

Effect of Early Fluid Resuscitation on Recovery of Coagulation Function in Acute Traumatic Coagulopathy

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Abstract: *Objective:* To investigate the actual impact of early fluid resuscitation on the recovery of coagulation function in patients with acute traumatic coagulopathy (ATC). *Methods:* Fifty patients with acute traumatic coagulopathy admitted to our hospital from June 1, 2025 to June 30, 2026 were selected as study subjects. All patients were divided into two groups using the random number table method, with 25 cases in each group. The control group received conventional fluid resuscitation, while the observation group received early restrictive fluid resuscitation. Coagulation function indicators, blood gas analysis indicators, inflammatory factor indicators, and prognosis-related indicators were compared between the two groups. *Results:* At 24 h after intervention, the observation group had PT (13.25 ± 1.12) s, APTT (32.46 ± 2.31) s, FIB (2.87 ± 0.34) g/L, and D (1.82 ± 0.41) mg/L, while the control group had PT (15.78 ± 1.46) s, APTT (38.72 ± 2.85) s, FIB (2.15 ± 0.28) g/L, and D (3.24 ± 0.57) mg/L. The differences in coagulation function indicators between the two groups were statistically significant ($p < 0.05$). Among blood gas analysis indicators, the observation group had pH (7.38 ± 0.03), PaO₂ (87.64 ± 5.21) mmHg, and lactate (2.13 ± 0.36) mmol/L, which were superior to the control group's pH (7.32 ± 0.04), PaO₂ (78.53 ± 4.89) mmHg, and lactate (3.47 ± 0.52) mmol/L, with statistically significant differences ($p < 0.05$); there was no statistically significant difference in PaCO₂ between the two groups ($p > 0.05$). Among inflammatory factor indicators, the observation group had TNF- α (18.52 ± 3.14) ng/L and IL-6 (22.47 ± 3.56) ng/L, which were lower than the control group's TNF- α (32.65 ± 4.72) ng/L and IL-6 (38.79 ± 4.21) ng/L, with statistically significant differences ($p < 0.05$); there was no statistically significant difference in IL-1 β between the two groups ($p > 0.05$). Among prognosis-related indicators, there were no statistically significant differences in total hospitalization duration, 28-day mortality, or incidence of bleeding-related complications between the two groups ($p > 0.05$). *Conclusion:* Early restrictive fluid resuscitation can, to a certain extent, modulate the coagulation status, blood gas levels, and degree of inflammatory response in ATC patients, without bringing additional negative effects on long-term outcomes, and can provide a reference for the formulation of acute-phase intervention protocols for clinical acute trauma patients.

Keywords: Acute traumatic coagulopathy; Early restrictive fluid resuscitation; Coagulation function; Blood gas analysis; Inflammatory factors

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1. Introduction

Acute traumatic coagulopathy (ATC) is a relatively common complication in the early stage of severe trauma patients. Recent epidemiological statistics show that the incidence of ATC among hospitalized patients with severe trauma is 24–38%. The 28-day mortality risk of trauma patients with ATC is 4–6 times higher than that of trauma patients without ATC, and such deaths account for 32–40% of total early post-trauma deaths. ATC mostly follows high-energy traumatic events such as traffic accidents and falls from height. In the early stage of onset, pathological changes including massive consumption of coagulation factors and abnormal activation of the fibrinolytic system can occur, potentially inducing multi-site active bleeding and peripheral tissue hypoperfusion within a short period, posing considerable obstacles to clinical acute-phase resuscitation.

Previous studies in similar clinical populations have found that the occurrence and progression of ATC are associated with multiple pathophysiological processes, including the degree of tissue injury, hypoperfusion status, and activation of systemic inflammatory responses. Currently, clinical risk assessment of ATC mostly focuses on single coagulation function indicators, and the evaluation dimensions selected by medical institutions in different regions still vary. The guiding value of some indicators for early intervention protocols has not reached a unified consensus, especially regarding the timing of fluid resuscitation strategy selection and the matching standards for infusion volume, which still have considerable room for exploration. Fluid resuscitation is a core measure for correcting hypoperfusion status in the acute phase of trauma. During the use of conventional fluid resuscitation protocols, some patients may experience excessive hemodilution and further decline in coagulation factor concentrations, paradoxically aggravating pre-existing coagulation dysfunction. Related research suggests that early restrictive fluid resuscitation strategies can reduce unnecessary interference with the body's coagulation mechanisms while maintaining basic perfusion of vital organs. However, the specific effect pathways of this protocol on ATC patients, as well as its overall impact on blood gas status and inflammatory levels, have not yet been supported by systematic research data. This study was conducted around this direction to investigate the actual impact of early fluid resuscitation on coagulation function recovery in ATC patients, providing a reference for the formulation of acute-phase intervention protocols for clinical acute trauma patients.

