Case Report

Recovery after Radiotherapy from Severe Interstitial Pneumonia due to Paraquat Poisoning

Masashi Shirahama, Takanobu Sakemi, Shinichiro Osato, Toru Sanai, Osamu Rikitake and Susumu Wada*

A 51-year-old man was admitted 3 hours after ingesting approximately 50 ml of mixture of paraquat and organophosphate insecticide. His arterial oxygen pressure fell progressively to 44.6 mmHg. Diagnosed was paraquat-induced interstitial pneumonia. No improvement was observed after treatment with corticosteroid. The pneumonia, however, resolved after irradiation of both lungs and arterial oxygen pressure showed marked improvement. Radiotherapy to the lungs should be considered only in patients who showed progressive deterioration of respiratory function.

Key Words: Paraquat-induced interstitial pneumonia, Corticosteroid therapy, Radiation therapy

The accidental and suicidal poisoning by paraquat, a commonly used herbicide, occurs often. Paraquat is a multisystem poison causing toxicity to the lungs, kidneys, liver, heart, and muscle. Although paraquat damages many tissues, important clinical toxicity usually involves the lungs and kidneys. Renal failure is common but reversible. Progressive pulmonary damage with respiratory failure, such as pulmonary edema which appears within the first few days after ingestion and pulmonary fibrosis within one to two weeks, has caused most of the fatalities. Because of the high mortality from paraquat poisoning, a multidirectional approach to the therapy has been recommended.

We describe a patient whose pulmonary function deteriorated progressively despite the intensive conventional treatment, showed marked improvement after radiotherapy to the lungs.

CASE REPORT

A 51-year-old farmer had suffered from schizophrenia for more than 20 years. He was admitted to a near hospital because of drinking 50 ml of mixture of paraquat (containing 24% paraquat) and organophosphate insecticide to attempt suicide. Because he developed a generalized convulsion after 3 hours' gastric lavage, he was transferred to our hospital. On admission, he was delirious. Pulse rate was 75/min, blood pressure, 120/70 mmHg and respiratory rate, 24/min on clinical examination. The result of urine test for paraquat by alkaline dithionite method was highly positive. Blood paraquat level was undetectable during the course of the patient's illness according to the method of absorption spectrometry (under 2.5 μg/ml). Other abnormal findings were elevation of GOT level and reduction of cholineesterase level, presumably due to the toxicity of organophosphate. The chest roentgenogram was normal. PaO₂ was 117 mmHg and PaCO₂ was 25.6 mmHg in room air. On admission, gastric lavage was performed. This was followed by 800 ml of mannitol and aluminium silica which was fed by nasogastric tube every 6 hours for 5 days. Forced diuresis was administered through a central line by massive infusion of saline with furosemide. Charcoal hemoperfusion was started 3 hours after admission and continued for 6 hours. The result of urine test for paraquat was still slightly positive after 6 hours' hemoperfusion and negative on the 3rd day.

On the 3rd day serum creatinine concentration increased to 1.6 mg/dl, but returned to normal by the 6th day. Liver damage detected on admission also improved by the 7th day. On the 4th day, however, he complained of dyspnea. Fine inspiratory crackle was audible at both lung bases. A
chest roentgenogram showed reticular shadows mainly in the left lobe. The PaO₂ was 63.8 mmHg in the room air at this time. On the 5th day, the PaO₂ fell to 54.6 mmHg and on the chest roentgenogram a reticular interstitial pattern spread to both lungs in spite of the treatment with daily administration of one gram of methylpredonisolone (Fig. 1). Radiotherapy was started: both lungs were irradiated with 4 MV X-ray. The total dose was 960 rad (160 rad per day for 6 days). On the 6th day, the PaO₂ continued to decrease to 44.6 mmHg in room air (Fig. 2). There was an improvement in the PaO₂ from the 7th day, associated with the resolution of pulmonary infiltrates. The PaO₂ increased to 83.7 mmHg on the 13th day when the 6th irradiation was carried out. One gram of methylpredonisolone was given intravenously daily from the first day until the 10th day, when improvement of pulmonary function was observed and thereafter the dosage was gradually tapered down. The patient simultaneously received Vitamine C, 3000 mg, Vitamine E, 1200

Fig. 1. Chest roentgenogram showing widespread interstitial shadowing on the 6th day after ingestion of paraquat.

Fig. 2. Clinical course after ingestion of paraquat.

Fig. 3. The biopsy specimen showing interstitial cellular infiltration and mild fibrosis in alveolar septum. (H.E. stain, x200)

Fig. 4. Chest roentgenogram is almost normal on the tenth month after ingestion of paraquat.
Radiotherapy to the Paraquat Lung

mg, glutathione, 2400 mg per day.

The transbronchial lung biopsy was performed on the 13th day. The biopsy specimen showed interstitial cellular infiltration and mild fibrosis in alveolar septum, consistent with the diagnosis of pulmonary damage due to paraquat (Fig. 3).

Ten months later, lung function test (% VC: 83.4%, % FEV_{1.0}: 84.6%, Dl_{CO}: 12.4 ml/min/mmHg) showed only impairment of diffusion capacity. The PaO₂ was 91.4 mmHg and chest roentgenogram was almost normal (Fig. 4). Now he is doing well as a farmer without exertional dyspnea.

DISCUSSION

This report describes a patient who recovered from paraquat poisoning, severe enough to death from pulmonary complication. Recently, successful treatments of paraquat poisoning have been reported. These reports have described the importance of early gastric lavage, hemoperfusion, and administration of corticosteroid and immunosuppressive drugs. Nevertheless there were many cases resistant to these treatment, which developed progressive pulmonary damage with respiratory failure similar to our case. Our patient was seriously affected and was on the point of requiring treatment with oxygen. Radiotherapy was indicated since no improvement was observed and death appeared inevitable. Webb et al reported the efficacy of radiotherapy in treating paraquat lung disease resistant to charcoal hemoperfusion, corticosteroid and immunosuppressive drugs. In our case as same as Webb’s, reticular infiltrates which had spread on both lungs on chest roentgenogram resolved after radiotherapy. Total radiation dose of our case was less than theirs (right lung 1125 rad and left lung 1500 rad) and was decided on the base of the report by Rowist et al² (in an adult 2000 rad and in a child 1400~1500 rad as the tolerant dose of whole lung irradiation) and the body size of Japanese people.

Concerning the mechanism of the efficacy of radiotherapy in treating the pulmonary fibrosis, Webb et al described that low dose irradiation prevented the proliferation of fibroblasts which was presumably responsible for the formation of pulmonary fibrosis. There are criticisms on the efficacy and application of radiotherapy for the paraquat-induced pulmonary fibrosis³. (1) The concomitant giving of corticosteroid and other drugs makes it difficult to decide that radiotherapy itself is effective for the patient survival. (2) Since the treatment with irradiation, though low dose, causes the pulmonary fibrosis, there are claims that radiotherapy should await studies confirming its efficacy on animals.

In the review of published case reports where recovery of pulmonary damage has been noted⁴⁻¹¹, the PaO₂ has been more than 60 mmHg and pulmonary improvement has been observed during 3 days steroid pulse therapy. Progressive deterioration of pulmonary function occurs in our case, despite the intensive treatment with daily one gram of methylpredonisolone. Improvement of pulmonary function was observed more rapidly than expected during radiotherapy. In the present situation, lack of any proved effective treatment, we considered that low dose irradiation may be useful in the treatment of paraquat lung only when progressive deterioration of pulmonary function occurred.

REFERENCES