A case of 2, 4- Dichlorphenoxyacetic acid (2,4-D) ingestion masquerading anticholinesterase poisoning

Kumar H, Kala V, Jain R, Priyanka, Panjeta P, Punia N

ABSTRACT

2, 4-Dichlorphenoxyacetic acid (2, 4-D) is an agricultural herbicide widely used to kill broad-leaved weeds in wheat cultivation. Most of the reported cases of documented poisoning are from Europe and North America. However, less than fifty cases of poisoning with this compound have been reported from India. We report a young adult who ingested it with suicidal intent and developed neurological, cardiac and respiratory toxicities ultimately leading to death.

Keywords: dichlorphenoxyacetic acid, herbicide, poisoning, anticholinesterase

INTRODUCTION

2,4-Dichlorphenoxyacetic acid (2,4-D) is widely used as agricultural herbicides against broad-leaf weeds in cereal crops as well as on pastures and lawns, in parks, and on golf courses. It is used extensively in the northern part of India where wheat is mainly cultivated. The common formulations are either solid alkali salt concentrate salt miscible solution or as ester based emulsifiable concentrate. 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/liter. Its toxic effects involve heart, central and peripheral nervous system, liver, kidneys, muscles, lungs and endocrine system.[1] It is rarely reported as an agent used for attempting suicide. Most of the reported cases of documented poisoning due to 2, 4-D are from Europe and North America.[2,3] To the best of our knowledge, less than fifty cases of documented poisoning with this compound have been reported from India.[4,5]

CASE REPORT

A 19 year young man was admitted with alleged history of ingestion of about 50ml of 2,4-D solution with suicidal intent. He became markedly restless and drowsy two hours after its ingestion. He had then multiple episodes of vomiting without any blood. He was given gastric lavage, injection atropine 6mg and intravenous fluid in a private set up and was then referred to our hospital. On physical examination, he was restless and drowsy and hemodynamically stable. Blood pressure was 128/84 mmHg; pulse was 68/min, regular and good volume. He was tachypnoeic with respiratory rate of 32/min. His pupils were dilated and showed sluggish reaction to light. Diffuse ulceration was noticed in the oral cavity along with brownish discoloration of the lips. There was no icterus, no cyanosis and neck veins were not distended. Chest had bilateral vesicular breathing. Abdomen was soft without any organomegaly. On cardiac auscultation, first and second heart sounds were normal in intensity without any audible murmur. Complete haemogram showed hemoglobin of 13.5 gm/dl, TLC 7800 and DLC revealed P74 L22 M2 E2. Platelets were normal (1.97lac/mm^3). Biochemical parameters showed bilirubin 0.8 mg/dl, SGOT 73 IU (normal 20-40), SGPT 43 IU (normal 20-40), alkaline phosphatase 231 IU, amylase 88 IU (normal 0-82), Na+ 140 mEq/l, K+ 4.9 mEq/L, urea 26 mg/dl, creatinine 1.1 mg/dl. Arterial blood gas analysis revealed PaO_2 70.3, $PaCO_2$ 19; pH 7.475, HCO_3 13.5, O_2 sat 95.6%. Urine examination and chest x-ray was normal. He was treated with intravenous fluids and antibiotics for suspected aspiration pneumonia. However, his sensorium continued to deteriorate and 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. Police was informed for further forensic evaluation.

DISCUSSION

Although 2, 4-Dichlorphenoxyacetic acid (2,4-D) has been available as a herbicide for many years, there are few reported cases of occupational exposure and its use as a suicidal agent.[2-5] It can be absorbed via the oral, dermal, and inhalation routes. 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] Nausea, vomiting, abdominal pain, diarrhea and, occasionally, gastrointestinal hemorrhage are early effects following ingestion of these compounds. Hypotension, which is common, is due predominantly to intravascular volume loss, although vasodilatation and direct myocardial toxicity may also contribute. Its toxic effects involve heart, central and peripheral nervous system, liver, kidneys, muscles, lungs and endocrine system. It also produces hematological and biochemical disturbances. Mechanisms of toxicity includes dose-dependent cell membrane damage, uncoupling of oxidative phosphorylation and disruption of acetylcoenzyme-A metabolism.[3]

The initial clinical manifestations of its poisoning are very similar to poisoning with anticholinesterase compounds, making it even more difficult for the treating physician to suspect this poisoning. Our patient was also treated as a case of anticholinesterase poisoning by a private practitioner. He developed respiratory distress either due to CNS depression or due to respiratory muscle paralysis and was put on mechanical ventilation. 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. However, in the present case, there was no evidence suggestive of myotoxicity and renal failure. Metabolic acidosis, increased aminotransferase activity, pyrexia and hyperventilation have also been reported.[3]

The reported neurological effects are impaired coordination, unconsciousness and coma. Unconsciousness or coma may result from a direct CNS depressant action or a number of metabolic derangements in these patients. Hypertonia, hyperreflexia, ataxia, nystagmus, meiosis, hallucinations, convulsions, fasciculation and paralysis may present at variable intervals during the course of systemic toxicity.[5] Our patient developed respiratory distress which was attributed to respiratory muscle involvement.

There is no specific antidote available for 2,4-D poisoning. Management is supportive in the form of maintaining hydration, assisting respiration and preventing arrhythmias and aspiration. Since myoglobinuria produces nephrotoxicity, alkaline diuresis may be helpful in preventing renal damage.[6] Urine alkalinisation with high-flow urine output may possibly enhance its elimination and should be considered in all seriously ill patients. Although the exact mechanism is not clear, haemodialysis may help by eliminating the 2,4-D.[7]

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 in identify cases with 2, 4-D poisoning and guide the physician for proper management of these cases.

AUTHOR NOTE

Hitender Kumar, Assist. Prof., Medicine
(Corresponding author)
dr.hitenderkaushik@yahoo.in
Vikram Kala, Assistant Professor, Medicine
Rajeev Jain, Senior Resident, Medicine
Priyanka, Demonstrator, Biochemistry
BPS Government Medical College for Women, Khanpur Kalan, Sonepat
Prerna Panjeta, Final Yr. Resident of Biochemistry Pt. B.D. Sharma University of Health Sciences, Rohtak
Neetu Punia, Senior Resident, Gynecology & Obstetric, BPS Government Medical College for Women, Khanpur Kalan, Sonepat

REFERENCES

1. Environmental Health Criteria. 2,4-dichlorphenoxyacetic acid (2,4-D) 29. Geneva: WHO 1984.
2. Keller T, Skopp G, Wu M, Aderjan R. Fatal overdose of 2,4-dichlorphenoxyacetic acid (2,4-D). *Forensic Sci Int.* 1994;65:13-8.
3. Bradberry SM, Proudfoot AT, Vale JA. Poisoning due to chlorphenoxy herbicides. *Toxicol Rev.* 2004;23:65-73.
4. Singh S, Yadav S, Sharma N, et al. Fatal 2,4 D (ethyl ester) ingestion. *J Assoc Physicians India.* 2003;51:609–10.
5. Bhalla A, Suri V, Sharma N, Mahi S, Singh S. 2,4 D (Ethyl ester) poisoning : experience at a tertiary care centre in Northern India. *Emerg Med J.* 2008;25:30-2.
6. Flanagan RJ, Meredith TJ, Ruprah M, et al. Alkaline diuresis for acute poisoning with chlorphenoxy herbicides and ioxynil. *Lancet.* 1990;335:454–8.
7. Durakovic Z, Durakovic A, Durakovic S, et al. Poisoning with 2,4-dichlorphenoxyacetic acid treated with haemodialysis. *Arch Toxicol.* 1992;66:518–21.