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ORIGINAL RESEARCH

Dynamic changes of serum cytokines in acute paraquat poisoning and changes in patients' immune function

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Abstract

Acute paraquat poisoning is due to the extremely severe toxicity of paraquat. After paraquat enters the human body, it will cause rapid changes in the human body system. Since paraquat poisoning will quickly invade the organs of the whole body, it may cause damage to the functions of multiple organs in the poisoned patient. The liver organ is the most important detoxification site for the human body, so the damage to the liver of the patient is more obvious. This article discovers and observes the structure of paraquat and the dynamic changes of serum cytokines in patients with paraquat poisoning through the clinical phenomenon of paraquat poisoning, and the related changes of human serum cells after the subjects took paraquat and the changes of cell dynamic factors after different doses of paraquat entered the human body were analysed. At the same time, the changes in the immune function of the body of different groups of people were also observed. The experimental results in this article show that according to the intake of paraquat, the severity of poisoning patients will be mild, moderate, severe and outbreak poisoning. Among them, the dose for adults who cannot be treated for prognosis is 10 ml.

1 | INTRODUCTION

The increase of paraquat (PQ) poisoning patients in recent years is mostly due to the emergence of accidental or self-administration. The lack of effective treatments after poisoning can lead to high mortality. Therefore, this article explores the changes in the immune function of patients with PQ poisoning through the study of paraquat and determines the changes in serum adhesion factors and cytokines in patients with paraquat poisoning. After being taken orally, it will quickly damage the human lungs and other organs, and then cause damage to the human body's various systems. In this article, we mainly observe the dynamic changes of serum factors after acute paraquat poisoning and the changes of the patient's immune function, then conduct clinical examinations on the patients, and prove the factors of paraquat poisoning by comparing them with health examiners.

Signs of fibrosis in the lungs of patients with paraquat poisoning and the obvious increase of neutralising cytokines in the patients' serum indicate that adhesion factors and cytokines are closely related to the process of pulmonary fibrosis caused by paraquat poisoning. In short, a significant increase in serum and cytokine levels in patients with paraquat poisoning may form a complex system, and this system plays an important role in the process of paraquat-induced pulmonary fibrosis. Therefore, when applying this theory to the study of acute paraquat, it is necessary to monitor the immune function of patients during clinical treatment and the combined use of inhibitors, especially after acute paraquat poisoning. Then, in order to promote the rapid recovery of patients, the risks and benefits of blood purification treatment are fully evaluated in combination with the patients' psychological, social, economic and other factors, so as to formulate a more reasonable blood purification treatment plan. In this regard, Wang J conducted a prospective cohort study design, collecting peripheral blood samples in the experimental centre, and then using flow cytometry to detect the expression of lymphocytes to compare the dynamic changes of cellular immune function at different time points after injury and the differences between different groups' differences and the relevance of acute physiology and chronic health assessment were assessed. Finally, by observing the dynamic changes of the immune function of the serum

cells of different poisoned patients and evaluating the effects of different drugs in it [1]. At the same time, Liu J Q conducted a case study while studying the dynamic changes of serum cytokines in acute paraquat poisoning and related changes in the patient's immune function. Through the comparison between suspended patients and patients who have been cured by radical cure, the coagulation function and the changes of lymphocytes during the operation period and the clinical significance were detected and discussed. He finally concluded the changes in blood coagulation function, cytokines and T lymphocytes of patients after paraquat poisoning and their clinical significance [2]. For related research on different cells, Zhang C explored the clinical significance of cytokines in acute graft-versus-host disease (aGVHD), and he detected the concentration of 8 cytokines and 19 conventional biochemical markers in the serum of aGVHD and non-GVHD patients during the allogeneic haematopoiesis. He also used 27 indicators to build a predictive model and validated the model through prospective experiments. Since most of these tests have low specificity for aGVHD, it is easy to ignore these changes in clinical work. However, by combining cytokine and biochemical tests, he established a prediction model that can greatly improve the ability of these biomarkers to predict the development of aGVHD one or two weeks in advance [3]. Not only that, Guojun studied the key role of mediated immune damage in the pathogenesis of hepatitis B virus-related acute and chronic liver failure (HBV-ACLF). He believes that in view of the high short-term mortality and the critical role of T cells in disease progression, it is necessary to study the dynamics of T cell cloning during HBV-ACLF. His ultimate goal is to study longitudinally the dynamic changes of T-cell receptor β chain library composition and perturbation, and to determine whether the T-cell receptor library characteristics are related to the results of HBV-ACLF patients, and then finally apply it in clinical medicine [4]. Regarding cell research, Huang M observed the dynamic changes of lymphocyte subsets in patients with acute paraquat poisoning by flow cytometry, trying to explore their role in pathogenesis and their impact on prognosis. Through the absolute counts and subgroups of peripheral blood lymphocytes in the acute phase, it was found that with the recovery of immune function, the fluctuation of the improved group rose to a normal level; the T lymphocyte count of the unimproved group remained at a low level or even continued to decline. Finally, the T lymphocyte count and its subgroups can be used to monitor immune function and predict the prognosis of patients with acute paraquat poisoning [5]. In addition, Rose A measures the genomic microbial community through the relevant observations of bacteria, and then uses the functional gene array to measure the changes of circulating genes. From this he found that the multi-directional comparison of the multifunctional group found different trends in time and space for different genes that perform the same cyclic action [6]. Then, Kisito's classification model based on conditional inference trees allows us to determine the main influencing factors. He conducts a clinical analysis on the dynamic changes of human serum cytokines and the changes of patients' immune function through the driving of professional activities and the related application of knowledge of physiology and biology; and to provide theoretical knowledge on the basis of the effective value while improving the understanding of usefulness and to obtain experimental data under the bidirectional premise of practice and cognition [7]. In addition, Diachenko-Bohun M found that with the deterioration of the quality of life, mental, physical and social human obstacles manifest the following aspects: reduce the level of adaptation to daily psychological and physical stress, affect the early development of neurosis, and cause children and adults disease. In this regard, one of the main strategies for the development of educational institutions is to organise health care activities. Under such circumstances, it is becoming more and more important to train future biological teachers to prepare for the implementation of medical and healthcare technologies in professional activities. He believes that the development of the theory and method basis of medical conservation activities and the need for demonstration determine the relevance of a given problem, and it is necessary to deeply reflect on the nature and content of the implementation of medical conservation technology education [8]. The different discourses about the study mainly focus on the introduction of paraquat poisoning, the clinical manifestations of the patients and the immune function. However, most of the content is about lymphocytes and other cells, and there is not too much research on serum cells, which makes the content of serum cells and clinical manifestations of patients with acute paraquat poisoning not convincing enough.

