

Effect of Ultra-Early Hemoperfusion on Emergency Treatment Outcomes in Patients with Severe Organophosphate Pesticide Poisoning

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Abstract: *Objective:* To analyze the emergency treatment effect of ultra-early hemoperfusion (HP) in patients with severe organophosphate pesticide poisoning (SOPP). *Methods:* Sixty SOPP patients treated in the emergency department between January 2022 and January 2024 were randomly divided into two groups using a random number table. The observation group (30 cases) received ultra-early HP treatment, while the reference group (30 cases) received conventional HP treatment initiated 6 hours post-poisoning. The groups were compared in terms of overall emergency efficacy, clinical indicators, serological markers, inflammatory factors, and complication rates. *Results:* The observation group had a higher total efficacy rate than the reference group, superior clinical indicators, and a lower complication rate ($P < 0.05$). After 24 hours of emergency treatment, serological markers and inflammatory factor levels in the observation group were lower than those in the reference group ($P < 0.05$). *Conclusion:* Ultra-early HP treatment provides better emergency outcomes for SOPP patients by shortening treatment time, improving serological markers and inflammatory factor levels, and offering higher safety. It demonstrates significant advantages in emergency care.

Keywords: Ultra-early hemoperfusion; Severe organophosphate pesticide poisoning; Emergency treatment outcomes

Online publication: February 17, 2025

1. Introduction

Organophosphate pesticides are rich in phosphorus elements, effective for pest control, and widely used in agricultural production. These pesticides are highly volatile, and their components can enter the body via the respiratory tract or skin and mucous membranes, leading to poisoning symptoms. Severe organophosphate pesticide poisoning (SOPP) represents a critical condition with a high mortality rate, often accompanied by complications and poor prognosis^[1]. Gastric lavage, catharsis, and anti-infection therapy constitute the basic treatments, alleviating poisoning symptoms and slowing disease progression. Combined hemoperfusion (HP) can

clear blood toxins, reduce pesticide residues in the body, purify the blood, and prevent multiple organ damage. However, the optimal timing for initiating HP remains controversial. Many scholars suggest that ultra-early HP initiation improves emergency efficiency, prevents adverse events related to the disease, and enhances clinical outcomes. Therefore, this study selected 60 SOPP patients to evaluate the emergency treatment efficacy of ultra-early HP.

2. Materials and methods

2.1. General information

A total of 60 patients with severe organophosphate pesticide poisoning (SOPP) admitted to the emergency department between January 2022 and January 2024 were included. They were randomly divided into two groups using a random number table. The observation group (30 cases) consisted of 19 males and 11 females, aged 34–76 years, with an average age of 52.65 ± 3.79 years. The reference group (30 cases) included 18 males and 12 females, aged 32–78 years, with an average age of 52.79 ± 3.80 years. There were no significant differences in baseline data between the two groups ($P > 0.05$).

Inclusion criteria: Diagnosed with organophosphate pesticide poisoning based on Practical Internal Medicine [2]; classified as severe; met the indications for HP treatment; poisoning-to-treatment time < 24 hours; complete clinical data; fully cooperative with emergency treatment.

Exclusion criteria: Coexisting heart, liver, or kidney diseases; immune diseases; hemoglobin ≤ 60 g/L or diastolic blood pressure ≤ 70 mmHg after fluid resuscitation; vegetative state; voluntary withdrawal of treatment by the patient or family.

2.2. Methods

Both groups received the same basic treatments, including gastric lavage with warm water or clean water until the lavage fluid was clear and garlic odor-free. Skin and hair were washed with soap water. Patients were given 250 mL of 25% mannitol (produced by Guangdong Nanguo Pharmaceutical, National Drug Approval Number H20103532) orally for catharsis. Early, adequate administration of atropine (produced by Jilin Jibang Pharmaceutical, National Drug Approval Number H20053923) was initiated with a dose of 5–10 mg every 5–10 minutes. The dosage was reduced or stopped 6 hours after symptom relief. Patients with respiratory failure underwent tracheal intubation and mechanical ventilation, with ventilator parameters adjusted based on their condition. Symptomatic treatments, including anti-infection, fluid replacement, and correction of electrolyte imbalances, were administered. Pralidoxime iodide (produced by Shanghai Huaihai Pharmaceutical Factory, National Drug Approval Number H31021788) was intravenously infused at an initial dose of 1.2–1.6 g, with a maximum of 2 g per hour and < 10 g per 24 hours, at an infusion rate of 0.4 g/h. The dosage was reduced or stopped after 6 hours of symptom relief.