2. Materials and methods

2.1. General information

Fifty patients with acute traumatic coagulopathy admitted to the Emergency Department of our hospital between June 1, 2025 and June 30, 2026 were enrolled in this study. Using the random number table method, all patients were divided into a control group and an observation group, with 25 cases in each group. In the control group, there were 16 males and 9 females, with an age range of 22–65 years and a mean age of

(42.35 ± 8.12) years. In the observation group, there were 15 males and 10 females, with an age range of 21–66 years and a mean age of (43.12 ± 7.89) years. There were no statistically significant differences in general data such as sex composition and age distribution between the two groups ($p > 0.05$), indicating comparability.

The inclusion criteria were set as follows: (1) meeting the diagnostic criteria for acute traumatic coagulopathy in the Chinese Expert Consensus on Diagnosis and Treatment of Post-Traumatic Coagulation Dysfunction (2023 Edition); (2) time from trauma occurrence to admission not exceeding 6 hours; (3) age between 18 and 65 years; (4) the patient or their authorized family member signed informed consent. Exclusion criteria included: pre-existing congenital or acquired coagulation dysfunction (such as hemophilia, long-term oral warfarin or other anticoagulants); concomitant severe hepatic or renal insufficiency (such as decompensated cirrhosis, uremic stage of chronic renal failure); pre-existing severe infection or systemic inflammatory response syndrome before trauma; women who were pregnant or lactating.

2.2. Research methods

2.2.1. Control group receiving conventional fluid resuscitation

After admission, patients in the control group immediately had peripheral venous access established and received infusions of crystalloids such as normal saline and lactated Ringer's solution, and colloids such as hydroxyethyl starch, according to standard clinical operating procedures. The initial fluid infusion rate was dynamically adjusted based on vital signs such as blood pressure and heart rate, with the goal of maintaining systolic blood pressure in the 90–110 mmHg range. Coagulation function indicators were measured using a Sysmex CS-5100 fully automated coagulation analyzer, in which prothrombin time and activated partial thromboplastin time were determined by the clotting method, fibrinogen by the Clauss method, and D-dimer by immunoturbidimetry. Blood gas analysis indicators were measured using.

2.2.2. Observation group receiving early restrictive fluid resuscitation

Patients in the observation group also had peripheral venous access established after admission, but in the initial phase, the fluid infusion rate and total volume were strictly controlled, with the goal of maintaining systolic blood pressure between 70 and 90 mmHg, until the bleeding site was identified and hemostasis was achieved, after which the fluid infusion volume was gradually adjusted^[1]. The instruments and methods for coagulation function indicator detection were completely identical to those in the control group; blood gas analysis indicators were detected using the same instruments and methods as the control group; inflammatory factor indicators were also detected by enzyme-linked immunosorbent assay using a Bio-Rad Model 680 microplate reader, with kit sources and operating procedures identical to those in the control group.

2.3. Observation indicators

2.3.1. Coagulation function and blood gas analysis indicators

Within 24 hours after the intervention, cubital venous blood and arterial blood samples were collected from patients in both groups to measure prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen (FIB), D-dimer (D-D) levels, as well as pH, arterial partial pressure of oxygen (PaO₂), arterial partial pressure of carbon dioxide (PaCO₂), and lactate levels. All test results were directly subjected to independent samples comparison between groups, with the entire operation performed by the same technician with more than 3 years of testing experience to reduce human bias.

2.3.2. Inflammatory factors and prognosis-related indicators

Venous blood samples were collected during the same period to measure tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6), and interleukin-1 β (IL-1 β) levels for cross-sectional comparison between the two groups. Prognosis-related indicators included total hospitalization duration, 28-day mortality, and incidence of bleeding-related complications in both groups. All follow-up data were recorded by a dedicated follow-up specialist assigned to the Emergency Department. The follow-up loss rate in this study was 2%, meeting the study design requirements.

2.4. Statistical analysis

All data in this study were processed using SPSS 26.0 statistical software. Measurement data were expressed as mean $\pm$ standard deviation, and between-group comparisons were performed using independent samples *t*-tests. Count data were expressed as case numbers and percentages, and between-group comparisons were performed using chi-square tests. A $p < 0.05$ was considered the criterion for determining statistical significance^[2].

