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Case Report Open Access

Paraquat-Induced Delayed Atrial Fibrillation

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Abstract

Introduction: Paraquat (1, 1'-dimethyl-4, 4'-dipyridylium) is a nonselective potent herbicide. But, it can cause dramatic toxicity when misused. When ingested, paraquat is toxic to the lung, kidney, heart, central nervous system, and gastrointestinal tract, etc.

Case report: A patient visited to our emergency room after accidental ingestion of paraquat herbicide. The patient was treated with cyclophosphamide, steroid therapy, and hemoperfusion three times consecutively. His condition had improved. Shortly following treatment, the patient presented with palpitation and dizziness. His systolic blood pressure was 60 mmHg and electrocardiogram showed atrial fibrillation with a rapid ventricular response. Synchronized electrical cardioversion was provided and amiodarone was administered. The heart returned back to normal sinus rhythm and the vital signs stabilized.

Discussion: The heart is one of the most severely affected organs in paraquat poisoning and cardiac disturbances can lead to fatal complications. Therefore, it is important to delve deeper into the cardiac manifestations of paraquat poisoning for proper management.

Keywords: Paraquat; Atrial fibrillation

Introduction

The ingestion of paraquat, a non-selective herbicide, can be fatal in humans. Especially, it is major public health problem in developing countries. It has a high mortality rate and no definitive treatment exists even though various treatment modalities have been attempted. Even with active treatment such as hemoperfusion, the recent mortality rate survival rate is as high as 40% [1]. Paraquat poisoning can lead to multiple organ failure, including progressive pulmonary fibrosis, acute tubular necrosis, centrilobular hepatic necrosis, focal hemorrhage, and death [2]. However, atrial fibrillation due to paraquat poisoning has not been reported in the literature. Here, we report the case of a 65-year-old man who presented with atrial fibrillation after paraquat intoxication.

Case Report

A 65 year-old-male without a significant medical history visited the emergency room. He was rushed to the hospital by relatives, approximately 50 minutes after accidental ingestion of paraquat. He presented as intoxicated and was noted by his family as having ingested the toxin with the alcohol. His total paraquat consumption was unknown. When he arrived at the emergency room, his vital signs were stable. He complained of a sore throat and epigastirc pain. On physical examination, he presented with excoriation of the lips and mouth (Figure 1). His initial laboratory data, chest radiograph, and Electrocardiogram (ECG) were normal. His sodium dithionite urine test result was positive (3+) for paraquat. As soon as he arrived in emergency room, he was treated with gastric lavage, with large amounts of normal saline, followed by infusion of 1 g/kg activated charcoal via a nasogastric tube. He was provided with hemoperfusion three consecutive times and immunosuppressive therapy with pulse therapies of cyclophosphamide (15 mg/kg/day) for 2 days and methylprednisolone (1 g/day) for 3 days, simultaneously, followed by dexamethasone (20 mg/day) for 14 days. About 10 days after the treatment, his Blood Pressure (BP) abruptly fell to 60/40 mmHg with heart rate of 114 beats per minute. There were no evidences that cause atrial fibrillation such as ischemic heart disease, emotional stress, hyperthyroidism, and electrolyte imbalance. Upon examination, he was confused and became progressively aggravated

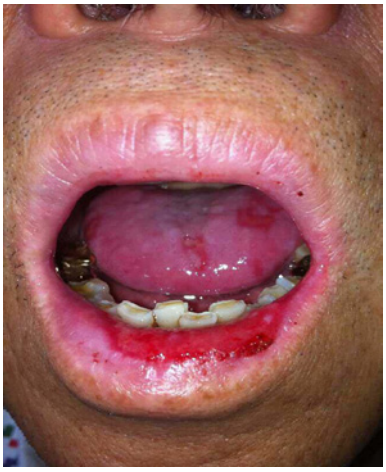

Figure 1: The patient complained of a sore throat and epigastirc pain. The abnormal physical findings were excoriation of the lips and mouth.

over time. An ECG showed atrial fibrillation with rapid ventricular rhythm. Immediately, synchronized electrical cardioversion using an energy level of 200 J was performed. After cardioversion, cardiac enzyme level revealed serum troponin I of 0.448 ng/mL (normal <0.16 ng/mL) and creatine kinase-myocardial band of 4.36 ng/mL

