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Anticoagulation therapy for intra-abdominal hypertension in patients with acute pancreatitis

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Abstract

Background Intra-abdominal hypertension (IAH) is a common complication in patients with acute pancreatitis (AP) and can lead to multiple organ failure. The efficacy of anticoagulant therapy for IAH in patients with acute pancreatitis is unclear. The objective of this study aimed to investigate the effects of anticoagulant therapy on IAH in patients with acute pancreatitis.

Materials and methods A total of 49 AP patients with IAH were included in this retrospective study, with 34 patients in the anticoagulant group and 15 patients in the non-anticoagulant group. The effects of anticoagulant therapy on intra-abdominal pressure, inflammation markers, disease severity, and imaging indices were compared between the two groups.

Results The anticoagulant group had significantly lower intra-abdominal pressure at 60 h of therapy compared to the non-anticoagulant group. In addition, inflammation markers and modified Marshall score were significantly improved in the anticoagulant group after 7 days of therapy. There was no significant difference in imaging indices, length of stay and mortality in hospital between the two groups. No adverse bleeding events or allergic reactions occurred during the treatment period. No significant difference was found in the rate of splanchnic vein thrombosis and portal hypertension between the two groups six months after the onset of the disease.

Conclusion Anticoagulant therapy may be a potential treatment option for IAH in patients with acute pancreatitis.

Keywords Acute pancreatitis, Intra-abdominal hypertension, Anticoagulant therapy, Prognosis

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Background

Intra-abdominal hypertension (IAH), can occur in patients with acute pancreatitis and is a known risk factor for the development of multiple organ failure and increased mortality [1]. Elevated intra-abdominal pressure (IAP) can result in decreased blood flow to the organs, leading to ischemia and impaired organ function [2]. Inflammation, coagulation, and endothelial dysfunction interact and promote disease progression [3]. Obstruction of pancreatic microcirculation and the formation of microthrombus are also thought to play a role.

The use of anticoagulants in the treatment of acute pancreatitis has long been controversial. Anticoagulation therapy, including the use of unfractionated heparin (UFH) and low molecular weight heparin (LMWH), is commonly used to prevent blood clots in patients with various medical conditions, including acute pancreatitis [4]. Currently, the decision to use anticoagulants in patients with acute pancreatitis with abdominal hypertension is made on a case-by-case basis, taking into account the patient's individual history, severity of the condition and other factors. Although the use of heparin is gaining acceptance in patients with hypertriglyceridemia acute pancreatitis [5, 6], more evidence is still needed for anticoagulant therapy in the overall pancreatitis population. And so far, there is no study on the relationship between anticoagulant therapy and IAP in acute pancreatitis.

The objective of this study was to determine the role of anticoagulant therapy in acute pancreatitis with IAH.

Methods

Study subjects

This is a retrospective study and the enrolled patients from January 1, 2020 to December 1, 2021 were divided into two groups. The non-anticoagulant group was given conventional treatment, and the anticoagulant group was given UFH or LMWH in addition to conventional treatment. The efficacy, adverse reactions and complication rate of the two groups were compared.

Inclusion and exclusion criteria

Inclusion criteria:

(a) Adult patients aged 18–70 years diagnosed with acute pancreatitis meeting the 2012 Atlanta diagnostic criteria [7].
(b) Admission to emergency intensive care unit (EICU) within 72 h of symptom onset.
(c) Development of IAH within the first 72 h of admission.

Exclusion criteria:

(a) Allergic to UFH or LMWH.
(b) Active bleeding.
(c) History of intracranial hemorrhage.
(d) History of heparin-induced thrombocytopenia.
(e) Severe chronic dysfunction of the heart, lungs, kidneys, or liver.
(f) Hematologic diseases or autoimmune diseases.
(g) History of malignant tumor.
(h) Ongoing anticoagulant or antiplatelet therapy for underlying conditions.
(i) Pregnancy or lactation.
(j) Incomplete medical records or loss to follow-up.

Conventional treatment

Conventional treatment was administered in accordance with expert consensus and guidelines for the management of acute pancreatitis [8, 9].

Fluid resuscitation

A goal-directed strategy was employed, with an initial crystalloid infusion of 5–10 mL/kg, guided by dynamic parameters (heart rate, mean arterial pressure, urine output, hematocrit) to avoid fluid overload.

