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Successful Treatment of Multiorgan Toxicity Induced by Explosive Agent With Repeated Hemodialysis: A Case Report

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Abstract

Introduction: Acute poisoning by oral ingestion of mixed hazardous materials can lead to multiorgan toxicity and may represent a special challenge in clinical management, in particular if little is known about the effects of the involved agents. This is especially true when industrial chemical substances have been swallowed, as a huge variety of toxic substances must be taken into account.
Case Presentation: This case report describes the management of this potentially life-threatening situation in a 21-year-old man with multiorgan system failure, who developed gastrointestinal bleeding, anemia, renal dysfunction, altered visual acuity, seizure, jaundice, and ascites due to multiorgan toxicity. This patient was successfully treated with repeated hemodialysis, and survived. He was discharged from the hospital with an acceptable level of morbidity.
Conclusions: In cases of oral-based multiple hazardous substance toxicity without a specific antidote, hemodialysis seems to be the best treatment. The problems created by multiorgan toxicity and the issues involved in the decision-making process are discussed in the management of this extraordinarily overwhelming complication.

Keywords: Multiorgan Toxicity, Explosive Agents, Hemodialysis, Multiorgan Failure

1. Introduction

Acute poisoning by oral ingestion of mixed hazardous materials can lead to multiorgan toxicity (MOT) and represents a special challenge in clinical management, in particular if little is known about the effects of the involved agents. This is especially true when industrial chemical substances have been swallowed, as a huge variety of toxic substances must be taken into account (1). In cases for which there is no specific antidote, treatment is very difficult (2). MOT is the failure of different organs that have been damaged by known or unknown compounds. The reported case is an adult who survived multiorgan failure caused by oral ingestion of Aklilsorang, an explosive agent used in a traditional ceremony. MOT from this agent has not been previously reported. After an extensive search in the literature, Aklilsorang was found to contain various compounds with different known or unknown toxicities, including Pb_3O_4, sulfur, aluminum powder, magnesium, ammonium, barium nitrate, HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine), lead nitrate, PETN (pentaerythritol tetranitrate), sodium picrate, RDX (1,3,5-trinitro-1,3,5-triazacyclohexane), tetryl (2,4,6-trinitrophenylmethyl-nitramine), potassium nitrate, boric acid, calcium chloride, and silica (3-7). We present this case of a 21-year-old man who swallowed Aklilsorang, and one day later experienced severe epigastric pain and hematuria. He progressed to multiorgan system failure, but ultimately recovered after a prolonged hospitalization.

2. Case Presentation

A 21-year-old man was admitted to Baharloo hospital after attempting suicide by exogenous intoxication with Aklilsorang, an explosive agent used in a traditional Iranian ceremony on the last Wednesday of the year. He was admitted two days ago for attempting suicide by exogenous intoxication with tramadol. The patient had experienced feelings of guilt and hopelessness for the previous two weeks.

At the time of admission, his complaints were severe epigastric pain and bloody urine. He denied tiredness, headache, or any symptom of poisoning, such as nausea, vomiting, or altered consciousness. His past medical history was negative and his family history was unreliable. On physical examination, his blood pressure was 125 mmHg, pulse rate was 80 beats/min, and respiratory rate was 16 breaths/min. His physical and neurological examinations were normal except for generalized abdominal tenderness and an upward Babinski reflex. Initial laboratory tests in-

cluded a hemoglobin (Hb) of 12.7 g/dL, white blood cell (WBC) count of 20.6 $\times$ 10^3/L, urea of 80 mg/dL, and creatinine (Cr) of 10.4 mg/dL. Urine analysis showed proteinuria and many erythrocytes.

