Analysis of the Protective Effect of Interleukin-5 On Sepsis Mice

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IL-5 can stimulate the growth and differentiation of EOS, inhibit its apoptosis and stimulate the proliferation and differentiation of B cells with other cytokines, and act on mature B cells to promote the synthesis of IgA and IgM. Therefore, this paper takes C57BL / 6 mice as the experimental object to explore the protective effect of interleukin-5 on sepsis mice. The experimental results show that the serum cxcl-1 in sepsis model group is significantly higher than that in control group at each time point, reaching the peak at 12 h, while cxcl-1 in IL-5 intervention group is significantly lower than that in control group. IL-5 can reverse the liver injury induced by CLP in septic mice, the mechanism may be to reduce the production of inflammatory factors in the liver, and the liver function of mice was not damaged 12 hours after sepsis. IL-4 and IL-13 of type 2 inflammatory response factors can promote the conversion of macrophages to anti-inflammatory macrophages, inhibit the inflammatory reaction, and play a protective role in sepsis.

Keywords: Sepsis Mice, Interleukin-5, Histopathological Changes, Inflammatory Factors, Animal Experiments
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Sepsis is a kind of systemic malignant inflammatory reaction caused by infection. Pathogens proliferate in the blood after local or invasive blood flow, and spread to other tissues or organs in the host body through the blood flow to produce new lesions. At the same time, the components of the bacteria or toxins released stimulate the innate and adaptive immune system to produce a large number of inflammatory mediators. Sepsis is a common clinical critical illness.

The production of a large number of inflammatory mediators will stimulate the rapid release of anti-inflammatory factors in the body, resulting in the alternation of the peak values of pro-inflammatory and anti-inflammatory mediators in the blood circulation [1]. Sepsis occurs when the infection exceeds local tissue control and causes a series of dysfunctional physiological reactions, leading to organ dysfunction. Some patients with sepsis progress to septic shock with severe circulatory, cellular and metabolic abnormalities, and high mortality. Historically, organ dysfunction and lethality caused by sepsis have been attributed to a complex interaction between the initial inflammatory response and the subsequent anti-inflammatory response. With the progress of intensive care medicine and goal-directed interventions, the early 30-day sepsis mortality rate has decreased, and it only increases steadily long after the acute event "recovers". Since many septic survivors later died of persistent, recurrent, hospital and secondary infections, Delano turned his attention to changes in cellular immune function caused by long-term sepsis. Sepsis significantly changes the innate and adaptive immune response in a period of time after clinical recovery, which is represented by immunosuppression, chronic inflammation and the persistence of bacteria. Delano understands that sepsis associated immune cell defects are associated with long-term mortality, and more research has focused on the potential of

