Case report
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Paraquat 중독 환자에서 혈액관류 요법의 임상 경험

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Clinical experience of hemoperfusion treatment in children with paraquat poisoning

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Paraquat is a fatal, rapid-acting, nonselective herbicide. Despite the restriction of production and sales of the herbicide in Korea since 2012, already purchased paraquat can incur poisoning. This article describes a 25-month-old boy who accidentally ingested old paraquat on a rural road. After initial decontamination at 2 outside hospitals, he was transferred to the intensive care unit. Given the urine drug concentration of 20,000 ng/mL 2 hours after the intoxication, he received charcoal hemoperfusion and continuous venovenous hemofiltration to enhance the excretion of the herbicide. In 36 hours after intoxication, it was not detected in the urine. The boy was discharged uneventfully on day 8. This case highlights the importance of hemoperfusion in paraquat poisoning.

Key words: Accidents; Child; Hemoperfusion; Paraquat; Poisoning

Introduction

Paraquat (1,1'-dimethyl-4,4' bipyridinium dichloride) is a nonselective herbicide that had been widely used in agriculture, but its use was restricted in the United States, Europe, and several Asian countries due to the high fatality rate associated with intentional poisoning. The major problem with paraquat poisoning is that even an amount of 7-8 mL can have fatal effects on the major organs, such as the lung, kidney, liver, and heart, eventually leading to death. Another problem is the absence of antidote for the poisoning. Thus, early and intensive decontamination is critical in the poisoning. In Korea, production and sales of the herbicide have been restricted since 2012. However, even after the restriction, the poisoning may occur due to already purchased paraquat. The authors report a Korean toddler who was hospitalized due to accidental paraquat poisoning and treated with charcoal hemoperfusion. This study was approved by the institutional review board of Kyungpook National University Hospital with waiver for informed consent (IRB no.

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Case

A previously healthy, 25-month-old boy was found by his parents drinking an old paraquat bottle on a rural road. It was unclear how much volume had been ingested. He was brought to a local hospital emergency department immediately, and underwent orogastric lavage and activated charcoal treatment. After the decontamination, he was transferred to another local hospital. At the hospital, paraquat concentration tests, which were performed 2 hours after the intoxication, were found to be 20,000 ng/mL in the urine and less than 100 ng/mL in the blood. He was transferred to the intensive care unit for further treatment.

The initial vital signs were as follows: blood pressure, 114/67 mmHg; heart rate, 133 beats/minute; respiratory rate, 24 breaths/minute; temperature, 36.7°C; and oxygen saturation, 100% on room air. His weight was 10 kg (3rd percentile). He was alert, and appeared well. No crackle or wheezing was found in both lungs, and he had no respiratory symptoms.

After confirming his alertness and stable vital signs, fluid therapy with nil per os was started while evaluating the manifestations of paraquat poisoning. The initial venous blood gas analysis findings were as follows: pH, 7.38; PCO_2, 33.3 mmHg; HCO_3, 19.9 mEq/L; base deficit, 5.4 mEq/L; and lactate, 1.9 mmol/L. Initial laboratory findings were within normal limits. Initial chest radiograph showed no abnormal findings.

The above-mentioned plasma concentration of paraquat at the outside hospital was lower than the previously reported values in survivors ranging from 1,100 to 2,640 ng/mL within 24 hours after intoxication[1,2]. In contrast, the urine concentration at the outside hospital was higher than the known mortality-related urine concentration of 1,000 ng/mL within 24 hours[3]. Thus, we decided to perform charcoal hemoperfusion using 367 mL as the volume of a kit (Adsorba 300 C HP cartridge; Baxter, Hechingen, Germany) to help excrete already absorbed drug, and consequently to prevent complications. The hemoperfusion was performed for a total of 4 hours. Subsequently, continuous venovenous hemofiltration was performed for 12 hours to further remove drug, which had already been absorbed into the tissue and released into the bloodstream. Intravenous dexamethasone, cefotaxime, and ampicillin-sulbactam were administered concurrently. Twenty hours after the intoxication, no drug was detected in the blood and 2,600 ng/mL in the urine. Thirty-six hours after the intoxication, it was not detected in either blood or urine.

On day 7, chest computed tomography showed only mild ground-glass opacity without fibrotic change. During hospitalization, the boy had no respiratory symptoms and was discharged on day 8. At 3-month follow-up, computed tomography showed mild pulmonary interstitial fibrosis, but he had no respiratory symptoms.

Discussion

This case highlights the importance of intensive decontamination with hemoperfusion in the acute stage of paraquat poisoning in children. Although paraquat is no longer produced in Korea, this report is meaningful because poisoning can occur with already purchased paraquat as in this case.

The leading cause of death in paraquat poisoning is respiratory failure associated with oxidative damage. Few hours after ingestion, paraquat-related reactive oxygen species are generated, and bind with strong affinity to the alveoli causing direct damage[4]. The reactive oxygen species cause inflammatory damage to the other organs, such as the liver, kidney, and heart.

Although more than 90% of paraquat poisoning is intentional in adults, accidental exposure accounts for a large proportion of the poisoning in children[5]. A study analyzing 5 years of pediatric paraquat poisoning in China found that 67.5% of cases were accidental ingestion and 32.5% were intentional

ingestion with suicidal intent[6]. Since the ingested amount in accidental ingestion is smaller than that in intentional one[5], the prognosis may depend on the type of treatment and plasma concentration[7]. A plasma concentration above 5,000 ng/mL usually results in a 100% mortality, while a concentration below 5,000 ng/mL is critical but curable depending on the treatment[8]. For this reason, it is important to select an appropriate treatment option, and to start the treatment early in plasma concentration lower than 5,000 ng/mL. Although the plasma concentration is most important in prognosis, urine concentration is also relevant[3]. Absorbed paraquat reaches the peak plasma concentration 2 hours after ingestion, and is deposited in the organs, and about 90% of the drug is excreted through the kidneys within 12-24 hours[9]. Paraquat is characterized by a rebound phenomenon, in which it accumulates in the tissues and then gradually gets released into the blood. Due to this phenomenon, studies have shown that the combination of hemoperfusion and continuous renal replacement therapy is effective in reducing mortality[8].

Various treatment options have been tried for paraquat poisoning. Intensive decontamination protocol includes: 1) reducing absorption of the drug via orogastric lavage and activated charcoal and 2) promoting the removal of already absorbed drugs via hemoperfusion or hemodialysis[6]. Hemoperfusion is known to be effective to enhance excretion[10,11]. In intensive care units, additional continuous renal replacement therapy after hemoperfusion is effective to remove the drug that has already been absorbed into the tissue and is slowly released into the blood[8,12]. Antioxidants, such as acetylcysteine, vitamin C, vitamin E, and glutathione, might be beneficial through free radical scavenging. Anti-inflammatory agents and immunosuppressive agents, such as methylprednisolone, dexamethasone, and cyclophosphamide, have been used to minimize damage to the organs already exposed to paraquat[13].

In the present case, the boy received intensive decontamination for paraquat poisoning, and showed good recovery in the acute phase. Although mild pulmonary interstitial fibrosis remained, he recovered without any respiratory symptoms. Because there were fewer poisoning cases in children than in adults, there is a paucity of reports regarding treatment of pediatric paraquat poisoning. It is important to be aware of treatment options because prognosis depends on the choice of therapeutic modality.

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Conflicts of interest

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