The patient was admitted to the toxicology department for suspected tramadol and/or other intoxication. On the second day, he developed jaundice and ascites. His blood test results showed increased direct bilirubin (hyperbilirubinemia, with a total bilirubin of 4.6 mg/dL and direct bilirubin of 0.8 mg/dL), high LDH (3332.1 U/L), leukocytosis (21.1 $\times$ 10^3/L), and increased Cr of 8.3 mg/dL. He was transferred to the intensive care unit (ICU) of the toxicology department. On the 2th day, he affirmed his attempted suicide by Aklilsorang. After an extensive search of the literature, the structure of this substance was determined to contain different mixed compounds (3-7), including lead as a main substance, which was considered a suspicious compound for poisoning. Hence, his blood was assessed for heavy metals, and quantitative measurements showed a blood lead level 63 μg/dL, while screening for aluminum and other heavy metals was negative or within acceptable limits. Chelation therapy was initiated with dimercaprol (75 mg/mL) for 24 hours. However, after 48 hours of chelation therapy, our patient's condition deteriorated and he developed tachycardia, upper gastrointestinal bleeding, and coma. Therefore, after two days of chelating therapy, this treatment was stopped. G6PD was ruled out as a suspicious factor. The patient's blood panel revealed anemia (Hb of 7.8 g/dL), high LDH (2060), and a creatine phosphokinase (CPK) of 3170 on the third day. On the fourth day of admission, hemodialysis was considered because of evident renal dysfunction and a blood Cr level of 8 mg/dL. During the hospitalization, this treatment was extended for 10 days.

A large gastric ulcer in the body of the stomach was confirmed by upper gastric endoscopy. Treatment with pantoprazole (20 mg daily) was initiated. Intravenous infusion of packed red blood cells and fresh frozen plasma was also prescribed based on the patient's symptoms. After seven days, he experienced one episode of tonic-clonic seizure, as well as altered visual acuity. Fundoscopic examination revealed mild pupil edema. Further evaluations, including computed tomography of the brain and a chest X-ray, were normal. Evaluation of the blood lead level confirmed a level of 19 mg/dL on the ninth day.

By day 13, after 11 days of initiation of dimercaprol and other therapies, the patient's symptoms had gradually faded, and he was transferred back to the toxicology ward. Due to continued leukopenia and hypochromic anemia, a bone marrow biopsy was proposed after ruling out dimercaprol-induced leukopenia, as well as anemia. Considerable declines in daily counts of white and red blood cells were observed after these events, with WBC and Hb levels falling to 1.2 $\times$ 10^3/L and 6.08 g/dL, respectively, after 21 days. On day 22, hemodialysis was performed. The blood lead level was 41.6 μg/dL by the next day. On day 31, after seven days of follow-up, the patient was discharged with his responsible family members but was instructed to keep a follow-up visit at the internal medicine clinic. The patient was examined one and six months later, and physical exams and routine tests, such as CBC, BUN, Cr, and electrolytes, were normal.

3. Discussion

This report describes the clinical history of a patient poisoned with a mixed hazardous substance, who was treated successfully with multiple sessions of hemodialysis. MOT due to mixed various agents is rare and requires emergency medical management, including rapid lavage, hemodynamic balance, and even continual hemodialysis in the acute condition. In this case, several compounds with unknown effects were involved and the clinicians were faced with a very difficult decision.

The main cause of the patient's deterioration was his delayed presentation to the hospital, which led to multiorgan damage. The first impaired target organ was the stomach, which showed a severe ulcer. Previous studies have shown that the main materials used in explosive agents are lead, sulfur, ammonium nitrate, magnesium, HMX, and tetryl (3-7), which are strong gastric irritants that can have destructive effects on gastrointestinal mucous membranes and cells (4). Although stomach ulcerations may confirm acute lead poisoning, other factors to consider are reactions between Pb_3O_4, aluminum, and magnesium, which have a volcanic reaction that leads to more stomach damage. Hence, it seems that complications and adverse effects of these mixed agents could be reduced by early appropriate lavage. Our patient also showed proteinuria and many erythrocytes in the urine, confirming nephrotoxicity and renal failure as the second step of organ toxicity. The symptoms of acute lead and chemical agent poisoning may be multiple and can have tremendous effects on different organs, but may have obvious effects when they enter into blood circulation. Hemodynamic disorders in our patient, such as hemolysis, leukocytosis, and imbalanced electrolytes, could be assumed to be due to the entrance of these hazardous substances into the bloodstream, rupturing the blood cells. As reported by other authors, hypoactivity of aminolevulinic acid dehydrase, which involves heme biosynthesis, was observed after lead poisoning (8). The significantly decreased Hb in our case could be explained partly by this mechanism. Also, the direct toxicity

