Retrospective Cohort Study

Portosplenomesenteric vein thrombosis in patients with early-stage severe acute pancreatitis

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AIM
To investigate the incidence and risk factors of portosplenomesenteric vein thrombosis (PSMVT) in the early stage of severe acute pancreatitis (SAP).

METHODS
Patients with SAP in a tertiary care setting from January 2014 to December 2016 were retrospectively reviewed.
All contrast-enhanced computed tomography (CT) studies were reassessed and reviewed. Clinical outcome measures were compared between SAP patients with and without PSMVT in the early stage of the disease. Univariate and multivariate logistic regression analyses were sequentially performed to assess potential risk factors for the development of PSMVT in SAP patients. A receiver operating characteristic (ROC) curve was generated for the qualifying independent risk factors.

RESULTS
Twenty-five of the one hundred and forty (17.86%) SAP patients developed PSMVT 6.19 ± 2.43 d after acute pancreatitis (AP) onset. PSMVT was confirmed by contrast-enhanced CT. Multivariate stepwise logistic regression analyses showed that Balthazars CT severity index (CTSI) scores [odds ratio (OR): 2.742; 95% confidence interval (CI): 1.664-4.519; P = 0.000], hypoalbuminemia (serum albumin level < 25 g/L) (OR: 32.573; 95%CI: 2.711-391.353; P = 0.006) and gastrointestinal wall thickening (OR: 4.367, 95%CI: 1.218-15.658; P = 0.024) were independent risk factors for PSMVT developed in patients with SAP. The area under the ROC curve for Balthazar's CTSI scores was 0.777 (P = 0.000), the sensitivity was 52%, and the specificity was 93% at a cut-off value of 5.5.

CONCLUSION
High Balthazars CTSI scores, hypoalbuminemia and gastrointestinal wall thickening are independent risk factors for PSMVT developed in the early stage of SAP.

Key words: Vascular complication; Portosplenosenteric vein thrombosis; Severe acute pancreatitis; Early stage; Risk factors; Contrast-enhanced computed tomography

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Core tip: Studies on portosplenosenteric vein thrombosis (PSMVT) occurring in the early stage of acute pancreatitis (AP) were rare. We found that 17.86% of severe AP patients developed PSMVT 6.19 d after AP onset. High Balthazars computed tomography severity index (CTSI) scores, hypoalbuminemia and gastrointestinal wall thickening are independent risk factors for the development of PSMVT, and Balthazars CTSI scores can predict the occurrence of PSMVT with a high degree of specificity which indicated that a low Balthazars CTSI score could be a good indicator that PSMVT would not occur.

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INTRODUCTION
Portosplenosenteric vein thrombosis (PSMVT) involves the portal vein (PV), splenic vein and superior mesenteric vein (SMV), either separately or in combination. In most cases, PSMVT does not cause any additional symptoms, but may sometimes generate localized portal hypertension, leading to gastrointestinal and intra-abdominal hemorrhage, ascites, splenomegaly, and splenic infarction, among other problems[1,2]. The previous studies reported that PSMVT mostly occurs late during acute pancreatitis (AP)[3,4], and is usually detected incidentally on radiological imaging performed to evaluate the severity of AP[5].

Until now, the detailed natural history of PSMVT has remained unknown, and the pathogenesis underlying PSMVT in patients with AP has remained unclear and may involve many clinical factors. There is little data regarding the risk factors for this complication in the early stage of AP. The results about the risk factors of PSMVT in AP patients vary among studies[3,5,7-10]. Therefore, in this study, we analyzed the data to explore the incidence of PSMVT in the early stage of severe AP (SAP), the risk factors for the development of PSMVT, and the clinical outcomes of PSMVT.

MATERIALS AND METHODS

**Patients**
Consecutive adult patients (age ≥ 18 years old) with SAP who were admitted to the First Affiliated Hospital of Nanchang University between January 2014 and December 2016 were enrolled in this study. Exclusion criteria include the following: (1) Admission > 6 d after AP onset; (2) history of AP, chronic pancreatitis, pancreatic malignancy or cysts; (3) no contrast-enhanced CT examination ≤ 10 d after AP onset; (4) pregnancy; and (5) cirrhosis or coagulopathy disorder diseases or other systemic tumors.

