Abdominal paracentesis drainage improves outcome of acute pancreatitis complicated with intra-abdominal hypertension in early phase

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ABSTRACT

Background: Intra-abdominal hypertension (IAH) is an important risk factor for organ dysfunction, and it occurs in the early phase of severe acute pancreatitis (SAP). We have reported a novel step-up approach and shown the benefit of performing abdominal paracentesis drainage (APD) ahead of percutaneous catheter drainage (PCD) when treating Patients with SAP with fluid collections. This study aimed to evaluate the efficacy of APD in Patients with SAP complicated with IAH in the early phase.

Methods: In the present study, 206 AP patients complicated with IAH in the early phase were enrolled in hospital between June 2017 and December 2020. The patients were divided into two groups: 109 underwent APD (APD group) and 97 were managed without APD (non-APD group). We retrospectively compared the outcomes of the APD and non-APD groups for IAH treatment. The parameters including mortality, infection, organ failure, inflammatory factors, indications for further interventions, and drainage-related complications were observed.

Results: The demographic data and severity scores of the two groups were comparable. The mortality rate was lower in the APD group (3.7%) than in the non-APD group (8.2%). Compared with the non-APD group, the intra-abdominal pressure and laboratory parameters of the APD group decreased more rapidly, and the mean number of failed organs was lower. However, there was no significant difference in incidence of infections between the two groups.

Conclusions: Application of APD is beneficial to AP patients. It significantly attenuated inflammation injury, avoided further interventions, and reduced multiple organ failure.

Key Indexing Terms: Acute pancreatitis; Abdominal paracentesis drainage (APD); Intra-abdominal hypertension (IAH); Abdominal compartment syndrome (ACS); Intra-abdominal pressure (IAP). **[Am J Med Sci 2023;365(1):48–55.]**

INTRODUCTION

Acute pancreatitis (AP) is a life-threatening inflammatory disease. The overall mortality of AP is about 5% and can reach 20–30% in patients with severe AP (SAP) due to the development of pancreatic and extrapancreatic necrosis, their subsequent infection and multisystem organ failure (MOF).[1,2] SAP develops in two phases.[3] During the first 1–2 weeks, a proinflammatory response occurs that results in systemic inflammatory response syndrome (SIRS); a sterile response in which sepsis or infection rarely occurs. Peripancreatic fluid collections are common in the early phase of SAP. More and more fluid collections in the cavity increase the intra-abdominal pressure (IAP) and result in intra-abdominal hypertension (IAH). The incidence of IAH in patients with SAP is 60–80%.[4] IAH is an important risk factor for organ dysfunction and occurs in the early phase of SAP.[5] IAH contributes to organ dysfunction and leads to the development of abdominal compartment syndrome (ACS). ACS resulting from sustained increase of IAP can cause early multiple (respiratory, cardiovascular, renal and hepatic) organ failure, and it is also defined as a state of serious organ dysfunction.[6]

IAH and ACS are now recognized as being dynamic processes characterized by a constantly changing continuum of physiological events. Patients with SAP developing ACS defined by IAP >20 mmHg, associated with new organ failure, need to undergo decompression laparotomy. AP patients are at risk for IAH because of the large volume of intra-abdominal and peripancreatic inflammatory fluid,[7]

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so we think the first line treatment would be the percutaneous drainage of the intraperitoneal exudates, which could lead to a significant drop in IAP.

In our previous study, we reported a novel step-up approach and showed it was beneficial to perform abdominal paracentesis drainage (APD) ahead of percutaneous catheter drainage (PCD) when treating AP patients with fluid collection.[8] To further improve the outcome of SAP, we have performed APD in patients with IAH since 2017. In the current study, we collected data for our patients from June 2017 to December 2020 and we retrospectively compared patients with and without APD. The results indicate that it is advisable to conduct APD because it can reduce the mortality rate of AP patients and reduce costs, mainly by eliminating inflammatory materials (cytokines), postponing further intervention, and decreasing the incidence of new onset of organ failure.