immunomodulatory therapy to improve the long-term prognosis of patients. The focus of these efforts is to better define and effectively reverse persistent immune cell dysfunction associated with long-term septic mortality [2]. Lemierre syndrome, or retro membranous sepsis, first appeared in the early part of this century and is characterized by pharyngitis, followed by high fever and stiffness, cervical adenosis, thrombophlebitis of the internal jugular vein, distant abscess formation, and jaundice, associated with the isolation of Fusobacterium necrotic in the blood. Leugers described a case of retro vascular sepsis and reviewed the medical literature on retro vascular sepsis using the key word Fusobacterium from MEDLINE database. The characteristics of Lemierre syndrome have hardly changed since the initial description, but with the development of antibiotics, the prognosis has improved greatly. Appropriate treatment includes rapid administration of an anaerobically coated antibiotic, continuous drainage of the abscess, and continued antibiotic treatment until the abscess is treated with radiation. The study of leugers shows that although Lemierre syndrome is a relatively rare disease, primary health care doctors need to understand its clinical characteristics and treatment methods in order to carry out appropriate treatment [3]. Song was designed to investigate the concentration of soluble triggering receptor expressed by myeloid cell-1 in acute abdominal pain and its diagnostic significance. Song collected plasma samples from 68 patients with abdominal sepsis, 60 patients with systemic inflammatory response syndrome (SIRS) and 60 healthy people. The sepsis group was divided into survival group and death group according to 28-day outcome. Song's study found that the plasma sTREM-1 concentration was positively correlated with APACHE II score in sepsis group, and the plasma sTREM-1 concentration could be used as a sensitive index for rapid diagnosis of abdominal sepsis [4]. The definition of acute sepsis and renal injury are consistent, and the occurrence of both cases can be identified as septic AKI. Septic AKI is the most common AKI syndrome in ICU, accounting for about half of all AKI. The pathophysiology of AKI is still unclear, but animal models and lack of histological changes suggest that septic AKI may be a functional phenomenon accompanied by microvascular shunt and tubular cell stress. Bellomo's study showed that starch containing liquids had nephrotoxicity and reduced renal function, and suggested that chlorine rich liquids may also have adverse effects on renal function. Vasoactive drugs have different effects on renal function in patients with septic AKI. Norepinephrine is the main drug, but vasopressin may also play a role. If sepsis subsides, most patients can recover their renal function. However, even one septic AKI may increase the risk of chronic kidney disease [5]. The impact of early resuscitation on the prognosis of sepsis in developing countries is unclear. Andrews studied whether early resuscitation with intravenous infusion, vasopressin, and blood transfusion reduced mortality in adult patients with sepsis and hypotension in Zambia compared with routine care. Andrew's research shows that in the case of limited resources, compared with conventional care, the use of intravenous infusion and vasopressin early resuscitation program increases the in-hospital mortality [6].

IL-5 is a small molecule cytokine produced by activated T cells and monocytes, and usually exists in the form of homodimer [7]. Mesenchymal stem cells (MSCs) have been studied as a new method to treat a variety of diseases. However, the role of placental derived mesenchymal stem cells in asthmatic children remains unclear. Based on the culture of asthmatic and non-asthmatic children, Lin evaluated the effects of placenta derived MSCs on T cell immune response and cytokine IL-5 levels. Lin's study showed that MSCs derived from placenta can inhibit the IL-5 level of different subgroups of asthmatic children, and can reduce the level of IL-5 [8]. Asthma is a very common chronic respiratory disease, about 10% of asthma patients will experience severe asthma. The new understanding of the pathogenesis of asthma provides the possibility for the development of new drugs, and also brings hope for patients with asthma. IL-5 and IL-5 receptor subunit α play an important role in the development, maturation and operation of eosinophils, and are the primary therapeutic targets of these new drugs. Patients

treated with anti-IL-5 and IL-5-r α at the same time had significantly reduced disease deterioration. Bagnasco discussed the main clinical trials of anti-IL-5 and il-5-r α in asthma and other diseases, especially the safety and efficacy results[9]. Gu evaluated the efficacy and safety of anti-IL-5 in the treatment of asthma. Gu research shows that anti-IL-5 mAb can reduce the risk of exacerbation of asthma patients, improve the quality of life of patients with good tolerance [10]. Experimental studies have shown that interleukin-5 can protect atherosclerosis by stimulating the expression of innate immunoglobulin M antibody. In Knutsson's study, Knutsson found no association between baseline IL-5 and the risk of coronary events or stroke during a 15.7 ± 6.3-year follow-up of 696 subjects randomly selected from the Malmo Diet and cancer study. However, the presence of plaques at carotid bifurcation is related to the decrease of IL-5 level. The lack of IL-5 leads to the increase of plaque development at blood flow oscillation site in mice, suggesting that IL-5 plays a protective role in plaque formation [11]. Chambliss focuses on early evidence that attempts to understand its pathogenesis through the British and Dutch hypotheses. Chambliss's study suggests that while patients with ACOS or overlapping symptoms may be an exception, overall, there seems to be more evidence to support that asthma and COPD are different processes. The treatment of eosinophils and anti-IL-5 seems to be an exciting approach, and the latest data support its application in chronic obstructive pulmonary disease [12].

