ORIGINAL RESEARCH

Value of Thymosin α1 Combined With Blood Purification to Increase Successful Rescues of Shock Patients

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ABSTRACT

Context • Septic shock (SS) can pose a high risk of death if rescue efforts in an emergency room aren't started in a timely manner. Thus, rapid and efficient treatment is of great significance to the SS patients' survival. T-α1 can enhance the cellular immune function of patients, and blood purification (BP) can improve the hemodynamics of SS patients by clearing inflammatory mediators in the blood.

Objective • The study intended to explore the effects of Thymosin α1 (T-α1) plus blood purification (BP) on SS patients under the emergency green channel (GC), a fast and efficient service system that hospitals provide for acutely and critically ill patients.

Design • The research team designed a randomized controlled study.

Setting • The study took place in the Emergency Department at the Second Affiliated Hospital of Xi'an Jiaotong University in Xi'an, Shaanxi, China.

Participants • Participants were 86 SS patients who came to the hospital for treatment between June 2019 and January 2021.

Intervention • The research team numbered the patients in sequence according to the admission time of the patients, and then randomly numbered them by the computer, and assigned participants to an intervention or a control group, with 43 participants in the intervention group receiving T-α1 plus BP therapy and 43 participants in the control group receiving BP treatment only.

Outcome Measures • The study measured preparation time before treatment, symptom-onset-to-door (SOTD), duration of shock, length of stay in the intensive care unit (ICU), and incidence of adverse reactions. The study also assessed changes between baseline and postintervention in inflammatory cytokines (ICs), immunological function, and myocardial-function markers. Finally, the research team conducted a one-year follow-up to determine participants' prognostic survival.

Results • The groups showed no significant differences in the preparation time before treatment, SOTD, rescue success rate, and incidence of adverse events ($P > .05$), while the intervention group showed a significantly shorter duration of shock and length stay in the ICU and a significantly higher overall response rate ($P < .05$). The research team observed significant improvements in the T-lymphocyte subsets, ICs, and myocardial function in both groups postintervention, but the changes in the intervention group were significantly greater ($P < .05$). Follow-up results showed no significant differences in overall survival between the intervention and control groups ($P > .05$), but the average LC was significantly higher in the intervention group ($P < .05$).

Conclusions • For SS patients, the combination of T-α1 and BP under the emergency GC can effectively improve their immunological and myocardial function, reduce inflammatory reaction, and prolong their LCs, which provides a greater guarantee of the effectiveness of treatment for SS patients in the future. (*Altern Ther Health Med.* 2022;28(7):146-152).

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Sepsis, as a systemic inflammatory-response syndrome that infection can cause, is an important public health issue.[1] Sepsis can develop into severe sepsis and septic shock (SS), the latter of which is also called infectious shock and is a common, clinically critical and acute illness, with shock as its prominent manifestation.[2]

Septic shock (SS) can be attributed mainly to the external stimulation, production, and release of massive amounts of inflammatory cytokines (ICs) and endotoxins in vivo, which

can lead to ischemia and hypoxia of tissues and cells. After invading the blood-circulation system, pathogenic microorganisms and their toxins activate the body's immune system and produce the cytokines and inflammatory mediators.[3] The uncontrolled inflammatory response can greatly reduce the effective circulating blood volume, causing an insufficient perfusion of tissues and organs throughout the patient's body as well as a compromised microvascular-contraction function, which vascular endothelial cell damage can cause.[4] These factors all contribute to the occurrence of SS.

The inflammatory response can cause systemic diseases of multiple organs and systems, resulting in the metabolism and dysfunction of local tissues and cells and even inducing multiple organ failure that can be life-threatening in serious cases.[3] The mortality rate of SS patients, with complications from multiple organ failure, can reach up to 40-60%, among which 15% die from refractory heart failure.[5] Russell et al have indicated a significant increase in the number of sepsis patients worldwide every year, and the fatality rate is high.[6]

SS patients are more severely ill at the onset of sepsis, with a condition that progresses rapidly.[7] As a rapidly developing disease, SS can pose an high risk of death if rescue efforts aren't started in a timely manner. Thus, rapid and efficient treatment is of great significance to the SS patients' survival.