METHODS

Patient selection

Patients diagnosed with SAP and moderately severe AP (MSAP) in the General Hospital of Western Theater Command (Chengdu Military General Hospital) were enrolled in this retrospective study, from June 2017 to December 2020. The diagnoses were based on clinical findings, biochemical parameters, and computed tomography severity index (CTSI).[3] This study was performed according to the principles of the Declaration of Helsinki (modified 2000), and ethical approval was given by the Medical Ethics Committee of the General Hospital of Western Theater Command. All of the patients were informed of the possible complications of surgical treatment, and those who underwent APD were informed of possible discomfort associated with puncture. Informed consent was obtained before the surgical procedure and not for anything specifically related to this study.

Inclusion criteria were: patients age >18 years with a first episode of MSAP or SAP, complicated with IAH in the early phase (IAP >12 mmHg, 1 mmHg=0.133 kPa),[9,10] admitted to hospital within 72 h of disease onset, and ultrasound or CT showed a lot of fluid collection (maximal diameter >3 cm) in the abdominal or pelvic cavity. Exclusion criteria were: patients complicated with immunodeficiency, previous abdominal surgery, or suspected carcinoma of the pancreas, or AP subsequent to endoscopic retrograde cholangiopancreatography (ERCP) or abdominal trauma.

Patients were divided into two groups: APD and non-APD, according to whether APD was performed in the early phase of SAP.

General management

Patients were treated by our standard management of pancreatitis protocol and practice guidelines for AP, with gastrointestinal decompression, trypsin inhibitors, H_2 receptor antagonists, fluid resuscitation, antibiotics, redress of water–electrolyte and acid–base imbalance.[11-13] Computed tomography (CT) and transabdominal ultrasound were performed within 48 h of admission. The images obtained were double-checked by two experienced radiologists to determine CTSI. Acute physiology and chronic health evaluation II (APACHE II) score, modified Marshall score and CTSI were calculated for each patient at the time of admission and 1 week after APD intervention.

IAP measurement and monitoring

IAP was measured immediately upon admission, followed by modification of the low cost transvesical technique described by Basu.[14] Before and after determination of IAP, severe acute pain should be controlled by analgesics, such as paracetamol, tramadol hydrochloride and morphine. We inserted a Foley catheter into the bladder and instilled 25 mL sterile saline (1 mm Hg=1.36 cmH_2O) to measure IAP. IAH was defined as consistently increased IAP ≥12 m Hg recorded by two readings during at least 8 h.[10] ACS was defined as sustained IAP >20 mmHg (measured by two readings at least 4 h apart) that was associated with organ dysfunction or failure.[10]

APD

AP patients who underwent APD fulfilled the following conditions: complicated with IAH in early phase (IAP >12 mmHg); ultrasound or CT showed a lot of fluid collection (max diameter >3 cm) in the abdominal or pelvic cavity; and there was a feasible pathway under imaging examination. APD was performed as early as possible in the early phase of AP, within 1 week of disease onset. Initially, an 8–12 Fr catheter was used to drain fluid from the abdominal cavity for 3–5 days. If the catheter drainage was not competent, we placed additional catheters or gradually replaced the catheter with a larger drainage tube (up to 22 Fr) over the next 5–10 days (Fig. 2). The drainage catheter was removed in the following situations: (1) catheter output <10 ml/day of nonpurulent fluid for 48 h; and (2) IAP <10 mm Hg for 48 h (in absence of any acute organ dysfunction).[10] Follow-up was scheduled in the outpatient department after the patients were discharged.