The effect of IL - 5 on inflammatory response in sepsis is still unclear. Although experimental studies have found and confirmed that sepsis causes oxidative stress in vivo and ROS has a direct damage to the function of various tissues and cells, there are few clinical studies on IL - 5 on sepsis and sepsis. Therefore, this paper studies the changes of inflammatory factors in mice, and to explore the effect of IL - 5 on organ function, and to explore new methods and strategies for the treatment of sepsis.

LNTERLEUKIN-5 AND SEPSIS

Sepsis

Sepsis can be caused by any type of infection, among which bacterial infection is the most common, and there are many infection sites that induce sepsis. Because bacterial endotoxins and exotoxins can also induce sepsis, not all patients with sepsis have positive blood culture results of pathogenic microorganisms, and patients with severe diseases are often accompanied by sepsis. The main pathogens causing sepsis are Gram-negative bacteria. Sepsis caused by G-bacteria infection is not only bacteremia caused by bacteria invading blood, but also endotoxemia caused by bacterial endotoxin release. LPS is a complex of lipids and polysaccharides existing in the cell wall of G-bacteria. It is the main component of G-bacteria cell wall and has a wide range of biological activities. A large amount of LPS released from the death of G-bacteria can activate monocyte macrophages and release many bioactive substances, such as interleukin. At the same time, it can also activate coagulation and fibrinolysis system, trigger disseminated intravascular coagulation, which leads to a series of pathophysiological reactions. Lipoteichoic acid and wall acyl dipeptides on the cell wall of G + bacteria are associated with the microbial mode of disease. At the same time, another important sign of G + bacteria infection is the production of soluble exotoxins, which can cause tissue damage and activate immune response process.

When the body is stimulated to cause inflammatory response, inflammatory cells and inflammatory factors can invade into the brain through the blood-brain barrier from the peripheral, which can induce the activation of inflammation in the brain and release a large number of pro-inflammatory factors.Compared with other parts of the brain, hippocampus has a high density of inflammatory mediators receptors, which is more vulnerable to inflammatory reaction damage. Notch signal is activated by the binding of Notch receptors and ligands in two adjacent cells. The transmembrane protein of notch is hydrolyzed twice in succession. Firstly, it is hydrolyzed by tumor necrosis factor - α - invertase, and then hydrolyzed by γ - secretase. After releasing the intracellular segment of notch, it is transferred into the nucleus. Its RAM domain and ankyrin repeat sequence bind with the DNA binding protein CSL

on the nucleus to form transcription factors, the bHLH gene family includes the transcription of HES and hes related inhibitory proteins. The nuclear bHLH protein encodes its downstream target genes and regulates the occurrence of nervous system. Therefore, NiCd can be used as one of the markers of Notch signaling pathway activation. Hesland Hes5 are inhibitive bHLH transcription factors, which are highly expressed on NPCs. They inhibit neurogenesis by inhibiting proneural genes, thus inhibiting the differentiation of NPCs into neurons. On the other hand, activated b*HLH* transcription factors can promote the differentiation of neurons, while *Hesl* and *Hes5* can inhibit the activity and expression of *Mashl* and *NeuroD*. Notch signal maintains the stable state of neural stem cells self-renewal and inhibits directional differentiation; however, when Notch signal is activated, neural precursor cells can irreversibly differentiate into astrocytes.

Pathophysiological Mechanism of Sepsis

LPS is released into the blood circulation after g-bacterial digestion. After entering the blood circulation, LPS itself cannot cause a series of hemodynamic manifestations related to endotoxemia, but it will interact with the host immune system. Lipid A in the LPS structure will combine with the amino terminal of lipopolysaccharide binding protein in blood, and then the carboxyl end of LBP molecule will bind to ncdl4 to form lbp-lps-cdl4 ternary complex. After LPS stimulation, macrophages can produce a variety of cytokines, and can quickly activate cells in multiple tissues, thus causing changes in neuroendocrine and metabolism, resulting in abnormal cell function, progressive failure of tissues and organs, and eventually lead to septic shock. LPS is the source of sepsis and the key substance of septic shock.