The innovation of this article is (1) By observing the changes of adhesion molecules and cytokines in the serum of patients with paraquat poisoning, timely serum collection for different cases of paraquat poisoning patients. Then, through timely detection of the patient's serum cytokine levels and dynamic changes, the patient's human body functions such as the lungs are functionally studied, and finally the patient's whole body observation is carried out. (2) By analysing the clinical data of surviving patients with paraquat poisoning and observing the dynamic changes of lung function and other indicators, and according to the current clinical analysis, there is no way to treat the current oral dose of paraquat poisoning patients at 10 ml and above. This article combines theory with practice, making the research more practical and guiding.

2 | THE DYNAMIC CHANGES OF SERUM CYTOKINES IN ACUTE PARAQUAT POISONING AND THE PATIENT'S IMMUNE KINETIC ENERGY

2.1 | Acute paraquat poisoning

The chemical composition of paraquat (PQ) is dimethyl, the chemical name of paraquat is 1-1-dimethyl-4-4-bipyridyl cation salt, the main component is 1,1′dimethyl 4,4′dichlorodipyridine, and its main component is dichloride and bipyridine methyl sulphate salt, which has a very strong effect in weeding and has a wide range of applications worldwide. It is also because of the

high efficiency in this aspect that its strong toxicity is also very harmful to the human body. After acute paraquat poisoning, most of the functions of multiple organs of the human body, such as fibrosis, will decline. Due to its strong toxicity, the clinical course of the disease is progressing rapidly, which will lead to an extremely high fatality rate and there is no effective antidote at present [9].

(1) Biochemical reaction equation

Symptoms of nausea and vomiting can occur early after paraquat poisoning, which is not significantly different from other poisons. Therefore, early diagnosis cannot be made through clinical manifestations. For patients who have been poisoned, it is necessary to ask the patient's history of exposure to poisons in detail and strive for early diagnosis and treatment.

In the process of constructing the paraquat model, we mainly used several typical biochemical reaction types to study this kind of reaction. The first is the process of the mass reaction equation for the combination and separation of different molecules:

$$X + Y \underset{K_1 \bullet K_2}{\longleftrightarrow} M + N \tag{1}$$

where X and Y represent the substrate of the reaction, M and N represent the product of the reaction, and then K_1 and 2 represent the kinetic parameters of the reaction and the kinetic parameters of the reverse reaction, respectively [10].

In addition to the above-mentioned biochemical reaction, there is also a Michaelis-Menten equation to describe a process promoted by enzymes as catalysts for different reactions:

$$E + P \underset{}{\overset{K_1 \bullet K_2}{\longleftrightarrow}} PS \overset{K_3}{\rightarrow} E + S \tag{2}$$

In the above formula, E represents an enzyme as a catalyst, P represents a substrate, Photoshop represents a complex of enzyme and substrate, and S represents a product of a reaction. Among them, K_1, K_2 and K_3 represent a kind of kinetic parameter of enzyme-promoted reaction.

According to the above-mentioned two kinds of reaction processes with respect to molecules, it is still insufficient. Finally, a transition equation about the qualitative state can be used to describe the transport of different molecules between the cytoplasm and the nucleus and a transition process of other molecular states:

$$A_s \xrightarrow{K_s P} A_N \tag{3}$$

Briefly introduced three kinds of chemical reactions between different molecules can form an interactive model for operation, by observing the transformation of equation (2) while the enzyme promotes the reaction, different experimental results can be recorded in time for comparison and observation when two different reactions occur. In the process of these three interactions, the transduction process between different signals can be realised, and the different movements of the molecules can be better realised.

(2) Paraquat reaction process

Paraquat causes the generation of a large number of active oxygen free radicals in the body [11, 12], which will cause oxidation reactions after entering the human body, and then damage the tissues of the human body to a certain extent. Especially the oxidation reaction of the lung tissue, and then due to the cascading reaction, it is also easy to stimulate the release of inflammatory cells in the body's functional tissues, etc. For the understanding of paraquat, it is not easy to volatilise, it will take effect very quickly after contact with the soil, and it has almost no side effects on the environment. The molecular structure of paraquat is shown in Figure 1:

Figure 1 shows the chemical structure of paraquat. When studying the process of the interaction between the various molecules of the cell after the acute paraquat enters the human function, it is necessary to record the data of the dosage of the drug. In its interaction with protein molecules, the equilibrium theoretical formula of the solution in which each molecule is fused can be calculated [13], and then the binding point constant of the interaction between various molecules can be obtained by the following formula:

$$m = \sum_{i=1}^{r} \frac{n_i K_i [S_g]}{1 + K_i [S_g]} \tag{4}$$

In equation (4), m represents the number of moles of small molecules that can be bound by the total moles of albumin macromolecules, $[S_g]$ represents the concentration of free small molecules, and i refers to the binding constant. Then the K_i at point i represents the binding site, which can be calculated by linear sum.