Etiological treatment

For obstructive acute pancreatitis, emergency endoscopic retrograde cholangio-pancreatography was performed within 48 h of symptom onset. Percutaneous transhepatic gallbladder drainage served as a rescue therapy if endoscopic treatment failed or was unavailable. For alcoholic acute pancreatitis, alcohol intake was strictly prohibited. For hyper-triglyceridemic acute pancreatitis, fibrates were administered for lipid control.

Maintenance of organ functions

Respiratory support ranged from nasal cannula oxygen to mechanical ventilation, guided by arterial blood gas analysis. Renal function was supported with intermittent renal replacement therapy (IRRT) when indicated for renal failure.

Management of intra-abdominal hypertension

A stepwise approach was implemented. Interventions included nasogastric/rectal decompression, negative fluid balance, and sedation/analgesia. For cases progressing to abdominal compartment syndrome, hemofiltration and percutaneous catheter drainage were performed.

Application of pancreatic enzyme inhibitors

Ulinastatin was administered at a dosage of 900,000 units per day.

Enteral nutrition support

Initiated within 3–5 days using a nasojejunal tube with a short-peptide formula, starting at 30 mL/h and gradually advanced as tolerated to a target of 25–30 kcal/kg/day.

Antibiotic strategy

The initial treatment plan was formulated based on the clinical judgment of the attending physician, and was subsequently adjusted according to culture results.

Traditional chinese medicine

Used as an adjunctive therapy. External application of mirabilite over the abdomen and administration of Dachengqi Decoction were employed to relieve bowel dysfunction.

Pain management

Achieved through the use of opioids, such as Fentanyl or Pethidine.

Intra-abdominal pressure measurement

Abdominal pressure was measured every 12 h for at least three days. The measurement of IAP was performed using a bladder pressure measurement [10]. The patient was placed in the supine position, the catheter inserted in a sterile procedure was connected to the pressure tube through the tee. Exhaust the air so that the pressure measuring tube is connected with the atmosphere, and adjust the tee so that the urine tube is connected with the pressure measuring tube. A maximum of 25 ml sterile saline was injected into the bladder through the catheter. When the liquid level of the pressure measuring tube fluctuates slightly and does not fall any more, the scale number on the concave surface of the liquid level in the patient's end expiratory pressure measuring tube is the IAP.

Abdominal hypertension is defined as an IAP greater than 12 mmHg. Grading: Grade I: IAP between 12 and 15 mmHg; Grade II: IAP between 16 and 20 mmHg; Grade III: IAP between 21 and 25 mmHg; Grade IV: IAP greater than 25 mmHg.

Anticoagulant therapy

In the anticoagulant treatment group, LMWH was used at 1 mg/kg/day for more than 7 days after IAH was found. The use of UFH was as follows (Fig. 1): first, administer the corresponding supplementation based on whether the patient's fibrinogen level is less than 1.3 g/L. If platelet $\leq 30 \times 10^9$/L, the initial dose of UFH is 3 U/kg/h; if platelet $> 30 \times 10^9$/L, intravenous infusion was continued at a rate of 5 U/kg/h. After 6 h, the dose of UFH was adjusted according to the changing trend of fibrinogen degradation product (FDP) and D-dimer. If the FDP and D-dimer began to decrease, it indicated that the heparin dose had reached the therapeutic level and no dose adjustment was required. If there was no decrease or even increase, the UFH dose was increased by 1–2 U/kg/h and titrated repeatedly according to the above method until FDP and D-dimer began to decrease. Anticoagulant therapy was discontinued when any contraindications (including allergy, active bleeding, cerebral hemorrhage, heparin-induced thrombocytopenia) were observed or if intra-abdominal pressure normalizes after one week of UFH administration. The anticoagulation standard established during the treatment was for acute pancreatitis complicated with IAH, and it was applicable in cases where there are no absolute contraindications for anticoagulation therapy. The ultimate decision on whether to use anticoagulation therapy primarily relies on the clinical experience of the attending physician.

Clinical variables

All data were extracted from Ruijin Hospital electronic database. Baseline demographic information, daily trends in abdominal pressure and stool volume during anticoagulant therapy were collected. Inflammatory indicators, biochemical indicators, coagulation indicators, disease severity 7 days after the start of anticoagulant therapy, side effects of anticoagulant therapy, and in-hospital mortality were included. The changes of modified computed tomography severity index (MCTSI) [11] score and splanchnic vein thrombosis were analyzed by contrast-enhanced computed tomography (CECT) before and after anticoagulant therapy. The presence of splanchnic vein thrombosis and portal hypertension was assessed by CECT six months after onset.

