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Clinical Characteristics and Efficacy of Percutaneous Catheter Drainage in Children with Acute Lymphoblastic Leukemia Complicated by Asparaginase-Associated Pancreatitis and Ascites

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Abstract

Objective

To summarize the clinical characteristics of children with acute lymphoblastic leukemia (ALL) complicated by asparaginase-associated pancreatitis (AAP) and ascites, and to evaluate the efficacy of percutaneous catheter drainage (PCD) using an ascites depth > 30 mm as the intervention threshold in patients with mild AAP.

Methods

A retrospective analysis was conducted on 47 children diagnosed with ALL who developed AAP following asparaginase chemotherapy at Children's Hospital of Soochow University between January 2022 and December 2024. AAP severity was classified according to the modified Atlanta criteria and the Pediatric Sequential Organ Failure Assessment (pSOFA) score into mild (n = 40), moderately severe (n = 3), and severe (n = 4) groups. Abdominal ultrasound with a maximum ascites depth > 30 mm was used as the indication for PCD. Clinical outcomes, including fasting duration, total hospital stay, and pancreatic enzyme recovery time, were compared between the PCD group (n = 18) and the conservative treatment group (n = 10) among mild AAP patients with ascites. Statistical analyses were performed using the Mann-Whitney U test and Fisher's exact test.

Results

Among the 47 AAP patients, 28 (59.6%) were male and 19 (40.4%) were female, with a median age of 6.5 years (range: 1.5–14.5 years). Mild AAP was identified in 40 patients (85.1%), moderately severe AAP in 3 (6.4%), and severe AAP in 4 (8.5%). Ascites was present in 35 patients (74.5%), and all 7 patients with moderately severe or severe AAP had ascites. Among the 28 mild AAP patients with ascites depth > 30 mm, 18 underwent PCD and 10 received conservative treatment, with no significant differences in baseline characteristics between the two groups ($P > 0.05$ for all). The PCD group had significantly shorter fasting duration (median 7.0 days vs. 10.5 days, $P = 0.031$) and total hospital stay (median 26.0 days vs. 31.0 days, $P = 0.043$) compared to the conservative group. No significant differences were observed in the recovery times of serum amylase, urinary amylase, or lipase between the two groups ($P > 0.05$ for all). One patient (5.6%) in the PCD group developed puncture site oozing, while in the conservative group, two patients (20.0%) required temporary puncture due to progressive ascites accumulation and one (10.0%) developed a pancreatic pseudocyst. All 7 patients with moderately severe or severe AAP underwent PCD, and all 4 patients with severe AAP died.

Conclusion

PCD using an ascites depth > 30 mm as the intervention threshold significantly shortened fasting duration and total hospital stay in children with mild AAP, with favorable safety profile. This approach may serve as an effective intervention strategy for AAP complicated by significant ascites.

Background

Acute lymphoblastic leukemia (ALL) is the most common malignancy in children. With the optimization of chemotherapy protocols, the 5-year overall survival rate has exceeded 90% [1]. Asparaginase (ASP) and its long-acting formulation pegaspargase (PEG-ASP) are core components of ALL chemotherapy regimens, exerting their effects by depleting plasma asparagine to selectively inhibit protein synthesis in leukemia cells [2]. However, asparaginase-associated pancreatitis (AAP) is one of the most serious adverse reactions, with an incidence of approximately 2–10% [3, 4].

Ascites is a common complication of acute pancreatitis (AP). Studies have demonstrated that AP patients with ascites tend to have more severe disease, with significantly higher risks of organ failure, pancreatic necrosis, and mortality compared to those without ascites [5, 6]. The accumulation of a large volume of intra-abdominal fluid can elevate intra-abdominal pressure (IAP), thereby exacerbating intra-abdominal hypertension, impairing respiratory function and intestinal barrier integrity, and even promoting systemic inflammatory response syndrome [7]. Nevertheless, there remains considerable controversy regarding whether patients with mild-to-moderate AAP and ascites should undergo drainage. Some experts argue that ascites in mild-to-moderate AAP is often reactive and self-limiting, and that invasive procedures may introduce unnecessary risks with unclear benefits [8]. Consequently, high-quality evidence regarding the indications and clinical benefits of PCD in mild-to-moderate AAP patients with ascites is lacking. In the pediatric AAP population in particular, there is no consensus on the indications, timing, or efficacy of PCD.