Data collection

The following data were collected: mortality rate; days in hospital and intensive care unit (ICU); hospitalization cost; incidence of organ failure (including ACS); further interventions (including decompression laparotomy and necrosectomy); level of IAP; laboratory parameters [C-reactive protein (CRP), interleukin (IL)–6, IL-10 and tumor necrosis factor (TNF)-α]; severity scores (APACHE II, CTSI and Marshall score); and presence of infective complications. The level of IAP and laboratory parameters and

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severity scores were collected upon admission in both groups, and 1 week after initial APD (8–14 days from onset of AP) in the APD group or 2 weeks after disease onset in the non-APD group. Organ dysfunction/failure was defined as modified Marshall score ≥2 for that particular organ system.[15]

Statistical analysis

Statistical analyses were performed using SPSS version for Windows 13.0 (SPSS, Munich, Germany). Continuous variables are presented as he median (interquartile range) or mean±SD and the proportions are expressed as numbers (%). Student's *t*-test or Mann–Whitney *U* test was used for data analysis according to the continuous data with normal or non-normal distributions. Differences in categorical variables were analyzed by χ^2 and Fisher's exact tests. A two-tailed $P<0.05$ was considered statistically significant.

RESULTS

General information

Between June 2017 and December 2020, 549 patients with AP were admitted to our department, and 334 were without IAH. Therefore, 215 patients complicated with IAH in early phase were included, 88 had MSAP and 127 had SAP according to the revised Atlanta severity classification. Nine patients were excluded; five subsequent to ERCP, one complicated with immunodeficiency, and three had a history of previous abdominal surgery. The remaining 206 patients were enrolled in the study; 109 were in the APD group and 97 in the non-APD group (Fig. 1).

Baseline data

There was no difference in the demographic data of the APD and non-APD groups, including age, sex and etiology. The most important etiology of AP in each group was mainly from bile duct problems, followed by hyperlipidemia. The laboratory parameters (CRP, IL-6, IL-10 and TNF-α) and severity scores (APACHE-II, CTSI and Marshall score) of the APD group were similar to those of the non-APD group ($P>0.05$). The mean level of IAP was higher in the APD group than in the non-APD group ($P>0.05$) (Table 1).

Characteristics of APD

We used 196 catheters in 109 patients. The median duration of APD was 11.6 days. The diameter of the APD catheters varied from 8 to 22 Fr; the size most frequently used was 12 Fr. During interventions under imaging guidance, either in the radiology suite or at the patient's bedside, the maximum catheter diameter used was 12 Fr initially, followed by upsizing to 16–22 Fr, such as in