Statistical analysis

The clinical data were analyzed using SPSS 26.0 statistical software (SPSS, Inc., Chicago, IL). The enumeration data were presented as means($\bar{x}$) ± standard deviation (SD) and compared using t-test or Mann-Whitney U test. Categorical data were expressed in frequency and percentage terms and compared using chi-square tests or Fisher's exact test. P-value < 0.05 was considered statistically significant.

Results

Characteristics of the study population

Among the 127 AP patients aged 18–70 admitted to the EICU within 3 days of onset, 71 patients presented with IAH within 3 days of onset. 22 patients with IAH were excluded due to the exclusion criteria. Finally, 49 patients were included in the analysis. There were 34 patients in the anticoagulant group and 15 patients in the non-anticoagulant group. The incidence of IAH during this period was 55.91% (71/127). The baseline characteristics of the two groups were shown in Table 1, and there were no significant differences in age, gender, etiology,

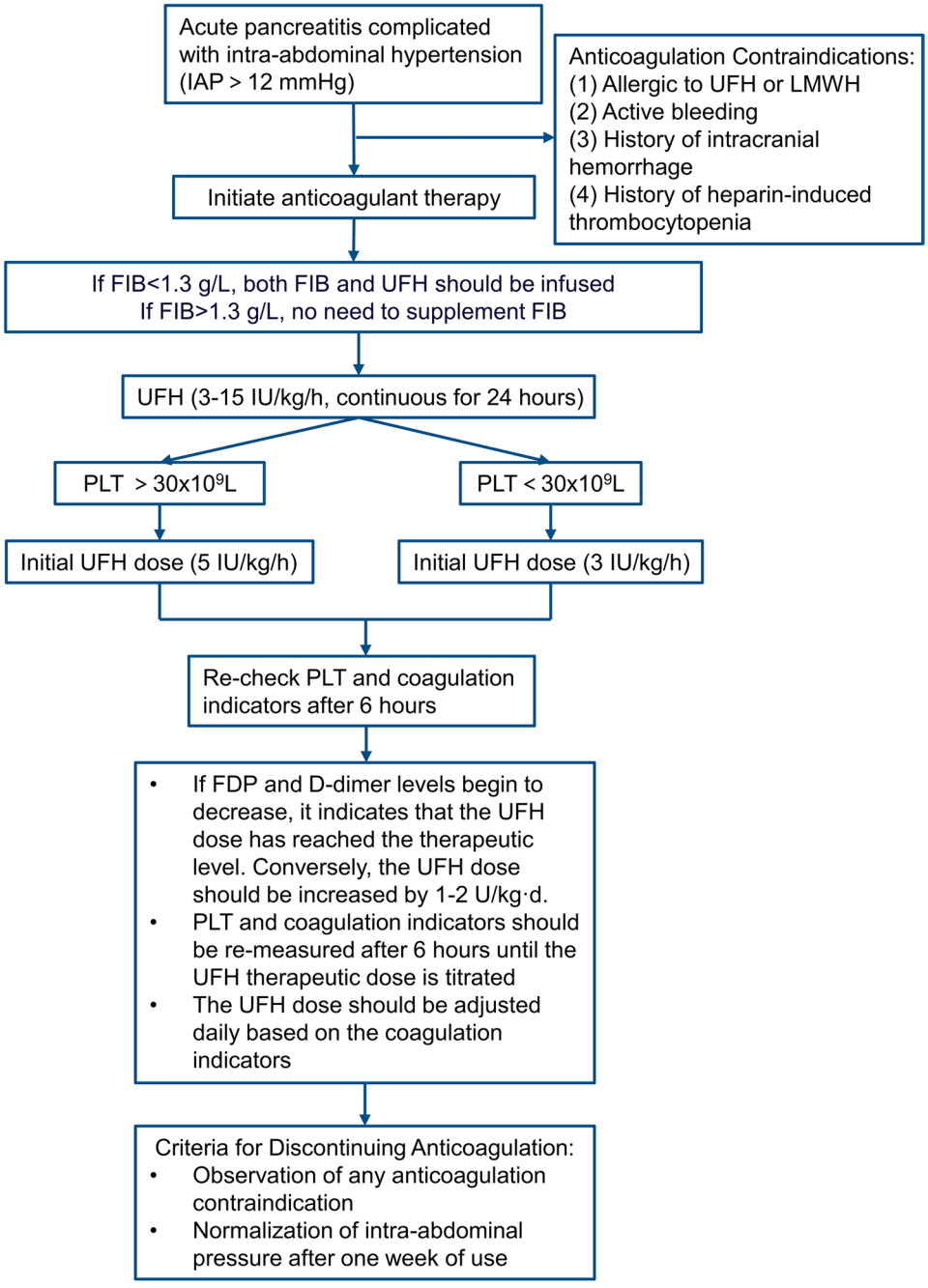

Fig. 1 Unfractionated heparin anticoagulation procedure for acute pancreatitis complicated with intra-abdominal hypertension. Note: UFH: Unfractionated heparin; LMWH: low molecular weight heparin; FIB: Fibrinogen; PLT: Platelet; FDP: Fibrin degradation products

comorbidities, inflammatory markers, biochemical markers, coagulation index, or disease severity.

Trends of abdominal pressure and stool volume between anticoagulant group and non-anticoagulant group

IAP in the anticoagulant group was significantly different at 60 h of anticoagulant therapy compared to the non-anticoagulant group (Fig. 2A). There was no significant difference in stool volume during the 7 days observed (Fig. 2B).

Changes in clinical indicators after treatment between anticoagulant group and non-anticoagulant group

After 7 days of treatment, inflammation markers White blood cell (WBC) and C-reactive protein (CRP) were significantly improved in the anticoagulant group compared to the non-anticoagulant group ($P < 0.05$) (Table 2). In the