Therefore, this study used "maximum ascites depth > 30 mm" as a quantitative threshold to retrospectively analyze the clinical characteristics of 47 children with AAP and ascites at our center, and to compare the efficacy of PCD versus conservative treatment in mild AAP patients, with the aim of providing reference for clinical practice.

Methods

Study population

This study included children with ALL treated according to the CCLG-ALL-2020 protocol at Children's Hospital of Soochow University who developed AAP between January 2022 and December 2024. Inclusion criteria were: (1) age 1 month to 18 years; (2) diagnosis of ALL confirmed by immunophenotyping, cytogenetics, and molecular biology; (3) development of AAP during chemotherapy containing asparaginase or within 28 days after discontinuation; and (4) meeting the diagnostic criteria for AAP.

Exclusion criteria were: (1) pre-existing pancreatitis at initial diagnosis; (2) pancreatitis caused by biliary obstruction, trauma, or hyperlipidemia (present before asparaginase administration); (3) pre-existing ascites before the onset of pancreatitis; and (4) incomplete clinical data with > 20% missing key variables.

This study was approved by the Ethics Committee of Children's Hospital of Soochow University (Ethics Approval No. 2026CS098). Informed consent was waived due to the retrospective design.

Diagnosis and severity classification of AAP

AAP was diagnosed according to the 2012 revised Atlanta classification [9], requiring at least two of the following three criteria: (1) acute, persistent, severe epigastric pain; (2) serum lipase or amylase ≥ 3 times the upper limit of normal; and (3) imaging findings (ultrasound, CT, or MRI) showing pancreatic enlargement, exudation, or necrosis.

Severity was classified based on the Atlanta criteria [9] and the Pediatric Sequential Organ Failure Assessment (pSOFA) score [10] as follows: (1) **Mild**: no organ failure, no local complications on imaging, or only pancreatic parenchymal edema/peripancreatic exudation; (2) **Moderately severe**: organ failure lasting < 48 hours or presence of local complications (e.g., pseudocyst, walled-off necrosis); (3) **Severe**: persistent organ failure (≥ 48 hours) and/or infected necrosis. The pSOFA score was assessed daily across six systems (respiratory, cardiovascular, coagulation, hepatic, renal, and neurological), with a score ≥ 2 defined as organ failure.

Ascites assessment and PCD indication

All AAP patients underwent abdominal ultrasound within 24 hours of diagnosis, and the maximum ascites depth was recorded. The indications for PCD were: (1) abdominal ultrasound showing maximum ascites depth > 30 mm; and (2) no absolute contraindications to puncture (platelet count < 10×10^9/L, coagulation disorders, etc.). Patients meeting the criteria were recommended to undergo ultrasound-guided percutaneous catheter drainage. The actual decision to perform puncture was made by the attending physician in consultation with the family.

Puncture procedure

Under ultrasound guidance, an 8-French pigtail drainage catheter was placed using the Seldinger technique. Drainage was continued until the effluent became clear, the daily drainage volume was < 50 mL/24 h, and follow-up ultrasound showed significant reduction in ascites (< 10 mm), at which point the catheter was removed. Patients in the conservative treatment group received only standard care including fasting, gastrointestinal decompression, somatostatin, and octreotide. Both groups received identical standard supportive care, including fluid resuscitation, pain management, and nutritional support.

Data collection

The following data were collected from the electronic medical record system: (1) **Baseline data**: age, sex, BMI, ALL immunophenotype (B-ALL/T-ALL), risk stratification (low/intermediate/high), cumulative ASP dose, time from last ASP administration to onset, clinical symptoms (abdominal pain, vomiting, shock), ICU admission, and vasoactive agent use; (2) **Laboratory parameters**: serum amylase, urinary amylase, lipase, liver function, and renal function; (3) **Imaging data**: ascites depth and pancreatic morphology; (4) **Treatment course**: fasting duration, total parenteral nutrition (TPN) duration, PCD performance, drainage duration, and ascites biochemistry; (5) **Outcome measures**: recovery time of serum amylase, urinary amylase, and lipase; recovery time of pancreatic imaging; time to clinical symptom improvement; total hospital stay; complications; and prognosis.

Statistical analysis

Statistical analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk test was used to assess normality. Continuous variables with non-normal distributions were presented as median (interquartile range, IQR) and compared between groups using the Mann-Whitney U test. Categorical variables were presented as frequencies (percentages) and compared using Fisher's exact test or the chi-square test. A P-value < 0.05 was considered statistically significant.