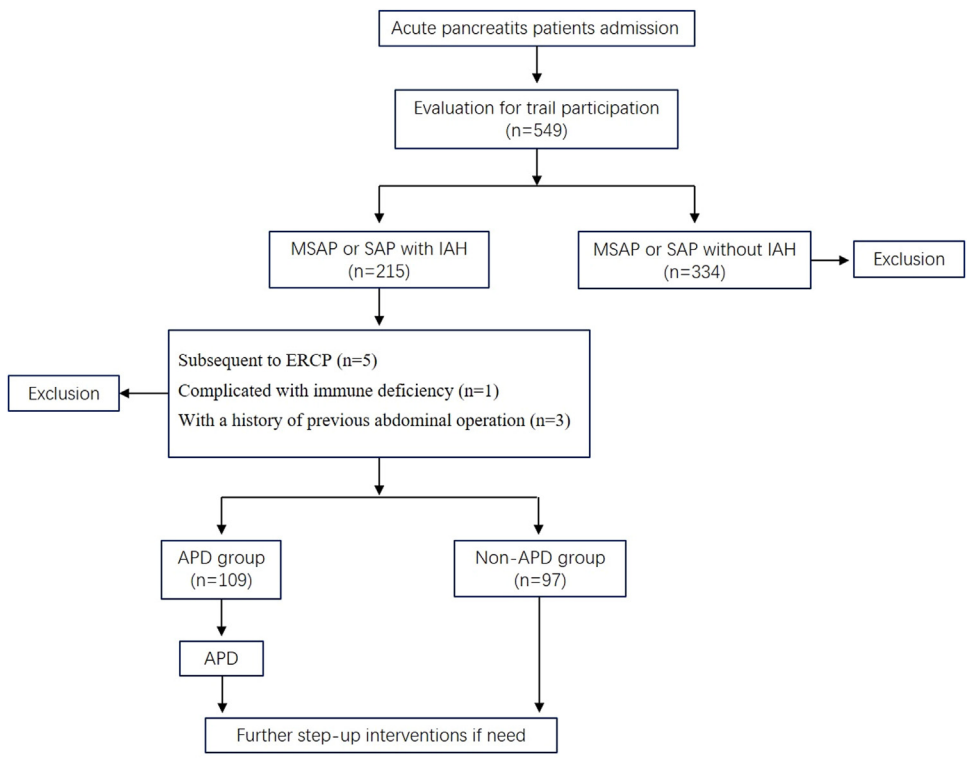

FIG. 1. Study flowchart. This flowchart shows the process of patient's selection of acute pancreatitis (AP). APD: abdominal paracentesis drainage; IAH: intra-abdominal hypertension.

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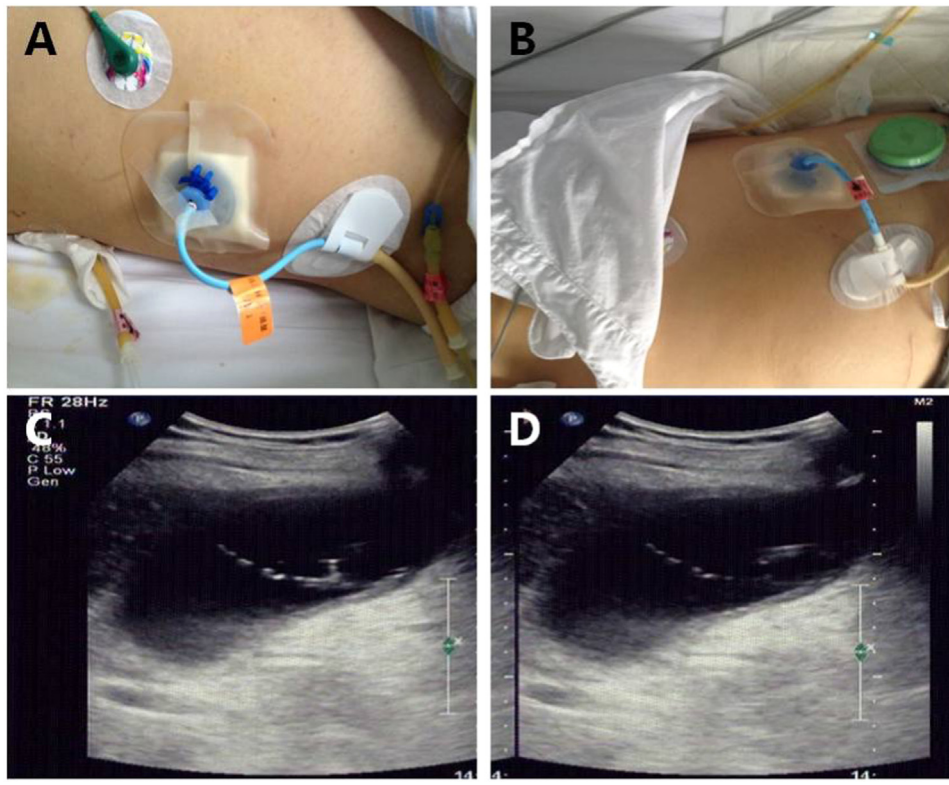

FIG. 2. The placement of catheters under APD. (A-B) two catheters respectively in the right paracolic sulci (RPCS) and left paracolic sulci (LPCS); (C-D) in ultrasound images, the catheter in the RPCS.

patients with gross ascites and ACS. Catheters were upsized in 39 patients in the APD group.

Mortality rate, further interventions and medical costs

Mortality rate in the non-APD group was 8.25% (8/97), compared with 3.67% (4/109 patients) in the APD group ($P<0.05$). The causes of death were mainly multiple organ dysfunction syndrome (MODS) and severe infection in the late phase. Significantly more patients accepted decompression laparotomy or necrosectomy in the non-APD group than in the APD group ($P<0.05$). There was no difference in days in ICU between the groups ($P>0.05$). The average total cost during hospitalization was significantly higher in the non-APD group than in the APD group ($P<0.05$) (Table 2).