Table 1 Comparison of baseline demographic and clinical characteristics between anticoagulant group and non-anticoagulant group on admission (n%; $\bar{x}\pm s$)

	Anticoagulant group (*n*=34)	Non-anticoagulant group (*n*=15)	*P* value
Mean age (years)	43.62 ± 15.25	45.67 ± 13.38	0.655
Gender (male%)	23 (67.65%)	8 (53.33%)	0.338
Body Mass Index (kg/m^2)	27.20 ± 3.62	27.13 ± 3.13	0.951
Etiology			0.246
Biliary	8 (23.53%)	7 (46.67%)	
Hypertriglyceridemia	21 (61.76%)	8 (53.33%)	
Alcohol	4 (11.76%)	0 (0.00%)	
Others	1 (2.94%)	0 (0.00%)	
Comorbidities			
Hypertension (%)	11 (32.35%)	4 (26.67%)	0.691
Diabetes mellitus (%)	12 (35.29%)	3 (20.00%)	0.284
Indicators			
White blood cell (×10^9/l)	12.86 ± 6.15	13.69 ± 4.88	0.646
C-reactive protein (μg/ml)	235.32 ± 103.47	227.47 ± 103.76	0.808
Procalcitonin (ng/ml)	5.61 ± 7.83	16.31 ± 23.61	0.107
Serum amylase (U/L)	1111.12 ± 1052.42	1238.53.43 ± 845.54	0.681
Alanine aminotransferase (U/l)	60.03 ± 153.59	54.80 ± 70.35	0.901
Total bilirubin (μmol/l)	22.43 ± 13.16	21.49 ± 12.61	0.817
Blood urea nitrogen (mmol/l)	7.21 ± 5.74	9.71 ± 4.82	0.148
Serum creatinine (μmol/l)	101.44 ± 110.09	159.60 ± 126.41	0.110
Lactic acid (mmol/l)	2.85 ± 1.78	3.51 ± 1.37	0.210
Platelet (×10^9/l)	188.00 ± 67.99	219.27 ± 83.50	0.171
Activated partial thromboplastin time (s)	32.29 ± 8.20	37.35 ± 20.35	0.218
Prothrombin time (s)	14.18 ± 1.82	15.38 ± 2.63	0.071
Fibrinogen (g/L)	5.20 ± 1.77	4.59 ± 1.70	0.266
Fibrinogen degradation products (mg/L)	10.86 ± 7.06	11.61 ± 5.23	0.711
D-Dimer (mg/L)	3.29 ± 2.39	3.93 ± 2.67	0.414
Modified Marshall score	2.09 ± 2.08	2.60 ± 2.06	0.430
BISAP score	2.18 ± 1.40	2.00 ± 0.93	0.606
APACHE Ⅱ score	10.62 ± 6.23	11.20 ± 5.03	0.751

BISAP score Bedside Index for Severity in Acute Pancreatitis score, *APACHEII score* Acute Physiology and Chronic Health Evaluation Ⅱ score

*$P < 0.05$ was considered statistically significant

disease severity, the modified Marshall score decreased significantly in the anticoagulant group ($P < 0.05$). However, there were no significant differences in PCT, Bedside Index for Severity in Acute Pancreatitis (BISAP) score, Acute Physiology and Chronic Health Evaluation Ⅱ (APACHE Ⅱ) score, biochemical index and coagulation index between the two groups.

