion rather without pleural effusion also had higher APACHE II scores (6.00; range, 3.50-10.00 vs 5.00; range, 3.00-7.00, $P=0.182$) and MODS scores (1.00; range, 1.00-3.00 vs 1.00; range, 0.00-2.00, $P=0.077$), but these differences were not statistically significant. There was no difference in the incidence of systemic inflammatory response syndrome between the 2 groups, and incidence of pancreatic necrosis was negligible in both groups.

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Table 2. Comparison of laboratory parameters between patients without and with pleural effusion.

	No pleural effusion (n=197)	Pleural effusion (n=25)	P value
Serum amylase (U/L)	773.00 (249.03-1944.59)	340.00 (151.58-1360.00)	0.109
WBC (10^9/L)	11.62 (8.72-15.64)	10.89 (7.69-15.46)	0.404
ALT (U/L)	50.15 (18.93-228.80) (n=188)	70.45 (24.2-154.90) (n=22)	0.780
AST (U/L)	45.55 (22.63-140.10) (n=188)	54.10 (29.90-99.90) (n=23)	0.594
Total bilirubin (μmol/L)	27.10 (17.23-50.90) (n=188)	32.80 (18.68-69.60) (n=22)	0.384
Triglycerides (mmol/L)	1.19 (0.77-2.93) (n=171)	1.25 (0.79-3.22) (n=23)	0.714
Blood glucose (mmol/L)	6.88 (5.26-9.83) (n=196)	7.00 (4.69-13.30) (n=25)	0.730
Hematocrit (%)	41.20 (37.15-45.10)	40.90 (36.85-45.30)	0.933
Hypocalcemia	39 (19.80) (n=196)	11 (44.00) (n=25)	

Data are presented as median (interquartile range) or n (%). ALT – alanine aminotransferase; AST – aspartate transaminase; WBC – white blood cell.

Table 3. Comparison of disease severity between patients without and with pleural effusion.

	No pleural effusion (n=197)	Pleural effusion (n=25)	P value
Pancreatic inflammation	139 (70.56)	23 (92.00)	**0.023**
Pancreatic fluid collection	26 (13.20)	9 (36.00)	**0.008**
Pancreatic necrosis	2 (1.02)	0 (0.00)	1.000
APACHE II score	5.00 (3.00-7.00)	6.00 (3.50-10.00)	0.182
MODS score	1.00 (0.00-2.00)	1.00 (1.00-3.00)	0.077
BISAP score	1.00 (0.00-1.00)	2.00 (1.00-3.00)	**<0.001**
Modified CTSI	2.00 (0.00-2.00)	4.00 (4.00-6.00)	**<0.001**
Severity			**<0.001**
Mild	161 (81.73)	12 (48.00)	
Moderate/severe	36 (18.27)	13 (52.00)	
SIRS	47 (23.86)	8 (32.00)	0.374

Data are presented as median (interquartile range) or n (%). APACHE II – Acute Physiology and Chronic Health Evaluation II; BISAP – Bedside Index for Severity in Acute Pancreatitis; CTSI – computed tomography severity index; MODS – multiple organ dysfunction syndrome; SIRS – systemic inflammatory response syndrome.

Renal Function Parameters

A significantly higher level of BUN (5.16; range, 3.91-7.81 vs 3.98; range, 3.14-5.36 mmol/L, *P*=0.003) and lower eGFR (53.61; range, 46.11-70.87 vs 66.76; range, 55.51-80.34 mL/min×1.73 m^2, *P*=0.044) were detected in the pleural effusion group than in the non-pleural effusion group. In addition, patients with AP who had pleural effusion had higher levels of serum creatinine (93.35; range, 78.85-112.73 vs 84.10; range, 72.95-98.00 μmol/L, *P*=0.092) and a higher incidence of RF (12.00% vs 3.55%, *P*=0.131) than those without pleural effusion, but these differences were not significant (**Table 4**).

Outcomes

Outcome measures including the incidences of use of ventilators (12.00% vs 0.51%, *P*=0.005) and vasopressors (8.00% vs 0%, *P*=0.012) were significantly higher in the pleural effusion

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Table 4. Comparison of renal function parameters between patients without and with pleural effusion.

	No pleural effusion (n=197)	Pleural effusion (n=25)	P value
BUN (mmol/L)	3.98 (3.14-5.36) (n=194)	5.16 (3.91-7.81) (n=24)	**0.003**
Serum creatinine (μmol/L)	84.10 (72.95-98.00) (n=185)	93.35 (78.85-112.73) (n=22)	0.092
eGFR [mL/(min×1.73 m^2)]	66.76 (55.51-80.34) (n=185)	53.61 (46.11-70.87) (n=22)	**0.044**
Renal failure	7 (3.55)	3 (12.00)	0.131

Data are presented as median (interquartile range) or n (%). BUN – blood urea nitrogen; eGFR – estimated glomerular filtration rate.

Table 5. Comparison of outcome parameters between patients without and with pleural effusion.

	No pleural effusion (n=197)	Pleural effusion (n=25)	P value
Use of ventilation	1 (0.51)	3 (12.00)	**0.005**
Use of vasopressor	0 (0.00)	2 (8.00)	**0.012**
ICU admission	3 (1.52)	2 (8.00)	0.099
Mortality	0 (0.00)	1 (4.00)	0.113
Hospital duration (days)	8 (5.00-10.50)	10 (6.00-11.00)	0.305

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

than in the non-pleural effusion group; however, the incidence of ICU admission and hospital length of stay did not differ significantly between the 2 groups. Mortality rates were very low in both groups, with only a single death in the pleural effusion group and none in the non-pleural effusion group (**Table 5**).