Organ failure

There were significant differences in the mean number of failed organs and the MOF rate between the two groups ($P<0.05$) (Table 3). The mean duration of organ failure in the non-APD group was higher than in the APD group, although the difference was not significant ($P>0.05$). The number of patients who developed ACS was significantly higher in the non-APD group than in the APD group ($P<0.05$). The number of patients who developed persistent SIRS was significantly higher in the APD group than in the non-APD group ($P<0.05$). These results indicate that the time necessary for reversal of organ failure was significantly higher in the non-APD group than in the APD group ($P<0.05$) (Table 3).

Clinical and laboratory parameter data

Clinical and laboratory parameter data were collected 1 week after initial APD (8−14 days from onset of AP) in the APD group or 2 weeks after disease onset in the non-APD group. CRP, IL-6, IL-10 and TNF-α were analyzed. Compared with the non-APD group, the serum levels of the above parameters (except TNF-α) in the APD group were significantly decreased, suggesting the alleviating effect of APD treatment on AP-induced inflammatory response. The APACHE II, Ranson and Marshall scores were significantly lower in the APD group compared with those in the non-APD group ($P<0.05$) (Table 3). The mean level of IAP was significantly lower in the APD group compared with that in the non-APD group ($P<0.05$) (Table 4).

Infective complications

There were more patients in the APD group (85/109, 78.0%) who had polymicrobial infections compared with those in the non-APD group (74/97, 76.3%). However,

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TABLE 1. The baseline characteristics of 206 patients enrolled in this study.

Characteristic	APD group	non-APD group	*P*
Number of patients	109	97	
Demographic data			
Age (mean ± SD)	58 ± 11.5	56 ± 10.3	>0.05
Male:female	59:50	52:45	>0.05
Etiology			>0.05
Gallstone	51 (46.8%)	47 (48.5%)	
Alcohol abuse	32 (29.4%)	28 (28.9%)	
Hyperlipemia	20 (18.3%)	17 (17.5%)	
Other	6 (5.5%)	5 (5.1%)	
Classification			>0.05
MSAP (84)	44	40	
SAP (122)	65	57	
Laboratory parameters			
CRP (mg/L)	138.94 ± 18.73	140.23 ± 17.45	>0.05
IL-6 (pg/L)	387.32 ± 62.31	376.65 ± 65.33	>0.05
IL-10 (pg/L)	128.41 ± 38.52	131.97 ± 36.68	>0.05
TNF-α(pg/L)	18.24 ± 5.33	19.36 ± 5.49	>0.05
Severity scores			
Initial APACHII score (mean ± SD)	15.8 ± 4.33	16.2 ± 3.91	>0.05
Initial CTSI score (mean ± SD)	7.9 ± 2.76	8.3 ± 2.90	>0.05
Marshall scores	4.1 ± 0.95	4.4 ± 1.07	>0.05
IAP	13.9 ± 3.2	14.3 ± 3.3	>0.05

Abbreviations: MSAP, moderately severe acute pancreatitis; SAP, severe acute pancreatitis; APACHE II, acute physiology and chronic health evaluation II; CTSI, computerized tomography severity index; IAH, intra-abdominal hypertension; IAP, intra-abdominal pressure; CRP, C-reactive protein; IL-6, interleukin-6; IL-10, interleukin-10; TNF-α, tumor necrosis factor-α.
The data are expressed as the mean ± SE. There was no difference in baseline characteristics between APD group and non-APD group groups.

there was no significant difference between the groups. The incidence of pneumonia was significantly higher in the non-APD group (11/97, 11.3%) compared with that in the APD group (9/109, 8.3%), but not sepsis or bacteremia (Table 5).