However, if there is only one binding site for the small drug molecule on the protein, that is, $i = 1$, then the formula (4) can be simplified as:

$$\frac{m}{S_g} = nK - mK \tag{5}$$

In formula (5), m represents the number of moles of small molecules bound by protein macromolecules [14]. $[S_g]$ represents the total concentration of various small molecules, and the binding constant is K. By using this equation, the concentration of the free ligand in the reaction system can be obtained. When the concentration of different cells in the human body is known, the patient's clinical data can be analysed in time to observe the dynamic changes of serum cytokines.

In short, subarachnoid hemorrhage treatment has obvious differences in the effects of paraquat poisoning and normal samples on the DNA stability maintenance mechanism. The disordered Double Data Rate SDRAM response to the samples of poisoned patients may cause the accumulation of DNA damage and the increase of genomic instability, which will

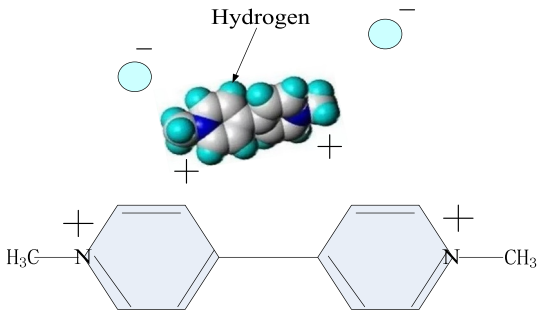


FIGURE 1 Molecular structure of paraquat

affect many downstream related pathways and open the irreversible apoptosis process. As shown in Figure 2:

According to Figure 2, the oxidative damage and the apoptosis of lung epithelial tissue after paraquat poisoning can be observed, and then the apoptosis-related genes can be regulated through the endoplasmic reticulum stress pathway in clinical patients.

(3) Fluorescence study of cell molecular movement

Because in fluorescence experiments, it is usually susceptible to interference from other lights, some interferences can be eliminated by using the modified Stern–Volmer equation:

$$\frac{S_0}{\Delta S} = \frac{1}{fK[Q]} + \frac{1}{f} \tag{6}$$

In the formula, the relative binding strength of protein macromolecules is S_0, the difference in the relative binding strength of protein macromolecules is ΔS, and the effective constant is K. Then the linear relationship is obtained by inputting the concentration and value of different molecules.

In the process of interaction between protein macromolecules and small drug molecules, through the dynamic changes of small molecules, the modified formula for obtaining the binding constant under this condition is:

$$\frac{1}{\Delta S} = \frac{1}{\Delta S_{max}} + \left(\frac{1}{K[e]}\right)\left(\frac{1}{S_{max}}\right) \tag{7}$$

In the above formula, $\Delta S_{max} = S_\infty - S_0$, $\Delta S = S_x - S_0$, and the initial dynamic change of protein macromolecules is expressed by S_0.

2.2 | Dynamic changes of serum cytokines caused by acute paraquat poisoning

(1) Changes in serum cytokines

Protein macromolecules and small drug molecules are non-covalently bound [15, 16]. Regardless of the magnitude of the force between the molecules, they are all interacting. Among them, the thermodynamic parameters that play a vital role in the binding force of protein and drug molecules mainly include enthalpy change (ΔH), entropy change (ΔS) and so on.

At a certain reaction temperature, the relationship between the reaction binding constant and the change of free energy ΔG in the system is expressed as:

$$\frac{\Delta G}{K} = -RLogK_i \tag{8}$$

Under the condition of equal pressure, the partial differentiation is obtained:

$$\left(\frac{\partial\left(\frac{\Delta G}{K}\right)}{\partial K}\right)_i = -R\left(\frac{\partial LogK_i}{\partial K}\right)_i \tag{9}$$

According to the Gibbs–Helmholtz equation:

$$\left(\frac{\partial\left(\frac{\Delta G}{K}\right)}{\partial K}\right)_i = \frac{\Delta H}{K^2} \tag{10}$$

We get the following equation:

$$\left(\frac{\partial LogK_i}{\partial K}\right)_i = \frac{\Delta H}{RK^2} \tag{11}$$

It can be seen from the equation that for the endothermic reaction of $\Delta H > 0$, K_i will increase as the temperature rises; conversely, K_i will decrease as the temperature rises. The above equation expresses the rate of change of the logarithm of the binding constant with temperature. By integrating them, two relations of binding constants at different temperatures can be obtained. After simplification, it is found that if the thermal effect ΔH of the reaction equal pressure does not change with temperature; or the temperature interval is not much different, and the equal pressure heating effect also changes very little, it can be ignored; or the average thermal effect of the two temperatures is used, and the integral Time ΔH can be regarded as a constant:

$$\int_{K_1}^{K_2} d\ln K = \frac{\Delta H}{R}\int_i^1 \frac{dT}{T^2} \tag{12}$$

$$Ln\frac{K_2}{K_1} = \frac{\Delta H}{R}\left(\frac{1-i}{i}\right) \tag{13}$$

Using the above two formulas, the thermal effect value of the reaction can be obtained through the binding constant values at different temperatures, and then the indefinite integral of the above formula can be obtained:

