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증 례

파라쿼트로 발생한 폐 섬유화증에서 pulse therapy로 호전된 환자 1례

이화여자대학교 의학전문대학원 응급의학교실

홍기훈 · 정진희 · 어은경

A Case of Moderate Paraquat Intoxication with Pulse Therapy in the Subacute Stage of Pulmonary Fibrosis

Ki Hun Hong, M.D., Jin Hee Jung, M.D., Eun Kyung Eo, M.D.

Department of Emergency Medicine, School of Medicine, Ewha Womans University, Seoul, Korea

In South Korea, attempted suicide by paraquat (PQ) intoxication is fairly common, and is lethal by pulmonary fibrosis and hypoxemia. However, the treatment of PQ poisoning is primarily supportive management. To increase the survival rate associated with PQ intoxication, many treatments have been developed. Here, we treated a case of PQ intoxication with steroid pulse therapy.

A 23-year-old man was admitted to the hospital because of PQ intoxication. He drank two mouthfuls of Gramoxon (24% commercial paraquat). His vital signs were stable, but he had a throat infection, and navy blue urine in the sodium dithionite test.

Standard treatment, including gastric lavage with activated charcoal was performed, and emergent hemoperfusion with a charcoal filter was initiated 11 h after PQ ingestion. Pharmacotherapy was initiated 18 h after PQ ingestion with the administration of 5 mg dexamethasone. On day 10, chest PA showed pulmonary fibrosis. Therefore, we initiated steroid pulse therapy, with 1 g methylprednisolone in 100 mL of D5W administered over 1 h repeated daily for 3 days, and 1g cyclophosphamide in 100 mL of D5W administered over 1 h daily for 2 days. On day 15, dexamethasone therapy was initiated. On day 30, pulmonary fibrosis was improved.

Thus, if pulmonary fibrosis becomes exacerbated after dexamethasone therapy during the subacute stage, pulse therapy with methylprednisolone and cyclophosphamide could be helpful.

Key Words: Paraquat, Pulmonary fibrosis, Steroid, Pulse therapy

INTRODUCTION

In South Korea, attempted suicide by paraqaut (PQ) intoxication results in a relatively high rate of mortality, caused primarily by pulmonary fibrosis and hypoxemia. However, the treatment of PQ poisoning has remained mainly supportive and typically includes gastric lavage, activated charcoal, and emergent hemoperfusion. To increase the survival rate associated with PQ intoxication, treatments have been developed using antioxidant, immunosuppressant drugs, and anti-inflammatory agents[1-5]. Some reports have demonstrated improved survival rates using steroid pulse therapy that was usually administered at the time of patient admission[6,7]. Here we describe a case of survival following PQ intoxication using steroid pulse therapy administered at the subacute stage.

책임저자: 어 은 경
서울특별시 양천구 목6동 911-1
이화여자대학교 의학전문대학원 응급의학교실
Tel: 02) 2650-5296, Fax: 02) 2650-5060
E-mail: liz0803@ewha.ac.kr

CASE REPORT

A 23-year-old man experienced stress related to a family problem and drank two mouthfuls of Gramoxon (24% commercial paraquat) with alcohol. After 9 h he was admitted to the hospital because of increasing oral pain and five instances of vomiting.

His blood pressure was 143/90 mmHg, pulse rate was 123 beats/min, respiration rate was 16 breaths/min, and body temperature was 37.5°C. Upon initial physical examination, the patient was mentality alert, and had throat injection without exudates and ulceration. His chest was clear to auscultation, and the heart rate was regular with no murmurs detected. Bowel sounds were normoactive, and the abdomen was soft and not tender and was without organomegaly. On laboratory tests, the sodium dithionite test of urine showed navy blue color. Arterial blood gas analysis produced a pH of 7.428, P_{CO2} of 33.7 mmHg, and Pa_{O2} of 99.3 mmHg. At blood cell count, the WBC was 15,800/mL with leukocytosis noted. Other laboratory findings showed a normal blood cell count with Hb 14.2mg/dL, hematocrit 41.0%, PLT 250k/mL, normal renal function with BUN 11 mg/dL and creatinine 0.8 mg/dL, normal liver function with AST 21 U/L and ALT 22 U/L. Chest radiography showed no active lung lesions.