DISCUSSION

IAH and ACS have been increasingly recognized as causes of significant morbidity and mortality in SAP during the last decade. The World Society of the Abdominal Compartment Syndrome has recently developed consensus definitions outlining the standards for IAP measurement, IAH and ACS.[16] Elevated IAP induces splanchnic hypoperfusion, which decreases intestinal perfusion. Intestinal ischemia is believed to cause MODS mediated by the inflammatory response. Sustained IAH can induce significant dysfunction of cardiovascular, respiratory, renal, gastrointestinal and central nervous systems.[10]

TABLE 2. The mortality rate, further interventions and medical economics between two groups.

Characteristic	APD group	non-APD group	*P*
Number of patients	109	97	
Mortality	4 (3.7%)	8 (8.2%)	<0.05*
Disease specific	3 (2.8%)	7 (7.2%)	<0.05*
Unrelated to disease	1 (0.9%)	1 (1.0%)	<0.05
Further interventions			
Decompression laparotomy	3 (2.8%)	7 (7.2%)	<0.05*
Necrosectomy	31 (28.4%)	39 (40.2%)	<0.05*
Medical economics (mean±SD)			
Days in hospital	50.9 ± 27.46	57.5 ± 31.39	<0.05*
Days in intensive care unit (ICU)	7.8 ± 4.13	8.1 ± 4.54	>0.05
Total cost during hospitalization (Dollars)	8658.7 ± 3465.37	11,943.5 ± 6358.76	<0.05*

Abbreviation: APD, abdominal paracentesis drainage.
* Significant difference. The data are expressed as the mean ± SE. * p <0.05 vs the non-APD group.

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TABLE 3. The organ failure related parameters between two groups.

Characteristic	APD group	non-APD group	*P*
Number of patients	109	97	
Organ failure			<0.05*
No organ failure	35 (32.1%)	24 (24.7%)	
Single-organ failure	41 (37.6%)	36 (37.1%)	
Multiple-organ failure	33 (30.3%)	37 (38.1%)	
RDS	38 (34.9%)	45 (46.4%)	<0.05*
ACS	2 (1.8%)	5 (5.5%)	<0.05*

Abbreviations: ACS, abdominal compartment syndrome; APD, abdominal paracentesis drainage; ARDS: acute respiratory distress syndrome.
* Significant difference. The data are expressed as the mean ± SE. * p <0.05 vs the non-APD group.

TABLE 4. Severity scores and IAP after treatment in early phase between two groups.

Characteristic	APD group	non-APD group	*P*
Number of patients	109	97	
Laboratory parameters			
CRP (mg/L)	74.52 ± 14.25	94.18 ± 20.33	<0.05*
IL-6 (pg/L)	95.27 ± 26.27	128.64 ± 37.19	<0.05*
IL-10 (pg/L)	43.82 ± 13.46	68.67 ± 19.54	<0.05*
TNF-α(pg/L)	11.07 ± 3.87	12.15 ± 3.64	>0.05
Severity scores			
APACHE Ⅱ score (mean ± SD)	8.4 ± 2.04	11.3 ± 3.41	<0.05*
Ranson score (mean ± SD)	2.3 ± 0.94	2.8 ± 1.03	<0.05*
Marshall scores	2.8 ± 0.88	3.4 ± 0.97	<0.05*
IAP	9.3 ± 2.81	12.7 ± 3.34	<0.05*

Abbreviations: APD, abdominal paracentesis drainage; CRP, c-reactive protein; IAP, intra-abdominal pressure; IL, interleukin; TNF, tumor necrosis factor.
* Significant difference. The data are expressed as the mean ± SE. * p <0.05 vs the non-APD group.

TABLE 5. Infected complications between two groups.

