Splenic Vein Involvement or Pancreatic Portal Hypertension in Acute Pancreatitis: A Retrospective Characteristic Study

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Abstract: This study analyzed the characteristics of acute pancreatitis (AP) complicated by splenic vein involvement, with or without pancreatic portal hypertension (PPH). Methods: A retrospective review was performed on 225 AP cases that met specific inclusion and exclusion criteria. Patients were classified into three groups: the PPH group, the splenic vein nvolvement group (patients with splenic vein inflammation or thrombosis, without PPH), and the normal splenic vein group (patients without splenic vein involvement or PPH). Imaging and clinical features were documented for each group, including pancreatic necrosis, local complications, ascites, splenomegaly, MCTSI, gender, age, smoking and drinking history, prior history of AP, time from onset to CT scanning, and length of hospital stay. Statistical analyses included chi-square tests, analysis of variance, or the Kruskal-Wallis test. Results: The PPH and splenic vein stenosis or thrombosis groups exhibited more pronounced indirect signs, a higher proportion of critically ill patients, and longer hospital stays (all $P<0.001$). Recurrent pancreatitis was more common in the PPH group. Differences in the remaining indicators were not statistically significant. Conclusion: Analysis of clinical and imaging findings provided clinically significant for assessing the severity of splenic vein involvement or PPH in acute pancreatitis.

***Keywords*:** tomography, x-ray computer, acute pancreatitis, pancreatic portal hypertension

1. Introduction

Pancreatic portal hypertension (PPH) is characterized by preserved liver function and the presence of isolated gastric varices[1]. Esophageal varices or splenomegaly can also be observed. PPH frequently arises to pancreatitis or pancreatic tumors and typically presents with subtle or nonspecific symptoms. Approximately 10% of patients experience life-threatening hemorrhage due to ruptured varices, resulting in a poor prognosis[2].

Recently, the incidence of acute pancreatitis (AP) has increased, leading to greater clinical focus on PPH as a complication[3-5]. Splenic vein stenosis or embolism may progress to PPH. The underlying pathogenesis frequently involves stasis caused by necrotic material or fluid, vascular compression, or endothelial injury adjacent to the pancreas[6]. The disease course is dynamic. Studies have reported diverse risk factors for peripancreatic vascular complications or PPH in AP[7-9], which may complicate clinical decision-making.

Therefore, this study aimed to compare and analyze the clinical and imaging characteristics of PPH, splenic vein stenosis or embolism in AP patients. A comprehensive understanding of disease progression may inform diagnosis, treatment strategies, and risk mitigation.

2. Data collection

2.1 Inclusion and exclusion criteria

This retrospective study analyzed 225 AP patients who were hospitalized at Guang'an People's Hospital, a tertiary care referral center in China, between January 2020 and November 2025 (Table 1). All patients met the 2012 revised Atlanta criteria[10]. Exclusion criteria included: 1) pancreatic or peripancreatic malignancies, prior surgical procedures, or liver disease; 2) acute exacerbation of chronic pancreatitis; 3) autoimmune pancreatitis; and 4) incomplete medical history, lack of patient follow-up, or missing imaging data. The study was approved by the Ethics Committee, which waived the requirement for informed consent signatures.

2.2 Diagnostic criteria

Splenic vein stenosis or thrombosis was diagnosed by CT-enhanced imaging showing roughened vessel walls, luminal narrowing, or filling defects. The diagnostic criteria for PPH with varicose veins included[11, 12]: 1) gastroduodenal vein diameter over 6 mm; 2) gastric coronary vein diameter over 6 mm; 3) gastric short vein diameter over 5 mm; 4) gastric fundus varices over 5 mm.

2.3 Clinical data

Data collected included patient gender, age, smoking and drinking history, prior history of AP, time from onset to CT scanning, and length of hospital stay.