Although 9 h passed after PQ ingestion, the patient was complaint of greenish vomitus just before admitting to the hospital. Therefore, the standard supportive treatment, including gastric lavage and activated charcoal was performed. And emergent hemoperfusion with charcoal filter (adsorba® 300 cc, Centrysystem 3, Gambro) was initiated 11 h after paraquat ingestion. Hemoperfusion was completed in 6 h, and pharmacotherapy was initiated 18 h after PQ ingestion with the administered of 5 mg dexamethasone every 6 h by intravenous injection. Renal function tests, liver function tests, arterial blood gas were checked regularly. The creatinine level was 2.3 mg/dL on day 3 and 3.3 mg/dL on day 5, but there was no decrease in the patient' s urine volume. On day 10, we checked chest PA regularly, and reticular patterns of infiltration suggested pulmonary fibrosis. On chest CT, ground glass and reticular opacity in

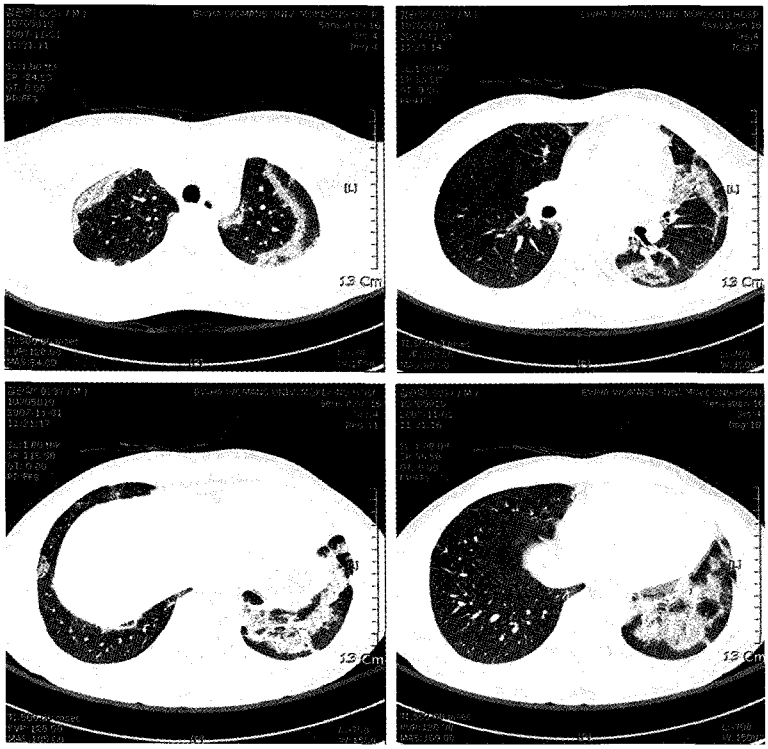

Fig. 1. The chest CT showed ground glass and reticular opacities in both peripheral lungs, suggesting acute or subacute stage of pulmonary fibrosis.

both peripheral lungs suggested acute or subacute pulmonary fibrosis (Fig. 1). Therefore, we initiated steroid pulse therapy, with 1g methylprednisolone in 100mL of 5% dextrose water administered over 1 h and repeated daily for 3 days and 1g cyclophosphamide in 100 mL of 5% dextrose water administered over 1 h daily for 2 days. On day 13, hypoxemia developed with Pa_{O2} of 69.9 mmHg, which by day 14 to Pa_{O2} of 91.6 mmHg. On day 15, dexamethasone therapy was initiated and continued for 2 weeks. On day 30, radiology including chest PA and chest CT revealed an improvement of the infiltrated lesions (Fig. 2). The patient was discharged on day 32, with outpatient follow-up. At 6 month post discharge, the patient remained symptom free.