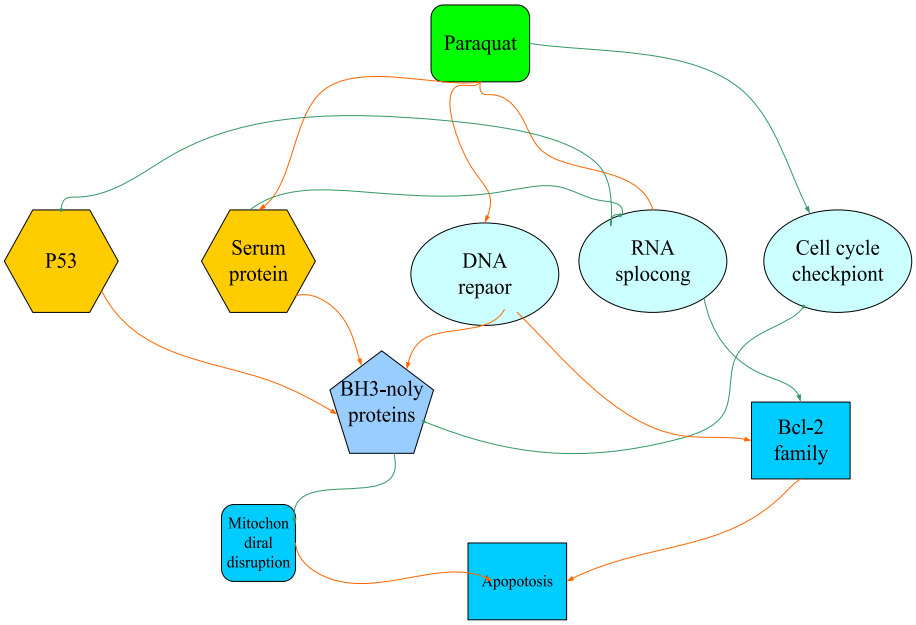


FIGURE 2 Model structure diagram of apoptosis induction mechanism after paraquat poisoning

$$\mathrm{LnK_i} = \frac{\Delta H}{R}\left(\frac{1}{K}\right) + C \tag{14}$$

It can be seen from the equation that the value of the above constant will become a linear relationship under the condition that the thermal effect ΔH of the isobaric reaction does not change with temperature [17]. And refer to the relevant occupational disease diagnostic standards to formulate paraquat poisoning diagnostic grading standards, as shown in Table 1.

The main clinical manifestations in Table 1 analyse the related content of different indexes of mild, moderate and severe for rapid semi-quantitative urine and gastrointestinal symptoms, and different dose changes need to be considered.

(2) Mechanism of acute paraquat

On the one hand, acute paraquat induces an increase in protein expression by activating serum protein factors, which causes an increase in intracellular calcium ions and an increase in cyclin expression, and promotes the transition in different phases [18]. On the other hand, acute paraquat causes an increase in the production of calcium ions, which may cause the break of the protease double chain. In order to perform related inspections and repairs, a large amount of broken DNA cannot be repaired effectively, which will eventually lead to cell death. In addition, the acute paraquat treatment and the inhibition of cyclin expression may have other regulatory mechanisms involved. For example, the mechanism of serum protein stimulating cell cycle regulation and cell death after acute paraquat poisoning is shown in Figure 3:

Through the signal of the cell molecule, and then transform it, we can get:

$$\mathrm{K}\left[\mathrm{p}_1(t)\right] = \frac{1}{\pi}\int_{+\infty}^{\infty}\frac{p_1(t)}{t^{\prime}-t}dt \tag{15}$$

Then the transformed part of the formula can get a simplified function

$$Z_i(t) = C_i(t) + jH[C_1(t)] \tag{16}$$

Among them, the instantaneous amplitude and phase [19, 20] are:

$$A_i(t) = C_i^2(t) + H^2[C_i(t)] \tag{17}$$

$$\theta_i(t) = \arctan\frac{H[C_i(t)]}{C_i(t)} \tag{18}$$

The function expression after deriving the formula is $\omega_i(t) = \frac{d\theta_i(t)}{dt}$. After performing the transformation on all serum cytokines, the initial dynamic changes of the cells can be expressed as:

$$\mathrm{Z(t)} = RK\sum_{i=1}^{n} a_i(t)e^{\theta_i(t)} \tag{19}$$

The amplitude and frequency obtained by the transformation of dynamic cytokines [21] are all functions of time change. Then the obtained series of changed data are arranged continuously, and the time function of the amplitude obtained in a certain period of time is:

$$\mathrm{k}(\omega) = \int_0^T K(\omega, t)dt \tag{20}$$

TABLE 1 Diagnosis and grading standard of paraquat poisoning

Performance	Mild poisoning	Moderate poisoning	Severe addiction
Quick semi-quantitative urine	<10μg/ml	10–30μg/ml	>30μg/ml
Gastrointestinal symptoms	+	++	++++
GB225-2013	Acute mild	Acute moderate	Acute severe
GBZ73-2010		Chemical pneumonia	Respiratory distress syndrome, etc.
GBZ77-2009			Poisoning or severe liver disease
GBZ59-2002			Multiple organ dysfunction syndrome