The reference group began HP treatment 6 hours after poisoning, while the observation group initiated HP immediately upon admission to the emergency department. A hemoperfusion machine (model JF-800A, produced by Jianfan Biological Technology, Zhuhai) and disposable hemoperfusion cartridges (model YTS-200, produced by Aier Medical Technology, Langfang) were used. The femoral vein was punctured, and the hemoperfusion machine and cartridge were connected, with a blood flow rate of 150–200 mL/min. Heparin sodium injection (produced by Shandong Lukang Chenyang Pharmaceutical, National Drug Approval Number H20043156) was

administered at an initial dose of 100 mL, followed by a maintenance dose of 6–8 mL per hour. Heparin sodium was discontinued 1 hour before the end of HP. Each HP session lasted 90–120 minutes, followed by a 12–24 hour pause, for a total treatment duration of 72 hours.

2.3. Observation indicators

- (1) Clinical indicators: Observations included atropine dosage, time to cholinesterase normalization, time to regain consciousness, and hospitalization duration.
- (2) Complications: The incidence of intermediate syndrome, pulmonary edema, respiratory failure, cerebral edema, and gastrointestinal bleeding was recorded.
- (3) Serological indicators: Venous blood samples (5 mL, fasting) were collected before emergency treatment and 24 hours afterward. Samples were centrifuged for 10 minutes at 3000 r/min, and an automatic biochemical analyzer (model Hitachi 3100, produced by Hitachi, Japan) was used to measure the following: (a) Alanine aminotransferase (ALT); (b) Amylase (AMS); (c) Cardiac troponin I (cTnI).
- (4) Inflammatory factors: Venous blood samples collected at the same time points were analyzed using enzyme-linked immunosorbent assays (ELISA) to measure the following: (a) Interleukin-6 (IL-6); (b) C-reactive protein (CRP); (c) Transforming growth factor-beta1 (TGF-β1).

2.4. Criteria for efficacy evaluation

- (1) Significant efficacy: Chest X-rays show no exudation, lung consolidation, or fibrosis; uniform density; all blood indicators are normal.
- (2) Partial efficacy: Chest X-rays show lung interstitial changes; blood indicators are basically normal.
- (3) No efficacy: Chest X-rays show exudation, lung interstitial changes, and fibrosis; blood indicators are significantly abnormal.

2.5. Statistical analysis

Data were processed using SPSS 28.0. Measurement data were expressed as mean ± standard deviation (SD) and analyzed using *t*-tests. Count data were expressed as numbers and percentages [*n* (%)] and analyzed using χ^2 tests. Statistical significance was set at $P < 0.05$.

3. Results

3.1. Comparison of overall emergency treatment efficacy

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

Table 1. Comparison of overall emergency treatment efficacy [*n* (%)]

| Group | <i>n</i> | Significant efficacy | Partial efficacy | No efficacy | Total efficacy (%) |
|-------------------|----------|----------------------|------------------|-------------|--------------------|
| Observation group | 30 | 14 | 13 | 3 | 27 (90.00%) |
| Reference group | 30 | 9 | 10 | 11 | 19 (63.33%) |
| χ^2 | | | | | 5.963 |
| <i>P</i> | | | | | 0.015 |

3.2. Comparison of clinical indicators

The observation group showed significantly better clinical indicators, including lower atropine dosage, shorter time to cholinesterase normalization, time to regain consciousness, and hospitalization duration compared to the reference group ($P < 0.05$), as shown in **Table 2**.

