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Therapeutic Efficacy of Reduced Glutathione in Emergency Treatment of Organophosphorus Pesticide Poisoning and Its Impact on ALT, AST, CRP, and IL-6 Levels

Yuezhou Wang*

Lianyungang Municipal Oriental Hospital, Lianyungang 222042, Jiangsu Province, China

***Corresponding author:** Yuezhou Wang, 672400503@qq.com

Abstract: *Objective:* To analyze the efficacy of reduced glutathione (GSH) in the emergency treatment of patients with organophosphorus pesticide poisoning (AOPP). *Methods:* A total of 100 AOPP patients admitted to the emergency department between January 2022 and January 2024 were selected and randomly divided into two groups. The observation group (n = 50) received GSH combined with conventional treatment, while the reference group (n = 50) received conventional treatment alone. The overall treatment efficacy, serum indicators, and adverse reaction rates were compared. *Results:* The observation group exhibited a higher overall treatment efficacy compared to the reference group ($P < 0.05$). Post-treatment, serum indicator levels in the observation group were lower than those in the reference group, and the adverse reaction rate was also lower in the observation group ($P < 0.05$). *Conclusion:* GSH can improve the overall treatment efficacy in AOPP patients, protect liver function, reduce inflammatory responses in the body, and minimize post-treatment adverse effects, thus accelerating recovery and demonstrating significant therapeutic advantages.

Keywords: Reduced glutathione; Organophosphorus pesticide poisoning; Emergency treatment; Therapeutic efficacy

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1. Introduction

Organophosphorus pesticide poisoning (AOPP) refers to the inhalation or contact with pesticides containing high levels of phosphorus-containing organic compounds. Symptoms include mental disorders, excessive sweating, and respiratory paralysis. AOPP is predominantly acute, with a rapidly progressive course and a high risk of mortality, necessitating prompt emergency treatment [1]. Common emergency treatments include administering pralidoxime and atropine, along with early gastric lavage, to slow disease progression, eliminate toxins, and reduce disease severity. However, these treatments do not adequately protect liver function or

reverse liver damage and inflammatory processes, resulting in a suboptimal prognosis.

Therefore, this study utilized reduced glutathione (GSH) as a treatment, which can counteract free radical generation, protect liver and kidney function, detoxify effectively, and enhance clinical outcomes. Given the high feasibility of this therapy, 100 AOPP patients were included to evaluate the efficacy of GSH.

2. Materials and methods

2.1. General information

A total of 100 patients with acute organophosphorus pesticide poisoning (AOPP) treated in the emergency department between January 2022 and January 2024 were selected and randomly divided into two groups. The observation group included 50 patients (26 males and 24 females) aged 25–75 years, with a mean age of 54.18 ± 3.95 years. The time from poisoning to admission ranged from 1 to 4 hours, with a mean time of 1.58 ± 0.61 hours. The reference group also included 50 patients (28 males and 22 females) aged 23–74 years, with a mean age of 54.92 ± 4.14 years. The time from poisoning to admission ranged from 1 to 3 hours, with a mean time of 1.53 ± 0.58 hours. No significant differences were observed in baseline characteristics between the two groups ($P > 0.05$).

Inclusion criteria: Patients met the diagnostic criteria for AOPP as outlined in the "Guidelines for the Diagnosis and Differential Diagnosis of Acute Organophosphorus Pesticide Poisoning" [2]; exhibited typical symptoms such as abdominal pain, generalized muscle spasms, and nausea; were diagnosed with AOPP based on cholinesterase activity and toxicological tests; had complete baseline data; and provided informed consent to participate.

Exclusion criteria: Patients with infectious diseases such as AIDS or syphilis; allergic to emergency medications; had coagulation disorders; were diagnosed with severe comorbidities such as malignant tumors; or withdrew during the study.

2.2. Methods

Reference group: Patients received standard treatment:

(1) Gastric lavage: A gastric tube was inserted, and 10–20 liters of clean water were infused to wash the stomach until the gastric effluent was clear.

(2) Atropine treatment: Patients were administered atropine sulfate injection (Jilin Jibang Pharmaceutical Co., Ltd., National Drug Approval Number H20053923, 1 mL: 5 mg). The initial dose was 5 mg via intravenous injection, followed by additional injections of 1–5 mg every 10 minutes until signs of atropinization appeared.