The Emergency Green Channel (GC)

The traditional admission process for emergency departments at hospitals is complicated, which easily can delay rescue efforts.[8] In China, the emergency green channel (GC) is a fast-and-efficient service system that hospitals provide for acutely and critically ill patients. The emergency GC allows medical staff to clearly allocate work among departments in the emergency process and promote the smooth progress of the emergency work.

The emergency GC is also conducive to improving the rescue and emergency response capabilities of nursing staff. It prompts them to constantly improve their professional level and nursing skills, so that they can fearlessly deal with complex injuries, cooperate with anesthesiologists and surgeons to complete operations in an orderly manner, and improve the success rate of surgical rescue for patients with traumatic shock.

After they open an emergency GC for a patient, the medical staff can prioritize the relevant examinations, laboratory tests, treatments, and operations and ensure that relevant departments can complete relevant diagnoses and treatment examinations in the shortest possible time, thus shortening the preparation time for emergency treatment.[9]

Thymosin α1 (T-α1)

Conventional anti-infectives, antibiotics, fluid resuscitation, and other therapeutic measures can't effectively control the toxic and side effects and inflammatory mediators in patients.[10] Dalimonte et al indicates that researchers have been committed to exploring and finding a new and more effective treatment for SS in recent years.[11]

Some studies have found that T-α1 can have excellent effects in treatment of pneumonia, immunity and other diseases.[12] T-α1 is a small-molecule, active peptide that is capable of activating the body's cellular conduction pathways and enhancing systemic immunoreaction.[13]

Recently, Minasyan found that immune dysfunction is an important predisposing factor for SS.[14] As a biological agent, T-α1 can enhance the cellular immune function of patients, promote the differentiation of immune cells and make them mature quickly, with ideal effects and high safety.[15]

Other studies have pointed out that T-α1 can block the secretion and synthesis of ICs to play a role in reducing inflammatory indicators.[16-18] Bellet et al found that T-α1's benefits are closely related to its inhibitory effect on lymphocyte apoptosis and dendritic-cell activation.[19]

Costantini et al and Yang et al found that that T-α1 also has the effect of stabilizing the cell membrane, which can reduce inflammatory exudation of capillaries during inflammatory infection and reduce tissue edema, thus playing a role in organ preservation.[20,21] Liu et al found that T-α1 can affect the rescue of SS patients by shortening shock duration and ICU admission time.[22] Dominari et al also found that T-α1 can exert a positive effect on SS treatment.[23]

The leading cause of death in SS is known to be refractory heart failure.[24] Therefore, an improvement in patients' myocardial function with T-α1 may protect them from death.

Blood Purification (BP)

In addition to T-α1, blood purification (BP) is also one of the main means of clinical treatment of sepsis,[25] and it can improve the hemodynamics of SS patients by clearing inflammatory mediators in the blood, helping patients recover their immune function, and accelerating their rehabilitation.[26]

Current Study

Based on the above information, the current research team hypothesized that T-α1 plus BP might provide beneficial results in treating SS, with half the effort. Zhang et al found that the combination could ensure efficiency, smoothness, and standardization in the rescue process, to provide more rescue time for patients, extend their prognostic survival, and improve their prognoses and quality of life.[27]

The current study intended to explore the effects of T-α1 plus BP)for SS patients under an emergency GC, to provide a reliable theoretical and experimental basis for future clinical management of SS patients.

METHODS

Participants

The research team designed a randomized controlled study. The study took place in the Emergency Department at the Second Affiliated Hospital of Xi'an Jiaotong University in Xi'an, Shaanxi, China. Potential participants were of various diseases and then experienced SS patients who came to the hospital for treatment between June 2019 and January 2021.

The research team included potential participants if they: (1) were older than 18 years of age, (2) met the diagnostic criteria for SS,[28] (3) had a score >12 points on the Acute Physiology and Chronic Health Enquiry II (APACHE II), (4) had complete medical records available at the hospital, and (5) agreed to voluntarily participate in the study.

The research team excluded potential participants if they: (1) had an autoimmune deficiency disease, blood disease, malignant tumor, or mental diseases; (2) had recently used immunosuppressants, hormones, or other immune stimulants; (3) were pregnant or lactating; (4) had drug contraindications; (5) were referrals; (6)death during treatment (if the patient died before the treatment was completed we cannot confirm whether T-α1 plays a role in the end, so such patients are excluded).