of lead, sulfur, and other chemical components can be involved in the induction of hemolysis and renal failure. The efficacy of chelating therapy for the remission of acute lead nephropathy is unclear, but anemia and renal failure have been observed in severe lead poisoning; however, studies on the role of lead as a renal toxic agent are scarce (8-10). We do not have access to succimer, a dimercaprol analogue, for the treatment of lead toxicity. In our patient, proteinuria, hematuria, decreased hemoglobin, increased urea, and increased Cr emphasized that the nephropathy might be created alone or by synergistic effects between lead and other compounds, such as barium nitrate and HMX (5, 11). Previous studies have implied that treatment with chelating agents may reverse acute lead nephropathy (9). Hence, it was considered that the chelating therapy with dimercaprol might have induced leukopenia and nephropathy, and the patient's situation improved after discontinuing this therapy. There are some controversial studies related to the adverse effects of EDTA therapy in patients with renal insufficiency (9). Hence, we did not use EDTA for chelating therapy in this case. Moreover, kidney damage from Aklilsorang might be due to the barium nitrate and HMX used in this compound, which have known nephrotoxic effects (5, 11).

Hepatitis, liver cancer and biliary tract obstruction were ruled out by complementary tests and sonography in our patient. Acute liver exposure to different substances with various harmful properties may have been the main cause of jaundice, ascites, hyperbilirubinemia, and high LDH in this case. Hepatocellular damage and pathological changes in the liver and kidney have been observed due to tetryl, HMX, and ammonium nitrate in animal studies (4-6). Therefore, it seems that the efficacy of the multiple hemodialysis treatments could be due to the elimination of such harmful agents from the circulating blood. Hepatocellular damage with an increased rate of hemolysis in our patient showed that the pathology was located within the liver. Generally, kidney diseases such as hemolytic uremic syndrome can also lead to coloration, but in our case, this was ruled out (12, 13). One step that was not performed in this case was an analysis of the ascitic fluid, which is recommended in poisoned patients. Also, the patient's visual problems might have been due to unknown effects of the involved substances, but we did not find any reports on this in our extensive literature search.

Finally, this patient's MOT may have been induced by imbalances in electrolytes, hemodynamic disorders, and/or neuropathy, which could have induced the seizure and led to coma. The convulsion effects of barium nitrate, HMX, RDX, Pb3O4, sulfur, aluminum powder, magnesium, ammonium, and lead nitrate have been reported in previous studies (3-6, 11). Therefore, the seizure that occurred after Aklilsorang ingestion in this case could have been due to the presence of these substances.

3.1. Conclusion

Multiple hemodialysis treatment had the greatest effectiveness in the management of this patient. Hence, in multi-substance toxicity without specific antidotes, hemodialysis seems to be the best therapy and can be proposed as the first-line treatment in such cases.

Footnotes

Authors' Contribution: Study concept and design: Mohammad Arefi; analysis and interpretation of data: Hamidreza Mohammadi and Mohammad Arefi; drafting of the manuscript: Hamidreza Mohammadi; critical revision of the manuscript for important intellectual content: Hamidreza Mohammadi and Mohammad Arefi; statistical analysis: Hamidreza Mohammadi; administrative, technical, and material support: Hamidreza Mohammadi and Mohammad Arefi; study supervision: Mohammad Arefi.

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