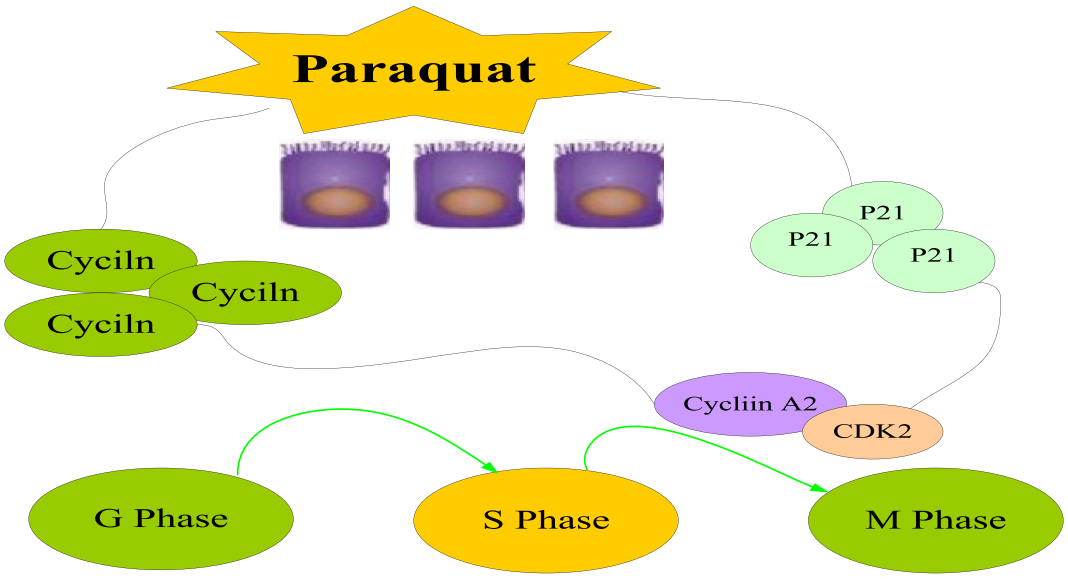


FIGURE 3 Cell cycle regulation after acute paraquat stimulation

2.3 | Acute paraquat poisoning and changes in the Patient's immune function

(1) The effect of blood purification methods on patients with paraquat poisoning

After paraquat enters the human body, it is mainly through the circulation of blood to transmit the dynamic changes of various cells to various parts of the body, and then how to clean up the effects of paraquat poisoning patients is mainly through blood purification. The toxic substances in the blood are metabolised by using the adsorption effect of the drug during the blood transfer process. This method is mainly used in the treatment of acute poisoning. Because it can not only adsorb protein macromolecules but also take into account the binding of some plasma proteins, it is widely used in the treatment of acute poisoning caused by poisons and drugs.

The dose of poison is an important factor affecting the prognosis of patients with paraquat poisoning. The concentration of paraquat in the blood is directly positively correlated with the dose of poison, and it has long been used as an important indicator for predicting the prognosis of poisoned patients. An oral dose of 30–40 mg/kg can cause death. According to research, an adult of 60 kg only needs to take 10 ml of poison to be fatal. Relevant doctors suggest that the oral dose of paraquat be used as the standard for assessing the severity of poisoning. At the same time, when the oral dose of paraquat reaches more than 50 ml, neither conventional treatment nor combined with blood purification treatment [22, 23] cannot improve the patient's prognosis.

PQ in the acute phase often causes the death of epithelial cells, causing serious tissue and organ damage. In order to further study the mechanisms involved in PQ causing cell death, and to clarify the types of cell death caused by PQ. The treatment of human bronchial epithelial cells with increasing doses of PQ showed that as the dose of PQ increased, the toxicity of PQ to cells increased significantly, and the lactate dehydrogenase assay showed that the release of lactate dehydrogenase [24] increased significantly; in addition, the cell proliferation caused by PQ was also significantly slowed down, and the measurement showed that the number of viable cells decreased in a dose-dependent manner. As shown in Figure 4:

(2) Changes in immune function of patients with acute paraquat poisoning

In current clinical studies, related observations on the cellular immune function of patients with acute paraquat

poisoning have found that poisoned patients will produce a large number of oxidative free radicals in the body [25]. It will produce very toxic free radicals after a biochemical reaction in the human body, and after oxidation reaction, it will produce a large amount of cytokines, and finally activate the immune function of the living cells in the human body. The next research is to try to determine whether the cell cycle arrest and cell death induced by PQ are related to the production of reactive oxygen species (ROS) in the cell and the conduction of calcium signals, as shown in Figure 5.

The mechanism of acute paraquat poisoning on the human body may behave as follows: oxygen free radical theory, free radical is an intermediate product of normal metabolism, and its reaction ability is very strong, which can oxidise many substances in cells and damage biofilm. It can also cross-link large molecules such as proteins and nucleic acids, affecting their normal functions. Paraquat damages human organs and tissues because it can induce the production of a large number of free radicals, which leads to organs and tissues lesions, with lung lesions being the most serious. It is precisely because of paraquat that the poisoning mechanism of various organ damages may involve more complicated aspects and it is limited by the medical level of today's society. The pathological mechanism of paraquat poisoning on the human body is still not completely clear, so clinically, no specific antidote for paraquat has been developed. This is why the mortality rate of paraquat poisoning patients has been high. As shown in Figure 6.

After the patients are treated, the levels of various elements in the serum of paraquat poisoning patients will be higher than that of the control group to a certain extent. Respiratory failure caused by pulmonary fibrosis is the main cause of death from paraquat poisoning. Current research believes that the product metabolised by paraquat by reducing coenzyme [26] acts as an electron acceptor and is actively transported into the cells by type I and type II alveolar cells, which will trigger lipid peroxidation and mitochondrial damage. As shown in Figure 7.

(3) Clinical significance in patients with acute paraquat poisoning

Various organs of patients after acute paraquat poisoning will be damaged, which will cause damage between tissues in various organs of the human body, and will rapidly deteriorate among other organs in a short period of time. In terms of clinical treatment, there are few very precise drugs that can provide fundamental treatments and provide good preventive measures. Therefore, after patients take this kind of paraquat, it will cause a high probability of death. The main discussion of this study is to analyse the clinical significance of patients with acute paraquat poisoning [27, 28]. Mainly observe the changes in the relationship between the lungs and the poisoned patients in different periods, as shown in Figure 8.

Paraquat patients usually spread the toxicity to all parts of the human body through oral administration, and after entering the oral cavity, paraquat is absorbed by the human body. However, how to determine a clear poisoning phenomenon has not been found in the clinic. After passing through the digestive system and central nervous system, it enters the body of the human body and destroys the internal functions of the patient's body, resulting in changes in the patient's biochemical indicators and fibrosis [29], and these factors are exactly the cause of the death of the patient. Of course, after taking paraquat into the human body, there will be changes in related cells, as shown in Figure 9.

For the above-mentioned related studies, it is known that patients with acute paraquat poisoning can destroy all body functions. At the same time, it also leads to the decline of the body's immune function, especially the related destruction of serum protein dynamic factors, which will affect the functional damage of the lungs and related livers to a certain extent. Real-time dynamic monitoring of immune function during the treatment of patients, and then related immunosuppressive therapy can promote the patient's early recovery to a relatively large extent.