To validate the independent effect of PCD on prognosis, sensitivity analyses were performed using two multiple linear regression models following the univariate analyses, with "PCD performance" as the primary independent variable and "fasting duration" and "total hospital stay" as dependent variables, respectively. Baseline ascites depth and age were included as covariates based on clinical relevance and P < 0.10 in univariate analyses. Regression results were presented as unstandardized coefficients (B) with 95% confidence intervals (CI).

Results

Overall clinical characteristics of the 47 AAP patients

Among the 47 AAP patients, 28 (59.6%) were male and 19 (40.4%) were female, with a median age of 6.5 years (range: 1.5–14.5 years). ALL risk stratification included low-risk in 22 patients (46.8%), intermediate-risk in 23 (48.9%), and high-risk in 2 (4.3%). AAP severity included mild in 40 patients (85.1%), moderately severe in 3 (6.4%), and severe in 4 (8.5%).

Clinical characteristics: Abdominal pain was the presenting symptom in 46 patients (97.9%), and vomiting occurred in 32 (68.1%). The median time from last ASP administration to onset was 15 days (range: 3–19 days), and the median cumulative ASP dose was 4,000 U/m² (range: 2,000–18,000 U/m²). At diagnosis, the median serum amylase was 321.5 (IQR: 230–498) U/L, and the median lipase was 514.8 (IQR: 310–1,012) U/L. Ascites was present in 35 patients (74.5%), with a median ascites depth of 47 (IQR: 30–117) mm. Among mild AAP patients, 28 (59.6%) had significant ascites (depth > 30 mm), of

whom 18 underwent PCD (PCD group) and 10 received conservative treatment due to family refusal. All 7 patients with moderately severe or severe AAP underwent PCD. Detailed clinical data of all 47 patients are presented in Table 1.

Table 1
Clinical characteristics of the 47 children with asparaginase-associated pancreatitis

Characteristic	Value
Sex [n (%)]	
Male	28 (59.6)
Female	19 (40.4)
Age [years, median (range)]	6.5 (1.5–14.5)
BMI [kg/m², median (IQR)]	15.6 (14.1–17.9)
ALL risk stratification [n (%)]	
Low-risk	22 (46.8)
Intermediate-risk	23 (48.9)
High-risk	2 (4.3)
Cumulative ASP dose [U/m², median (IQR)]	4,000 (2,000–6,000)
Time from last ASP to onset [days, median (IQR)]	15 (3–19)
Abdominal pain [n (%)]	46 (97.9)
Vomiting [n (%)]	32 (68.1)
Serum amylase [U/L, median (IQR)]	321.5 (230–498)
Lipase [U/L, median (IQR)]	514.8 (310–1,012)
Urinary amylase [U/L, median (IQR)]	1,022 (480–2,400)
Ascites [n (%)]	35 (74.5)
Ascites depth [mm, median (IQR)]	47 (30–117)
Ascites depth > 30 mm [n (%)]	35 (74.6)
AAP classification [n (%)]	
Mild	40 (85.1)
Moderately severe	3 (6.4)
Severe	4 (8.5)
ICU admission [n (%)]	15 (31.9)
Vasoactive agent use [n (%)]	4 (8.5)
Fasting duration [days, median (IQR)]	8.0 (5.0–11.0)

Characteristic	Value
TPN duration [days, median (IQR)]	4.0 (3.0–11.0)
Total hospital stay [days, median (IQR)]	27.0 (17.0–34.0)
Serum amylase recovery time [days, median (IQR)]	8.0 (5.0–11.0)
Lipase recovery time [days, median (IQR)]	9.0 (6.0–12.0)
Death [n (%)]	5 (10.6)

IQR, interquartile range; BMI, body mass index; ALL, acute lymphoblastic leukemia; ASP, asparaginase; AAP, asparaginase-associated pancreatitis; ICU, intensive care unit; TPN, total parenteral nutrition

Baseline characteristics of mild AAP patients with significant ascites: PCD vs. conservative groups

Among the 28 mild AAP patients with significant ascites, 18 underwent PCD and 10 received conservative treatment. There were no statistically significant differences between the two groups in age, sex, BMI, ALL risk stratification, cumulative ASP dose, time from last ASP to onset, ascites depth, or baseline pancreatic enzyme levels ($P > 0.05$ for all), indicating comparability between groups (Table 2).

