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Effect of continuous hemofiltration on severe acute pancreatitis with different intra-abdominal pressure

A cohort study

Yongle Xie, Master of Medicine[a,*], Yuan Yuan, Doctor of Medicine[b], Wentao Su, Bachelor of Medicine[a], Nan Qing, Bachelor of Medicine[a], Hongwei Xin, Bachelor of Medicine[a], Xiaoying Wang, Bachelor of Medicine[a], Jing Tian, Bachelor of Medicine[a], Yun Li, Bachelor of Medicine[a], Junnian Zhu, Bachelor of Medicine[a]

Abstract

Background: The clinical efficacy and timing of continuous veno-venous hemofiltration (CVVH) in the treatment of severe acute pancreatitis (SAP) remain uncertain. In this prospective cohort study, patients with SAP were classified according to intra-abdominal pressure (IAP).

Methods: Seventy-four patients with SAP admitted to the intensive care unit were randomly divided into group A (IAP ≥20 mm Hg) and group B (with IAP ≤20 mm Hg). Then, according to whether CVVH was administered or not, groups A and B were divided into 4 subgroups: group A1 and B1 (non-CVVH treatment), group A2 and B2 (CVVH treatment). Changes in clinical and laboratory indicators were recorded before and on the seventh day after treatment, and clinical outcomes were analyzed.

Results: Before treatment, there was no significant difference in general conditions between subgroups A1 and A2, and between subgroups B1 and B2. After CVVH treatment, the indicators recorded in group A2 were significantly improved compared to those in group A1 ($P < .05$). In group A2, the 28 day operation rate was lower ($P < .05$), as mechanical ventilation, gastric decompression, and intensive care unit treatment time were shorter ($P < .05$). However, there was no statistically significant difference in any of the above indicators between subgroups B ($P > .05$). Groups A2 and B2 had more days of negative fluid balance within 1 week of admission than groups A1 and B1 ($P < .05$).

Conclusions: For SAP, patients with IAP ≥20 mm Hg can benefit from treatment with CVVH, but for patients with IAP ≤20 mm Hg, the efficacy is not clear, and monitoring IAP may be an indicator to decide whether or when to initiate CVVH. Negative fluid balance caused by CVVH treatment may be one of the reasons for the benefit of this group of patients.

Abbreviations: ACS = abdominal compartment syndrome, AKI = acute kidney injury, AP = acute pancreatitis, APACHE II = acute physiology and chronic health evaluation score II, CVVH = continuous veno-venous hemofiltration, IAH = intra-abdominal hypertension, IAP = intra-abdominal pressure, ICU = intensive care unit, MV = mechanical ventilation, SAP = severe acute pancreatitis.

Keywords: continuous veno-venous hemofiltration, intra-abdominal pressure, severe acute pancreatitis

1. Introduction

Severe acute pancreatitis (SAP) is a severe systemic inflammatory response disorder that is initiated by pancreatic autodigestion. Inflammatory cells are activated and release massive cytokines, and the resulting cytokine-level chain reaction is an important reason for the aggravation of SAP.[1] Inflammatory mediators play an essential role in the development of acute pancreatitis (AP) and systemic complications, which are the dominant causes

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The study was approved by our hospital, and ethics approval was obtained from the medical ethics committee. Signed informed consent documents were obtained from all the patients.

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The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

[a] *Department of Intensive Care Unit, the First Hospital of Tianshui City, Gansu, China,* [b] *Department of Intensive Care Unit, Gansu Provincial People's Hospital, Gansu, China.*

[*] *Correspondence: Yongle Xie, Department of Intensive Care Unit, the First Hospital of Tianshui City, No. 105 Jianshe Road, Qinzhou Dist, Tianshui City, Gansu Province, China (e-mail: ylxie007@163.com).*

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of patient mortality.[2] Continuous hemofiltration therapy can effectively reduce the levels of inflammatory factors in patients with SAP, improve their biochemical and physiological indicators, and have a high clinical application value.[3,4] Accordingly, continuous blood purification has been widely used for the treatment of SAP.[5–8] However, its efficiency and safety are uncertain,[9] and there is no recognized standard[10,11] for the opportunity of hemofiltration in patients with SAP. In particular, there is no clear guidance and suggestions for the implementation of renal replacement therapy in patients with severe metabolic disorders that are not life-threatening.[12] At present, for SAP, there is no effective indicator to judge whether continuous veno-venous hemofiltration (CVVH) should be implemented and when to implement CVVH. Therefore, this prospective cohort study of SAP patients with intra-abdominal hypertension (IAH) has the following 3 purposes: What is the effect of CVVH treatment? Is the curative effect of CVVH different for patients with different intra-abdominal pressure (IAP) values? Can IAP be used as a clinical monitoring indicator to initiate CVVH?