3 | EXPERIMENT AND ANALYSIS

3.1 | Acute paraquat poisoning

Based on laboratory examinations and observations of the lungs after paraquat poisoning, combined with clinical manifestations and various relevant diagnostic criteria, formulate diagnostic grading standards for paraquat poisoning, and formulate diagnostic grading standards for paraquat poisoning with reference to relevant occupational disease diagnostic standards. The details are shown in Table 1 in Section 2.2.

After the analysis of the above table, it can be seen that after acute paraquat poisoning, the body system will be damaged in many ways, especially some damages to the lungs are very worth recording.

Then according to the physical load tolerance, the common pulmonary dysfunction can be pulmonary ventilation dysfunction and diffusion dysfunction [30], and then the degree of classification can be seen in Table 2.

In Table 2, first, the related effects of different lungs are mainly pulmonary ventilation dysfunction and diffusion dysfunction. According to research data, it is found that a very small amount of paraquat poisoning has almost no effect on the lungs, but a slightly large amount of paraquat poisoning can cause serious damage to the lungs. The symptoms are: expectoration, blood in sputum, difficulty breathing, shock and even death; diffuse fibrosis of the lungs caused by paraquat, even if actively treated, it is still incurable, and the damage to the lungs is irreversible.

3.2 | Clinical situation of acute paraquat poisoning

Among the 48 surviving patients of acute paraquat poisoning by oral administration, 34 were males and 30 were females, aged 16–55 years old, with an average age of (21.9 ± 10.32)

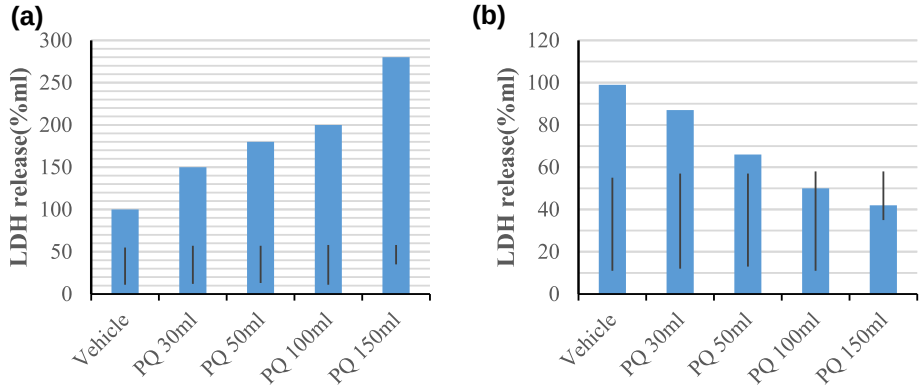


FIGURE 4 Participation mechanism of death caused by PQ

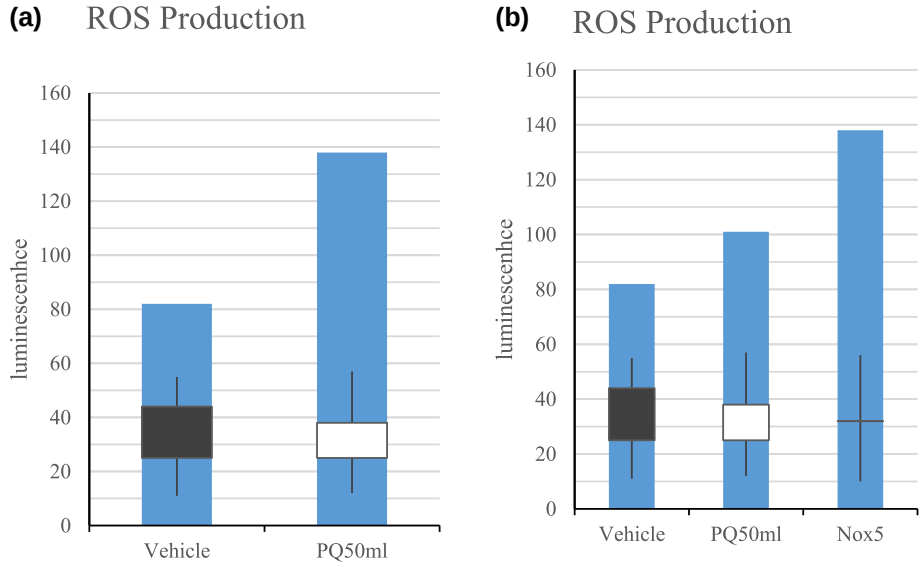


FIGURE 5 Production of extracellular reactive oxygen species (ROS) after PQ treatment

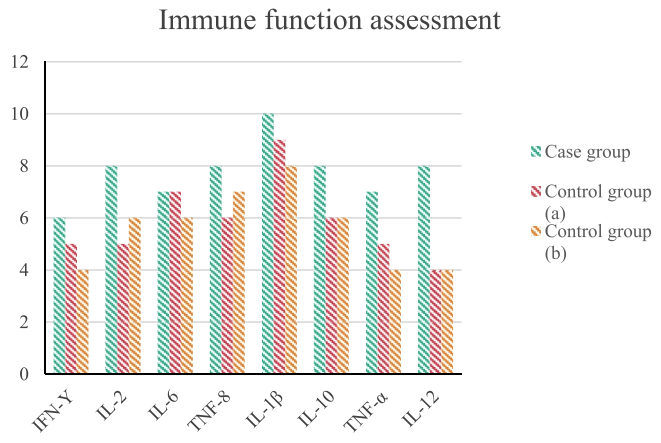


FIGURE 6 Serum cytokine expression levels in patients with acute paraquat poisoning and healthy controls

years old, with a dose of 10–100 ml and an average dose of 30–50 ml; survival at 18 weeks is the standard, including 10 mildly poisoned patients, 6 males, and 4 females, aged 15–45 years old, with an average age of (25.33 ± 10.12) years old, with a dose of 10–40 ml and an average dose of 20 ml; there were 38 severely poisoned patients, including 23 males and 15 females. Aged 16–59 years old, with an average age of (28.36 ± 12.12) years old, the dose of poison is 50–100 ml, with an average of 60 ml. The specific situation can be seen in Table 3.