Table 2
Baseline characteristics of mild AAP patients with significant ascites: PCD vs. conservative groups

Variable	PCD group (n = 18)	Conservative group (n = 10)	P-value
Age [years, median (IQR)]	6.8 (4.5–10.5)	7.0 (5.5–11.0)	0.68
Sex [male/female, n]	10/8	6/4	0.72
BMI [kg/m², median (IQR)]	15.4 (14.1–17.2)	15.9 (14.5–18.0)	0.41
ALL risk stratification (low/intermediate) [n]	8/10	5/5	0.63
Cumulative ASP dose [U/m², median (IQR)]	4,000 (2,000–6,000)	4,000 (2,000–6,000)	0.90
Time from last ASP to onset [days, median (IQR)]	14 (10–19)	15 (11–20)	0.63
Ascites depth [mm, median (IQR)]	52 (40–68)	48 (38–65)	0.44
Serum amylase [U/L, median (IQR)]	320 (228–425)	298 (210–410)	0.57
Lipase [U/L, median (IQR)]	505 (312–850)	488 (290–795)	0.62

IQR, interquartile range; BMI, body mass index; ALL, acute lymphoblastic leukemia; ASP, asparaginase

Comparison of outcomes between PCD and conservative groups

The PCD group had significantly shorter fasting duration (P = 0.031) and total hospital stay (P = 0.043) compared to the conservative group. Clinical symptom improvement time showed a trend toward shorter duration in the PCD group, but the difference did not reach statistical significance (P = 0.12). No significant differences were observed between the two groups in recovery times of serum amylase, urinary amylase, lipase, or pancreatic imaging (P > 0.05 for all). TPN duration was similar between the two groups (P = 0.48). Detailed results are presented in Table 3.

Table 3
Comparison of outcomes between PCD and conservative groups in mild AAP patients with significant ascites [median (IQR)]

Outcome measure	PCD group (n = 18)	Conservative group (n = 10)	P-value
Fasting duration (days)	7.0 (5.0–9.0)	10.5 (7.0–14.0)	0.031
TPN duration (days)	4.0 (3.0–5.5)	4.5 (3.0–6.0)	0.48
Total hospital stay (days)	26.0 (19.0–31.0)	31.0 (24.0–39.0)	0.043
Clinical symptom improvement time (days)	5.0 (3.0–7.0)	6.0 (4.0–8.0)	0.12
Serum amylase recovery time (days)	9.0 (6.0–11.0)	11.0 (7.0–14.0)	0.18
Urinary amylase recovery time (days)	6.0 (5.0–8.0)	8.0 (6.0–11.0)	0.13
Lipase recovery time (days)	9.0 (7.0–12.0)	11.0 (7.5–15.0)	0.21
Pancreatic imaging recovery time (days)	12.0 (9.0–16.0)	15.0 (10.0–21.0)	0.09

IQR, interquartile range; TPN, total parenteral nutrition

Sensitivity analysis

To control for potential minor confounding in baseline characteristics, multiple linear regression analysis was performed with baseline ascites depth and age as covariates. The results showed that PCD remained an independent factor associated with shorter fasting duration (B = -2.9, 95% CI: -5.6 to -0.2, P = 0.035) and shorter total hospital stay (B = -4.8, 95% CI: -9.5 to -0.1, P = 0.046). These findings were consistent with the univariate analysis, suggesting that the clinical benefits of PCD were independent of age and ascites depth.

Safety evaluation of PCD

Among the 18 patients in the PCD group, 1 (5.6%) developed puncture site oozing that resolved spontaneously with compression. No cases of abdominal infection, intestinal perforation, or catheter-related sepsis occurred. The median ascites fluid amylase was 276 (range: 106–716) U/L, and the median white blood cell count was 3,496 (range: 1,060–18,431) × 10^6/L. In the conservative group, 2 patients (20.0%) required temporary puncture due to progressive ascites accumulation, and 1 patient (10.0%) developed a pancreatic pseudocyst. The overall incidence of adverse events did not differ significantly between the two groups (5.6% vs. 30.0%, P = 0.09).

Clinical characteristics of moderately severe/severe AAP

All 7 patients with moderately severe or severe AAP had significant ascites (depth > 30 mm), with a median ascites depth of 68 (range: 45–117) mm, and all underwent PCD. All 3 patients with moderately severe AAP survived, while all 4 patients with severe AAP died. Causes of death included septic shock/multiple organ dysfunction syndrome (MODS) in 3 patients and leukemia relapse in 1 patient. Clinical characteristics and outcomes are presented in Table 4.