2. Materials and methods

2.1. Patients and groups

The study period was from June 2015 to December 2019. Selected patients who were admitted to the intensive care unit (ICU) of Tianshui People's Hospital in Gansu Province within 72 hours after the onset of the disease and diagnosed with SAP, all met the revised Atlanta criteria for SAP.[13] The severity of SAP was evaluated according to the modified Marshall score, acute physiology chronic health evaluation score II (APACHE II), and CT severity index.

After admission to the ICU, all patients were catheterized and connected to a sensor to monitor IAP, and mean values of 12 IAP measurements were obtained 12 hours later. Patients with mean IAP $\geq$20 mm Hg were included in group A, and $\leq$20 mm Hg in group B. Patients were then divided into 4 subgroups by the random number table method according to whether CVVH was administered or not, without CVVH in groups A1 and B1, and with CVVH in groups A2 and B2. Patients who were already enrolled in A1 or B1, if they had to be administered CVVH because of obvious renal failure, oliguria, and aggravation during the treatment, they were excluded from the study. Finally, 17 patients were excluded, and 74 patients were included in the study. There were no significant differences in the general characteristics of patients between groups A1 and A2 and between groups B1 and B2, including sex, age, body mass index, modified Marshall score, CT severity index score level, blood lactic acid, number of patients with acute respiratory distress syndrome and mechanical ventilation (MV), and cause of disease ($P > .05$) (Table 1).

2.2. Treatment methods

All the patients were treated with routine internal medicine therapy. Vital signs of patients were closely monitored, and patients were given gastrointestinal decompression and fasting, with oxygen inhalation or MV to achieve an oxygen saturation of >95%. In the early stage, controlled liquid resuscitation was adopted, and omeprazole sodium for injection was intravenously injected to protect the gastric mucosa and indirectly inhibit pancreatic enzyme secretion. Octreotide was injected at a rate of 0.025 mg/h to inhibit pancreatic enzyme secretion. All patients received systemic antibiotic therapy to prevent infectious complications. Nutritional support was initially started with total parenteral nutrition, which was later combined with enteral feeding by placing a nasointestinal tube, as soon as gastrointestinal peristaltic movement was confirmed, and the dose of enteral nutrition was adjusted according to the feedback of the examinations. Patients (oxygen saturation continuously <90%) who developed acute respiratory distress syndrome underwent tracheal intubation to enable ventilatory support according to the protective-ventilation strategy. Symptomatic and supportive treatments were performed according to the condition of the patients. At 12 hours after admission, patients in groups A2 and B2 were administered CVVH, while those in groups A1 and B1 were not.

2.3. Continuous veno-venous hemofiltration

For vascular access, a double coaxial lumen 14-Fr catheter was inserted percutaneously through the right femoral vein using the

Table 1

Patients' general characteristics of subgroup A and subgroup B.

Characteristics	Group A1 (n = 15)	Group A2 (n = 21)	P	Group B1 (n = 16)	Group B2 (n = 22)	P
Sex (male/female)	11/4	12/9	.319‡	10/6	17/5	.321‡
Average age (yrs)	47.2 ± 8.5	50.2 ± 9.2	.330†	45.6 ± 7.4	46.8 ± 8.6	.653†
Body mass index (kg/m^2)	35.87 ± 6.14	37.43 ± 7.18	.500†	35.06 ± 6.61	37.45 ± 5.25	.222†
Modified Marshall score	3.53 ± 0.83	4.05 ± 1.17	.559†	2.44 ± 0.81	2.59 ± 1.18	.113†
CT severity index	6.80 ± 1.37	7.52 ± 1.50	.149†	6.00 ± 1.37	5.50 ± 0.91	.246*
ARDS (yes/no)	7/8	12/9	.535‡	5/11	8/14	.743‡
Mechanical ventilation (yes/no)	10/5	17/4	.329‡	8/8	12/10	.782‡
Blood lactic acid (mmol/L)	4.08 ± 0.91	4.41 ± 1.14	.358†	3.08 ± 0.79	3.47 ± 0.90	.185†
Cause of disease [n (%)]			.992‡			.923‡
Cholelithiasis	7 (46.7%)	9 (42.9%)		6 (37.5%)	9 (40.9%)	
Hyperlipemia	3 (20.0%)	5 (23.8%)		3 (18.8%)	3 (13.6%)	
Alcoholism	2 (13.3%)	3 (14.3%)		3 (18.8%)	3 (13.6%)	
Others	3 (20.0%)	4 (19.0%)		4 (25.0%)	7 (31.8%)	

ARDS = acute respiratory distress syndrome.