Comparison of imaging indexes before and after treatment between anticoagulant group and non-anticoagulant group

There was no significant difference in the improvement of the MCTSI scores between the anticoagulation group and the non-anticoagulation group before and after treatment. However, there was a significant increase in the post-treatment MCTSI scores in both groups. When comparing the difference in MCTSI scores before and after treatment, there was no significant difference in the magnitude of the increase between the two groups (Table 3).

Effect of anticoagulant therapy on prognosis

There was no significant difference between anticoagulant group and non-anticoagulant group in length of stay and mortality in hospital. The incidence of Splanchnic vein thrombosis during treatment was similar. According to the enhanced CT imaging examination, there was no significant difference in the rate of splanchnic vein thrombosis and portal hypertension between the two groups six months after the onset of the disease (Table 4).

Side effects of anticoagulant therapy

No adverse bleeding events or allergic reactions occurred during the treatment period. In one patient, the platelet count dropped below 100*10^9/L after treatment.

Discussion

IAH and abdominal compartment syndrome (ACS) are common complications in patients with acute pancreatitis and are associated with increased mortality [12]. IAH and ACS may be caused by severe intestinal barrier dysfunction and high endotoxin levels in patients with acute pancreatitis, and IAH may be caused by a proinflammatory response resulting in effusion, ascites, and retroperitoneal edema, as well as by excessive rehydration [13]. Although there have been case reports that acute pancreatitis can induce both splanchnic vein thrombosis and IAH [14], the association between them has not been confirmed. And most of the time, microthrombsis is not detectable on enhanced computed tomography. Early detection and prevention of IAH can improve the prognosis of acute pancreatitis [15]. Although anticoagulant therapy to reduce intra-abdominal pressure is not recommended in current guidelines and management [2, 16], it has been shown to have a beneficial effect on improving the prognosis of patients with acute pancreatitis [17, 18]. This effect is thought to occur through a variety of mechanisms, including anticoagulation, anti-inflammation and endothelial protection [4].

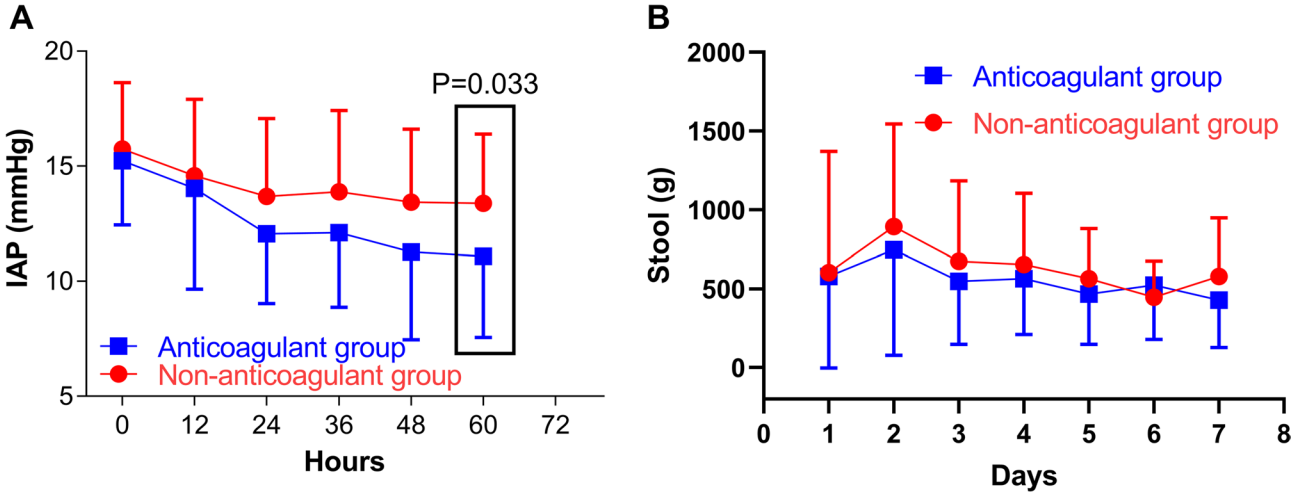

Fig. 2 The changing trends of intra-abdominal pressure (IAP) (Fig. 2**a**) and stool volume (Fig. 2**b**) between anticoagulant group and non-anticoagulant group. Data visualization is expressed in terms of mean and error. $P < 0.05$ was considered statistically significant