2.4 Image analysis

CT images were independently evaluated by two senior diagnostic physicians using a double-blind method. In cases of disagreement, consensus was achieved through discussion. The evaluation indicators included all cases of PPH, splenic vein stenosis or thrombosis in AP patients, and the varicose branch involved in PPH. Additional indicators comprised pancreatic necrosis, local complications such as peripancreatic fluid collection or necrotic collections, pancreatic pseudocyst or walled-off necrosis, ascites, and splenomegaly. The Modified CT Severity Index (MCTSI) was applied to assess disease severity.

3. Statistical analysis

Statistical analyses were performed using SPSS version 26.0. Categorical variables were described as counts (n) and percentages (%). The Kolmogorov-Smirnov test assessed the normality of continuous variables. Normally distributed data were expressed as mean ± standard deviation, while non-normally distributed data were presented as median and interquartile range. Categorical variables were compared using the chi-squared test. Continuous variables were analysed using analysis of variance or the Kruskal-Wallis test, with pairwise comparisons within groups adjusted using the Least Significant Difference method or Bonferroni correction.

4. Results

Of the 225 enrolled patients (Table 1), 25 (11.1%) exhibited PPH. Among these, 16 cases (64.0%) had splenic vein stenosis, 9 cases (36.0%) had splenic vein occlusion, and 20 cases (80.0%) had splenomegaly. Gastroduodenal varices were observed in 22 cases (88.0%), gastric short varices in 20 cases (80.0%), gastric coronary varices in 16 cases (64.0%), and gastric fundic varices in 13 cases (52.0%). Additionally, 40 cases (17.8%) presented with AP accompanied by splenic vein inflammation or thrombosis without variceal manifestations; splenomegaly was present in 24 of these cases (60.0%). A total of 160 cases (71.1%) demonstrated neither splenic vein involvement nor PPH.

No statistically significant differences were identified among the three AP patient groups regarding gender, age, smoking history, alcohol consumption history, time from onset to CT scanning ($P>0.05$). In contrast, significant differences were found in pancreatic necrosis, local complications, ascites, MCTSI scores, history of previous pancreatitis, and length of hospital stay ($P<0.05$). Both the AP with PPH group and the splenic vein inflammation or thrombosis group demonstrated higher rates of pancreatic necrosis, local complications, and ascites compared to the normal splenic vein group, as well as a greater proportion of severe AP cases (60.0% vs. 75.0% vs. 13.1%) and longer hospital stays. However, the differences between these two groups were not statistically significant. The AP with PPH group also had a higher prevalence of prior pancreatitis compared to the AP with splenic vein inflammation or thrombosis group and the normal splenic vein group (80.0% vs. 20.0% vs. 21.2%). (Table 2)

Table 1. The varicose branch involved in PPH

	PPH (n=25)
gastroduodenal vein, n(%)	22 (88.0)
gastric short vein, n(%)	20 (80.0)
gastric coronary vein , n(%)	16 (64.0)
gastric fundus, n(%)	13 (52.0)

Table 2. Comparison of clinical and imaging characteristics among three AP patient groups

	PPH (n=25)	Splenic vein involvement (n=40)	Normal splenic vein (n=160)	χ^2/F/Z	P
Gender					
Male, n(%)	18 (72.0)	22 (55.0)	98 (61.3)	χ^2=1.876	0.398
Famle, n(%)	7 (38.0)	18 (45.0)	62 (38.7)		
Age, year	43.16±10.30	48.90±12.18	47.68±13.57	F=1.629	0.199
Smoke, n(%)	20 (80)	33 (82.5)	123 (76.9)	χ^2=0.647	1.195
Drinking, n(%)	18 (72.0)	33 (82.5)	129 (80)	P=0.762	0.0.580
Prior history of AP, n(%)	20 (80)	8 (20.0)	34 (21.2)	χ^2=38.776b	<0.001