TNF - α is a particularly important early inflammatory mediator in the process of sepsis. It plays a direct role in the occurrence and outcome of sepsis and can lead to septic shock. LPS stimulation of monocytes and macrophages can promote the rapid synthesis and release of TNF - α, and further lead to the production of other inflammatory factors, forming a cascade reaction of cytokines such as TNF - α and IL-1, and thus trigger the "waterfall effect" of inflammatory factors. These cytokines have a very complex network effect in the process of inflammatory response. The interaction of various cytokines can cause uncontrolled inflammatory reaction, even lead to shock and death. When the body has tissue damage, it will cause cell necrosis and release histone to the periphery of cells, which is called extracellular histone. Extracellular histone is significantly increased in many inflammatory diseases of sepsis, and can aggravate the damage of tissue cells and even cause death. When sepsis occurs, a variety of cell apoptosis or necrosis, chromatin degradation, and will release histone into the peripheral blood. Extracellular histone itself can also activate TLR-2 and TLR-4 receptors in TLR family located on the surface of macrophages and endothelial cells. TLR can further regulate the expression and release of inflammatory factors by activating its downstream MyD88 protein and then activating NF - κ B.

Adhesion of neutrophils to endothelial cells is an early phenomenon of acute inflammation. PMN-EC adhesion is a complex process of cell surface molecule interaction. It mainly depends on the binding of cdl1 / cdl8 on PMN membrane and ICAM-1 on EC membrane. In normal state, leukocytes adhere to endothelial cells very little. Inflammatory reactions such as ischemia-reperfusion can cause leukocytes and endothelial cells to release cytokines, which in turn stimulate endothelial cells and increase their adhesion to leukocytes. Intercellular adhesion molecule-1 (ICAM-1) is a single chain receptor type transmembrane glycoprotein composed of 507 amino acids. It is a proinflammatory cytokine in vivo, and participates in the adhesion and aggregation of various leukocytes. Its ligands on PMN are CDlla / cdl8 and cdllb / cdl8. Their main functions are to mediate the adhesion of PMN to vascular endothelial cells and epithelial cells, participate in inflammation. There was only a small amount of ICAM-1 expression in normal pulmonary microvascular endothelial cells. The direct stimulation of LPS could significantly promote the expression of ICAM-1 in PMVEC in a time and dose-dependent manner, and the change of adhesion rate of pmvec-pmn was almost

synchronous with that of ICAM-1. Endothelial cells regulate vascular reactivity by releasing vasoactive substances. No is an endogenous vasodilator secreted by vascular endothelial cells. No is produced under the action of NOS with L-arginine as substrate. There are three types of NOS, which are nerve cell-derived NOS, endothelial cell-derived NOS and inducible Nos. Endothelial cells can express two types: eNOS and iNOS. Endothelial cells in quiescent phase mainly express eNOS, which catalyzes the production of low concentration of no to relax blood vessels and inhibit platelet aggregation, which is very important for maintaining normal endothelial cells and endothelial integrity. When vessels respond to external injury and activate endothelium to produce various cytokines, eNOS expression will be reduced, while iNOS will be activated to induce high concentration of NO synthesis, this may be the last common pathway of sepsis[13].

Interleukin-5

IL-5 is mainly produced by activated Th2 cells, which is a kind of strongest eosinophil chemotactic factor. It acts on B lymphocytes, T lymphocytes, eosinophils and other cells to induce the proliferation and differentiation of B cells. After 11-2 activation, T cells can produce IL-5, eosinophils, mast cells and B cells. IL-5 is a glycoprotein composed of homodimers linked by disulfide bonds. The molecular weight of IL-5 is 40000-45000.The structure of IL-5 provides the determinants of receptor recognition and the molecular basis of signal transduction.