Table 4
Clinical characteristics and outcomes of patients with moderately severe/severe AAP

Characteristic	Moderately severe (n = 3)	Severe (n = 4)	Total (n = 7)
Age [years, median (range)]	7.0 (6.5–11.0)	10.0 (5.5–12.5)	8.5 (5.5–12.5)
Sex (male/female)	2/1	3/1	5/2
Cumulative ASP dose [U/m², median (range)]	4,000 (2,000–6,000)	6,000 (2,000–18,000)	5,000 (2,000–18,000)
Ascites depth [mm, median (range)]	60 (48–95)	72 (58–117)	68 (45–117)
Peak serum amylase [U/L, median (range)]	597 (284–698)	786 (575–981)	641 (284–981)
ICU admission [n (%)]	3 (100)	4 (100)	7 (100)
Vasoactive agent use [n (%)]	2 (66.6)	4 (100)	6 (85.7)
Fasting duration [days, median (range)]	10 (8–13)	12 (11–14)	11 (8–14)
Total hospital stay [days, median (range)]	32 (27–38)	49 (39–58)	39 (27–58)
Survival [n (%)]	3 (100)	0 (0)	3 (42.9)
Death [n (%)]	0 (0)	4 (100)	4 (57.1)

Discussion

This study systematically reviewed the clinical data of 47 children with ALL complicated by AAP, with a focus on the characteristics and clinical impact of ascites. To our knowledge, this is the first study to use "maximum ascites depth > 30 mm" as a quantitative threshold to evaluate the efficacy and safety of PCD in pediatric patients with mild AAP. The results showed that the incidence of ascites in AAP reached 74.5%, and all 7 patients with moderately severe or severe AAP had ascites. This incidence is significantly higher than the 20–50% reported in previous adult or pediatric pancreatitis literature [11, 12]. Several factors may account for this discrepancy: (1) routine abdominal ultrasound screening within 24 hours of AAP diagnosis in this study improved the detection rate of ascites; (2) asparaginase-associated pancreatitis has unique pathophysiological characteristics with more pronounced pancreatic enzyme leakage, predisposing to the formation of significant ascites; and (3) the smaller abdominal cavity volume in children allows even small amounts of fluid to be clearly identified by ultrasound.

All 7 patients with moderately severe or severe AAP in this cohort had ascites and underwent PCD, but all 4 patients in the severe group ultimately died of MODS. This observation suggests that for this high-risk subgroup, drainage alone is insufficient to reverse the extremely poor prognosis. Previous studies have indicated that once asparaginase-associated pancreatitis progresses to severe disease, persistent organ failure is the key driver of mortality [13]. Therefore, there is an urgent need to establish more comprehensive and intensive treatment protocols. Beyond timely abdominal decompression, aggressive sustained life support, multidisciplinary collaboration, and precision management targeting organ failure may be necessary strategies to improve survival outcomes [14].

This study focused on comparing the clinical outcomes of early PCD versus conservative treatment in mild AAP patients with ascites, and found that drainage significantly shortened fasting duration and total hospital stay. Previous literature has largely focused on the diagnosis and comprehensive medical management of AAP, with a lack of pediatric evidence regarding the optimal management strategy for ascites as a common local complication, particularly the necessity of early PCD. In case reports of ALL patients with necrotizing pancreatitis, ascites may arise from pancreatic necrosis involving surrounding tissues, and conservative treatment is often inadequate [15]. However, these experiences derive from uncontrolled studies or adult populations, leaving a gap in evidence for drainage benefits in pediatric mild AAP with ascites. Our retrospective controlled study demonstrated that for mild AAP patients with ascites depth > 30 mm, the PCD group had significantly superior outcomes in terms of fasting duration and total hospital stay compared to the conservative group, suggesting that early intervention can accelerate gastrointestinal function recovery and shorten hospitalization. This finding supports the notion that even in cases with relatively mild inflammation, decompression of significant ascites may alleviate intra-abdominal hypertension and promote pancreatic recovery, with mechanisms similar to those by which abdominal drainage reduces systemic inflammation in adult pancreatitis.