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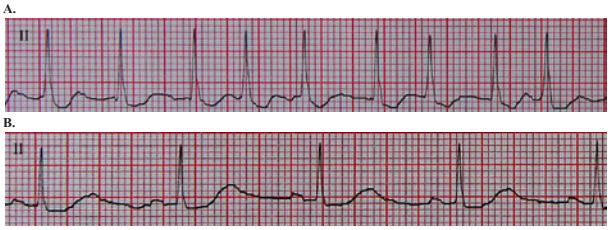

Figure 2: An electrocardiogram showed atrial fibrillation with a rapid ventricular rhythm (A). At once, DC cardio version was performed. An amiodarone infusion was also added to his treatment protocol in order to keep his cardiac rhythm under control (B).

(normal <2.88 ng/mL). Cardiac enzyme was normalized after 7 days. Amiodarone (loading dose 15 mg/minute for 10 minutes, followed by 1 mg/minute for 6 hours; maintenance dose 0.5 mg/minute for 18 hours) was added to his treatment regimen to maintain his heart rhythm (Figure 2). After sinus conversion, his BP recovered to 110/70 mmHg and his general condition improved. He completely recovered and had no further episodes of atrial fibrillation noted in follow up.

Discussion

Paraquat is a highly toxic bipyridyl herbicide acting as a nonselective herbicide with somewhat unique properties [3]. It is used in many countries without restriction including South Korea [1]. There have been numerous fatalities, mainly caused by accidental or voluntary ingestion of the herbicide [4]. The toxic effect of paraquat is mediated by radicals, which are the products of paraquat reduction in cells. An ingestion of paraquat may cause severe and fatal poisoning. These herbicides produce corrosive lesions as a local effect, but their systemic effects are much more fatal [5]. When ingested, paraquat is toxic to the lung, kidney, heart, central nervous system, and gastrointestinal tract, etc. Following the ingestion of 30 mg/kg or 50 ml of a 21% (w/w) solution of paraquat as the base, hepatic, cardiac or renal failure or death may occur. Smaller doses (greater than or equal to 4 mg/kg of paraquat base) may cause respiratory distress, renal dysfunction or, occasionally, jaundice or adrenal cortical necrosis [6]. Paraquat is usually localized in the lung tissue, with its concentration in the lung found to be 10 times greater than that of plasma, and is found to be retained in lung tissue; even blood paraquat levels have decreased [4]. Therefore, the lung is a major target organ in paraquat poisoning and respiratory failure from lung injury is the most common cause of death [7]. Cardiac manifestations are not typical hallmark symptoms of paraquat poisoning but they can frequently be expressed and the clinical picture of cardiac involvement has a diverse spectrum with varying degrees of severity, ranging from minimal changes in the ECG to acute and extensive myocardial necrosis. Histopathological studies found that edema, congestion and hemorrhage occurred in the myocardium after the ingestion of paraquat [8]. However, to our knowledgement, there was no literature accurately described the mechanism of the atrial fibrillation due to paraquat. More detailed study is needed about the cardiac toxicity by praraquat poisoning. Until now, there is no effective treatment because paraquat poisoning has a high morbidity and mortality rate. Immediate gastric lavage or whole-gut irrigation for drug removal from the gastrointestinal tract are considered first [6]. The combined use of diuretics and therapy with glucocorticoid and cyclophosphamide may be effective in preventing respiratory failure and reducing mortality [9,10]. Moreover, hemoperfusion is known to be the most effective treatment for paraquat elimination [10,11]. Therefore, early diagnosis and initiation of the appropriate treatment as early as possible is the key to save the patient's life.

In this case, first detected atrial fibrillation by paraquat poisoning was occurred with unstable vital signs and recovered after proper management. The heart is one of the most severely affected organs in paraquat poisoning and cardiac disturbances can lead to fatal complications. Therefore, it is important to delve deeper into the cardiac manifestations of paraquat poisoning for proper management.

Conclusion

Paraquat is toxic to multiple organs, including the lung, kidney, heart, gastrointestinal tract and central nervous system. Cardiac manifestations are not typical hallmark symptoms of paraquat poisoning but they can frequently be expressed with varying grees of severity, ranging from minimal changes in the ECG to acute and extensive myocardial necrosis. We report on patient with atrial fibrillation that presented following paraquat intoxication.

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