**Data collection**
The following data were collected: (1) General characteristics, including age, gender, etiology of AP, modified Marshall score, systemic inflammatory response syndrome score, acute physiology and chronic health evaluation II score, and Balthazars computer tomography severity index (CTSI)[11] at admission; (2) clinical outcomes, including microbiological outcomes, ascites, and intra-abdominal pressure measured with a catheter inserted into the bladder according to the guidelines[12], complications including gastrointestinal hemorrhage, intra-abdominal hemorrhage, multiple organ dysfunction syndrome, and infected pancreatic necrosis, and clinical treatments including minimally invasive and surgical interventions and hospital and intensive care unit lengths of stay and mortality; and (3) laboratory markers,
Persistent organ failure, that was, organ failure for
on contrast-enhanced CT images. SAP was defined by
as a filling defect within the lumen of the vessel seen
the contrast-enhanced CT. Thrombosis was defined
The diagnosis of PSMVT was based on the results of
diaphragmatic domes to the pubic symphysis.
fixed 60 s delay. The scanned area extended from the
thoracolumbar junction had reached 180 HU with a
injected at a rate of 3 mL/s with a high-pressure injector.
Schering Pharma, Berlin, Germany) was intravenously
obtained, 1.3 mL/kg iopromide (Ultravist 370; Bayer
Erlangen, Germany). After noncontrast scans were
(PSMVT: Portosplenomesenteric vein thrombosis.
PSMVT , which occurred within 10 d after AP onset, was
detected. All contrast-enhanced CT examinations were
analyses using SPSS software (v17.0; SPSS Inc.,
statistically significant in the univariate logistic
regression analysis were introduced into a multivariate
logistic analytic model (stepwise regression) to identify
independent risk factors with odds ratios (ORs) and
95% confidence intervals (CIs). Furthermore, receiver
operating characteristic (ROC) curves were generated for
each of the qualified independent risk factors. A P-value
was considered statistically significant. Data
were analyzed using SPSS software (v17.0; SPSS Inc.,
Chicago, IL, United States).
RESULTS
As shown in the flow chart in Figure 1, 140 eligible
patients were enrolled in this study. Twenty-five patients
developed PSMVT (17.86%), and all the PSMVT
cases were confirmed 6.19 ± 2.43 d after AP onset by
contrast-enhanced CT. Supplementary Table 1 shows
the demographic characteristics of all patients. The
average age of all patients was 54.59 ± 15.90 years,
and the study included 67 males (47.85%) and 73
females (52.14%). The PSMVT and non-PSMVT groups
were matched by age, sex, AP etiology, Marshall score,
systemic inflammatory response syndrome score, and
acute physiology and chronic health evaluation II score.
Balthazar’s CTSI score was higher in the PSMVT group
than in the non-PSMVT group (7.20 ± 2.65 vs 4.63 ± 1.45,
P = 0.000).
The most commonly involved vessel was the splenic
including hypoalbuminemia (serum albumin level < 25
g/L), hematocrit, cholesterol, triglycerides, inflammation
markers including leucocytes, C-reactive protein and procalcitonin, coagulation tests including platelets, prothrombin time, activated partial thromboplastin
time, fibrinogen and D-dimer during the 24 h following
admission. Anticoagulation therapy was not included in
the manage protocol of PSMVT at early stage (within 10
d after AP onset).
Imaging protocols
All contrast-enhanced CT studies were reassessed and
reviewed by a radiologist who specialized in abdominal