Although no statistically significant differences were observed between the PCD and conservative groups in the recovery times of serum amylase, urinary amylase, or lipase, the PCD group achieved

significant improvements in fasting duration and total hospital stay—two core indicators reflecting overall recovery. This finding reveals the clinical benefit mechanisms of PCD in pancreatitis with ascites, which operate through two interrelated pathways: abdominal decompression and inflammatory mediator clearance.

From the perspective of abdominal decompression

Following the onset of AAP, large amounts of pancreatic enzymes leak into the abdominal cavity, forming pancreatitis-associated ascitic fluid rich in proteins and inflammatory mediators. Given the limited abdominal cavity volume in children, significant ascites rapidly elevates IAP. In patients with severe acute pancreatitis, the incidence of intra-abdominal hypertension reaches 45.7%, and patients with intra-abdominal hypertension have higher rates of organ failure, longer ICU stays, and significantly higher mortality [13]. For patients with pancreatitis-associated ascites, PCD is the preferred non-surgical intervention for reducing IAP. Singh et al. demonstrated in a study of 105 acute pancreatitis patients that IAP decreased significantly after PCD, and patients with a > 40% reduction in IAP from baseline at 48 hours post-PCD had significantly better survival [16]. Bansal et al. systematically reviewed and clearly stated that indications for ascites drainage include intra-abdominal hypertension, persistent or new-onset organ failure, and compressive symptoms, and that PCD is comparable to open decompression in reducing IAP, potentially avoiding open surgery in up to 81% of patients [17]. Therefore, for mild AAP patients with ascites depth exceeding 30 mm, early PCD can effectively reduce IAP,□□ the negative effects of intra-abdominal hypertension on respiratory function, intestinal barrier integrity, and venous return, thereby preventing progression to severe disease.

From the perspective of inflammatory mediator clearance

Another important mechanism of PCD is the removal of accumulated inflammatory mediators from the abdominal cavity, thereby attenuating systemic inflammation. In acute pancreatitis, large amounts of activated pancreatic enzymes leak into the abdominal cavity, stimulating peritoneal macrophages to release large quantities of proinflammatory cytokines. Studies have confirmed that the concentrations of proinflammatory cytokines (including IL-1β, IL-6, IL-8, IL-18, and TNF-α) in ascitic fluid are significantly higher than paired serum levels, forming a local "inflammatory pool" [18]. These inflammatory mediators can enter the systemic circulation through peritoneal lymphatic pathways and splanchnic blood circulation, triggering systemic inflammatory response syndrome and exacerbating multi-organ dysfunction. Animal experiments have revealed that removal of pancreatitis-associated ascitic fluid significantly reduces serum TNF-α and IL-6 levels while increasing the anti-inflammatory cytokine IL-10; moreover, expression of cyclooxygenase-2 and inducible nitric oxide synthase in pancreatic tissue is reduced, and hepatic mitochondrial dysfunction is alleviated [19]. Thus, early drainage to remove accumulated proinflammatory mediators from the abdominal cavity can effectively attenuate systemic inflammation and promote organ function recovery.

Consequently, the clinical benefit of PCD does not derive from accelerating the clearance of pancreatic enzymes from the blood—pancreatic enzymes that leak from the pancreas are primarily metabolized

through lymphatic and circulatory pathways, and abdominal drainage can only remove a small portion that has already accumulated in the abdominal cavity—but rather from reducing IAP, relieving gastrointestinal compression, promoting intestinal function recovery, while simultaneously removing proinflammatory mediators from the abdominal cavity and inhibiting sustained activation of systemic inflammatory response syndrome, thereby improving systemic metabolic status and organ function. This is the fundamental reason why the PCD group, despite having similar pancreatic enzyme recovery times to the conservative group, achieved significantly shorter overall hospitalization and fasting duration.

Regarding safety, only 1 patient (5.6%) in the PCD group developed puncture site oozing, with no serious complications such as abdominal infection, intestinal perforation, or catheter-related sepsis, suggesting that ultrasound-guided PCD is safe and feasible. In contrast, 30% of patients in the conservative group experienced adverse events (2 with progressive ascites requiring temporary puncture, 1 developing pancreatic pseudocyst). Although the difference in overall adverse event rates between the two groups did not reach statistical significance (P = 0.09), the conservative group had a higher proportion requiring "rescue intervention," suggesting that a "drain first" strategy for significant ascites may be superior to "observe first, rescue later." Whether the formation of pseudocysts in the conservative group was entirely attributable to the lack of drainage requires validation with larger samples. Overall, for mild AAP with ascites depth > 30 mm, active drainage does not additional risk and may reduce the incidence of local complications.