Table 2 Changes in clinical indicators after treatment between anticoagulant group and non-anticoagulant group (n%; $\bar{x}\pm s$)

Clinical indicators	Anticoagulant group (*n*=34)	Non-anticoagulant group (*n*=15)	*P* value
Inflammatory markers			
White blood cell ($\times10^9$/l)	14.66 ± 5.30	18.46 ± 6.56	0.037*
C-reactive protein (μg/ml)	138.47 ± 77.70	208.87 ± 76.11	0.005*
Procalcitonin (ng/ml)	1.78 ± 3.78	4.84 ± 8.48	0.199
Biochemical markers			
Alanine aminotransferase (U/l)	28.79 ± 28.06	30.33 ± 38.83	0.876
Total bilirubin (μmol/l)	26.79 ± 28.31	31.31 ± 29.73	0.614
Blood urea nitrogen (mmol/l)	8.72 ± 7.78	11.10 ± 9.07	0.352
Serum creatinine (μmol/l)	109.88 ± 138.98	208.27 ± 210.58	0.113
Lactic acid (mmol/l)	2.42 ± 0.80	2.14 ± 0.80	0.267
Coagulation index			
Platelet ($\times10^9$/l)	216.18 ± 61.75	226.27 ± 65.15	0.607
Activated partial thromboplastin time (s)	30.31 ± 5.61	35.29 ± 17.16	0.131
Prothrombin time (s)	14.06 ± 1.50	14.45 ± 1.53	0.407
Fibrinogen (g/L)	4.44 ± 1.02	4.79 ± 1.20	0.301
Fibrinogen degradation products (mg/L)	26.37 ± 13.52	35.60 ± 20.19	0.066
D-Dimer (mg/L)	8.68 ± 5.22	11.83 ± 9.19	0.134
Disease severity score			
Modified Marshall score	1.18 ± 2.11	2.73 ± 2.76	0.036*
BISAP score	2.03 ± 1.45	2.45 ± 1.30	0.320
APACHE Ⅱ score	7.06 ± 6.36	9.53 ± 7.91	0.250

BISAP score Bedside Index for Severity in Acute Pancreatitis score, *APACHE II score* Acute Physiology and Chronic Health Evaluation Ⅱ score

*$P < 0.05$ was considered statistically significant

Table 3 Comparison of imaging indexes before and after treatment between anticoagulant group and non-anticoagulant group

		Anticoagulant group (*n*=34)	Non-anticoagulant group (*n*=15)	Z value	*P* value
MCTSI score	Before treatment	6.24 ± 1.69	5.73 ± 2.12	−0.718	0.473
	After treatment	7.29 ± 1.55	7.20 ± 1.47	−0.157	0.875
	Z value	−2.757	−2.138		
	P value	0.006*	0.033*		
ΔMCTSI		1.06 ± 1.41	1.47 ± 2.07	−0.566	0.571

MCTSI score Modified computed tomography severity index score

*$P < 0.05$ was considered statistically significant

Table 4 Effect of anticoagulant therapy on prognosis (n%; $\bar{x}\pm s$)

	Anticoagulant group	Non-anticoagulant group	*P* value
	(*n*=34)	(*n*=15)	
Length of hospital stay (days)	41.47 ± 38.31	48.13 ± 26.50	0.084
In-hospital mortality	4 (11.76%)	2 (13.33%)	0.605
Splanchnic vein thrombosis			
During hospitalization	(*n*=33)	(*n*=15)	
	7 (21.21%)	3 (20.00%)	0.623
Six months after onset	(*n*=30)	(*n*=13)	
	4 (13.33%)	0 (0.00%)	0.222
Portal hypertension			
Six months after onset	(*n*=30)	(*n*=13)	
	1 (3.33%)	2 (15.38)	0.213

UFH and LMWH are anticoagulants that help prevent blood clots from forming. In patients with acute pancreatitis, microcirculatory disturbances and microthrombosis may worsen the course of the disease [17]. Similarly, the occurrence of splanchnic vein thrombosis is associated with disease severity and mortality [19]. Blood clots that form in the veins and small blood vessels around the pancreas may cause an increase in abdominal pressure. Anticoagulation therapy helps prevent these clots and keep microcirculation open [20]. The recovery of microcirculation promotes the peristalsis of the intestine and increases the volume of defecation. In this study, anticoagulant therapy was found to significantly reduce IAH in patients with acute pancreatitis compared to controls. But there was no significant increase in stool volume, probably because other combined measures to unclog the bowel were strong enough.