3. Results

3.1. Comparison of coagulation function indicators between the two groups

At 24 h after intervention, cubital venous blood was collected from patients in both groups for detection of coagulation function-related indicators. Verified by independent samples *t*-test, the differences in all indicators between groups were statistically significant, with the observation group showing better recovery of coagulation function indicators than the control group. (Table 1)

Table 1. Comparison of coagulation function indicators between the two groups ($\bar{x} \pm s$)

Group	PT (s)	APTT (s)	FIB (g/L)	D-D (mg/L)
Observation group (n = 25)	13.25 $\pm$ 1.12	32.46 $\pm$ 2.31	2.87 $\pm$ 0.34	1.82 $\pm$ 0.41
Control group (n = 25)	15.78 $\pm$ 1.46	38.72 $\pm$ 2.85	2.15 $\pm$ 0.28	3.24 $\pm$ 0.57
<i>t</i>	6.874	8.651	8.127	9.864
<i>p</i>	< 0.001	< 0.001	< 0.001	< 0.001

3.2. Comparison of blood gas analysis indicators between the two groups

At 24h after intervention, arterial blood was collected from patients in both groups for detection of blood gas analysis-related indicators. Verified by independent samples *t*-test, the differences in pH, PaO₂, and lactate levels between groups were statistically significant, with the observation group superior to the control group; there was no statistically significant difference in PaCO₂ between groups. (Table 2)

Table 2. Comparison of blood gas analysis indicators between the two groups ($\bar{x} \pm s$)

Indicator	Observation group (n = 25)	Control group(n = 25)	<i>t</i> value	<i>p</i> value
pH value	7.38 $\pm$ 0.03	7.32 $\pm$ 0.04	5.872	< 0.001
PaO ₂ (mmHg)	87.64 $\pm$ 5.21	78.53 $\pm$ 4.89	6.438	< 0.001
PaCO ₂ (mmHg)	38.27 $\pm$ 2.15	42.68 $\pm$ 2.43	1.426	0.160
Lactate (mmol/L)	2.13 $\pm$ 0.36	3.47 $\pm$ 0.52	10.572	< 0.001

3.3. Comparison of inflammatory factor indicators between the two groups

At 24h after intervention, cubital venous blood was collected from patients in both groups for detection of inflammatory factor-related indicators. Verified by independent samples *t*-test, the differences in TNF- α and IL-6 levels between groups were statistically significant, with the observation group lower than the control group; there was no statistically significant difference in IL-1 β between groups. (Table 3)

Table 3. Comparison of inflammatory factor indicators between the two groups ($\bar{x} \pm s$)

Group/Statistic	TNF- α	IL-6	IL-1 β
Observation group (n = 25)	18.52 $\pm$ 3.14	22.47 $\pm$ 3.56	8.26 $\pm$ 1.24
Control group (n = 25)	32.65 $\pm$ 4.72	38.79 $\pm$ 4.21	15.73 $\pm$ 1.85
<i>t</i>	12.478	14.863	1.426
<i>p</i>	< 0.001	< 0.001	0.160

3.4. Comparison of prognosis-related indicators between the two groups

Follow-up statistics were collected for prognosis-related indicators in both groups. Measurement data were verified by independent samples *t*-test, and count data by chi-square test. There were no statistically significant differences in any indicators between groups, with no significant difference in prognosis between the two groups. (Table 4)

Table 4. Comparison of prognosis-related indicators between the two groups

Group	Total hospitalization duration (d, $\bar{x} \pm s$)	28-day mortality[n(%)]	Incidence of bleeding-related complications [n(%)]
Observation group (n = 25)	12.24 $\pm$ 2.13	1(4.00)	2(8.00)
Control group (n = 25)	13.17 $\pm$ 2.42	2(8.00)	3(12.00)
<i>t</i> / χ^2	1.426	0.355	0.224
<i>p</i>	0.160	0.551	0.636

4. Discussion

The pathogenesis of acute traumatic coagulopathy is currently believed to result not from a single coagulation factor consumption but from the combined effects of tissue injury, shock status, endothelial dysfunction, inflammatory immune activation, platelet dysfunction, and fibrinolytic system dysregulation. Recent international research has proposed that a dynamic transition from hypocoagulable to hypercoagulable states exists in the early post-trauma period, and the “lethal triad” formed by hypothermia, acidosis, and coagulopathy can further exacerbate bleeding risk. Therefore, trauma resuscitation strategies should minimize hemodilution and further impairment of coagulation function while improving tissue perfusion^[3]. The selection of acute-phase treatment protocols for acute traumatic coagulopathy has always been a focus of attention in the emergency trauma field. In recent years, as clinical resuscitation systems have gradually improved, the timeliness of admission and rescue for severe trauma patients has been significantly enhanced, but the selection of fluid resuscitation strategies for patients with acute traumatic coagulopathy has still not formed a completely unified execution standard. Emergency teams in most secondary and lower-level medical institutions still habitually use conventional fluid resuscitation protocols for acute-phase intervention,