Characteristic	APD group	non-APD group	*P*
Number of patients	109	97	
Incidence of infections			<0.05*
Polymicrobial infections	85 (78.0%)	74 (76.3%)	
Monomicrobial infections	13 (11.9%)	12 (12.4%)	
No infection	12 (11.1%)	11 (11.3%)	
The incidence of pneumonia	9 (8.3%)	11 (11.3%)	<0.05*
The incidence of bacteremia	51 (46.8%))	47 (48.5%)	>0.05
The incidence of sepsis	32 (29.4%)	30 (30.9%)	>0.05

Abbreviation: APD, abdominal paracentesis drainage.
* Significant difference. The data are expressed as the mean ± SE. * p <0.05 vs the non-APD group.

In the early phase of SAP, IAH may play an important role in the process of organ failure and SIRS and induce high mortality. Abdominal fluid collection is the most important factor that results in IAH.[17] In particular, fluid collections can affect the development of AP and clinical outcome for patients. Abdominal fluid collection contains a lot of inflammatory mediators such as cytokines, chemokines and reactive oxygen species, which results in SIRS and MODS, converting AP to a severe form.[18,19] So, release of abdominal fluid collections to decrease IAP is particularly important in the early phase.

Surgical decompression laparotomy may be the optimal solution, but mortality remains high and is accompanied by an increased risk of enteric fistula, hemorrhage, infection and necrosis.[20] Through this retrospective study, we found that APD in the early phase of SAP can reduce IAP, relieve organ dysfunction, decrease mortality and decrease the need for further interventions, especially surgical decompression laparotomy.

In our study, the mortality rate in patients managed with APD was 1.9% (4/109), compared with 6.8% (2/29) in those without APD. APD to treat AP patients with IAH

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resulted in a better curative effect, for the following possible reasons: (1) APD alleviated organ failure by lowering IAP; (2) APD decreased infections by eliminating inflammatory factors; and (3) APD reduced the need for further intervention, especially decompression laparotomy.

The lack of effective circulating blood volume and the increased IAP can significantly reduce renal perfusion, resulting in renal and cardiac dysfunction and failure in the early phase of AP. APD in the early phase can relieve IAP in patients with IAH, increasing venous return of the inferior vena cava. In our study, the incidence of renal failure was only 21% in the APD group, which was significantly lower than 32% in the non-APD group. APD can also reduce IAP to make the diaphragm descend, lower intrathoracic pressure, and increase total lung capacity, reducing the incidence of respiratory distress syndrome. In this study, the incidence of respiratory distress syndrome was significantly lower in the APD group (14%) than in the non-APD group (27%) ($P<0.05$).

Secondary infection of fluid collections is the leading cause of mortality in AP.[21,22] The infection rate in the APD group was similar to that of the non-APD group in our study. Moreover, the incidence of pneumonia was lower in the APD group than that in the non-APD group. It was different from other report that extra puncturation could increase the incidence of infection.[23] APD avoids additional infections because it eliminates fluid collection, thus removing the necessary conditions for microorganisms to thrive. The early phase of SAP, which usually lasts for about 1 week, is characterized by a complex inflammatory reaction. The cytokine cascades are activated by pancreatic inflammation and SIRS.[24,25] APD may reduce inflammatory factors, such as CRP, IL-6 and IL-10, reduce severity of SIRS, and benefit AP patients. Although extra puncturation could increase the incidence of infection, the infective complications rate in the APD group was similar to that in the non-APD group.

Although APD seems to be beneficial to AP patients, there are some factors in association with IAH which may result in poor therapeutic effect, such as decreased abdominal wall compliance and increased intraluminal contents.[17] Some retroperitoneal fluid collection can cause difficulty, and CT-guided APD would improve the therapeutic results. APD also carries the risk of introducing additional infection; therefore, we should try to avoid secondary infection caused by interventions.

AUTHOR CONTRIBUTIONS

Yi Wen, WQZ and HYL contributed equally to this work. YW and WQZ participated in the writing of the main manuscript. ZLL participated in the study conception and design. HYL, FZT and TW participated in statistical data analysis, and interpretation. HYL, ZH and LC participated in preparing all the figures. ZLL and LJT participated in the revision of the manuscript and final approval.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interests regarding the publication of this paper.

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