* Mann–Whitney test.

† Independent samples *t* test.

‡ Chi square test.

Seldinger technique. CVVH was performed using the Prismaflex continuous blood purification system (produced by Gambro, Sweden) with the M100 set via an AN69 filter, and 4000 mL of potassium-free hemofiltration base fluid from Chengdu Qingshan Likang, according to the specific situation of each patient, 10 to 20 mL of 10% potassium chloride was injected into each bag. The parameters were as follows: therapeutic dose, 30 mL/kg per hour; blood flow velocity, 180 mL/min; dilution mode, pre-dilution 50%; frequency of filter replacement, 48 to 72 hours (depending on transfilter pressure). Anticoagulation regimen: heparin, the first dose, 20 μ/kg, sustained dose, 5 to 15 kg/h. Activated partial thromboplastin time was monitored every 4 to 6 hours to maintain activated partial thromboplastin time prolongation to 1.5 to 2.5 times. Patients with active bleeding or high bleeding risk were treated with regional citrate anticoagulation (4% sodium citrate and 10% calcium gluconate); detection range for free Ca^{2+} ions before the filter, 0.25 to 0.35 mmol/L; and detection range for free Ca^{2+} ions after filter,1.12 to 1.20 mmol/L. Continuous treatment was performed for 1 to 7 days. The ultrafiltration rate was adjusted according to diuresis and fluid balance.

CVVH was stopped when the following 2 conditions were met: Oliguria or anuria disappeared; Modified Marshall score (Table 2) was decreased by at least 1 point, which was calculated daily (using the worst values of physiological variables for the day for a particular organ system) and was used to follow-up the response of patients to therapy and individualize day to day hospital management of the patient (organ failure as defined by the Atlanta Symposium was used only to determine the severity of AP).

2.4. IAP measurement

The intra-vesicular pressure was used as the IAP. IAP measurements were performed using an 18 Fr standard 3-way bladder catheter connected to the insertion of a transducer to a 24-hour bedside monitor. The connection between the flushing port and the transducer is effectuated with a triple tap. The zero point for fixation of the transducer was established at the level of the mid-axillary line. After the system was set to 0, the measured data were easily read off from the bedside monitor. The IAP was measured in the supine position at end-expiration when 25 mL of sterile saline was injected retrogradely into the bladder, and the catheter was clamped immediately distal to the connection.[14] The actual IAP value appeared directly in mm Hg and required no further conversion. IAP measurements are subject to significant volatility and uncertainty. Therefore, all IAP values in this study were the average values of 12 or 24 hour, which were measured under the condition of quiet or sedative analgesia after control of severe acute pain by optimal use of analgesics (including non-steroidal anti-inflammatory drugs, pethidine hydrochloride, fentanyl, and morphine as necessary in individual cases) to minimize the possible confounding effect of pain on IAP measurement. Pain control was assessed using the numeric rating scale targeting values $\leq$5 (on a scale from 1–10) in awake patients or the Richmond Agitation Sedation Scale $\leq$0 in intubated patients.

2.5. Daily fluid balance

Daily fluid input included oral, enteral, and intravenous fluids. Daily fluid output included urine volume, ultrafiltration, and fluid loss from drains and tubes. Calculating the difference between the 2 results in a positive or negative daily fluid balance.

2.6. Observed indicators

Before and on the seventh day after treatment, the clinical and laboratory indicators of the 4 groups were observed and collected, including oxygenation index, respiratory rate, heart rate, mean arterial pressure, APACHE II score, white blood cell, and changes in serum levels of alanine transaminase, triglyceride, creatine, procalcitonin, interleukin-6, and C-reactive protein. After 4 weeks of hospitalization, the MV time, ICU length of stay, gastric decompression, 28 day fatality rate, and 28 day safety rate of the 4 groups were statistically analyzed. Furthermore, the number of days with negative fluid balance within 1 week of admission for each patient in the 4 subgroups was recorded.