(3) Pralidoxime therapy: Patients received pralidoxime iodine (Tianjin Pharmaceutical Group Xinzheng Co., Ltd., National Drug Approval Number H20066022, 20 mL: 0.5 g). The initial dose was 1.0 g via intravenous injection, followed by a supplementary dose of 0.5 g every 12 hours.

Observation group: Patients received standard treatment combined with GSH therapy. Reduced glutathione (GSH) for injection (Chongqing Yaoyou Pharmaceutical Co., Ltd., National Drug Approval Number H20051600, 2.0 g) was administered at a dose of 2.0 g mixed in 100 mL of normal saline via intravenous infusion once daily.

The treatment duration for both groups was 7 days.

2.3. Observation indicators

(1) Serological markers: Venous blood samples (5 mL) were collected before treatment and on the 7th day of treatment. Samples were centrifuged for 10 minutes at 3000 rpm. Serum alanine transaminase (ALT) and aspartate transaminase (AST) levels were measured using an automatic biochemical analyzer. C-reactive protein (CRP) was determined using an immunoturbidimetric method, and interleukin-6 (IL-6) levels were measured using an enzyme-linked immunosorbent assay (ELISA).

(2) Adverse reaction rate: The incidence of intermediate syndrome, respiratory depression, and pulmonary edema was recorded.

2.4. Efficacy evaluation criteria

(1) Markedly effective: Poisoning symptoms completely resolved after 7 days of treatment, and ALT and AST levels returned to normal.

(2) Effective: Poisoning symptoms alleviated after 7 days of treatment, with ALT and AST levels reduced by > 50%.

(3) Ineffective: No improvement in poisoning symptoms after 7 days of treatment, with ALT and AST levels reduced by ≤ 50% or increased.

2.5. Statistical analysis

Data were processed using SPSS 28.0 software. Measurement data were expressed as mean ± standard deviation (SD) and analyzed using *t*-tests. Count data were expressed as frequencies and percentages [*n* (%)] and analyzed using χ^2 tests. A *P*-value < 0.05 was considered statistically significant.

3. Results

3.1. Comparison of overall treatment efficacy between the two groups

Table 1 shows that the overall treatment efficacy in the observation group was significantly higher than in the reference group ($P < 0.05$).

Table 1. Comparison of overall treatment efficacy between the two groups [*n* (%)]

Group	Cases	Markedly effective	Effective	Ineffective	Overall effective rate
Observation	50	29	18	3	47 (94%)
Reference	50	25	15	10	40 (80%)
χ^2	-	-	-	-	4.332
P	-	-	-	-	0.037

3.2. Comparison of serological indicators between the two groups

Before treatment, there were no significant differences in serological indicators between the two groups ($P > 0.05$). After 7 days of treatment, the levels of serological indicators in the observation group were significantly lower than those in the reference group ($P < 0.05$), as shown in **Table 2**.

Table 2. Comparison of serological indicators between the two groups before and after treatment (mean ± SD)

Group	Cases	ALT (U/L)		AST (U/L)		CRP (ng/mL)		IL-6 (pg/mL)	
		Before	After	Before	After	Before	After	Before	After
Observation	50	79.84 ± 6.91	49.18 ± 4.32	76.95 ± 6.86	48.95 ± 4.11	16.35 ± 2.98	3.77 ± 0.52	117.35 ± 19.42	62.48 ± 5.11
Reference	50	79.92 ± 7.12	61.77 ± 4.83	77.15 ± 6.91	59.84 ± 4.18	16.42 ± 2.93	6.48 ± 1.47	116.84 ± 20.37	67.94 ± 5.83
t	-	0.057	13.738	0.145	13.136	0.118	12.290	0.128	4.980
P	-	0.955	0.000	0.885	0.000	0.906	0.000	0.898	0.000

3.3. Comparison of adverse reaction rates between the two groups

Table 3 shows that the adverse reaction rate in the observation group was significantly lower than that in the reference group ($P < 0.05$).