This study was conducted in strict compliance with the Declaration of Helsinki, and was approved by the ethics committee. The immediate family members of all participants signed an informed consent.

Procedures

Groups. The research team according to the random number table method participants to an intervention or a control groups, with 43 participants in the intervention group receiving T-α1 plus BP therapy and 43 participants in the control group receiving BP treatment only

Establishment of the emergency GC. The hospital set up a special telephone for emergency GC in all relevant departments to improve the GC process, and the first diagnosis responsibility system was implemented (Physicians must be responsible for the examination, diagnosis, treatment, consultation, referral, transfer to department, hospital transfer and other clinical diagnosis and treatment work for the patients they receive, especially for critical, urgent and severe patients).

After a patient arrives at the emergency room, the receiving physician quickly clarifies the patient's condition, opens a GC as needed, and then notifies the operating room to be ready for surgery at any time. Afterward, the emergency-room staff notifies the doctors of the corresponding departments by telephone to allow them to assemble quickly, which ensures that all the relevant doctors will arrive within 10 minutes.

After the patient's admission, the medical staff instructs him or her to remain supine and establishes ECG monitoring after setting up oxygen inhalation for the patient. The emergency department's nurses quickly create venous access, and the emergency doctors prepare first-aid medicines after receiving the notice, especially those medicines that will correct the shock, in accordance with the patient's specific situation.

During the first aid, the roving nurses closely observe any changes in a patient's condition, ensure that the patient's infusion and blood transfusion channels are unobstructed, and assist the physicians in completing the first aid.

Postadmission treatment. Both groups received fluid replacement, vasoactive drugs, and correction of water-electrolyte and acid-base imbalances as well as routine treatments such as respiratory and nutritional support.

In addition, the medical staff mixed 50 mL of normal saline with 300 000 U of ulinastatin (Guangdong Techpool Bio-Pharma, SFDA Approval No.: H19990133, Guangzhou, Guangdong, China) for an intravenous drip, which all participants received once every 12h for 30 min each time.

Outcome measures. The study measured preparation time before treatment, symptom-onset-to-door (SOTD), duration of shock, length of stay in the intensive care unit (ICU), and incidence of adverse reactions. At baseline and postintervention, participants gave fasting, venous blood samples that the research team divided into two parts. They used the first part for flow-cytometry detection of T lymphocyte subsets CD3+, CD4+, and CD8+, to measure immunological function.

The team used the other part for serum collection, with 30 min of centrifugation (TXK4 blood type serum multi-purpose centrifuge, Yingtai, Changsha, Hunan, China) at a 1505 relative centrifugal force (xg), to measure inflammatory cytokines: high-sensitivity C-reactive protein (hs-CRP), tumor necrosis factor-α (TNF-α), and procalcitonin (PCT) using an enzyme-linked immunosorbent assay (ELISA). The team purchased the ELISA kit from Solarbio (Beijing, China).

The team also used the serum to measure myocardial-function markers: brain natriuretic peptide (BNP), creatine kinase isoenzyme (CK-MB), and lactate dehydrogenase (LDH), using an automatic biochemical analyzer (BS-2000, mindray, Shenzhen, Guangdong, China).

Finally, the research team conducted a one-year follow-up to determine participants' prognostic survival.

Follow-up investigation. The research team followed the successfully rescued participants for one year until February 1, 2022, to record their survival time and plot the survival curve.

Intervention

Both groups were treated continuously for 10 days.

Control group. The control group received BP treatment, as in the intervention group.

Intervention group. The intervention group received the BP treatment and also received a subcutaneous injection of 1.6 mg of T-α1 (Sciclone Pharmaceuticals Italy, registered number of approval: H20080079, Rome, Lazio, Italy), twice a week, with an injection interval of three days.

Outcome Measures

Clinical indices. The indices included preparation time before treatment (The preparation time from the onset of the patient's condition to the official start of treatment), symptom-onset-to-door (SOTD) (The time from the onset of the patient's condition to being admitted to the emergency room), duration of shock, and length of ICU stay.

Rescue efficacy. The treatment's efficacy in both groups was evaluated at 10 days postintervention, to determine if the treatment had been effective, which refers to basic recovery of consciousness, with an improved condition. If the participant had regained consciousness, with a stable

condition, the research team considered the treatment to be markedly effective. Ineffective treatment corresponded to failure to meet those standards or an aggravation of illness.

