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Analysis of 1 cases of symptomatic epilepsy caused by paraquat poisoning and literature review

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Abstract: Objective: To explore an effective diagnosis and treatment scheme for paraquat poisoning and to improve the survival rate of patients. Methods: Through the case of a paraquat poisoning survivors combined with symptomatic epilepsy, we explore the treatment plan and review the recent literature related to the treatment of paraquat poisoning. Results: After 6 months of follow-up, the patients survived, liver function and renal function had no obvious abnormalities, lung CT showed mild interstitial lung, and the quality of life of the patients was good. Conclusion: Rational application of hemoperfusion, hormone and other supportive treatment methods and real-time adjustment of individualized medication according to the patient's condition can effectively improve the survival chance of paraquat poisoning patients.

Keywords: paraquat; poisoning; convulsions; pleural effusion; symptomatic epilepsy

Paraquat, the chemical name is 1-1-two methyl-4-4-bipyridine cationic salt, because it has strong herbicidal effect and is widely used in many countries. It is the largest organic heterocyclic contact defoliant and herbicide in the world. However, because of its strong toxic and side effects on human and animal, a small amount of oral administration can cause damage and failure of many organs such as liver, kidney, heart and lung, and so on[1]. In the data reported by many domestic and foreign literature, the mortality rate of oral paraquat is even more than 70% [2]. A case is now reported that a patient with severe paraquat poisoning (oral) was diagnosed and treated in the emergency department of the First Affiliated Hospital of China Medical University, with rare complication (symptomatic epilepsy) in the course of treatment and eventually survives.

1. Case report

A patient, male, aged 23 years old, took the “oral administration of paraquat for more than 1 hours” as the chief complaint. He had been treated with vomiting and gastric lavage and asked for medical history, the dosage of oral paraquat was about 40 milliliters. Physical examination: the mind is clear, and he can accurately answer, the temperature is 36.5 degrees Celsius, pulse 80 beats/min, 125/75 mmHg blood pressure, respiratory rate 20-25 breaths per minute, no conjunctival edema, no enlargement ofsuperficial lymph node in the whole body, a small amount of burn-like ulcer can be seen in the mouth, auscultation of lungs sound clear, and no obvious wet and dry rales, thoracic symmetry, regular heart rhythm, no pathological murmur, flat abdomen, no tenderness and rebound tenderness, no swelling of both the lower extremities was found, and the pathological reflex was not caused. In the first-time results of hospital admission, there was no significant change in the total number of white blood cells in the blood, and there was no obvious abnor-

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mality in liver function, renal function, and pancreas function. The myocardial enzyme spectrum was normal, and there was no obvious abnormality in lung CT.

Emergency jugular catheterization was performed immediately after the emergency, then the blood perfusion was carried out, 4 times of hemoperfusion in 24 hours of intoxication, a total of 8 hours of perfusion time, at the same time, use methylprednisolone (40 mg three times daily intravenous) vitamin C, ambroxol, Xuebijing and other drugs as the symptomatic support therapy. Biochemical tests, blood gas analysis, and monitoring of the degree of injury to the liver, kidney and pancreas were carried out every day. Lung HRCT examination was conducted every 2-3 days to monitor the degree of lung injury. On the first 9 days of treatment, the patient's condition was stable, no complaints such as dyspnea and other complaints. Diet and defecation were normal, the nutritional status was good, liver and kidney function and pancreas function were slightly damaged, and imaging showed mild interstitial change of lung. At the time of tenth days, the patient burst into convulsions when he was sober. The patient relieved himself after about ten seconds, and the number of episodes was 5 times on the day. Then the sedative drugs (Diazepam Injection 16 mg/h) were used, and there was no obvious pathological changes in the parallel brain CT and MRI examination. The diagnosis of Neurology consultation is “symptomatic epilepsy”, and then gradually reduced the use of hormone drugs and sedatives for 2 days, the patient’s convulsion symptoms did not appear again, but the main complaint was dyspnea, which was severe in the supine position and the seat was relieved. After lung CT examination, a large number of patients with bilateral pleural effusion were found, but there were no significant changes in pulmonary fibrosis. After the puncture of the pleural effusion, the pleural effusion was the leaking fluid. Adjust the plan at the same time, gradually reducing the use of methylprednisolone, and for patients to strengthen protein nutrition, twenty-third days after admission, the patients discharged from hospital. After 6 months of follow-up of patient, he has survived, prognosis, liver function, renal function had no obvious abnormalities, lung CT showed mild pulmonary interstitial, the patient's quality of life is good.

2. Conclusion

Paraquat, the chemical name is 1-1- two methy l-4-4-bipyridine cationic salt, is a rapid inactivated herbicide, which has the effect of touch and internal sucking. As a result of the advantage of high efficiency and low price, paraquat is widely used in domestic rural areas. However, paraquat is highly toxic, and has great threat to human and livestock, and there is no specific antidote. It can cause multiple organ failure such as liver and kidney, pulmonary fibrosis (irreversible) and respiratory failure. The mortality of oral poisoning can reach over 90%[3]. At present, it has been banned or strictly restricted by more than 20 countries. In July 2016, China also listed paraquat as a banned pesticide category. Paraquat poisoning has the characteristics of mild early symptoms, high mortality rate and no special antidote. It is believed that acute paraquat poisoning can cause multiple organ damage and even exhaustion. The main mechanisms of poisoning are oxidative damage, inflammatory reaction and cytokine network, apoptosis and mitochondrial damage, calcium overload and NO. The current consensus is that the mechanism of acute paraquat induced body damage is not a result of single factor, but is caused by a variety of factors that interact with each other[4]. Besides the oxygen free radical injury caused by paraquat itself, systemic inflammatory response is also involved in acute multiple system injury and is the main cause of death in acute phase. Early inhibition of inflammatory response and sepsis is the key to reducing mortality, in the later period, there is an irreversible pulmonary fibrosis due to the active uptake and accumulation of paraquat in the alveolar cells, and in this hypoxic state, if oxygen therapy is performed, the progression of pulmonary fibrosis is accelerated[5,6]. In recent years, although the mechanism of paraquat poisoning has been studied from molecular level or even gene level, its mechanism has not yet been fully understood. To explore the new mechanism of acute paraquat disease, it is also a problem that clinical medical personnel and researchers need to solve together.

