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Review Paper: Therapeutic Strategies in Managing Acute Paraquat Poisoning: A Review Study

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ABSTRACT

Background: Paraquat is an extremely toxic herbicide that causes such severe adverse effects as acute lung injury, pulmonary fibrosis, metabolic acidosis, cardiogenic shock, acute renal failure, and even death. Regarding the high prevalence and mortality rate without a specific antidote in paraquat poisoning, we explored managing acute paraquat poisoning.

Methods: In this study, required articles were searched using the following keywords: "paraquat poisoning" and "treatment" on an electronic database, such as Pubmed and Google Scholar from January 2013 to December 2020.

Results: This article examined pharmacokinetic, clinical, and laboratory findings, complications, diagnosis, prognostic factors, and the treatment of paraquat poisoning. Then, the medical management and therapeutic procedures of paraquat poisoning were discussed.

Conclusion: Although there exists no antidote for paraquat poisoning, numerous therapeutic methods have been suggested for treating paraquat poisoning. However, their efficacy remains undiscovered.

An interesting finding in one study indicating that endotracheal intubation is an inappropriate procedure for managing paraquat poisoning; further studies are required in this regard. For these reasons, preventing poisoning and using other herbicides with less toxicity than paraquat are recommended.

1. Introduction

Poisoning due to pesticide usually occurs intentionally, with suicidal ideations; in some cases, it occurs accidentally, and rarely occurs criminally [1]. Paraquat poisoning was recognized in rodents since the early 1960s. Since then, it is consumed in numerous agricultural countries [2]. Recent studies indicated that the mortality rate of pesticide poisoning was approximately 250000 to 370000 cases, annually. Additionally, the annual incidence of paraquat poisoning was ≥3.8 cases/100000 inhabitants/year [3, 4]. Paraquat is a highly toxic, non-selective, and dose-dependent herbicide.

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According to studies, suicide attempts with herbicides, like paraquat has become highly frequent in developing agriculture countries due to their availability and cost-effectiveness in individuals [5-8]. The main route of the involvement of paraquat poisoning is through the mouth [8]. Furthermore, the absorption of paraquat poisoning has been reported through other routes, such as dermal, mucus, and inhalation contacts [5].

Unlike some developing countries and due to high mortality, the production and distribution of paraquat have been stopped in the United States and the European Union since 1994 and 2007, respectively [5, 6]. Studies revealed that the minimum lethal dose of paraquat poisoning for adults equals 20-30 mg/kg. Besides, studies suggested that the lethal doses of paraquat poisoning are 20 mL of a 20% solution [8-11]. Mortality is very high in all centers, despite numerous improvements in treatment; however, the mortality rate varies from 50% to 90%. Especially in cases of an intentional poisoning with concentrated formulation, the mortality rate is 100% [5]. This review study was conducted given the importance of the topic and high mortality rate despite no antidote for paraquat poisoning.

Type of study

In this article, the pharmacokinetics, such as the mechanism of toxicity, clinical and laboratory findings, complications, as well as the diagnosis and prognosis factors of paraquat poisoning were studied. Then, medical management and therapeutic procedures in paraquat poisoning were discussed.

Evidence acquisition

In this study, required articles using the keywords "paraquat poisoning" and "treatment" were searched on electrical databases, such as Pubmed and Google Scholar from January 2013 to December 2020.

2. Discussion

Pharmacokinetic (the absorption, distribution, and mechanism) of toxicity

The main routes of the absorption of paraquat poisoning are through the mouth, skin, mucus, and respiratory system [5, 8]. The absorption of paraquat is very rapid and its maximum concentration occurs within one-hour post-ingestion. The oral bioavailability of paraquat in humans is <5%. Moreover, the half-life of the distribution is estimated at approximately 5 hours. Paraquat minimally binds to plasma proteins and is often rapidly distributed in all tissues. The maximum time for diagnosis of plasma paraquat is about 48 hours; however, it can be detected in urine for >30 days in surviving patients. With normal kidneys, 90% of paraquat is eliminated in the first 24 hours of intoxication; however, with acute renal failure, it is prolonged to more than 48 to 80 hours [12]. Paraquat can inhibit the reduction of NAD(P)+ to NAD(P)H, which produces oxygen radicals and inflammatory responses, resulting in cell damage, multiple organ failures, and even death. The mechanisms of action of paraquat poisoning are presented in Figure 1.