Rescue success rate. The rate used the number of participants having survived at the follow-up in each group.

Overall response rate (ORR). ORR = (markedly effective + effective cases) / total cases × 100%.

Safety. The research team recorded any adverse events that had occurred during treatment to count the incidence. Possible adverse events were abnormal liver function, abdominal distension and diarrhea, fatigue, dizziness and headache, and nausea.

Inflammatory cytokines (ICs). IC is a very representative detection index in the pathological process of SS, which reflects the inflammatory response and injury degree in the patient's body. The research team measured hs-CRP, TNF-α, and PCT, the higher the test result, the more serious the patient's injury.

Immunological function. T lymphocyte subsets reflect the body's immune and metabolic function, and reflect the body's ability to resist infection and damage self-repair, the research team determined participants' CD3+, CD4+, CD8+, and CD4+/CD8+. Normal value: CD3+ is 71.5%±6.2%, CD4+ is 45.7% ± 5.3%, CD8+ is 27.9% ± 5.0%, CD4+/CD8+ is 1.66 ± 0.33.

Myocardial function. The research team assessed BNP, CK-MB, and LDH. This is a marker of myocardial damage that reflects the normal functioning of the heart muscle. An elevated test result indicates a worsening degree of myocardial damage in the patient.

Survival. The research team counted the number of participants who survived for 28 days postintervention, and regarded a patient's survival as a successful rescue. Survival included overall survival (OS) as well as the shortest and the prognostic survival during the follow-up.

Statistical Analysis

The research team performed the statistical analysis using SPSS23.0 (NDTimes, Beijing China). The Chi-square test was used for categorical data, numbers and percentages, while the independent sample t-test and paired t-test were used for quantitative data, means ± standard deviations (SDs). The Kaplan-Meier method and Log-rank test were employed for the survival-rate calculation and comparison, respectively. Differences were of statistical significance when $P < .05$.

RESULTS

Participants

The study enrolled and analyzed the data of 86 SS participants, with 43 participants in the intervention group and 43 participants in the control group. As Table 1 shows, the intervention and control groups had no significant differences in age, gender, course of disease, or APACHE II score ($P > .05$).

Table 1. Comparison of Clinical Baseline Data for the Intervention and Control Groups

	Control Group n = 43 Mean ± SD n (%)	Intervention Group n = 43 Mean ± SD n (%)	*t* or χ^2	*P* Value
Age	66.77 ± 6.35	65.63 ± 7.55	0.758	0.451
Disease duration, min	42.21 ± 2.30	41.79 ± 11.33	0.238	0.812
APACHE II score	20.88 ± 1.78	20.58 ± 1.88	0.760	0.450
Gender			0.745	0.388
Male	24 (55.81)	20 (46.51)		
Female	19 (44.18)	23 (53.49)		
Disease Type			1.289	.863
SPN	16 (37.21)	17 (39.53)		
SPA	11 (25.58)	10 (23.26)		
BU	6 (13.95)	9 (20.93)		
NC	5 (11.63)	3 (6.98)		
Other	5 (11.63)	4 (9.30)		
Infection Site			1.149	.887
RI	21 (48.84)	22 (51.17)		
USI	9 (20.93)	8 (18.60)		
AI	4 (9.30)	6 (13.95)		
BI	5 (11.63)	5 (11.63)		
Other	4 (9.30)	2 (4.65)		
Smoking			0.050	.822
Yes	16 (37.21)	15 (34.88)		
No	27 (62.79)	28 (65.12)		
Drinking			0.212	.645
Yes	13 (30.23)	15 (34.88)		
No	30 (69.77)	28 (65.12)		
Family History Of Illness			2.048	.152
Yes	2 (4.65)	0 (0.00)		
No	41 (95.35)	43 (100.00)		

Abbreviations: AI, abdominal infection; APACHE, Acute Physiology and Chronic Health Enquiry II; BI, bloodstream infection; BU, burn; NC, necrotizing cholangitis; RI, respiratory infection; SPA, severe pancreatitis; SPN, severe pneumonia; USI, urinary system infection.