DISCUSSION

Severe pulmonary fibrosis plays a major role in the lethal hypoxemia of patients with PQ intoxication during the subacute period of intoxication. Hence, the survival rates of PQ-poisoned patients improved with pulse therapies that were usually administered at the time of admission[5,6]. Lin et al. used repeated pulse therapy if pulmonary fibrosis developed[6]. Here we used a conventional protocol with 5 mg dexamethasone every 6 h for 2 weeks without pulse therapy, and we initiated steroid pulse therapy only after the development of pulmonary fibrosis in the subacute phase. In some previous studies, mild pulmonary fibrosis improved without methylprednisolone pulse therapy, and it was reversible[8,9]. But, my patient exhibited more severe toxicity, by the navy blue reaction than patients in the previous studies. The reasons for moderate toxicity in our case were the follows: 1) the sodium dithionite test was navy blue; 2) the creatinine level was elevated at 3.3 mg/dL; 3) two mouthfuls of Gramoxon were consumed. Previous study[10-12] of PQ intoxication have shown that the plasma and urine concentration within first 24 h of intoxication were good predictors of outcome. Here, most plasma PQ concentrations were not checked owing to the limitations of our facilities, although the urine dithionite test was a reasonable indicator for severity of PQ poisoning.

In conclusion, we successfully treated a case of

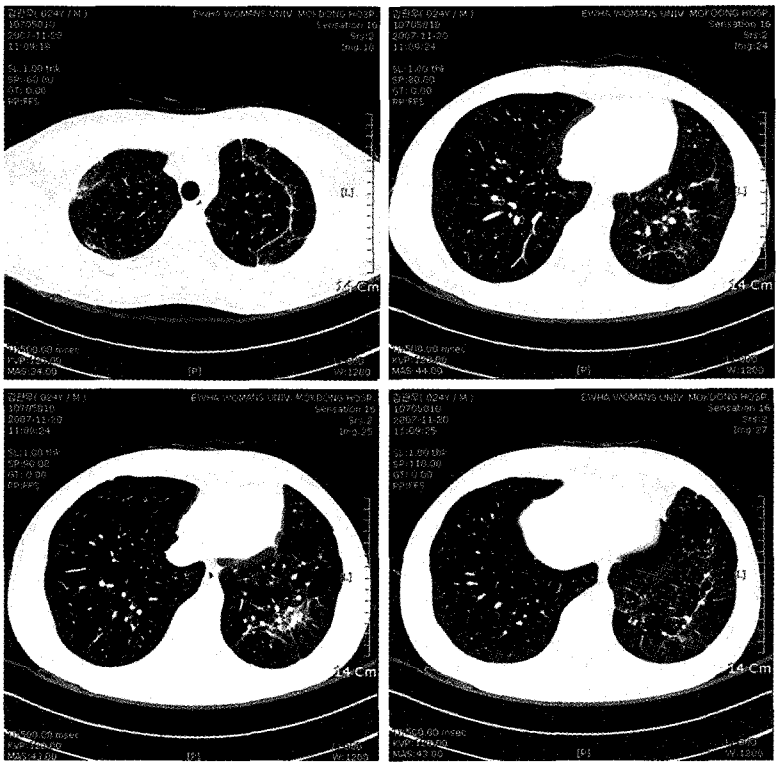

Fig. 2. The follow-up chest CT on day 31 showed much improved infiltrated lesions and suggested fibrotic stage.

early pulmonary fibrosis resulting from PQ intoxication using steroid pulse therapy and prolonged dexamethasone treatment. Our successful treatment suggests that if pulmonary fibrosis becomes exacerbated after dexamethasone therapy during the subacute stage, pulse therapy with methylprednisolone and cyclophosphamide could be attempted.

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