Table 2. Comparison of clinical indicators (mean \pm SD)

| Group | <i>n</i> | Atropine dosage (mL) | Cholinesterase normalization time (days) | Time to regain consciousness (days) | Hospitalization duration (days) |
|-------------------|----------|----------------------|--|-------------------------------------|---------------------------------|
| Observation group | 30 | 201.65 \pm 19.75 | 5.41 \pm 0.97 | 5.20 \pm 0.76 | 10.75 \pm 1.36 |
| Reference group | 30 | 315.29 \pm 23.44 | 8.04 \pm 1.53 | 7.83 \pm 1.48 | 15.29 \pm 2.24 |
| <i>t</i> | | 20.307 | 7.952 | 8.658 | 9.489 |
| <i>P</i> | | < 0.001 | < 0.001 | < 0.001 | < 0.001 |

3.3. Comparison of complication rates

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

Table 3. Comparison of complication rates [*n* (%)]

| Group | <i>n</i> | Intermediate syndrome | Pulmonary edema | Respiratory failure | Cerebral edema | Gastrointestinal bleeding | Incidence (%) |
|-------------------|----------|-----------------------|-----------------|---------------------|----------------|---------------------------|---------------|
| Observation group | 30 | 0 | 1 | 1 | 0 | 1 | 3 (10.00%) |
| Reference group | 30 | 1 | 3 | 3 | 1 | 2 | 10 (33.33%) |
| χ^2 | | | | | | | 4.812 |
| <i>P</i> | | | | | | | 0.028 |

3.4. Comparison of serological indicators

Before emergency treatment, there was no significant difference in serological indicators between the two groups ($P > 0.05$). After 24 hours of emergency treatment, the observation group had significantly lower levels of ALT, AMS, and cTnI than the reference group ($P < 0.05$), as shown in **Table 4**.

Table 4. Comparison of serological indicators (mean \pm SD)

| Group | <i>n</i> | ALT (IU/L) | | AMS (U/L) | | cTnI (ng/mL) | |
|-------------------|----------|----------------------------|------------------------------------|----------------------------|------------------------------------|----------------------------|------------------------------------|
| | | Before emergency treatment | 24 hours after emergency treatment | Before emergency treatment | 24 hours after emergency treatment | Before emergency treatment | 24 hours after emergency treatment |
| Observation group | 30 | 65.75 \pm 6.84 | 31.55 \pm 4.02 | 188.65 \pm 17.92 | 132.06 \pm 14.51 | 17.92 \pm 2.35 | 4.69 \pm 0.51 |
| Reference group | 30 | 65.19 \pm 6.91 | 39.42 \pm 4.87 | 187.53 \pm 19.02 | 150.63 \pm 16.77 | 17.88 \pm 2.43 | 6.70 \pm 0.76 |
| <i>t</i> | | 0.315 | 6.826 | 0.235 | 4.587 | 0.065 | 12.029 |
| <i>P</i> | | 0.754 | < 0.001 | 0.815 | < 0.001 | 0.949 | < 0.001 |

3.5. Comparison of inflammatory factors

Before emergency treatment, there was no significant difference in inflammatory factors between the two groups ($P > 0.05$). After 24 hours of emergency treatment, the levels of IL-6, CRP, and TGF- β 1 in the observation group were significantly lower than those in the reference group ($P < 0.05$), as shown in **Table 5**.

Table 5. Comparison of inflammatory factors (mean \pm SD)

| Group | <i>n</i> | IL-6 (pg/mL) | | CRP (mg/L) | | TGF- β 1 (ng/L) | |
|-------------------|----------|----------------------------|------------------------------------|----------------------------|------------------------------------|----------------------------|------------------------------------|
| | | Before emergency treatment | 24 hours after emergency treatment | Before emergency treatment | 24 hours after emergency treatment | Before emergency treatment | 24 hours after emergency treatment |
| Observation group | 30 | 151.65 \pm 19.77 | 75.45 \pm 6.92 | 81.55 \pm 9.12 | 45.09 \pm 4.67 | 3,315.62 \pm 186.41 | 762.56 \pm 37.84 |
| Reference group | 30 | 150.86 \pm 20.34 | 85.11 \pm 7.16 | 81.49 \pm 9.05 | 50.49 \pm 5.13 | 3,314.95 \pm 189.60 | 855.42 \pm 40.74 |
| <i>t</i> | | 0.153 | 5.314 | 0.026 | 4.263 | 0.014 | 9.147 |
| <i>P</i> | | 0.879 | < 0.001 | 0.980 | < 0.001 | 0.989 | < 0.001 |