2.7. Statistical analysis

SPSS 20 was used for data analysis (SPSS Inc., Chicago). Quantitative data were expressed as mean ± standard deviation. The normality of the data was assessed using the Shapiro-Wilk test. Data with a non-normal distribution were analyzed using a non-parametric test (Mann–Whitney), and data with a normal

Table 2

Modified Marshall scoring system for organ dysfunction score.

Organ system	Score				
	0	1	2	3	4
Respiratory (PaO_2/FiO_2)	>400	301–400	201–300	101–200	≤101
Renal* (serum creatinine, μmol/L)	≤134	134–169	170–310	311–439	>439
Cardiovascular† (systolic blood pressure, mm Hg)	>90	<90, and fluid responsive	<90, and not fluid responsive	<90, and PH <7.3	<90, and PH <7.2
For non-ventilated patients, the FiO_2 can be estimated from below:					
Supplemental oxygen, L/min	FiO_2 (%)				
Room air	21				
2	25				
4	30				
6–8	40				
9–10	50				

A score of 2 or more in any system defines the presence of organ failure.

* A score for patients with pre-existing chronic renal failure depends on the extent of further deterioration of baseline renal function. No formal correction exists for a baseline serum creatinine ≥134 μmol/L.

† Off inotropic support.

Table 3

Changes of patients' indicators in A subgroups before and after treatment of 7 days.

Variable	Before			After treatment of 7 days		
	Group A1	Group A2	*P*	Group A1	Group A2	*P*
IAP (mm Hg)	25.20 ± 2.96	26.14 ± 2.54	.312†	15.00 ± 2.93	12.43 ± 3.75	.023*
OI (mm Hg)	169.8 ± 40.5	159.8 ± 38.2	.451†	213.0 ± 48.5	253.1 ± 53.7	.019*
RR (beats/min)	30.4 ± 3.2	32.1 ± 4.6	.279*	26.0 ± 1.9	24.0 ± 3.4	.045†
HR (beats/min)	129.3 ± 7.1	132.6 ± 10.0	.274†	108.7 ± 12.1	106.1 ± 13.1	.543†
MAP (mm Hg)	67.2 ± 9.3	68.8 ± 9.1	.607†	72.3 ± 6.3	77.4 ± 7.8	.044†
ALT (U/L)	72.27 ± 23.17	84.86 ± 28.49	.487†	64.67 ± 13.85	73.76 ± 20.12	.140†
TG (mmol/L)	17.59 ± 4.86	15.09 ± 4.25	.110†	10.22 ± 1.98	8.23 ± 2.37	.012†
CR (mmol/L)	225.3 ± 62.8	244.7 ± 72.0	.407†	141.6 ± 39.4	117.6 ± 29.0	.011*
WBC (10^9/L)	12.04 ± 3.54	13.53 ± 3.48	.215†	10.96 ± 2.21	9.28 ± 2.33	.037†
PCT (ng/mL)	2.15 ± 1.27	2.59 ± 1.64	.400†	1.28 ± 0.68	0.83 ± 0.56	.049*
IL-6 (ng/L)	117.97 ± 30.30	122.69 ± 37.96	.751*	66.08 ± 22.54	46.85 ± 26.20	.016*
CRP (mg/L)	240.7 ± 76.6	255.6 ± 97.7	.615†	144.3 ± 55.0	93.9 ± 50.4	.007†
APACHE II	14.27 ± 2.91	15.57 ± 2.73	.294*	9.80 ± 1.78	8.29 ± 1.55	.016*

ALT = alanine transaminase, APACHE II = acute physiology and chronic health evaluation score II, CR = creatine, CRP = C-reactive protein, HR = heart rate, IAP = intra-abdominal pressure, IL-6 = interleukin-6, MAP = mean arterial pressure, OI = oxygenation index, PCT = procalcitonin, RR = respiratory rate, TG = triglyceride, WBC = white blood cell.

* Mann–Whitney test.

† Independent samples *t* test.

distribution were analyzed using a parametric test (independent sample *t* test). Categorical variables were presented as absolute numbers and proportions and were tested using the chi-square test or Fisher exact test. Statistical significance was set at $P < .05$.