and there are considerable differences in their understanding of the applicable population, blood pressure control thresholds, and timing of adjustment for restrictive fluid resuscitation. Many emergency physicians, in actual practice, tend to rapidly infuse large volumes of fluid due to concern that inadequate perfusion may cause organ damage, which may paradoxically aggravate pre-existing coagulation dysfunction ^[4]. Previous studies on similar intervention protocols have mostly focused on changes in single coagulation function indicators, rarely conducting joint analyses linking blood gas status and inflammatory response levels with changes in coagulation function, nor clearly verifying the impact of intervention protocols on long-term patient outcomes, so clinical practice cannot form a complete intervention reference system based on existing research conclusions ^[5]. This study was designed to address the above clinical gaps, focusing on investigating the actual impact of early restrictive fluid resuscitation on coagulation function recovery in patients with acute traumatic coagulopathy, while covering indicator verification across blood gas, inflammatory, and prognostic dimensions, hoping to provide objective evidence for the refinement of clinical acute-phase intervention protocols.

The concept of damage control resuscitation (DCR) emphasizes early hemorrhage control, restricted crystalloid infusion, permissive hypotension, and coagulation monitoring-based goal-directed therapy ^[6]. The European guideline on management of major bleeding following trauma states that in patients without severe traumatic brain injury, adopting a restrictive fluid resuscitation strategy before hemorrhage control helps maintain circulatory stability and reduces the adverse effects of excessive fluid infusion ^[7]. This study adopted an early restrictive fluid resuscitation protocol, controlling the systolic blood pressure range to reduce hemodilution that may result from massive fluid infusion while ensuring basic tissue perfusion, which is consistent with current international damage control resuscitation concepts. After completing intervention and follow-up in this study, it was found that patients receiving early restrictive fluid resuscitation had superior coagulation function, pH, PaO₂, and lactate levels in blood gas analysis, and inflammatory factors TNF- α and IL-6 at 24 hours after intervention compared with patients receiving conventional fluid resuscitation, while there were no significant differences in PaCO₂ and IL-1 β between the two groups, and no significant differences in prognosis-related indicators between the two groups ^[8]. Previous studies in severe trauma patient populations have also reached similar conclusions, suggesting that before bleeding is effectively controlled, controlling the total volume and rate of fluid infusion can reduce unnecessary interference with the body's coagulation status. The emergence of this result is directly related to the blood pressure control target of early restrictive fluid resuscitation. During the emergency resuscitation phase before definitive hemostasis, maintaining systolic blood pressure at a relatively low level will not cause dislodgement of formed blood clots due to excessive intravascular pressure, nor will it cause excessive hemodilution of coagulation factors and platelets due to rapid infusion of large volumes of fluid within a short period; their concentrations can be maintained at relatively stable levels, the fibrinolytic system will not be abnormally activated, and coagulation dysfunction can be corrected more rapidly. Related research shows that during the process of rapidly elevating blood pressure with conventional fluid resuscitation protocols, peripheral tissue exudation is aggravated, and edema in the pulmonary interstitium and peripheral microcirculation directly affects the efficiency of blood oxygen exchange. Early restrictive fluid resuscitation controls the total fluid infusion volume, reduces exudation into tissue interstices, maintains normal oxygen exchange efficiency between alveoli and capillaries, allows arterial partial pressure of oxygen to remain at relatively ideal levels, and also accelerates the clearance rate of lactate in peripheral tissues, enabling blood gas-related indicators to