The current consensus on the diagnosis and treatment of paraquat poisoning is that blood purification is effective for the rescue of paraquat poisoning. In this case, the patient’s blood irrigation time was timely, and sufficient blood perfusion time was given. It also accords with the “golden time” argument of paraquat poisoning in related research[7],

and protects all viscera with hormone, antioxidant and other symptomatic support treatments[8-10]. While this patient is to follow the above scheme for the diagnosis and treatment, the final clinical prognosis was satisfactory, and the patient was not only alive but with almost no sequelae, proving that the scheme could help patients with paraquat poisoning.

Paraquat poisoning has no specific antidote yet, but in the classic treatment of paraquat poisoning, blood purification and methylprednisolone are recognized effective treatment measures[11,12]. And during the treatment of paraquat poisoning patients, many complications often occur, such as liver dysfunction, renal impairment, pancreatic injury, hypoxemia, and pulmonary interstitial changes and other complications, the symptoms of symptomatic epilepsy and the clinical manifestations of the leaked pleural effusion in this case were rarely seen. Symptomatic epilepsy, also known as secondary epilepsy, refers to an epileptic seizure caused by abnormal discharge of neural network due to abnormal brain structure or function caused by some unexpected cause. It can be caused by a variety of causes such as drug poisoning, nutritional metabolism disorder and so on. After adjusting the dosage of hormone and observing the clinical manifestation of the patients later, the patient is considered to be a hormone sensitive constitution. In the tenth day of hormone application, there are side effects caused by hormone, neurological symptoms and substances, disorders of water and salt metabolism and other symptoms. After the punctured pleural effusion was leaked, the patient's pleural effusion decreased after discontinuous administration of albumin injection and enhanced protein nutritional support. And after 3 months' visit, the pleural effusion almost disappeared, but the body weight was significantly reduced, showing a negative nitrogen balance state. The symptoms of the patient were all obvious disorders of nutrition and metabolism. Therefore, we believe that although the early use of a strong dragon is helpful in the treatment of paraquat poisoning, however, if there is no obvious evidence that we must continue to use high dose hormone, we should gradually reduce the amount of hormone and discontinue use during the 5-7 days of treatment. The application of methylprednisolone can not only fully cover the acute period of paraquat poisoning, so as to achieve the aim of inhibiting paraquat injury in the acute period, but also reduce the chances of a number of side effects that hormones may bring.

The early symptoms of paraquat poisoning are not obvious or light, which often cause the neglect and delay of patients and their families. This is also a cause of high mortality of paraquat[13]. Rational application of hemoperfusion, hormone and other supportive treatment methods and real-time adjustment of individualized medication according to the patient's condition can effectively improve the survival chance of paraquat poisoning patients.

References

1. Dinis-Oliveira RJ, Duarte JA, Sanchez-Navarro A, *et al*. Paraquat poisonings: Mechanisms of lung toxicity, clinicalfeatures, and treatment. Toxicol 2008; 38:13-71.
2. Bertolote JM, Fleischmann A. Deaths from pesticide poisoning: A global response. The British Journal of Psychiatry 2006; 189(1): 201-203.
3. Luan Wang, Min Zhao. The mechanism and treatment progress of acute paraquat poisoning. Medical Review 2017; 23(14): 2790-2793.
4. Li S, Shen Y. Research progress of pathogenesis and treatment of acute paraquat poisoning. World's Latest Medical Information Digest 2015; 15(97): 49-50.
5. Liu M. Lung injury and systemic inflammatory response to paraquat poisoning. Journal of Clinical Pulmonary 2010; 15(12): 1779-1781.
6. Hampson EC, Pond SM. Failure of haemoperfusion and haemodialys is to prevent death in paraquat poisoning. Medical Toxicology and Adverse Drug Experience 1988; 3(1): 64-71.
7. Vadivelan M, Chellappan A, Suryanarayana BS. The golden hour in paraquat poisoning. Toxicology International 2014; 21(3): 339-340.
8. Sun Y, Dong X, Liu Z. Clinical analysis of 25 cases of paraquat poisoning. Chinese Journal of Critical Care Medicine 2008; 1(1): 37-38.
9. Koo JR, Kin JC, Yoon JW, *et al*. Failure of continuous venovenous hemofiltration to pervent death in parqual poisoning. American Journal of Kidney Diseases 2002; 39(1): 55-59.
10. Guo L, Wang X, Liu Y, *et al*. The treatment of acute paraquat poisoning with hemoperfusion and methylprednisolone. Chinese Journal of Industrial Hygiene and Occupational Diseases 2011; 29(2): 136-137.
11. Tian Y, Shi H, Tong F, *et al*. Standardized treatment of paraquat poisoning. Chinese Journal of Emergency Medicine 2007; 16(5): 559-560.

12. Wang Y, Zhou C, Fu J, *et al*. Therapeutic effect of different doses of methylprednisolone on paraquat poisoning. Chongqing Medicine 2017; 46(04): 509-511.
13. Dong X, Wang R, Sun D, *et al*. Epidemiological investigation of 2746 cases of acute poisoning. Journal of Clinical Emergency 2016; 17(03): 211-213.