Table 3. Comparison of adverse reaction rates between the two groups [*n* (%)]

Group	Cases	Intermediate syndrome	Respiratory depression	Pulmonary edema	Incidence rate
Observation	50	1	1	0	2 (4%)
Reference	50	3	4	1	8 (16%)
χ^2	-	-	-	-	4.000
P	-	-	-	-	0.046

4. Discussion

AOPP is a common condition in emergency medicine. It occurs when organophosphate pesticides enter the body via oral ingestion, respiratory tract, or skin, binding tightly with cholinesterase to produce phosphorylated cholinesterase. This process reduces the bioactivity of acetylcholinesterase, inhibits its breakdown capability, increases acetylcholine levels at neuromuscular and synaptic junctions, and enhances cholinergic excitation, leading to pronounced neurological symptoms [3]. Early symptoms of AOPP include miosis and confusion, while disease progression can result in respiratory failure and sudden death. Current emergency treatments, such as gastric lavage, help flush out pesticide residues from the stomach, preventing systemic absorption and reducing the toxic effects on various organs [4]. Atropine administration can act on muscarinic receptors to dilate blood vessels, relieve smooth muscle spasms, improve blood circulation, and shorten recovery time. Pralidoxime iodine can bind rapidly to organophosphate compounds in pesticides, preventing excessive inhibition of cholinesterase and restoring enzyme activity, thereby alleviating respiratory muscle paralysis and promoting toxin elimination. These measures stabilize the condition and alleviate symptoms but have limitations in protecting liver and kidney function.

GSH is present in the cytoplasm and participates in redox processes, maintaining cellular health. Under normal conditions, the body can continuously synthesize GSH, which has strong antioxidant properties [5]. However, during AOPP, endogenous GSH is insufficient to meet detoxification demands, necessitating exogenous supplementation. GSH promotes the inactivation of reactive oxygen species (ROS) by binding with free radicals, reducing oxidative stress, and mitigating organ damage caused by pesticide components.

Exogenous GSH improves metabolic function, increases GSH levels, and enhances enzyme activation. It binds to ROS and aldehydes in the liver, preventing excessive lipid peroxidation, modulating oxidative stress markers, and protecting liver cells from damage by improving their membrane stability and repairing injured hepatocytes [6].

As a naturally synthesized peptide, GSH contains abundant sulfhydryl groups and is widely distributed in body tissues to maintain cellular activity. It acts as a cofactor for enzymes involved in glycolysis and oxidative metabolism, binding with free radicals to generate harmless compounds, repair tissue damage, and enhance detoxification efficacy [7]. The results showed an overall treatment efficacy of 94.0% in the observation group compared to 80.0% in the reference group ($P < 0.05$), consistent with findings by Xie [4], indicating the reliability of this study.

AOPP causes pesticides to target liver tissue, resulting in significant hepatocyte damage, increased membrane permeability, and the release of ALT and AST into the plasma. Myocardial injury caused by poisoning can also elevate serum ALT levels [8]. CRP, an acute-phase protein, increases in response to inflammation triggered by poisoning, while IL-6 is a sensitive early marker of infection, both of which rise significantly during systemic inflammation [9]. GSH exerts potent free radical scavenging and detoxifying effects, forming low-toxicity compounds with a high metabolic rate that facilitates pesticide elimination. This reduces oxidative stress, cell membrane damage, and ALT/AST levels [10]. GSH also possesses strong anti-inflammatory properties, suppressing inflammatory mediators and alleviating systemic inflammation, thereby reducing CRP and IL-6 levels. In this study, the observation group showed significantly lower levels of all serological markers after 7 days of treatment compared to the reference group ($P < 0.05$), demonstrating the ability of GSH to improve liver function and mitigate inflammatory responses.

GSH exhibits stable pharmacokinetics, high bioavailability, sustained therapeutic effects, and minimal cumulative toxicity with prolonged use, resulting in fewer adverse reactions. The observation group had a lower adverse reaction rate than the reference group ($P < 0.05$), supporting these findings.

5. Conclusion

In summary, GSH provides effective treatment for AOPP by reducing ALT and CRP levels, minimizing adverse effects, and improving overall therapeutic outcomes, thereby supporting better clinical recovery.

Disclosure statement

The author declares no conflict of interest.

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