	PPH (n=25)	Splenic vein involvement (n=40)	Normal splenic vein (n=160)	χ^2/F/Z	P
Time from onset to CT scanning,day	8.77(7.20,12.49)	8.44(6.99,10.02)	8.01(7.17,9.10)	Z=3.314	0.191
Length of hospital stay, day	15.18(11.92,20.11)	17.60(13.53,21.49)	9.29(9.29,15.95)	Z=26.892a	<0.001
Pancreatic necrosis, n(%)	16 (64.0)	33 (82.5)	24 (15.0)	χ^2=79.298a	<0.001
Local complications, n(%)	21 (80)	34 (85.0)	66 (41.3)	χ^2=34.975a	<0.001
Ascites, n(%)	19 (84.0)	30 (75.0)	37 (23.1)	χ^2=38.826a	<0.001
Splenomegaly, n(%)	20 (80)	24 (60.0)	-	χ^2=2.814	0.075
MCTSI					
Mild, n(%)	0 (0.0)	0 (0.0)	52 (32.5)	χ^2=78.052a	<0.001
Moderately severe , n(%)	10 (40)	10 (25.0)	87 (54.3)		
Severe , n(%)	15 (60)	30 (75.0)	21 (13.1)		

Note: a The PPH group and the splenic vein inflammation or thrombosis group each showed statistically significant differences compared with the normal splenic vein group (P<0.001). b The PPH group showed statistically significant differences compared with both the splenic vein inflammation or thrombosis group and the normal splenic vein group (P<0.05).

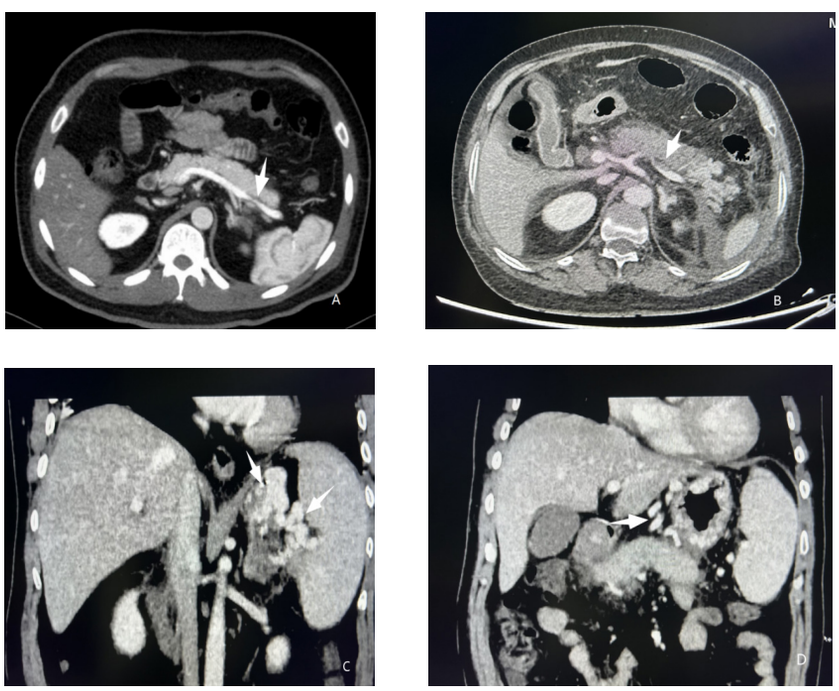

Figure 1. A: Male, 38 years old. Portal venous phase reveals localised stenosis of the splenic vein (white arrow). B: Female, 69 years old. Portal venous phase reveals splenic vein thrombosis (white arrow). C and D: Male, 52 years old. Portal venous phase reveals sequentially: gastric fundus vein, gastric short vein, and coronary varices (white arrow).