Multiple Logistic Regression Analysis

Multiple logistic regression analysis was used to explore whether pleural effusion was a risk factor for severe AP, RF, or poor outcomes. Patients with pleural effusion had a higher risk of pancreatic inflammation (OR 4.80, 95%CI 1.05-21.95, *P*=0.043); pancreatic fluid collection (OR 4.15, 95%CI 1.59-10.84, *P*=0.004); moderate-to-severe AP (OR 5.53, 95%CI 2.15-14.24, *P*<0.001); RF (OR 6.32, 95%CI 1.08-36.78, *P*=0.040); and use of ventilation (OR 25.36, 95%CI 2.52-255.66, *P*=0.006) (**Table 6**).

Discussion

In the present study, the incidence of pleural effusion in patients with AP was 11.26% on admission, which was consistent with the previously described range of 4% to 20% [28]. In addition, similar to previous studies [14,29], our data showed that pleural effusion was a risk factor for more severe disease (pancreatic inflammation, pancreatic fluid collection, and moderate-to-severe AP) and worse outcomes (use of ventilation) in patients with AP. However, considering that pancreatic necrosis, ICU admission, and death were rare in our study, associations between pleural effusion and these variables were difficult to assess.

Complications are predictors of disease severity and worse prognosis for AP [30,31]. Some prior studies have shown that pleural effusion is associated with complications of AP. Maruti et al reported that the prevalence of respiratory failure was higher in patients with AP who had pleural effusion on admission than in those without pleural effusion [28]. In addition, Giorgio et al found that pleural effusion on admission was a risk factor for pancreatic necrosis, which is a known local and lethal complication [32]. Moreover, a recent study suggested that the volume of pleural effusion was also a specific indicator for predicting organ failure in AP [14]. Nevertheless, the association between pleural effusion and renal impairment is unclear. Given that renal dysfunction is one of the most common complications and a leading cause of death in AP and the symptoms of renal dysfunction are not obvious in early-stage illness, increased knowledge about the risk factors for renal dysfunction in AP will help clinicians select the appropriate level of care and improve the prognosis for patients with this illness [6,9,33].

Our results suggest that renal function is impaired (higher level of BUN and lower eGFR) in patients with AP who have pleural effusion. In addition, the incidence of RF was higher in patients with than without pleural effusion, although the difference was

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Table 6. Logistic regression analyses of pleural effusion as a risk factor for severe acute pancreatitis, renal failure, and poor outcomes.

	OR (95% CI)	*P* value
Disease severity		
Pancreatic inflammation	4.80 (1.05-21.95)	**0.043**
Pancreatic fluid collection	4.15 (1.59-10.84)	**0.004**
Pancreatic necrosis	–	–
Moderate/severe AP	5.53 (2.15-14.24)	**<0.001**
SIRS	–	–
Renal parameters		
Renal failure	6.32 (1.08-36.78)	**0.040**
Outcomes		
Use of ventilation	25.36 (2.52-255.66)	**0.006**
Use of vasopressor	–	–
ICU admission	–	–
Mortality	–	–

Data were adjusted for age, sex, hypocalcemia, blood glucose, and white blood cell count. AP – acute pancreatitis; CI – confidence interval; ICU – Intensive Care Unit; OR – odds ratio; SIRS – systemic inflammatory response syndrome.

not statistically significant, which may be because of potential confounders between the 2 groups and the small sample size in the present study. Therefore, a multiple logistic regression analysis was performed to adjust for potential confounders. We found that pleural effusion was an independent risk factor for RF. Thus, to facilitate early recognition and timely treatment of renal dysfunction and even RF, renal function should be closely monitored in patients with AP who have pleural effusion on admission. To the best of our knowledge, this is the first study to assess the association between pleural effusion and impairment of renal function in patients with AP and to show the predictable effect of pleural effusion on RF in AP.

There are some possible explanations for the results of the present study. First, pleural effusion can cause increased intrathoracic pressure, which may subsequently lead to diminished cardiac output and reduced renal artery perfusion [7]. Decreased renal arterial blood supply is known to play a critical role in causing acute kidney injury in AP [6]. Second, pleural effusion also can lead to hypoxemia, secondary to decreased residual lung capacity. In this circumstance, renal tubular epithelial cells, which are sensitive to hypoxemia, are damaged. In addition, hypoxemia can cause renal vessel constriction, which eventually can lead to renal ischemic injury [6]. Third, pleural effusion is an indicator of disease severity in patients with AP. Hence, patients with pleural effusion may have higher levels of nephrotoxic substances, such as inflammatory cytokines, enzymes, and proteases, which can subsequently contribute to impaired renal function [6].

Our study has some limitations. First, because it was retrospective, the association between the volume of pleural effusion and impairment in renal function was difficult to assess. Second, some potential confounders, such as use of diuretics and volume of water intake, were not considered; thus, the assessment of renal function may have been somewhat affected by these factors. Third, the present study was single-center study and the sample size was limited. Therefore, our conclusions should be interpreted with caution; future studies with a larger sample size are needed to validate our findings.

Conclusions

Pleural effusion is associated with severe renal dysfunction, including high levels of BUN, low eGFR, and a high risk for RF in patients with AP. Therefore, renal function should be closely monitored in patients with AP who have pleural effusion on admission so that RF can be identified early and treated in a timely fashion.

Conflicts of Interest

None.

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