Interleukin - 5 is a cytokine secreted by helper T cells type 2 and type 2 innate lymphoid cells, which can regulate innate and adaptive immune responses. In bronchial asthma, allergic dermatitis and other diseases, IL - 5 activates eosinophils, promotes allergic inflammation and damages tissues. IL - 5 has a certain protective effect on diseases characterized by chronic inflammatory reaction. IL - 5 maintains the accumulation of eosinophils and anti-inflammatory macrophages in visceral adipose tissue. IL - 5 can promote B - 1 cells to secrete T15 / eo6 IgM antibody and reduce the uptake of ox LDL by macrophages, thus playing an anti-atherosclerotic role. IL-5 is a type 2 inflammatory response factor. After specific binding with IL-5 receptor α, IL-5 activates downstream pathways through receptor β subunit shared by granulocyte macrophage colony stimulating factor and IL-3.

EXPERIMENTS MATERIALS AND METHODS

Subjects and Groups

A total of 130 male C57BL / 6 mice aged 8 weeks, weighing (20 ± 3) g, were purchased from the animal center of the Provincial Academy of Medical Sciences and raised in the barrier environment of the animal house of the Provincial Academy of Medical Sciences. The mice were divided into three groups: control group (n = 20), sepsis model group (n = 55) and IL-5 group (n = 55).

Model Preparation and Statistical Analysis

Animals were bled from the retroorbital venous plexus of the mice at 3h, 6h, 12h, and 24h after the model was prepared. After 6h and 24h of the model, the cervical vertebrae of the mice were sacrificed, the right atrium was cut, and the apex of the heart was flushed with saline. After the liver was white, lung, heart and kidney tissues were taken and fixed with 4% paraformaldehyde. The lung (6h), heart (24h), and kidney (24h) tissues of 3 groups of mice were taken, the sections were dewaxed and hydrated, washed with distilled water for 5 minutes, and then stained with HE. The distilled water was rinsed and dehydrated.The lungs were observed under light microscope. Morphological changes of heart and kidney. GraphPad Prism 5.0 software was used to analyze the results. Shapiro-Wilk was used for normality test, P>0.05 was in line with normal distribution. For those who meet the normal distribution, the measurement data is represented by $(\bar{x} \pm s)$. One-way analysis of variance and Bonferroni test are used to compare each other. For those who do not meet the normal distribution, the measurement data is expressed by M (P25, P75), and the Kruskal-Wallis test and the dunns test are used to compare each other. The formulas of mean and standard deviation are as follows:

$$\mu = A_n = \frac{a_1 + a_2 + a_3 + ... + a_n}{n} (1)$$

$$\sigma = \sqrt{\frac{1}{N}\sum_{i=1}^{N}(x_i - \mu)^2}$$

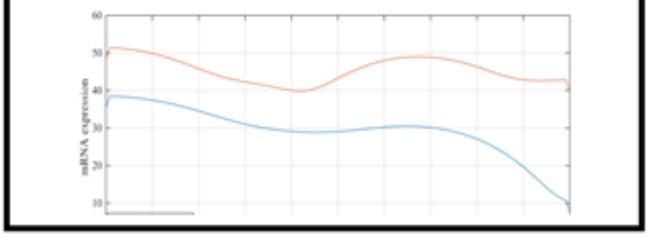
ANALYSIS OF THE PROTECTIVE EFFECT OF INTERLEUKIN-5 ON SEPSIS MICE

Analysis of Inflammatory Factor mRNA Level in Mouse Liver

In order to explore the mechanism of liver protective effect of ll-5 on sepsis mice, the expression of inflammatory factors mRNA in mice liver was determined. After 24 hours of operation, liver tissues of appropriate size were taken, and total RNA was extracted immediately after being placed in liquid nitrogen. The expression of IL-6 and TNF - α mRNA was quantitatively analyzed by reverse transcription. The mRNA expression level of inflammatory factors in liver of each group was analyzed as shown in Fiure 1.

Figure 1.