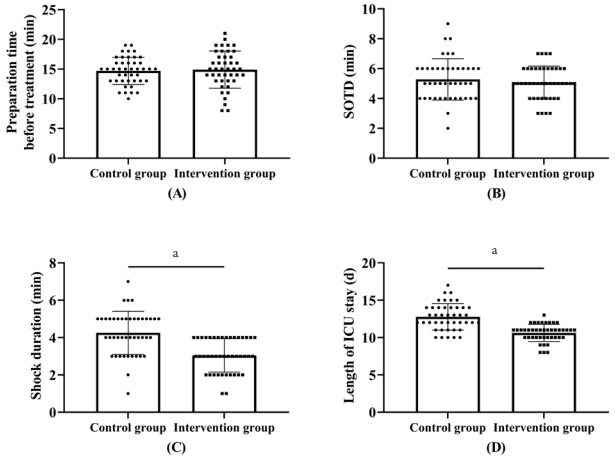
Clinical Indices

As Figure 1 shows, the groups showed no significant differences in preparation time before treatment or SOTD ($P > .05$), while the shock duration (3.05 ± 0.90 min) and length of ICU stay (10.63 ± 1.16 d) were significantly shorter in the intervention group than those in the control group ($P < .05$), showing that the intervention group had a faster recovery than the control group did.

Curative Effects

As Table 2 shows, the rescue success rate for the intervention group was 83.72%, but it was not significantly different from that of the control group at 74.42% ($P > .05$). For clinical efficacy in the intervention group, the study found the treatment was markedly effective 27.90% of

Figure 1. Comparison of Clinical Indexes Between the Intervention and Control Groups. Figure 1A shows the preparation time before treatment; Figure 1B shows the SOTD; Figure 1C) shows the shock duration; and Figure 1D shows the length of the ICU stay



[a]$P < .05$, indicating that the shock duration and length of ICU stay were significantly shorter in the intervention group than those variables in the control group

Abbreviations: ICU, intensive care unit; SOTD, symptom-onset-to-door

Table 2. Clinical Efficacy for the Intervention and Control Groups. As of February 1, 2022, the research team had successfully followed up 32 participants in the intervention group and 35 in the control group. Between postintervention and the follow-up, 11 deaths had occurred in the intervention group and 9 in the control group

	Markedly Effective n (%)	Effective n (%)	Ineffective n (%)	ORR n (%)	Rescue Success Rate n (%)
Control group, n = 43	12 (27.90)	14 (32.56)	17 (39.54)	26 (60.47)	34 (79.01)
Intervention group, n = 43	16 (37.21)	19 (44.19)	8 (18.60)	35 (81.40)	32 (74.42)
χ^2				4.568	1.124
P Value				0.033*	.289

*$P = .033$, indicating that the clinical efficacy was significantly better for the intervention group than for the control group

Abbreviations: ORR, overall response rate.

Table 3. Incidence of Adverse Reactions in the Intervention and Control Groups

	Abnormal Liver Function n (%)	Abdominal Distension and Diarrhea n (%)	Fatigue n (%)	Dizziness and Headache n (%)	Nausea n (%)	Overall Incidence n (%)
Control group, n = 43	2 (4.65)	1 (2.33)	3 (6.98)	3 (6.98)	2 (4.65)	11 (25.58)
Intervention group, n = 43	1 (2.33)	1 (2.33)	3 (6.98)	2 (4.65)	2 (4.65)	9 (20.93)
χ^2						0.261
P Value						.610

participants, effective for 32.56%, and ineffective for 39.53%, with an ORR of 81.40%, which was significantly higher than the 60.47% in the control group ($P < .05$). Therefore, the intervention group's treatment had significantly better curative effects than that of the control group.

Safety

As Table 3 shows, the number of participants in the intervention group who developed abnormal liver function, abdominal distension and diarrhea, fatigue, dizziness and headache, and nausea was 1, 1, 3, 2, and 2, respectively. That group's overall incidence of adverse events was 20.93%, a rate that wasn't significantly different from the 25.58% in the control group ($P > .05$).

Immunological Function

As Figure 2 shows, the CD3+, CD4+, CD8+ and CD4+/CD8+ levels were not significantly different between the groups prior at baseline ($P > .05$), and postintervention, the CD3+, CD4+ and CD4+/CD8+ levels increased while CD8+ decreased significantly in both groups. The changes were significantly greater in the intervention group than those in the control group ($P < .05$). The results suggest better immunological function in the intervention group than in the control group postintervention.