3. Results

In SAP patients with IAP ≥20 mm Hg (group A), there was no significant difference in various clinical and laboratory indicators between groups A1 and A2 before treatment. After 7 days of treatment, it was found that in group A2 with CVVH treatment, compared with group A1 without CVVH treatment, all indicators except heart rate and alanine aminotransferase were improved (Table 3). In terms of clinical outcome indicators, patients in group A2 had shorter ICU stays, gastric decompression and MV times, and lower 28-day operation rates than those in group A1, but there was no significant difference in 28-day mortality (Table 5). However, among SAP patients (group B) whose IAP was ≤20 mm Hg, there was no significant difference between groups B1 and B2 in all the observation indicators before and 7 days after treatment, including all clinical, laboratory, and clinical outcome indicators (Tables 4 and 5). In addition, groups A2 and B2 with CVVH treatment had more days of negative fluid balance within 1 week of admission than groups A1 and B1 without CVVH treatment, and the difference was statistically significant (Table 5).

4. Discussion

The incidence of IAH in patients with SAP is approximately 60%, while abdominal compartment syndrome (ACS) may occur in approximately 30% of patients. The mortality of patients with

Table 4

Changes of patients' indicators in B subgroups before and after treatment of 7 days.

Variable	Before			After treatment of 7 days		
	Group B1	Group B2	*P*	Group B1	Group B2	*P*
IAP (mm Hg)	14.20 ± 2.65	14.33 ± 2.57	.727*	9.40 ± 1.99	9.52 ± 1.89	.751*
OI (mm Hg)	240.0 ± 73.0	227.1 ± 59.7	.505*	268.3 ± 67.0	292.4 ± 51.0	.229†
RR (beats/min)	27.6 ± 2.6	29.2 ± 3.7	.158†	22.5 ± 2.9	23.7 ± 2.8	.214†
HR (beats/min)	123.3 ± 7.6	126.1 ± 8.4	.310†	103.9 ± 12.7	102.6 ± 12.6	.750†
MAP (mm Hg)	74.1 ± 9.2	72.1 ± 8.2	.477†	80.4 ± 6.6	78.3 ± 7.3	.366†
ALT (U/L)	78.94 ± 29.34	68.64 ± 26.05	.261†	59.06 ± 22.69	55.55 ± 18.81	.715*
TG (mmol/L)	13.65 ± 4.00	11.85 ± 3.02	.124†	8.09 ± 2.18	7.09 ± 2.02	.156†
CR (mmol/L)	183.8 ± 44.2	190.7 ± 61.6	.705†	136.6 ± 48.2	128.5 ± 38.0	.549*
WBC (10^9/L)	11.17 ± 2.16	12.04 ± 2.29	.246†	10.53 ± 1.88	9.87 ± 1.92	.300†
PCT (ng/mL)	1.03 ± 0.51	1.26 ± 0.79	.372*	0.94 ± 0.64	1.00 ± 0.64	.776†
IL-6 (ng/L)	104.77 ± 23.68	98.09 ± 31.82	.246*	60.27 ± 26.71	51.82 ± 22.86	.326*
CRP (mg/L)	136.3 ± 44.0	149.3 ± 43.6	.473*	104.3 ± 30.3	94.7 ± 17.2	.221†
APACHE II	11.94 ± 2.41	12.82 ± 2.40	.312*	5.88 ± 1.36	6.50 ± 1.60	.258*

ALT = alanine transaminase, APACHE II = acute physiology and chronic health evaluation score II, CR = creatine, CRP = C-reactive protein, HR = heart rate, IAP = intra-abdominal pressure, IL-6 = interleukin-6, MAP = mean arterial pressure, OI = oxygenation index, PCT = procalcitonin, RR = respiratory rate, TG = triglyceride, WBC = white blood cell.

* Mann–Whitney test.

† Independent samples *t* test.

Table 5

Comparison of clinical outcomes and negative fluid balance between the 4 groups.

Parameter	Group A1	Group A2	*P*	Group B1	Group B2	*P*
ICU length of stay (d)	15.2 ± 4.9	11.8 ± 3.3	.012*	11.7 ± 3.6	10.1 ± 3.1	.129*
Duration of MV (h)	280.1 ± 94.7	211.8 ± 93.7	.007*	146.8 ± 95.2	160.8 ± 116.9	.988*
28-d surgery rate [n (%)]	60.0%	23.8%	.028†	25.0%	36.4%	.457†
28-d fatality rate [n (%)]	26.7%	23.8%	.854†	6.2%	9.1%	.748†
Negative fluid balance (d)‡	2.4 ± 0.8	3.1 ± 0.9	.04*	1.8 ± 0.8	2.5 ± 0.9	.02*
Gastric decompression (d)	17.33 ± 4.70	13.52 ± 2.00	.004*	14.27 ± 3.61	12.82 ± 2.66	.166*

ICU = intensive care unit, MV = mechanical ventilation.