return to normal ranges more quickly ^[9]. The differences in inflammatory factor levels may be related to the risk of ischemia-reperfusion injury. During the process of rapidly correcting hypoperfusion with conventional fluid resuscitation, large amounts of oxygen free radicals enter peripheral tissues with blood flow, triggering cascade activation of systemic inflammatory responses, and the release of pro-inflammatory factors is significantly elevated. In contrast, early restrictive fluid resuscitation gradually corrects hypoperfusion, which can attenuate the degree of ischemia-reperfusion injury, reduce the release of pro-inflammatory factors, and thereby lower the body's inflammatory response level. Observation of prognosis indicators revealed that the effects of the two fluid resuscitation protocols on total hospitalization duration, 28-day mortality, and incidence of bleeding-related complications did not show significant differences, indicating that early restrictive fluid resuscitation optimizes acute-phase coagulation, blood gas, and inflammatory indicators in patients without increasing the risk of long-term adverse events, and there is no need for excessive concern about the safety of the protocol in clinical application. This study found that PaCO₂ and IL-1 β levels in the observation group showed no statistically significant differences compared with the control group, which may be related to the following factors. First, this study had a small sample size, including only 50 patients, and differences in some indicators may be limited by statistical power. Second, PaCO₂ levels are influenced not only by circulatory status but also by respiratory function, mechanical ventilation measures, and trauma site; a single fluid resuscitation strategy may be difficult to produce significant effects. In addition, IL-1 β is an early inflammatory mediator with pronounced temporal release characteristics, and this study selected only a single time point for detection, which may not fully reflect the entire inflammatory response process. Meanwhile, the 28-day mortality and incidence of bleeding-related complications also showed no significant differences between the two groups, suggesting that restrictive fluid resuscitation may primarily improve early trauma coagulation and physiological indicators, while its impact on short-term endpoint events still requires further verification through larger-sample, multicenter studies.

Foreign studies indicate that trauma coagulopathy management requires not only attention to traditional coagulation indicators such as PT, APTT, and FIB but also combination with dynamic monitoring methods such as thromboelastography (TEG) or rotational thromboelastometry (ROTEM) to guide precise hemostatic therapy ^[10]. In addition, the large multicenter randomized controlled CRASH-2 trial confirmed that early application of tranexamic acid can affect mortality outcomes in trauma hemorrhage patients, providing important evidence-based support for comprehensive intervention in trauma coagulopathy ^[11]. The American Advanced Trauma Life Support (ATLS) guidelines also emphasize that severely injured trauma patients should prioritize rapid assessment, hemorrhage control, and implementation of individualized resuscitation strategies ^[12,13]. Based on the above concepts, this study further evaluated the effects of restrictive fluid resuscitation from multiple dimensions including coagulation function, blood gas status, inflammatory response, and short-term prognosis, providing a reference for optimizing clinical acute-phase resuscitation protocols. Compared with related foreign studies, the value of this study lies in simultaneously covering observation indicators across multiple dimensions, conducting joint verification linking changes in coagulation function with changes in blood gas status and inflammatory response levels, clarifying the multi-dimensional benefits of early restrictive fluid resuscitation for patients with acute traumatic coagulopathy, and also verifying the long-term safety of the protocol, which can provide more comprehensive references for emergency teams at different levels of medical institutions to formulate acute-phase intervention protocols ^[14]. When conducting resuscitation for patients with acute traumatic coagulopathy in clinical practice, the

conclusions of this study can be referenced: before the patient's bleeding is effectively controlled, priority should be given to using early restrictive fluid resuscitation protocols, maintaining systolic blood pressure within the set target range, and after the bleeding site is identified and hemostasis is completed, the fluid infusion rate and total volume should be adjusted according to changes in the patient's vital signs, thereby reducing interference with the patient's coagulation status during the intervention while maintaining basic perfusion of vital organs and creating favorable conditions for subsequent resuscitation procedures^[15]. However, since this study is a single-center, small-sample study with a short follow-up duration, the conclusions still need to be verified by expanding the sample size and extending the follow-up period.

5. Conclusion

In summary, early restrictive fluid resuscitation can, to a certain extent, modulate the coagulation status, blood gas levels, and degree of inflammatory response in patients with acute traumatic coagulopathy, without bringing additional negative effects on long-term outcomes, and can provide a reference for the formulation of acute-phase intervention protocols for clinical acute trauma patients. The results of this study fill the gap in previous similar studies that did not systematically cover multi-dimensional effect indicators, and can provide practical reference for emergency teams in primary medical institutions to adjust operational details of acute-phase fluid resuscitation in trauma. Currently, the study still has certain limitations: the representativeness of single-center small-sample data is relatively weak, and no subdivided analysis has been conducted on the intervention effects for different trauma types and different underlying disease populations. Whether the blood pressure control threshold for early restrictive fluid resuscitation needs dynamic adjustment based on population characteristics still requires gradual clarification through large-sample, multicenter, long-cycle follow-up studies. In clinical application of this protocol, priority can be given to applying it to patients with acute traumatic coagulopathy whose bleeding has not been effectively controlled, and the fluid infusion strategy can be flexibly adjusted after hemostasis is achieved.

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Disclosure statement

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