Analysis of mRNA expression levels of liver inflammatory factors in mice of each group at 24 hours after operation



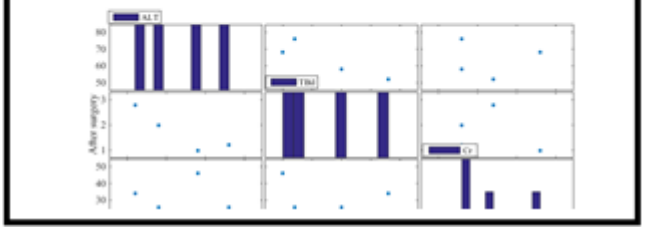
Compared with sham group and sham + rhIL-5 group, the expression of TNF - α and IL-6 mRNA in liver tissue of model group was up-regulated, and the difference was statistically significant; compared with CLP Group, the expression level of TNF - α and IL-6 mRNA in liver tissue of treatment group was down-regulated compared with CLP Group. Therefore, IL-5 can reverse CLP induced liver injury in mice with sepsis.

Comparative Analysis of the Functions of Various Organs in Mice

The comparison of alt, TBIL and Cr levels in each group was shown in Figure 2.

Figure 2.

Comparison of postoperative ALT, TBil and Cr levels of mice in each group



The results of serum CR level showed that there was no difference in serum CR level between each group and sham group. This indicated that the renal function of mice was not damaged 6 hours after sepsis. The results showed that there was no difference in serum TBIL level between the groups compared with sham group. This indicated that the liver function of mice was not damaged 12 hours after sepsis. The results of serum CK and LDH levels showed that there was no difference in serum CK level between sham group and sham group, and there was no difference in serum LDH level between each mouse and sham group. This indicated that the cardiac function of mice was not damaged 12 hours after sepsis. The results of serum Samy level showed that there was no difference in serum Samy level between the groups, which indicated that the pancreatic function of mice was not damaged 12 hours after sepsis.

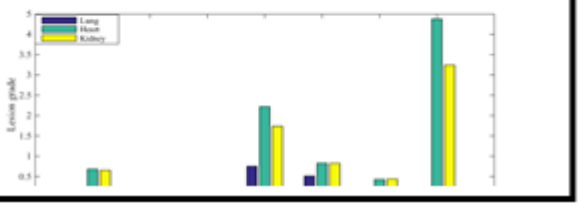
Analysis of Histopathological Changes in Three Groups of Mice

In the control group, the alveolar structure was clear, and there was no edema and inflammatory cell infiltration in the alveoli and pulmonary interstitium of the mice in the sepsis model group, a large number of inflammatory cells were found in the alveoli and pulmonary interstitium of the mice in the sepsis model group, with obvious congestion and edema of the alveoli and pulmonary interstitium, and the alveolar deformation and stenosis; while the degree of inflammatory cell infiltration and edema in the alveolar and pulmonary interstitium of the mice in the IL-5 intervention group was reduced, The degree of alveolar deformation and stenosis was reduced, and the histological structure was clearer in sepsis group. In the control group, the arrangement of myocardial fibers was normal, without

degeneration and inflammatory cell infiltration; in the sepsis model group, the arrangement of myocardial fibers was obviously disordered, with moderate degeneration of cells, interstitial edema and inflammatory cell infiltration. In the control group, the renal parenchyma structure was normal; in the sepsis model group, the renal parenchyma was slightly disordered, with glomerular tubular congestion and renal tubular epithelial cells swelling; in the IL-5 intervention group, the degree of glomerular renal tubular congestion was reduced. The grading results of lung, heart and kidney lesions in each group were shown in Figure 3.

Figure 3.

Lung, heart and kidney lesion grading results of mice in each group



In sepsis mice, it was found that IL-5 receptor α was expressed on the surface of neutrophils and monocytes. IL-5 could enhance the phagocytosis and viability of macrophages, thus enhancing the clearance of pathogenic microorganisms. These changes were also verified in patients with sepsis, and it was found that plasma IL-5 levels were higher in patients with sepsis than those who died. This effect of IL-5 is consistent with the study that GM-CSF can improve monocyte function in sepsis patients and sepsis animal models, suggesting that IL-5 and GM-CSF share the same signaling pathway. GM-CSF down regulated toll like receptor 1,2,4 in human monocytes in a time and dose-dependent manner. In addition, IL-4 and IL-13, which are both type 2 inflammatory response factors, can promote the conversion of macrophages to anti-inflammatory macrophages, inhibit the inflammatory reaction, and thus play a protective role in sepsis. Therefore, it is speculated that IL-5 may inhibit the inflammatory reaction in sepsis.