Clinical and laboratory findings and complications

The main clinical symptoms and signs of paraquat poisoning include nausea, vomiting, epigastric pain, mucosal lesions, inflammation of the oral cavity and pharynx, mild to moderate loss of consciousness, and fever. Moreover, the main laboratory findings include leukocytosis, anemia, acute hepatitis with the rise of alanine transaminase and aspartate transaminase, increased serum bilirubin, and creatinine [5, 14]. Significant complications of paraquat poisoning are pulmonary, cardiovascular, hepatic, renal, coagulation, and fibrinolysis dysfunctions due to cell damage [11, 15, 16]. Additionally, progressive lung injury and acute renal failure are the main causes of death in paraquat poisoning [11, 16].

Diagnosis

The diagnosis of paraquat poisoning is usually based on clinical suspicions; however, measuring blood level concentration and urine screen tests are used to confirm the diagnosis of paraquat poisoning [1, 11]. The evaluation of urinary paraquat (the level of urinary sodium dithionite) cannot accurately predict the outcome of paraquat poisoning in patients. However, in one study, patients with paraquat poisoning who had urinary paraquat concentration about 25-50 ppm (or especially above 50 ppm) after 24 hours of consumption experienced a mortality rate of about 90%. Serum or plasma paraquat levels are measured by various methods, including high-performance liquid chromatography (HPLC), gas chromatography-mass spectrometry (GC-MS), enzyme-linked immunosorbent assay (ELISA), and spectrophotometry. Serum or plasma paraquat levels are also used to assess the prognosis of paraquat poisoning. One study revealed that plasma paraquat concentrations less than 2 ppm within 10 hours of intoxication indicated a good prognosis; however, this study was not supported in subsequent studies [15].

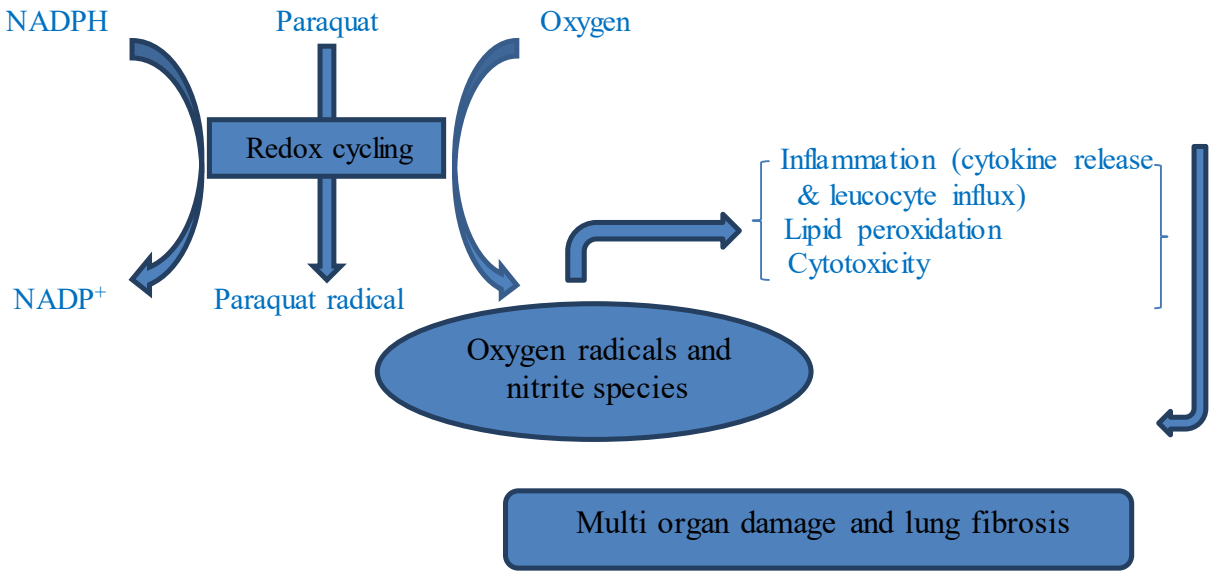

Figure 1. The mechanisms of action of paraquat poisoning [6, 12, 13]

Prognostic factors

Laboratory profiles and their effects on the prognosis of patients with paraquat poisoning were reported in limited studies [11, 16, 17]. Some studies demonstrated that increased leukocyte, prothrombin rate, the blood, and urine concentration of creatinine, pancreatic enzymes, and arterial lactate had a poor prognosis in paraquat poisoning [10, 11]. Moreover, other predictors of prognosis in paraquat poisoning include APACHE II1 and SOFA2 [11].