For the relevant studies of different poisoned patients, the factors of different age groups should be considered. The age structure of paraquat poisoned patients is shown in Figure 10:

According to Figure 10, it can be seen that the prevalence of acute paraquat poisoning is the young and middle-aged population, and most of the population is concentrated in the population between 35 and 45, which can also be seen that they take more in the oral dose., and it can be seen from the

figure that the dose taken is between 8 and 10. In a survey report on rural women's family pressure and its influencing factors, it was pointed out that compared to urban areas, rural women have a relatively low level of education, and they not only undertake the agricultural production of the family, the upbringing and care of their children and the elderly. At the same time, they are also facing the pressure of being lonely and the risk of being illegally violated. As a result, the psychological burden, mental pressure, and the pressure of raising and supporting such people are significantly increased, and they are prone to some excessive behaviours or negative emotions.

However, for patients with different levels of intoxication, how clinical decision-makers can formulate a reasonable haemoperfusion programme with the best medical economics effect is currently not a unified conclusion at home and abroad. Most scholars believe that the focus of choosing a reasonable blood purification programme is to assess the severity of poisoning. A survey found through further data analysis that patients with severe poisoning had significantly better effects of haemoperfusion therapy than those in the conventional drug treatment group. Because they took a smaller amount of paraquat, conventional treatment could take effect, and blood poisoning concentration is low, and it is difficult to cause greater damage to various organs. However, some patients tend to be in serious condition and take a larger amount of paraquat. The blood poison concentration is higher, and the speed of absorption of the poison into the blood and the speed of diffusion into the organs and tissues exceeds the speed of haemoperfusion to remove the poison. As a result, the poison is rapidly distributed in the spaces between various organs and tissues, causing serious damage to the lungs, liver, kidneys and other important organs of the human body, and even death due to functional failure. A retrospective analysis of paraquat poisoning patients showed that when the blood concentration

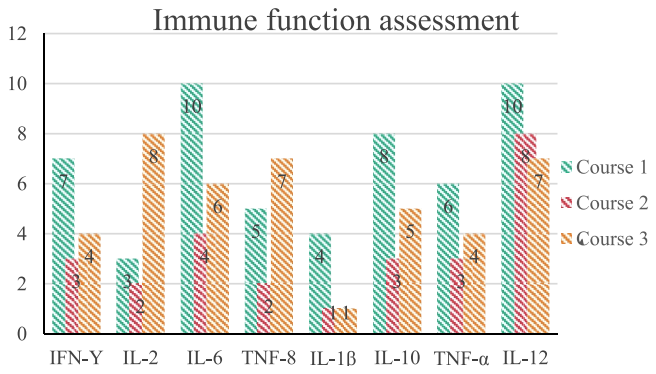


FIGURE 7 Change trend of serum factors in patients with paraquat poisoning who benefited from chemotherapy after receiving chemotherapy