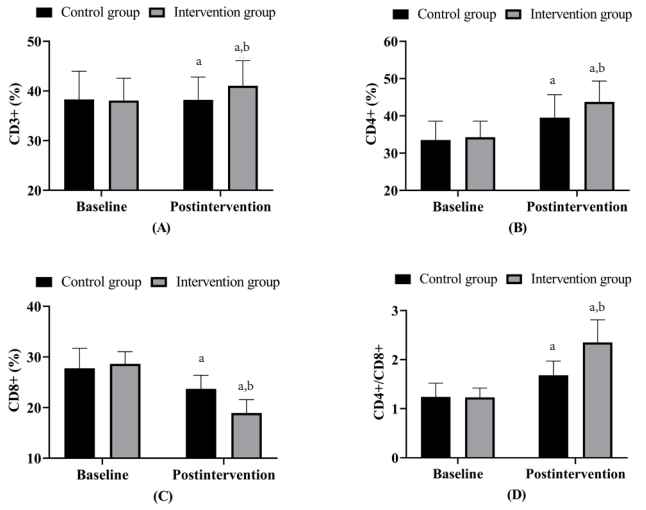
As Figure 3 shows, the ICs (hs-CRP, TNF-α and PCT at baseline weren't significantly different between the groups ($P > .05$), and the ICs had decreased significantly postintervention for both groups ($P < .05$). The PCT, TNF-α, and hs-CRP in the intervention group were 0.49 ± 0.09 ng/ml, 26.86 ± 6.93 ng/L and 13.62 ± 2.89 mg/L, respectively, postintervention. The reductions for the intervention groups were significantly greater than those in the control group ($P < .05$).

Myocardial Function

As Figure 4 shows, the BNP, CK-MB and LDH levels were not significantly different between the groups at baseline ($P > .05$), and the measures had decreased significantly postintervention for both groups ($P < .05$).

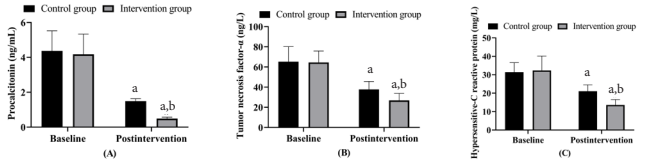
The BNP in the intervention group decreased to 344.64 ± 89.54 μg/L postintervention, which was significantly lower than that in the control group ($P < .05$). Similarly, the reductions in CK-MB and LDH in the intervention group were significantly greater than those of

Figure 2. Comparison of Immune Function at Baseline and Postintervention Between the Intervention and Control Groups. Figure 2A shows the CD3+; Figure 2B shows the CD4+; Figure 2C shows the CD8+; and Figure 2D shows the CD4+/CD8+



[a]$P < .05$, indicating that the CD8+ significantly decreased and the CD3+, CD4+, and CD4+/CD8+ significantly increased between baseline and postintervention for both groups
[b]$P < .05$, indicating that the CD8+ was significantly lower and the CD3+, CD4+, and CD4+/CD8+ were significantly higher for the intervention group postintervention than those variables for the control group

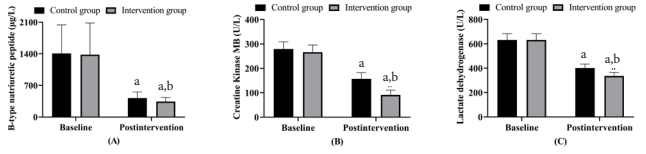
Figure 3. Comparison of Inflammatory Cytokines at Baseline and Postintervention Between the Intervention and Control Groups. Figure 3A shows the PCT; Figure 3B shows the TNF-α; and Figure shows the hs-CRP



[a]$P < .05$, indicating that the PCT, TNF-α, and hs-CRP significantly decreased between baseline and postintervention for both groups
[b]$P < .05$, indicating that the PCT, TNF-α, and hs-CRP for the intervention group were all significantly lower postintervention than those variables for the control group

Abbreviations: hs-CRP, high-sensitivity C-reactive protein; PCT, procalcitonin; TNF-α, tumor necrosis factor-alpha.