* Mann–Whitney test.

† Chi square test.

‡ Negative fluid balance days within 1 week after admission.

SAP who develop ACS is high at 50% to 75 %.[15–18] IAH, a sustained or repeated pathological elevation in IAP of 12 mm Hg or higher,[19] is categorized as grade I (IAP 12–15 mm Hg), grade II (16–20 mm Hg), grade III (21–25 mm Hg), and grade IV (>25 mm Hg). ACS, a sustained IAP >20 mm Hg that is associated with new organ dysfunction/failure, as defined by the World Society of the Abdominal Compartment Syndrome.[14]

IAH in SAP with organ failure is generally thought to reflect visceral edema due to the severity of the inflammatory process, potentially compounded by aggressive fluid resuscitation (volume overload). Pancreatic inflammation initiates a cascade of acute peripancreatic fluid collections, capillary leakage syndrome, and paralytic ileus, leading to elevated IAP.[20,21] Inflammatory cells are activated and release a large number of cytokines, and the resulting cascade reaction at the level of inflammatory cytokines is an important cause of SAP deterioration.[1] In SAP, several inflammatory mediators contribute to increased capillary permeability in various organs, which together with aggressive fluid resuscitation may result in visceral edema and the development of IAH, ultimately leading to the development of ACS.[22,23] One study showed a significant relationship between graded IAH and the severity of AP.[19] IAP should be measured in patients with AP, as it is associated with organ failure and mortality and can predict the severity of the disease.[24–28] Evidence is still scarce whether IAP measurements should be routine in all patients with AP or can some selectivity be maintained and how can the patient at risk for developing IAH and ACS be identified at the earliest. Routine transvesical pressure measurements in all patients with AP may not be necessary, but for patients with manifest organ failure or persistent systemic inflammatory response syndrome or APACHE II score ≥8 should be offered IAP surveillance.[29]

Many studies suggest that early administration of CVVH in SAP can effectively reduce IAP and pathogenic cytokines in patients with ACS and improve prognosis.[30,31] The application of CVVH helps to remove inflammatory cytokines from the blood and the extracavascular compartment, maintains negative fluid balance in the body, reduces fluid accumulation in the interstitial tissues, effectively reduces IAP, accelerates the recovery of liver and kidney functions, shortens hospital stay, avoids multiorgan failure, and reduces mortality.[30,32,33] For patients with acute kidney injury (AKI), starting CVVH treatment as early as possible can deal with the state of body fluids and electrolytes more effectively, correct acid-base imbalances more quickly, remove uremic toxins properly, and prevent subsequent complications caused by AKI.[34]

However, it remains unclear in many studies whether CVVH has an impact on reducing mortality or complications in patients with SAP.[9,35] The disadvantage of early initiation of renal replacement therapy is that it may expose patients with AKI to unnecessary complications related to renal replacement therapy, including hemodynamic instability, coagulopathy, bloodstream infections, and even inflammation or oxidative stress caused by biocompatible reactions with dialyzer membranes.[36]

Our study confirms the findings of many previous observers that AP is a risk factor for developing IAH and ACS.[37,38] There has been scepticism in past if IAP monitoring helped survival but recent evidence shows improvement in survival with evolving management of IAH and ACS.[39] For SAP patients with IAH, which patients will benefit from CVVH and which patients will not? What is the right time to implement CVVH? We conducted small-scale studies and obtained meaningful clinical results. We found that CVVH did not improve the prognosis of SAP patients with IAP ≤20 mm Hg. However, for SAP patients with IAP ≥20 mm Hg, CVVH showed improvement in a number of clinical indicators, with a statistical difference compared with the control group. CVVH was effective in a subset of patients with SAP and IAH. Therefore, monitoring IAP may be an indicator of whether or when to initiate CVVH. It was also found that patients receiving CVVH treatment had more negative fluid balance days within 1 week of admission, suggesting that negative fluid balance caused by CVVH treatment may be one of the reasons for the benefit of this group of patients.

Author contributions

Conceptualization: Yongle Xie
Data curation: Wentao Su, Nan Qing, Hongwei Xin, Jing Tian, Yun Li, Junnian Zhu.
Formal analysis: Xiaoying Wang
Investigation: Yuan Yuan.
Supervision: Yuan Yuan.
Writing – original draft: Yongle Xie.
Writing – review & editing: Yongle Xie, Yuan Yuan.

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