The 7 patients with moderately severe or severe AAP in this cohort all had significant ascites and underwent PCD, but all 4 patients in the severe group died. These data suggest that for severe AAP with persistent organ failure, simple ascites drainage is far from sufficient. The main causes of death were infected necrosis, abdominal compartment syndrome, and MODS. Such patients often require intensive care, vasoactive agents, continuous renal replacement therapy, and even surgical debridement—multidisciplinary comprehensive interventions. Therefore, early identification of risk factors for severe AAP and timely transfer to the ICU for comprehensive supportive care are more critical than relying solely on PCD.

Previous studies on the management of acute pancreatitis with ascites have largely focused on adults or severe cases, with a lack of standardized intervention indications and methods. In adult severe pancreatitis, ascites is considered an important trigger for MODS, and management strategies tend toward active drainage [20]. However, in children, particularly those with ALL who are immunosuppressed, the pathophysiology of AAP differs significantly from adult biliary or alcoholic pancreatitis, and mild cases account for a higher proportion. Previous literature on the management of ascites in pediatric AAP has largely extrapolated from adult severe pancreatitis experience, lacking evidence-based guidance for mild cases [21]. This study targeted the specific subgroup of mild AAP with ascites in ALL patients, using a relatively uniform drainage indication (ascites depth > 30 mm), filling the evidence gap regarding the impact of PCD on clinical outcomes in mild cases and providing reference for balancing conservative treatment and early drainage decisions in clinical practice.

Limitations

This study has several limitations. First, the retrospective single-center design may introduce selection bias; although drainage decisions were based on objective indicators, they were still influenced by family preferences. Second, the small sample size, particularly the moderately severe/severe subgroup with only 7 patients, limited the ability to perform multivariate analysis of prognostic factors. Third, the drainage threshold was set at 30 mm but was not compared with smaller (e.g., 20 mm) or larger (e.g., 40 mm) thresholds in a randomized fashion; the optimal threshold requires prospective validation. Fourth, the follow-up duration was limited, and long-term pancreatic exocrine insufficiency, diabetes, and ALL survival outcomes were not assessed. Fifth, IAP was not systematically measured, limiting the investigation of the relationship between ascites drainage and intra-abdominal hypertension. Sixth, propensity score matching or multivariate regression correction was not performed; although baseline characteristics were comparable between the two groups, selection bias cannot be completely excluded, and prospective studies are needed for validation. Future directions should integrate early warning scores, inflammatory marker monitoring, enhanced organ function support, and exploration of novel therapeutic targets.

Conclusions

PCD using an ascites depth > 30 mm as the intervention threshold significantly shortened fasting duration and total hospital stay in children with mild AAP, with a favorable safety profile. This approach may serve as an effective and safe intervention strategy for AAP complicated by significant ascites. However, for patients with severe AAP who have already developed persistent organ failure, simple ascites drainage is far from sufficient, and comprehensive intensive care and multidisciplinary management are essential.

Abbreviations

Abbreviation
Full term
ALL
Acute lymphoblastic leukemia
AAP
Asparaginase-associated pancreatitis
AP
Acute pancreatitis
ASP
Asparaginase
PCD
Percutaneous catheter drainage
PAAF

Pancreatitis-associated ascitic fluid
IAP
Intra-abdominal pressure
pSOFA
Pediatric Sequential Organ Failure Assessment
MODS
Multiple organ dysfunction syndrome
TPN
Total parenteral nutrition
ICU
Intensive care unit
IQR
Interquartile range
CI
Confidence interval
IL
Interleukin
TNF-α
Tumor necrosis factor-alpha
COX-2
Cyclooxygenase-2
iNOS
Inducible nitric oxide synthase

Declarations

Ethics approval and consent to participate: This study was approved by the Ethics Committee of Children's Hospital of Soochow University (Ethics Approval No. 2026CS098). Informed consent was waived due to the retrospective nature of the study.

Consent for publication: Not applicable.

Availability of data and materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no competing interests.