imaging and was blinded to the clinical data. Contrast-
enhanced CT was generally performed approximately
3-10 d after AP onset, with an average of 7 d. Thus,
PSMVT, which occurred within 10 d after AP onset, was
detected. All contrast-enhanced CT examinations were
performed using a 64-channel multidetector CT scanner
(Somatom Definition AS+, Siemens Medical Systems,
Erlangen, Germany). After noncontrast scans were
obtained, 1.3 mL/kg iopromide (Ultravist 370; Bayer
Schering Pharma, Berlin, Germany) was intravenously
injected at a rate of 3 mL/s with a high-pressure injector.
Then, contrasted arterial and portal phase scans were
obtained when the attenuation of the aorta at the
thoracolumbar junction had reached 180 HU with a
fixed 60 s delay. The scanned area extended from the
diaphragmatic domes to the pubic symphysis.
Definitions
The diagnosis of PSMVT was based on the results of
the contrasted-enhanced CT. Thrombosis was defined
as a filling defect within the lumen of the vessel seen
on contrast-enhanced CT images. SAP was defined by
persistent organ failure, that was, organ failure for longer
than 48 h[3]. Extrapancreatic necrosis alone was defined
as extrapancreatic morphological changes exceeding
fat stranding with complete enhancement of the
pancreatic parenchyma without signs of focal or diffuse
nonenhancement that could be determined on contrast-
enhanced CT images[13]. Pancreatic parenchymal necrosis
was defined as focal or diffuse nonenhancement of the
pancreatic gland. When pancreatic necrosis was present,
its location (head, neck, body, tail) and amount (< 30%,
30%-50%, > 50%) were noted. Acute peripancreatic
fluid collections (APFCs) were defined as peripancreatic
fluid collections with homogeneous liquefied components
and without well-defined walls. The anatomical locations
of APFCs included the anterior and posterior renal
spaces, perirenal space, great and lesser omentum,
paracolic sulci, mesenteric root, and transverse
mesocolon. APFCs were recorded when the diameter
was > 1 cm. Gastrointestinal wall thickening was defined
as the thickening and edema of the gastrointestinal wall,
with wall thickness greater than 4 mm, and as robust
enhancement of the mucosa with reduced enhancement
of the submucosa[13,14].
Statistical analysis
Continuous variables are described as the mean ± SD,
and categorical variables are described as the absolute
numbers and percentages. Continuous variables were
compared using t-tests, and categorical data were
analyzed with the chi-squared test. Variables found
to be statistically significant in the univariate logistic
regression analysis were introduced into a multivariate
logistic analytic model (stepwise regression) to identify
independent risk factors with odds ratios (ORs) and
95% confidence intervals (CIs). Furthermore, receiver
operating characteristic (ROC) curves were generated for
each of the qualified independent risk factors. A P-value
< 0.05 was considered statistically significant. Data
were analyzed using SPSS software (v17.0; SPSS Inc.,
Chicago, IL, United States).
In the third patient, no reason was found. Intra-abdominal hemorrhage was detected in eight patients by abdominal CT, digital subtraction angiography, or during invasive interventions and surgical processes. Patients with PSMVT were more severely ill, as evidenced by their greater need for minimally invasive interventions, higher rates of multiple organ dysfunction syndrome, infected pancreatic necrosis, and longer durations of hospital and intensive care unit stays.