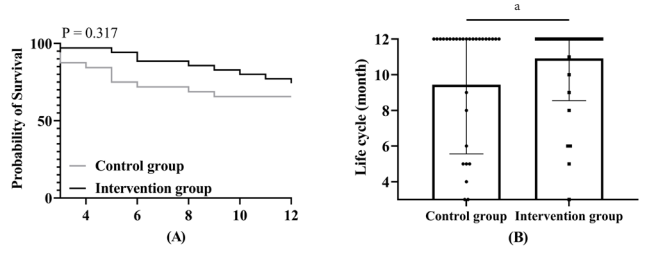
Figure 4. Comparison of Myocardial Function at Baseline and Postintervention Between the Intervention and Control Groups. Figure 3A shows the BNP; Figure 3B shows the CK-MB; and Figure 3C shows the LDH



*$P < .05$, indicating that the BNP, CK-MB, and LDH significantly decreased between baseline and postintervention for both groups
#$P < .05$, indicating that the BNP, CK-MB, and LDH for the intervention group were all significantly lower postintervention than those variables for the control group

Abbreviations: BNP, brain natriuretic peptide; CK-MB, creatine kinase isoenzyme; LDH, lactate dehydrogenase.

Figure 5. Comparison of Life Cycles Between the Intervention and Control Groups. Figure 5A shows the survival curves, and Figure 5B shows the life cycle.



[a]$P < .05$, indicating that the control group's life cycle was significantly shorter than that of the intervention group

the control group ($P < .05$), indicating greater improvement in myocardial function in the intervention group than in the control group.

LC

As of February 1, 2022, the research team had successfully followed up 32 participants in the intervention group and 35 in the control group. In the intervention group, 11 deaths had occurred and 9 had occurred in the control group, with no significant difference in the survival curves between the groups, as Figure 5A shows ($P > .05$).

The shortest LC was one month in the intervention group and 3 months in the control group, while the average LC in the intervention group was 10.91 ± 2.37 months, significantly higher than that of the control group ($P < .05$).

DISCUSSION

The current study found clinical indices found no significant differences in the preparation time before treatment and SOTD between the intervention group and the control group, but the shock duration and length of ICU stay were significantly shorter in the intervention group than in the control group.

In the subsequent comparison of clinical efficacy, the current study found a higher ORR in the intervention group

than in the control group, again demonstrating the excellent therapeutic effects of T-α1 on SS. Furthermore, the current study tested the T lymphocyte subsets at baseline and postintervention in the two groups, finding increased CD3+, CD4+ and CD4+/CD8+ and decreased CD8+ postintervention in both groups, which showed that patients' immune function had improved. Moreover, the intervention group showed more significant improvement in the ICs postintervention, which once again showed that T-α1 can have a good effect in alleviating inflammation in SS patients. However, we found that the experiments of Giacomini et al. showed that T-α1 had no effect on inflammatory factors in multiple sclerosis patients,[29] which was inconsistent with our results. We believe that it may be that SS patients themselves have more severe inflammatory response, so the reduction is significantly after T-α1 treatment; it may also be that the inflammatory response of multiple sclerosis itself is not obvious.[30] Of course, this needs to be confirmed by in vitro experiments as soon as possible.

After the T-α1 treatment, the intervention group in the current study also achieved greater improvement in myocardial function than the control group did. Finally, the current research team compared patient survival in the follow-up, and the average LC of the intervention group was longer than that of the control group, which also indicated that T-α1 had a more reliable guarantee of the SS patients' survival.

Despite the above findings, the current study had some limitations to be addressed. For example, the experimental period was short, the number of cases included in this study was small, and the follow-up time was too short. In addition, detailed grouping of the underlying causes of SS in patients is needed to determine a more precise application of T-α1. In the follow-up research, the current research team also needs to understand the specific influencing mechanism of T-α1 on SS through basic experiments, so as to provide more reliable and comprehensive reference opinions for clinical practice.

CONCLUSIONS

The use of T-α1 plus BP under the emergency GC can effectively improve the immunological and myocardial function of SS patients, reduce inflammatory reaction, and prolong patients' LCs, which provides a greater guarantee of the effectiveness of treatment for SS patients in the future.

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