4. Discussion

SOPP is one of the critical conditions encountered in emergency departments, characterized by symptoms such as dyspnea, abnormal blood pressure, and tachycardia, often accompanied by arrhythmias and multiple organ damage [2]. The primary routes of poisoning include inhalation, skin contact, and ingestion. Inhalation poisoning typically manifests as eye redness and breathing difficulties; skin contact poisoning as erythema, blisters, and a burning sensation; and ingestion poisoning as nausea and vomiting. This condition progresses rapidly, affecting multiple systems in a short time and increasing the risk of mortality.

Emergency treatments, including gastric lavage, catharsis, and detoxification, are the main approaches for SOPP patients. These methods remove acetylcholine from the body and alleviate poisoning symptoms but cannot eliminate organophosphorus compounds and toxins, leading to frequent complications. HP is an effective therapy for SOPP, leveraging the strong adsorptive properties of activated charcoal to remove organophosphorus compounds and efficiently clear blood toxins via systemic circulation, thus purifying the blood [3]. Ultra-early HP can shorten the onset time of emergency treatment, prevent further absorption of organophosphorus pesticide components by organs, and reduce the risks associated with SOPP.

This study shows that the overall emergency treatment efficacy in the observation group was higher than in the reference group. The observation group also required less atropine, and had shorter cholinesterase normalization time, consciousness recovery time, and hospitalization duration than the reference group ($P < 0.05$). The primary reason for this is that ultra-early HP treatment effectively removes toxic substances. The adsorbents used in HP, including resin materials and activated charcoal, have strong adsorption and lipophilic properties, allowing them to bind with large proteins, adsorb cytokines, inflammatory factors, and immune complexes, thereby protecting tissues and organs and improving emergency treatment efficacy [4]. Following ultra-early HP treatment, the poisoning symptoms in patients were significantly alleviated, cholinesterase levels and consciousness states recovered promptly, and treatment cycles were shortened.

The complication rate in the observation group was lower than in the reference group ($P < 0.05$). The primary

reason is that ultra-early HP treatment is highly proactive. It can be performed simultaneously with emergency treatments like gastric lavage or detoxification, preventing organophosphorus compounds from entering multiple organ tissues through the bloodstream. This inhibits the binding of organophosphorus to cholinesterase, reducing phosphorylated cholinesterase levels in the body, lowering acetylcholine content, and thereby preventing complications.

ALT is a sensitive indicator for evaluating liver function and predicting the extent of liver damage caused by organophosphorus pesticides. AMS is secreted by the salivary and pancreatic glands, and in cases of poisoning, significant spasms in intestinal smooth muscles can lead to pancreatic duct obstruction, thereby increasing AMS levels^[5]. cTnI is a common marker of myocardial injury, used to assess the extent of myocardial tissue damage caused by organophosphorus pesticides. After ultra-early HP treatment, the serological indicators (ALT, AMS, and cTnI) in the observation group were significantly lower than those in the reference group ($P < 0.05$). The main reason is that HP can adsorb both free and lipid- or protein-bound organophosphorus compounds, reducing pesticide-related damage to the central nervous system, alleviating respiratory muscle paralysis, and mitigating damage to cardiac and hepatic tissues. This also prevents pancreatic duct obstruction and lowers serological indicator levels^[6].

SOPP activates the monocyte-lymphocyte system, increasing the release of inflammatory factors and inducing systemic inflammatory response syndrome. The results show that after ultra-early HP treatment, the inflammatory factors in the observation group were significantly lower than those in the reference group ($P < 0.05$). The primary reason is that this treatment uses extracorporeal circulation to promptly remove exogenous toxins and efficiently adsorb inflammatory factors, preventing the systemic spread of inflammatory mediators through the blood system and effectively mitigating the inflammatory response.

5. Conclusion

In conclusion, ultra-early HP treatment is highly effective for SOPP. It enhances emergency treatment efficiency, improves patients' physiological functions, and leads to better prognoses.

Disclosure statement

The author declares no conflict of interest.

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