Table 1 shows the results of the univariate regression analysis of PSMVT. The following were potential risk factors: Higher Balthazar’s CTSI score (OR: 1.780, \( p = 0.000 \)); hypoalbuminemia (OR: 0.047, \( p < 0.01 \)); culture-positive drainage fluid (OR: 3.97, \( p < 0.01 \)); the APFC at the mesenteric root (OR: 2.765, \( p < 0.05 \)); extrapancreatic necrosis alone (OR: 0.314, \( p < 0.05 \)); peripancreatic and pancreatic parenchymal necrosis (OR: 6.021, \( p = 0.000 \)); necrosis located in the head (OR: 4.580, \( p < 0.01 \)), neck (OR: 4.413, \( p < 0.01 \)), body (OR:

| Variables | OR (95%CI) | \( p \) value |
|-----------|------------|---------------|
| Age       | 0.974 (0.948-1.002) | 0.065         |
| Sex       | 0.828 (0.347-1.976) | 0.670         |
| Etiology  | 0.775 (0.405-1.483) | 0.441         |
| APACHEII score | 1.03 (0.924-1.148) | 0.590         |
| Modified Marshall score | 1.06 (0.816-1.377) | 0.660         |
| SIRS score | 1.369 (0.873-2.147) | 0.171         |
| Balthazar’s CTSI score | 1.78 (1.419-2.232) | 0.000         |
| Hypoalbuminemia | 21.143 (2.25-198.68) | 0.008         |
| Hematocrit | 1.027 (0.974-1.083) | 0.331         |
| Leucocyte | 1.021 (0.955-1.092) | 0.539         |
| C-reactive protein | 1.001 (0.997-1.005) | 0.596         |
| Procalcitonin | 0.988 (0.951-1.022) | 0.485         |
| Platelet  | 1 (0.995-1.006) | 0.928         |
| Prothrombin time | 0.973 (0.875-1.081) | 0.606         |
| APFF      | 1.01 (0.981-1.040) | 0.594         |
| Fibrinogen | 0.984 (0.865-1.120) | 0.806         |
| D-dimer   | 1.008 (0.954-1.064) | 0.782         |
| Cholesterol | 1.034 (0.893-1.198) | 0.654         |
| Triglyceride | 1.006 (0.952-1.063) | 0.831         |
| APFEC     | 1.161 (0.947-1.423) | 0.151         |
| Highest value of IAP | 1.174 (0.985-1.400) | 0.074         |
| Culture positive of drainage fluid | 3.97 (1.566-10.067) | 0.004         |
| Culture positive of blood | 1.889 (0.766-4.655) | 0.367         |
| APFC on Mesenteric root | 2.765 (1.126-6.791) | 0.026         |
| Extrapancreatic necrosis alone | 0.314 (0.125-0.787) | 0.013         |
| Pancreatic parenchymal necrosis | 6.201 (2.357-15.379) | 0.000         |
| Location of necrosis | 6.021 (2.357-15.379) | 0.000         |
| Head      | 4.58 (1.514-13.855) | 0.007         |
| Neck      | 4.413 (1.624-11.997) | 0.004         |
| Body      | 6.43 (2.536-16.306) | 0.000         |
| Tail      | 8.5 (3.209-22.514) | 0.000         |
| Amount of necrosis |
| < 30%  | 0.722 (0.226-2.304) | 0.582         |
| 30%-50%  | 3 (0.668-13.482) | 0.152         |
| > 50%    | 24.889 (6.148-100.750) | 0.000         |
| Gastrointestinal wall thickening | 4.25 (1.725-10.474) | 0.002         |

1Hypoalbuminemia was defined as serum albumin level < 25 g/L; 2IAP was measured in the first three days from admission and the average and highest value of IAP was noted; 3The fluid was obtained by percutaneous paracentesis or drainage pancreatic necrosis. OR: Odds ratio; CI: Confidence interval; APACHE: Acute Physiology, Age, and Chronic Health Evaluation; SIRS: Systemic inflammatory response syndrome; CTSI: Computed tomography severity index; APTT: Activated partial thromboplastin time; IAP: Intra-abdominal pressure; APFC: Acute peripancreatic fluid collection.
6.431, \( p = 0.000 \)), or tail (OR: 8.500, \( p = 0.000 \)) of the pancreas; an extent of pancreatic necrosis > 50% (OR: 24.889, \( p = 0.000 \)); and gastrointestinal wall thickening (OR: 4.053, \( p = 0.000 \)).

Multivariate stepwise logistic regression analyses showed that higher Balthazar’s CTSI score (OR: 2.742; 95%CI: 1.664-4.519; \( p = 0.000 \)), hypoalbuminemia (serum albumin level < 25 g/L) (OR:32.573; 95%CI: 2.711-391.353; \( p = 0.006 \)) and gastrointestinal wall thickening (OR: 4.367, 95%CI: 1.218-15.658; \( p = 0.024 \)) were independent risk factors for PSMVT in patients with SAP (Table 2). The area under the ROC curve for Balthazar’s CTSI score was 0.777 (\( p = 0.000 \)), the sensitivity was 52%, and the specificity was 93% at a cut-off value of 5.5 (Figure 4).

**Table 2** Results of multivariate logistic regression analyses

| Variables                        | OR       | 95%CI               | \( p \) value |
|----------------------------------|----------|---------------------|--------------|
| Balthazar’s CTSI score           | 2.742    | 1.664-4.519         | 0.000        |
| hypoalbuminemia                  | 32.573   | 2.711-391.353       | 0.006        |
| Gastrointestinal wall thickening | 4.367    | 1.218-15.658        | 0.024        |

**DISCUSSION**

There is a lack of data on the prevalence and natural course of PSMVT in AP. It has been reported that the incidence of vascular abnormalities varies from 1.8% to 57% because of the different vascular abnormalities described, and differences in the severity of AP, and time or method of detecting vascular abnormalities\(^{3-5,10,15-18}\).