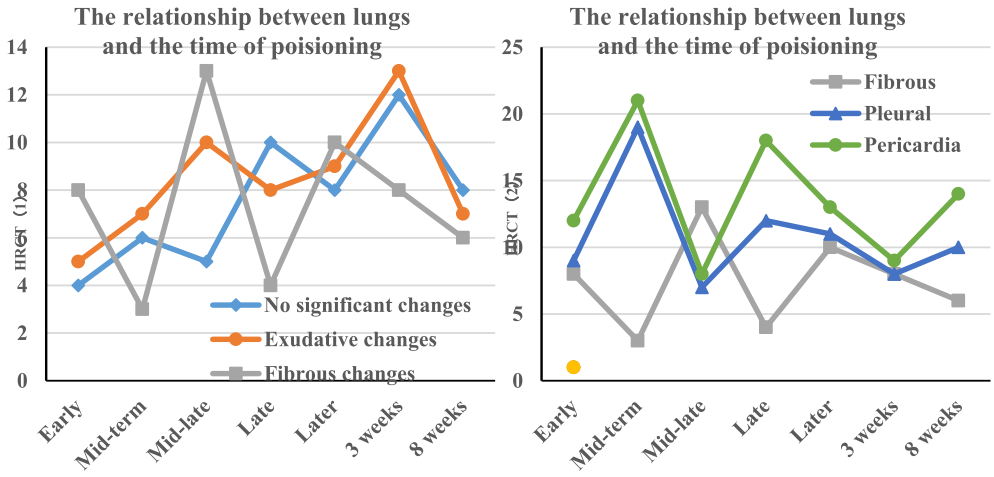


FIGURE 8 Time comparison between lungs and acute paraquat poisoning

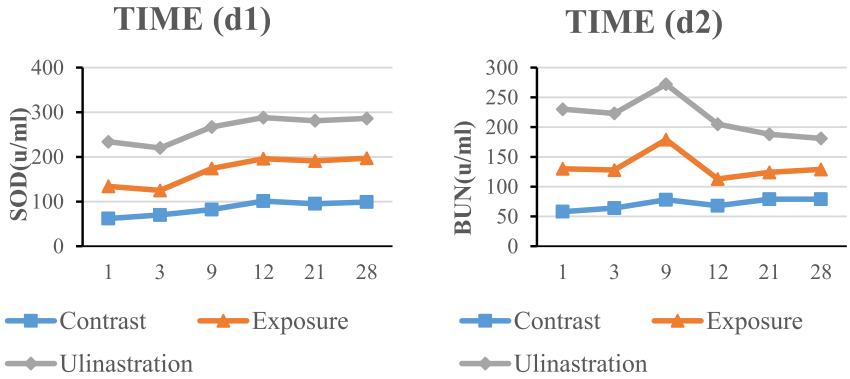


FIGURE 9 Changes in cell molecules taking different doses of paraquat

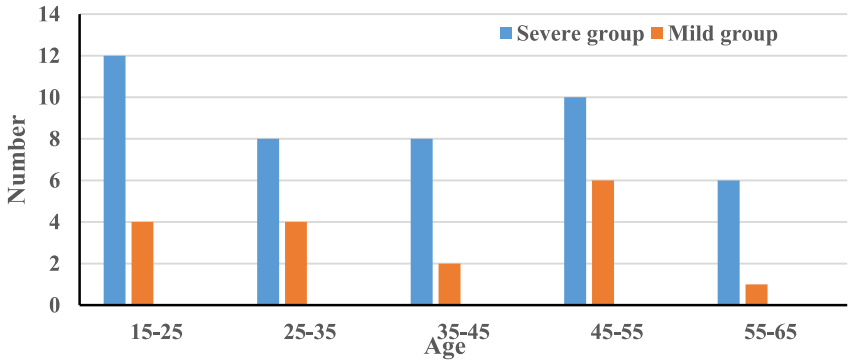
TABLE 2 Types of lung dysfunction and detection indicators

Types of lung damage	Reason	Detection indicator
	Restrictive	Lung volume expansion is limited, causing lung volume to decrease
Pulmonary ventilation		Airway obstruction or narrow airway causes gas
Obstacle	Obstructive	Traffic drop
	Hybrid	Both
Diffuse dysfunction		Diffusion function decreased

TABLE 3 Basic situation of the number of observers

Project poisoning classification	Male	Female	Average age (years)	Average dose of poison (ml)
Mild	6	4	25.33 ± 10.12	20
Severe	23	15	28.36 ± 12.12	60

FIGURE 10 Age structure of paraquat poisoned persons



of paraquat in the body is very high, no matter what blood purification method is used, the long-term mortality rate of patients cannot be reduced. Therefore, although haemoperfusion has gradually become a conventional treatment for paraquat poisoning, its curative effects are quite different for patients with different severity of poisoning. Only by developing an individualised haemoperfusion plan for the patient can the patient get the most benefit.

3.3 | The effects of acute paraquat poisoning on the body function of patients

Paraquat (PQ) is quickly distributed throughout the body after oral administration and is toxic to all organs of the body, so its toxicity is multi-system, multi-organ and multi-level.

PQ interferes with the redox reaction system of cells in the body, thereby producing ROS (reactive oxygen molecules) harmful to tissues, and then destroying the body's cell defence mechanisms to produce ROS (reactive oxygen molecules) harmful to tissues. It causes damage and necrosis of corresponding organs and tissues, remodelling of alveolar structure, and finally pulmonary fibrosis. Paraquat is characterised by chronic toxicity. The early clinical manifestations of paraquat poisoning patients are not specific, so it is difficult for clinicians to type it immediately. Some people make a classification based on the amount of paraquat drunk and the test results, as shown in Table 4.

As can be seen in Table 4, for different doses of paraquat, the damage to various structures inside the patient's body is different, including mild, poisoning and no treatment. Basically, when there is more than 10 ml, it will cause very serious damage, and there is no way to treat the patient.

Most patients in the acute phase tend to lose the protective mechanism formed on the cell membrane surface due to severe fibrotic oxidative biochemical reactions. When it actively absorbs paraquat in the blood, it will eventually lead to death through a series of oxidation reactions.

4 | DISCUSSION

Paraquat is usually used for weeding in people's daily life. Because it is very toxic and volatilises quickly when it comes into contact with the land, it is popular among people. However, it is precisely because of its strong toxicity that after oral administration, it will quickly spread into the human body and

TABLE 4 Types of paraquat poisoning

Degree of poisoning	Toxic dose	Clinical symptoms	Lung injury	Kidney function	Liver and spleen function	Prognosis
Mild	5ml	No or light	No or light	No or light	No or light	Can be restored
Moderate to severe	5–10 ml	Have	Have	Have	Have	Partial survival
Burst type	>10 ml	Have	Have	Have	Have	All dead

damage the liver and spleen and other organs. The main cause of death is that the patient's lungs will undergo severe fibrotic lesions, which leads to the decline of the patient's immune function. Another possibility is to treat the patient with immunosuppressive agents, which inhibits the function of immune cells to varying degrees and reduces the secretion function. For the study of the dynamic changes of serum cytokines in patients with acute paraquat poisoning and the immune function of patients, it can be found that the effects of different doses and different subjects are different. How to give timely treatment after discovering poisoned patients is of course very important, but there is no clinical experience that can be completely cured so far, so continuous efforts are needed to improve the probability of curing patients.

5 | CONCLUSIONS

Paraquat poisoning is a very serious problem, and it is not realistic to completely disable it. However, it is very important to prevent paraquat poisoning incidents and provide treatment assistance when the disease is discovered. Paraquat poisoning has no specific antidote. The treatment includes removing the poison and reducing the absorption of the poison. It can be treated by inducing vomiting, cleaning, gastric lavage, removing poisons, catharsis, and even blood purification, followed by anti-toxic and anti-oxidant therapy, and finally treating complications such as lung infection and organ damage. This article first introduces paraquat, and then studies the dynamic changes of serum cells and immune function in patients with paraquat poisoning based on the clinical analysis of different paraquat poisoning patients. Since paraquat enters the human body, it will quickly cause the decline of the lungs and liver, which will lead to the decline of the body's immune function. Therefore, the immunotherapy plan can be grasped by observing the dynamic changes of immune cells, and then the cure rate can be improved for the patient's recovery in the later treatment.

CONFLICT OF INTEREST

The author declares that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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