Treatment

Paraquat poisoning has no specific antidote; however, previous studies suggested some treatment approaches that can be effective in the management of paraquat poisoning. Recent proposed treatments and procedures of paraquat poisoning are presented in Table 1. Wu et al. (2018) in a prospective and observational clinical study evaluated 68 hospitalized patients with respiratory failure due to paraquat poisoning in Taiwan [18]. Then, a comparison was made between the do-not-intubate group and endotracheal intubated group; they unexpectedly concluded that the procedure of intubation in paraquat poisoning can be considered inappropriate treatment [18].

3. Conclusion

There exists no antidote for paraquat poisoning; however,numerous therapeutic strategies have been suggested for managing paraquat poisoning . The mechanisms of multi-organ failure, especially lung injury and the pulmonary fibrosis of paraquat poisoning are cell damage caused by oxygen radicals and inflammatory cytokines (Interleukin-1β (IL-1β), interleukin -6 (IL-6), and tumor necrosis factor-alpha (TNF-α)). Therefore, most proposed treatment methods are based on anti-inflammatory and anti-oxidative therapy. In addition, decreased absorption increased elimination of paraquat, and using procedures, such as extracorporeal and surgical procedures remain the main management methods of paraquat poisoning. However, their efficacy remains unestablished . Unexpectedly, a study suggested that endotracheal intubation is an inappropriate procedure for the management of paraquat poisoning. Thus, the prevention of poisoning and using other herbicides with less toxicity than paraquat is recommended.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles were considered in this article.

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Author's contributions

The main author contributed to preparing this article.

Table 1. Proposed treatments and the procedures of paraquat poisoning and their mechanisms of action

Mechanisms of Action	Recent Treatment Methods
Supportive therapy [11, 19]	Fluid infusion and pulmonary supports [19] Parenteral nutrition [11]
Reduce absorption [19-22]	Gastric lavage and the ingestion of charcoal [19] Lavage with sucralfate [20] Milk gargle and activated carbon retention enema [21] Gastric lavage with "Multani mitti" (Fuller's Earth) or (calcium montmorillonite) and Bentonite [22]
Eliminate and reduced absorption of paraquat [23]	Rhubarb [23]
Anti-inflammatory and anti-oxidative properties [19, 24-40]: Medicines [19, 24-37] Herbal treatment and traditional Chinese medicine [38-40]	Immunosuppressive (cyclophosphamide and corticosteroids) [19] Vitamins C, E and N-acetylcysteine [19] Naringin, Edaravone, Quercetin [19] Lysine acetylsalicylate (a salt of aspirin) [19] Anti-C5a antibodies such as IFX-1 [24] Type III procollagen peptide [25] Rapamycin [26] Procyanidin B2 [27] Doxycycline [28] Rosiglitazone [29] Silymarin [30] ω-3 fish oil emulsion [31] Ambroxol [32] Atorvastatin [33] Alpha lipoic acid [34] Sodium tauroursodeoxycholate [35] 1-methylhydantoin (MH) [36] Metformin [37] Xuebijing injection [38] Danshen injection [38] Rheum officinale Baill [38] Rehmannia glutinosa [39] Monoammonium glycyrrhizinate [40] Dandelion [40]
Procedures	
Extracorporeal removal techniques (even in unknown hepatitis viral marker status) [41-44]	
Hemodialysis	
Hemoperfusion, charcoal hemoperfusion, and resin hemoperfusion	
Hemodiafiltration and continuous venovenous hemofiltration	
Blood purification	
Surgical procedures [45-47]	
Interventional strategy for pulmonary salvage such as one-lung circumvention	
Lung transplantation with or without extracorporeal membrane oxygenation	
Other procedures [19, 48, 49]	
Lung radiotherapy	
Mesenchymal stem cells	
Whole lung lavage therapy	

Conflict of interest

The author declared no conflicts of interest.

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1. APACHE II: Acute physiology and chronic health evaluation score

2. SOFA: Sequential organ failure assessment

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