Case report: transdermal absorption of paraquat-induced poisoning combined with pemphigus vulgaris.

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Abstract

In this study, we report on a case of transdermal paraquat poisoning combined with Pemphigus Vulgaris (PV). The patient was a 68 year old male farmer. He was exposed to paraquat (50% concentration) in a closed environment for 3 h, when the skin and respiratory symptoms of paraquat poisoning occurred. He was admitted to the hospital and diagnosed with transdermal paraquat poisoning combined with PV. After symptomatic treatment using glucocorticoids, immunosuppressive agents, and antibiotics for more than 3 weeks, PV was controlled, and the patient was discharged. Exploring paraquat as an agent in the etiology or induction of PV and its pathogenesis are worthy of further investigation.

Keywords: Paraquat poisoning, Pemphigus vulgaris, Transdermal absorption.

Introduction

Pesticide poisoning is one of the major public health problems in developing countries, with approximately 2 million people hospitalized due to pesticide poisoning each year and even higher annual hospitalization and mortality rates [1]. Paraquat is a widely used herbicide, and is fatal when absorbed by the body. Paraquat Poisoning (PP) accounts for 34% of all pesticide poisoning cases, and is associated with the highest mortality [2]. Paraquat can be absorbed through the skin as well as the gastrointestinal and respiratory tracts, and systemic toxicity can occur from either long-term exposure or short-term exposure to high concentrations on the skin, particularly broken skin, scrotum, and perineum. Pemphigus vulgaris (PV) is an autoimmune disease characterized by skin or mucous membrane blisters, but the specific cause is still unknown; other factors such as genetic and environmental factors are likely to cause this disease [3]. The pathogenesis of PV is clearly related to immunity; however, the precise immune mechanisms of the occurrence and development of PV are still unclear.

It has not been reported whether skin absorption-induced PP can induce or cause PV. This study reports on one case of skin absorption-induced PP with PV, in which both conditions were controlled after appropriate and effective treatment. The relevant mechanisms are also discussed.
admitted to our department. The physical examination on admission revealed that the patient’s oral mucosa, as well as the skin on his head, chest, and back, had soybean-sized (approximate) blisters and erosions, with slightly turbid blister fluid. Figure 1 displays a flushing erosive surface on a site of a ruptured blister, together with minor exudate or crusting. The patient’s family members stated that his wound healing was slow and recurrent and he felt burning pain. Bilateral pulmonary respiration sounds were noted; however, the other results of the physical examination were normal. Computed Tomography (CT) of his lungs showed bilateral lung legions (Figure 2). The patient was previously healthy, with normal liver and kidney functions, and he had no history of skin disease or other immune system disease. He did not have contact with other chemicals or radioactive substances before and after PV onset. In addition, no problems were detected during a physical examination 2 months before PV onset, and on this basis, other immune system disorders were ruled out. In addition, no similar disease had occurred in any of the family members of the patient.

Following admission to our department, a smear of the basal tissue fluid of a fresh blister from the patient was taken for pathological examination and staining. It exhibited separated or clumped lytic free acanthocytes, with large and uniformly stained nuclei. Furthermore, one zona pellucida could be seen around the nucleus, and the surrounding cells were heavily stained; the spinous process disappeared, namely the lytic cells of the spinous process, which exhibited the characteristics of PV (a pathological section of the selected slice is shown in Figure 3). Indirect immunofluorescence revealed positive results for PV-circulating antibody (mean titer of 1:1280). The patient received glucocorticoid therapy, immunosuppressant agents, antibiotics, and supportive symptomatic treatment, after which his symptoms were controlled.

Discussion

PP occurs in a variety of ways including oral transmission, which is the most common, as well as skin exposure, mucosal contact, and through open cuts or wounds, which would also lead to systemic toxicity. In most cases, oral PP is associated with the following symptoms: oral burning, erosion, and ulceration of the oral mucosa and esophageal mucosa; and nausea, vomiting, abdominal pain, diarrhea, possible hematemesis and hematochezia or gastric perforation, and acute pancreatitis. A few cases of oral poisoning have hepatomegaly and/or jaundice with abnormal liver functions and possible liver failure. Regarding the Central Nervous System (CNS), some cases report dizziness and/or a headache, while only a small minority of patients had hallucinations, fear, convulsions, coma, and other CNS symptoms. Lung injury is very common and serious, manifesting as a cough, chest tightness, shortness of breath, cyanosis, dyspnea, diminution of respiration, and pulmonary dry and moist rales.

Most causes of death following oral ingestion of large quantities of paraquat were due to acute respiratory distress syndrome, with death in many of these cases occurring within a few days, since pulmonary edema and hemorrhage always occurred within 24 h. While subacute cases (only small quantities of paraquat were taken), with the main manifestations of chest tightness at 1 week and dyspnea at 2-3 weeks, usually died of respiratory failure. A few cases had complications such as pneumothorax, mediastinal emphysema, toxic myocarditis, and pericardial bleeding. This process changed gradually, and in the first week, the injury of the lung included marked thickening, interlobular fissures, and exudative changes or consolidation, which only occurred at the bottom or peripheral of the lung, sometimes with pleural
In conclusion, the patient in this study was admitted to our hospital for skin contact with paraquat in combination with PV. Because skin contact with paraquat plays an important role in the occurrence and development of PV, the possible mechanism may be concluded since PP reduces the patient's immune system. However, the in-depth mechanisms between paraquat and immune system still need further investigation and exploration. Therefore, PP, as a causative factor, should be highly questioned and investigated in relation to PV.

Conflict of Interest

All authors have no conflict of interest regarding this paper.

References


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