There are no standard anticoagulant recommendations for acute pancreatitis complicated with intra-abdominal hypertension. The initial dose chosen was based on our center's experience in anticoagulation for sepsis (Figure S1), although this has not been internationally recognized as well. Figure 1 provides a detailed description of our center's anticoagulation protocol for acute pancreatitis complicated with IAH using UFH.

UFH has been shown to have anti-inflammatory properties that help reduce swelling and inflammation in the pancreas. Ceranowicz et al. [21]. showed that heparin inhibits the development of acute pancreatitis and reduces plasma pro-inflammatory interleukin-1β and plasma D-dimer levels. In another animal study, heparin demonstrated an immunomedulatory function by reducing pancreatic necrosis, macrophage infiltration, and serum inflammatory cytokines (interleukin-6 and tumor necrosis factor-α) concentrations by targeting high-mobility group protein-B1 (HMGB-1) -related pathways [22]. Heparin can also reduce the infiltration of M1-macrophages and the secretion of inflammatory factors by inhibiting heparin-binding protein which is significantly increased in acute pancreatitis [23, 24]. In addition, inhibition of neutrophil activation improved pancreatic microcirculation [25]. In rat models, LMWH also showed anti-inflammatory and oxidative effects [26]. These results are consistent with our study. The decrease of these inflammatory markers including WBC and CRP might help to reduce capillary leakage in the abdominal cavity, thereby improving abdominal pressure.

In our research, following the use of anticoagulant therapy, we observed a decrease in IAH along with a decline in the modified Marshall Score. We hypothesize that there is a certain correlation between these two phenomena. Anticoagulant therapy, through its significant effects of improving microcirculation in various organs and reducing inflammation, directly contributes to the effective improvement of respiratory, circulatory, and renal functions. Meanwhile, the reduction in IAH further alleviates the compression on the diaphragm, helps to alleviate the hypoperfusion state of tissues and organs, and significantly increases renal blood flow. These combined effects result in a statistically significant difference in the modified Marshall Score between the two groups. However, the precise mechanism of this relationship still requires further clinical and experimental research for clarification.

The disorder of coagulation system plays an important role in the pathogenesis of severe acute pancreatitis (SAP). In addition to heparin, some anticoagulant drugs have entered clinical application and the prognosis of patients has been improved. In an animal study, Dawid et al. [27]. found that preconditioning with low doses of warfarin increased the international normalized ratio, reduced pancreatic injury and reduced pancreatic edema. These effects were accompanied by improvements in pancreatic blood flow and reductions in interleukin-1β levels and plasma D-dimer levels. In animal models, low dose of acenocoumarol contributed to ischemia/reperfusion recovery in rats with acute pancreatitis [28]. These results suggest that anticoagulation can promote recovery from acute pancreatitis. In our study, there was no statistically significant difference in coagulation indexes including D-dimer between the two groups after anticoagulation therapy, which might be related to the relatively short duration of anticoagulation.

Moreover, LMWH has been found to be effective in reducing abdominal fluid accumulation, which can lead to increased IAH in patients with acute pancreatitis. The study showed that LMWH reduced the Computed Tomography Severity Index and decreased the amount of fluid accumulated in the peritoneal cavity, leading to a decrease in IAH [29]. In our study, no matter in the anticoagulant group or the non-anticoagulant group, intra-abdominal fluid accumulation continued to increase after treatment. This was mainly because the treatment period was in the progressive stage of the disease, so the MCTSI score increased.