Serum Levels of cTnI, SCr, BUN, TNF - α, IL-6, IL-1 β and CXCL1 in the Three Groups were Measured

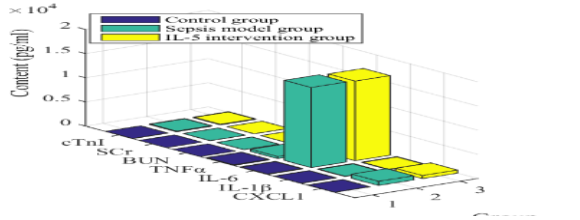
The serum levels of cTnI, SCr, BUN, TNF - α, IL-6, IL-1 β and CXCL1 in the three groups are shown in Table 2.

Table 1.
Serum cTnI, SCr, BUN, TNF-α, IL-6, IL-1β, CXCL1 levels in three groups of mice

	Control group	Sepsis model group	IL-5 intervention group
cTnI (pg/ml)	37.84±9.57	136.04±37.84	130.5±21.14
SCr (μmol/L)	8.65±0.54	14.32±0.57	14.35±0.56
BUN (mmol/L)	7.02±0.54	13.14±1.62	12.72±0.56
TNFα (pg/ml)	150.4±21.01	443.9±11.29	347.5±56.72
IL-6 (pg/ml)	102.5±2.49	16583±453.8	16476±806.6
IL-1β (pg/ml)	45.76±23.14	85.39±4.65	87.49±15.52
CXCL1 (pg/ml)	41.38±19.89	875.7±160.4	648.5±148.2

Compared with the control group, the serum cTnI, SCr and BUN in the sepsis model group were significantly increased at 3 h, 6 h, 12 h and 24 h; compared with the sepsis model group, the serum cTnI, SCr and BUN in the IL-5 intervention group were significantly decreased at 24 h, but there was no significant difference among the 3 h, 6 h and 12 h 2 groups. The serum levels of cTnI, SCr, bun, TNF - α, IL-6, IL-1 β and CXCL1 in the three groups were analyzed as shown in Figure 4.

Figure 4.
Analysis of serum cTnI, SCr, BUN, TNF-α, IL-6, IL-1β, CXCL1 levels in three groups of mice



Compared with the control group, the levels of

TNF α, IL-6, IL-1 β and CXCL1 in the sepsis model group were significantly higher than those in the control group at 3 h and 6 h, IL-1 β at 3 h and 12 h, and CXCL1 at all time points. Compared with the sepsis model group, the serum TNF - α of IL-5 intervention group was significantly decreased at 3 h, and CXCL1 was significantly decreased at 12 h. There was no significant difference between the two groups at other time points.

In the early stage of sepsis, most of the inflammatory factors and chemokines in blood and tissue usually reach the peak at 1 h ~ 3 h, some inflammatory factors reach the peak at 6 h ~ 12 h, and then gradually decrease.

CONCLUSIONS

Histopathology showed that a large number of inflammatory cells were infiltrated into the alveoli and pulmonary interstitium of mice in sepsis model group. After the intervention of IL-5, the above pathological changes were alleviated, and the levels of cTnI, SCR and BUN in serum were decreased.

The number of deaths in 7 days of IL-5 intervention group was less than that in sepsis model group. IL-5 has a protective effect on sepsis induced liver injury in mice, and the mechanism may be to reduce the production of inflammatory factors in the liver of septic mice. It is the first time that IL-5 can up regulate liver MDSCs and reverse sepsis induced liver injury through this mechanism.

This study found that IL - 5 can reduce the inflammatory reaction of septic mice, and has protective effect on sepsis mice. The anti-inflammatory mechanism of IL - 5 is not clear. Due to the limited research time, its mechanism cannot be explained by the current research results. Further research is expected to be more detailed combined with in vivo and in vitro experiments.

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