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References

1. Jelić M, Pavlović M, Mucavac L, et al. Causes of Death in Childhood Acute Lymphoblastic Leukemia: A Single-Center Experience. Med (Kaunas). 2025;61(7). 10.3390/medicina61071193.
2. Pieters R, Hunger SP, Boos J, et al. L-asparaginase treatment in acute lymphoblastic leukemia: a focus on Erwinia asparaginase. Cancer. 2011;117(2):238–49.
3. Gibson A, Hernandez C, Tejada FNH, et al. Asparaginase-associated pancreatitis in pediatric patients with acute lymphoblastic leukemia: current perspectives. Paediatr Drugs. 2021;23(5):457–63.
4. Rank CU, Wolthers BO, Grell K, et al. Asparaginase-associated pancreatitis in acute lymphoblastic leukemia: results from the NOPHO ALL2008 treatment of patients 1–45 years of age. J Clin Oncol. 2020;38(2):145–54.
5. Samanta J, Rana A, Dhaka N, et al. Ascites in acute pancreatitis: not a silent bystander. Pancreatology. 2019;19(5):646–52. 10.1016/j.pan.2019.06.004.
6. Zeng QX, Wu ZH, Huang DL, Huang YS, Zhong HJ. Association Between Ascites and Clinical Findings in Patients with Acute Pancreatitis: A Retrospective Study. Med Sci Monit. 2021;27:e933196. 10.12659/MSM.933196.
7. Rao JW, Li JR, Wu Y, et al. Ascites characteristics in acute pancreatitis: A prognostic indicator of organ failure and mortality. World J Gastroenterol. 2025;31(28):108926. 10.3748/wjg.v31.i28.108926.
8. Bush N, Rana SS. Ascites in Acute Pancreatitis: Clinical Implications and Management. Dig Dis Sci. 2022;67(6):1987–93. 10.1007/s10620-021-07063-6.
9. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis–2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62(1):102–11.
10. Smolka V, Karaskova E, Tyl J, et al. Etiology and classification of acute pancreatitis in children admitted to ICU using the Pediatric Sequential Organ Failure Assessment (pSOFA) score. Hepatobiliary Pancreat Dis Int. 2023;22(3):317–22.
11. Du X, Liu Z, Jia X, et al. Clinical analysis of asparaginase-associated pancreatitis in children. Pancreatology. 2022;22(6):706–12. 10.1016/j.pan.2022.06.003.
12. Thakur S, Kaur R, Bhatia L, et al. Acute Pancreatitis: Clinical Profile of 60 Patients. Cureus. 2023;15(12):e51234. 10.7759/cureus.51234.

13. Jena A, Singh AK, Kochhar R. Intra-abdominal hypertension and abdominal compartment syndrome in acute pancreatitis. Indian J Gastroenterol. 2023;42(4):455–66. 10.1007/s12664-023-01407-y.
14. Siebert M, Le Fouler A, Sitbon N, Cohen J, Abba J, Poupardin E. Management of abdominal compartment syndrome in acute pancreatitis. J Visc Surg. 2021;158(5):411–9. 10.1016/j.jviscsurg.2021.01.001.
15. Acer T, Malbora B, Ötgün İ, et al. Fat necrosis of transverse colon and necrotizing pancreatitis in a patient with acute lymphoblastic leukemia (ALL): cause of massive ascites and high fever. Turk J Pediatr. 2013;55(4):458–61.
16. Singh AK, Samanta J, Dawra S, et al. Reduction of intra-abdominal pressure after percutaneous catheter drainage of pancreatic fluid collection predicts survival. Pancreatology. 2020;20(4):772–7. 10.1016/j.pan.2020.04.012.
17. Bansal A, Gupta P, Singh H, et al. Drainage of pancreatic fluid collections in acute pancreatitis: A comprehensive overview. World J Clin Cases. 2022;10(20):6769–83.
18. Bezmarević M, van Dijk SM, Voermans RP, van Santvoort HC, Besselink MG. Management of (Peri)Pancreatic Collections in Acute Pancreatitis. Visc Med. 2019;35(2):91–6. 10.1159/000499631.
19. Souza LJ, Coelho AM, Sampietre SN, Martins JO, Cunha JE, Machado MC. Anti-inflammatory effects of peritoneal lavage in acute pancreatitis. Pancreas. 2010;39(8):1180–4.
20. Sharma V, Gorsi U, Gupta R, Rana SS. Percutaneous Interventions in Acute Necrotizing Pancreatitis. Trop Gastroenterol. 2016;37(1):4–18. 10.7869/tg.314.
21. de-Madaria E. Últimos avances en pancreatitis aguda [Latest advances in acute pancreatitis]. Gastroenterol Hepatol. 2012;35(Suppl 1):98–101. 10.1016/S0210-5705(12)70041-9.