The use of therapeutic anticoagulation in the early stages of moderately severe and SAP may be beneficial in reducing the risk of disease progression and pancreatic necrosis [29]. Qiu et al. [30]. found that LMWH treatment was associated with a reduced white blood cell count, C-reactive protein levels and computed tomography severity index in patients with SAP. The study followed 1077 and 1115 patients with SAP and found that those who received LMWH treatment had a significantly lower incidences of operation rate and mortality compared to conventional treatment group. Another meta-analysis also showed that LMWH reduced complications and length of hospital stay in SAP patients [31]. However, several clinical trials have shown that LMWH does not improve mortality in this patient population. Noor

et al. [32]. found that therapeutic anticoagulation could not benefit patients with splanchnic vein thrombosis in acute pancreatitis. In this meta-analysis, most studies used LMWH first, followed by sequential use of warfarin. Results showed an absolute risk difference of 2% for mortality (95% CI = −8-12%). Another study also found that LMWH did not reduce the risk of death in patients with AP [18]. This randomized controlled trial enrolled 100 patients with MSAP, and compared the outcomes of those who received LMWH plus standard care with those who received only standard care. The results showed that there was no significant difference in mortality between the two groups, and that LMWH did not provide any benefit in terms of improving patient outcomes. Our study also found no benefit in clinical outcomes and medium to long-term complications of splanchnic vein thrombosis and portal hypertension. The possible reason is that the clinical outcome of pancreatitis is caused by a variety of confounding factors, and short-term anticoagulant therapy could not play a decisive role.

Although anticoagulant therapy is used for the treatment of acute pancreatitis with splanchnic vein thrombosis, deep vein thrombosis, and other thromboembolic events and has been shown to have some benefits [33]. However, like all medical treatments, low molecular weight heparin is not without potential complications. The main concern comes from complications including bleeding, allergic reactions and thrombocytopenia. Existing studies have shown that the use of anticoagulants is safe, both prophylactic and therapeutic, and even after pancreatic surgery [18, 27, 34–36]. But none of these anticoagulants were used when IAP was elevated. In our study, no patients experienced adverse bleeding events during this treatment period. No statistical difference was found in serum creatinine levels and no allergic reaction occurred in either group. The only one patient in the anticoagulant group whose platelet loss was reanalyzed by specialists was more likely to have been caused by infection. Therefore, anticoagulant therapy is safe in the treatment of acute pancreatitis with IAH. But it is still important to closely monitor patients who receive anticoagulants for any adverse effects or complications.

Limitation

The retrospective study has certain bias. The number of cases in this study is small, which made further subgroup analysis difficult. This may limit the statistical power and broader applicability of our conclusions. Because the monitoring of IAP was stopped after the disease improved, the numerical changes of abdominal pressure could not be observed continuously for 7 days. Therefore, further prospective studies are needed to verify the efficacy of anticoagulant therapy for IAH in patients with acute pancreatitis.

Conclusion

IAH can have serious consequences in patients with acute pancreatitis, including reduced organ blood flow, impaired organ function, and increased risk of multiple organ failure. Therefore, it is important to monitor patients for signs of IAH and take appropriate steps to manage this complication when it occurs. Anticoagulant therapy might be a potential tool to benefit acute pancreatitis patients with IAH by reducing abdominal pressure.

Abbreviations

IAH	Intra-abdominal hypertension
IAP	Intra-abdominal pressure
AP	Acute pancreatitis
UFH	Unfractionated heparin
LMWH	Low molecular weight heparin
EICU	Emergency intensive care unit
FDP	Fibrinogen degradation product
MCTSI	Modified computed tomography severity index
CECT	Contrast-enhanced computed tomography
SD	Standard deviation
WBC	White blood cell
CRP	C-reactive protein
BISAP score	Bedside Index for Severity in Acute Pancreatitis score
APACHE Ⅱ score	Acute Physiology and Chronic Health Evaluation Ⅱ score
MCTSI score	Modified computed tomography severity index score
ACS	Abdominal compartment syndrome
SAP	Severe acute pancreatitis
HMGB-1	High-mobility group protein-B1

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12876-025-04404-x.

Supplementary Material 1.

Acknowledgements

Not applicable.

Authors' contributions

TTN, EQM and WSJ contributed to conception and design of the study. TTN and YG organized the database. TTN, WWS and BZ analyzed and interpreted the data. TTN, YG and LM wrote the first draft of the manuscript. YC, ZTY, EZC, and EQM revised the manuscript. All authors read and approved the submitted version.

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Data availability

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

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics committee of Ruijin Hospital affiliated to Shanghai Jiao Tong University School of Medicine and granted waiver of informed consent (2023235). The study strictly adheres to the ethical standards outlined in the Declaration of Helsinki and its later amendments.

Consent for publication
Not applicable in our study.

Competing interests
The authors declare no competing interests.

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