In the present study, PSMVT developed in 25 of 140 (17.86%) patients with early-stage SAP. It has been reported that most PSMVTs have been clinically confirmed in the late stage of AP onset\(^{3,5,10}\). This study is the first report to focus on PSMVT occurring as early as within 10 d (average 6 d) from AP onset and to indicate that high Balthazar’s CTSI, hypoalbuminemia and gastrointestinal wall thickening are independent prognostic factors of PSMVT in the early stage of the onset of SAP.

The development of PSMVT is associated with the presence, location, and extent of pancreatic necrosis\(^{3,10}\). In contrast, some papers showed a weaker association between Balthazar’s CTSI and superficial venous thrombosis (SVT) or PSMVT in AP\(^{3,15}\). In our study, Balthazar’s CTSI was an independent risk factor of PSMVT and the area under the ROC curve for Balthazar’s CTSI score was 0.777 at a cut-off value of 5.5. The high specificity indicated that a low Balthazar’s CTSI score could be a good indicator that PSMVT would not occur. The splenic vein runs behind the tail and the body of the pancreas, showing a filling defect within the lumen of the portal vein (up arrow), splenic vein (down arrow) and superior mesenteric vein (left arrow). CT: Computed tomography.
pancreas. Behind the neck of the pancreas, the splenic vein joins the SMV to form the hepatic PV. Necrosis could directly impact the vascular system, resulting in the formation of PSMVT. In addition, pancreatic necrosis is thought to be associated with a severe local inflammatory response and may surround the vein directly, causing impaired vasomotor function, reduced capillary perfusion, and enhanced thrombosis. However, the appropriate time for drainage is controversial, and it is recommended that drainage should be delayed until the fluid is well encapsulated or there is necrosis[20]. In our study, we found that PSMVT could form in the early stage of AP and hence, for these patients, early drainage might alleviate local inflammation and lower the risk of PSMVT.

Low albumin status reflects poor nutritional status. Low albumin levels are thought to be associated with increased venous thromboembolism in head and neck surgery patients[21], hepatocellular carcinoma patients[22], colorectal surgery patients[23], and thoracolumbar surgery patients[24]. A prospective study determined that lower serum albumin levels were more accurate than clinical scores for diagnosing DVT in patients with several comorbidities[25]. In our study, hypoalbuminemia with albumin levels < 25 g/L was a good predictor of PSMVT, with a very high OR (32.573). Thus, we speculate that correcting hypoalbuminemia during the early stage of AP may help prevent the occurrence of PSMVT. Low albumin levels may lead to peripheral edema and possibly to increased venous stasis. However, the exact mechanism needs further study.

This study revealed that gastrointestinal wall thickening was an independent risk factor for PSMVT. Gastrointestinal wall thickening is represented by the thickening and edema of the gastrointestinal wall, which means that the intestinal barrier is more vulnerable. There is speculation that intestinal bacteria and their products can infiltrate the blood circulation through the damaged intestinal barrier[26], causing severe local inflammation, which is related to venous thrombosis. The level of D-dimer is mostly used as an effective diagnostic tool to rule out deep vein thrombosis (DVT) and pulmonary embolism[27], and it has been reported to have great predictive power in the early phase of AP[28]. However, our study showed that coagulation tests including platelets, prothrombin time, activated partial thromboplastin time, fibrinogen and D-dimer were not independent risk factors in the logistic regression analysis, suggesting that coagulative disturbance may not be a direct cause of PSMVT, as reported before[29].

There were several limitations of our study. First, it was a retrospective study with a limited sample size. Second, we focused on the vascular complications during the early stage of AP; thus, whether more patients experienced PSMVT in later stages was unknown. Third, anticoagulation therapy was not included in the management protocol of PSMVT at the early stage (within 10 d after AP onset). However, when and whether anticoagulants and other treatments were given 10 d after AP onset depended on the decisions made by the attending doctors. Whether these interventions could have affected the long-term prognosis is unclear.

In conclusion, we demonstrated that the occurrence rate of PSMVT is high in SAP patients in the early stage, and high Balthazar’s CTSI scores, hypoalbuminemia and gastrointestinal wall thickening were independent prognostic factors.
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