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Case Report

A rare case of 2, 4-Dichlorphenoxyacetic acid (2, 4-D) poisoning

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ABSTRACT

2,4-D (2,4-dichlorphenoxyacetic acid) is a selective herbicide which kills broad leaved plants but not grasses. It is not toxic to beneficial insects and has moderate mammalian toxicity. It is used widely in agriculture and forestry as an herbicide in either ester or salt formulation. Human poisoning with 2,4- D is rare and is generally following suicidal ingestion. Here we report a case of 2,4- D poisoning.

Keywords: Alkaline diuresis, Dichlorphenoxyacetic acid, Poisoning

INTRODUCTION

2, 4-Dichlorophenoxyacetic Acid (2, 4-D) is an herbicide in chlorophenoxy group which is used as a weed killer. It is used extensively in the northern part of India where wheat is mainly cultivated. It can be absorbed via the oral, dermal, and inhalation routes. Its toxic effects involve heart, central and peripheral nervous system, liver, kidneys, muscles, lungs and endocrine system.[1] Acute poisoning with 2, 4-D may be fatal in case of large ingestions. It is rarely reported as an agent used for attempting suicide. There is no specific antidote for 2, 4-D herbicide poisoning. We report here a case of 2, 4-D poisoning.

CASE REPORT

A 14-year-young male was admitted with alleged history of ingestion of about 50ml of 2, 4-Dichlorphenoxyacetic acid (2, 4-D) solution with suicidal intent. He became markedly restless and drowsy two hours after the ingestion of this poison. He then had multiple episodes of vomiting without any blood. Patient was given gastric lavage, injection atropine, and intravenous fluids outside this institute and was then referred to this hospital. On examination, he was hemodynamically stable but has altered sensorium. Blood pressure was 100/60 mmHg and the pulse was 122/min, regular, and of good volume. He has tachypnea with respiratory rate of 34/min. His pupils were mid dilated and showed sluggish reaction to light. There was no icterus, no cyanosis, and neck veins were not distended. The chest had bilateral vesicular breathing; abdomen was soft and without any organomegaly. On cardiac auscultation, the first and second heart sounds were normal in intensity without any audible murmur. Complete haemogram showed haemoglobin of 10.3 gm/dl, TLC - 10,900; and DLC revealed P84.6, L12.6, M2.5, E2. Platelets were normal (2,87,000/mm3). Biochemical parameters showed bilirubin 0.2 mg/dl, SGOT - 88 IU (normal: 20-40), SGPT - 34 IU (normal: 20 - 40), alkaline phosphatase - 321IU, Na+ -137 meq/l, K+- 3.8 meq/l, urea-26mg/dl, creatinine-0.63 mg/dl. Arterial blood gas analysis revealed PaO_2 - 103, $PaCO_2$ - 20, pH - 7.57, HCO_3 - 18.5, O_2 sat-99%. Electrocardiogram revealed sinus tachycardia with heart rate of 122/min. Urine complete examination and chest x-ray were normal. He was started on forced alkaline diuresis along with antibiotics for suspected aspiration pneumonia. However, his sensorium continued to deteriorate and went into Coma stage 4 after that he

developed respiratory depression for which he was intubated and managed with mechanical ventilation. He continued to deteriorate despite supportive measures and died on the next day following a cardiac arrest.

DISCUSSION

2,4-D (Dichlorophenoxy acetic acid) which was used as a regulatory chemical for the growth of plant at the beginning was developed by Zimmerman and Hithoock in 1942 and was used as an herbicide by Hammer and Tukes in 1944. The common formulations are either solid alkali salt concentrate, salt miscible solution, or as ester-based emulsifiable concentrate.[1] It can be absorbed via the oral, dermal, and inhalation routes. The toxic and lethal levels of 2, 4-D in human blood and tissues are still not well defined. In general, the acute lethal levels of 2, 4-D in the plasma appear to lie between 447 and 826 mg/litre. Blood levels of 2, 4-D can be measured most accurately with gas liquid chromatography with electron-capture detection (GLC-EC).[1] Direct toxic effects on the gastrointestinal tract cause nausea, vomiting, abdominal or throat pain, and diarrhea. Severity of GI manifestation has varied depending on the dose with peak of 12 to 24 h after ingestion and may persist for a number of days.[2] Other reported clinical manifestations include myalgia, rhabdomyolysis, weakness, myopathy, myotonia, fasciculations, agitation, sedation, confusion, miosis, tachycardia, hypotension, renal toxicity, hypocalcemia, and hypokalemia. Hypotension, which is common, is due predominantly to intravascular volume loss, although vasodilatation and direct myocardial toxicity may also contribute. It also produces haematological and biochemical disturbances.[3]

Mechanisms of toxicity including dose dependent cell membrane damage, uncoupling of oxidative phosphorylation and disruption of acetyl-coenzyme A (acetyl-CoA) metabolism.[4] The initial clinical manifestations of 2, 4-D poisoning are very similar to poisoning with anticholinesterase compounds, making it even more difficult for the treating physician to suspect poisoning due to these compounds. Muscle involvement occurs in the form of muscle fibrillation, myotonia, loss of reflexes, and muscular weakness. Skeletal muscle damage results in increased levels of creatine kinase and myoglobinuria which, in turn, leads to renal failure.

Hypertonia, hyperreflexia, ataxia, nystagmus, miosis, hallucinations, convulsions, fasciculation, and paralysis may present at variable intervals during the course of systemic toxicity.

The management is mainly supportive in the form of maintaining hydration, assisting respiration, and preventing arrhythmias and aspiration, as no specific antidote is available. However, forced alkaline diuresis or hemodialysis can enhance its excretion.[5,6] Urine alkalinisation with high flow urine output may possibly enhance herbicide elimination and should be considered in all seriously ill patients.

CONCLUSION

To conclude, 2, 4-D is a rare agent used for attempting suicide. Initial manifestations with 2, 4-D poisoning are similar to anticholinesterase poisoning. The early recognition of signs of corrosive injury, tachycardia, muscle weakness, and CNS toxicity will help identify cases with 2, 4-D poisoning and guide the physician for proper management of these cases.

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