5. Conclusion

PPH remains the curable form of portal hypertension[13], highlighting the necessity of effective prevention strategies. Diagnosis of PPH relies on a combination of clinical evaluation and imaging assessments. It is confirmed by excluding hepatic disease and verifying adherence to diagnostic criteria for the underlying pancreatic condition. The assessment of primary pancreatic disease primarily depends on imaging examinations, which yield essential diagnostic and therapeutic information and inform prognosis.

Digital angiography is regarded as the gold standard for diagnosing post-pancreatic haemorrhage, although its clinical application is limited. Gastroscopy is the preferred diagnostic method but necessitates gastrointestinal preparation. CT is routinely used to assess the severity of pancreatitis and to identify peripancreatic vascular complications. Recent studies have examined independent risk factors for peripancreatic vascular complications or PPH in AP using enhanced CT scans[7-9].

However, comparative studies evaluating splenic vein thrombosis with or without post-pancreatic haemorrhage in AP remain scarce.

This study identified a detection rate of splenic vein stenosis or thrombosis of 17.8%, which is lower than the 22.9% reported by Ahmed [7], possibly due to a higher proportion of moderately severe and severe AP cases in the latter. The detection rate of PPH was 11.1%, which is 3.3% higher than that reported by Xie[12], and 12.9% lower than that of Li[8]. Xie[12] included only first-episode AP patients, whereas Li[8] enrolled patients with more severe disease and longer follow-up, thereby providing a broader diagnostic window. No cases of PPH were identified in patients with mild AP in this study, consistent with the low incidence (<1%) reported by Xie[12] and Li[8]. These findings indicate that PPH occurrence and progression may be associated with the number of episodes, timing of diagnosis, and disease severity. In 25 cases of postpartum haemorrhage, collateral vessels most frequently involved the mesenteric veins, consistent with previous research. This pattern differs from hepatic portal hypertension, where oesophageal varices are most common.

No significant differences in gender, age, smoking, or drinking history were observed among the three AP patient groups. This finding aligns with the results of Zhang[4], but contrasts with those of Zhao et al[5, 9, 12], who reported a higher prevalence of PPH in males. Regional population characteristics may explain these discrepancies.

Both the PPH group and the splenic vein stenosis or thrombosis group exhibited higher rates of pancreatic parenchymal necrosis, local complications, and ascites than the normal splenic vein group, indicating greater disease severity. No significant differences were observed between the PPH and splenic vein stenosis or thrombosis groups. Xie[12] reported that pancreatic haemorrhage predominantly occurs in acute necrotising pancreatitis. Ahmed[7] found that pancreatic necrosis and CTSI scores above 6 are more frequently associated with pancreatic complications in AP. These findings are consistent with the pathogenesis of post-pancreatic haemorrhage, which involves splenic vein thrombosis, mechanical compression from local peripancreatic complications, and vascular wall fibrosis due to recurrent inflammatory stimulation. Associated manifestations may include ascites and splenomegaly, often resulting in prolonged hospitalisation.

The incidence of recurrent pancreatitis in the PPH group (80.0%) was higher than in the splenic vein inflammation or thrombosis group (20%) and the normal splenic vein group (20.7%). Li[8] and Ru[9] identified local complications and recurrent acute pancreatitis (RAP) as independent risk factors for PPH. In this study, no significant differences in time of presentation or illness severity were observed between the PPH group and the splenic vein stenosis or thrombosis group. However, RAP was more likely to result in PPH in these groups, possibly due to fibrosis of the splenic vein vascular wall resulting from repeated inflammatory stimulation or compression.

In summary, this study identified both clinical and imaging indicators by characterising splenic vein involvement in AP, with or without PPH, thereby supporting clinical decision-making. However, the sample sizes among the three AP groups varied considerably. Statistical analysis was limited to splenic vein involvement with or without PPH, and the normal splenic vein group included a small number of cases with additional vascular involvement. These limitations may affect the reliability and accuracy of the findings. A larger sample size in the group with concomitant PPH is needed to enable further follow-up studies, risk factor analysis, and the development of predictive models.

Acknowledgments

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