Case Report
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Myotonia: A rare isolated clinical feature of systemic toxicity of 2, 4-Dichlorphenoxyacetic acid (2, 4-D) poisoning

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Abstract

There are variety of herbicides that are used to control the growth of weed in agriculture and forestry. 2, 4-Dichlorphenoxyacetic acid (2, 4-D) is one of these compounds. Despite its wide use, human poisoning with this compound is rarely reported. Acute poisoning with 2, 4-D compound may be fatal in large ingestion. Almost all of the reported cases of poisonings with this compound (most with suicidal intent) describe the multiorgan dysfunction. We report a case of poisoning with 2, 4-D compound having the myotonia as the isolated clinical feature of systemic toxicity and patient improving with conservative management.

Keywords: 2, 4-Dichlorphenoxyacetic acid (2, 4-D), Herbicides, Multiorgan failure, Myotonia, Forced alkaline diuresis.
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1. Introduction

There are many types of pesticides used in farming mainly insecticides, herbicides and rodenticides. 2, 4-Dichlorophenoxyacetic Acid (2, 4-D) is an herbicide, in Chlorophenoxy herbicide group. Human exposures to 2, 4-D herbicides can occur by ingestion, skin contact and inhalation. It is mainly used to control broadleaf weeds in cereal crops, pastures and lawns, in parks, and golf courses. Acute poisoning with 2, 4-D may be fatal in large ingestion.[1] There is no specific antidote for 2, 4-D herbicide poisoning.[2] We report here a rare case of 2, 4-D toxicity with myotonia as the only feature of systemic toxicity.

2. Case History

A 44 years male farmer was admitted with consumption of approximately 50 ml of 2, 4- D amine compound (MAHAN 580 58%) with suicidal intent. On admission after three hours, patient had burning sensation in throat and chest with five vomiting episodes. Initial vital signs were: Blood Pressure: 126/80 mmHg, Pulse Rate: 98 beats/min, respiratory rate 18 breaths/min, Axillary temperature: 37.4˚C and oxygen saturation 98% on ambient air. Physical examination revealed ulceration in the oral cavity with mild epigastric tenderness; rest of the physical and nervous system examination was normal. Patient was given gastric lavage with cleaning of skin contacts and removal of cloths and was started on intravenous fluids, pulse oximetry and cardiac monitoring. Investigations revealed Haemoglobin: 14.5 gm/dl; TLC: 8400/mm^3 with normal differential count. Platelets were normal (187000/mm^3). Blood biochemistry revealed Na+ 138 mEq/L; K+ 4.5 mEq/L; urea 30 mg/dl; serum creatinine: 1.1 mg/dl; serum bilirubin 0.7 mg/dl; SGOT 82 IU (normal 20-40); SGPT 31 IU (normal 20-38). ABG revealed PaO_2 97; $PaCO_2$ 33; pH 7.35 HCO3 22; O2 saturation 99%. Total CPK level was 109 mcg/L (normal10-120). Urine examination, ECG and chest x-ray were normal. Next morning, patient developed stiffness of limbs with difficulty in getting up from the bed and walking. On examination, patient had normal higher mental functions with normal cranial nerves. Motor

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system examination revealed normal muscle power with hypertonia and inability to release objects from hand suggestive of myotonia and percussion myotonia over thenar muscles. All the deep tendon reflexes were exaggerated and plantar responses were flexors. There was no evidence of sensory system and co-ordination defect and rest of the systemic examination was normal. EMG/NCV study confirmed myotonia (Figure 1). Repeat serum total CPK level was 227 mcg/L (normal 10-120) while rest of investigations were normal including serum electrolytes, serum creatinine and ECG. Patient was started on forced alkaline diuresis with sodium bicarbonate and all these features lasted for 48 hrs. Repeat EMG/NCV suggested resolution of myotonia. Further hospital course was uneventful and he was discharged on fourth day. Follow up after one week showed normal physical examination and investigations.

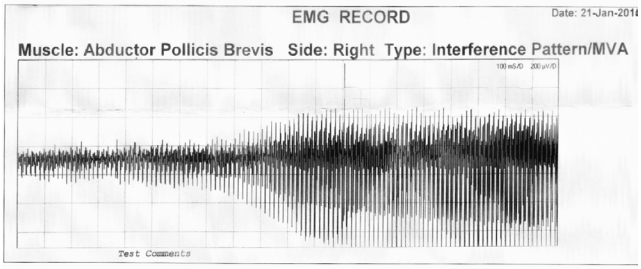


Figure 1. EMG record of Abductor Pollicis Brevis showing myotonia.

3. Discussion

Many kinds of herbicides are available in market such as: Bipyridyls (paraquat and diquat), Glufosinate, Glyphosate, Phenoxy compounds (2, 4- dichlorophenoxyacetic acid [2, 4-D], 4- chloro-2-methylphenoxyacetic acid [MCPA],2,4,5-trichlorophenoxyacetic acid [2,4,5- T], and mecoprop (MCPP; 2-[4-chloro-2- methylphenoxy] propionic acid. Triazine and Phenoxy compounds are selective herbicides; out of which 2, 4-D is widely used. Human poisoning is rare and generally follows suicidal ingestion or occupational exposures.[1, 2] Usually, 2, 4-D is well absorbed from gastrointestinal tract. It is not stored significantly in fat and excreted almost entirely in urine. There is limited biotransformation in the body and metabolites are highly protein bound. The average half-life of 2, 4-D in humans is 13 to 39 hours. Excretion is greatly enhanced in alkaline urine and half-life is prolonged to 70-90 hour in acidic urine.[3]

2, 4 D moderately irritates the skin and mucous membranes. Inhalation of sprays causes burning sensations in the nasopharynx and chest with coughing. Manifestations of systemic toxicity are known mainly from clinical experience with cases of deliberate suicidal ingestion of large quantities. Most reports describe occurrence of renal failure, metabolic acidosis, cardiac arrhythmia, rhabdomyolysis, electrolyte imbalance, and resultant multiple organ failure.[1, 2] Patients present within a few hours of ingestion with vomiting, diarrhoea, headache, confusion, and bizarre or aggressive behaviour. Muscle weakness and peripheral neuropathy have been reported after occupational exposure.[3] Convulsions may occur rarely. Mental status changes occur with progression to coma in severe cases.[3] With effective urinary excretion, consciousness usually returns in 48-96 hours.[3] Skeletal muscle injury can cause elevated creatine phosphokinase, and sometimes myoglobinuria. Muscle involvement occurs in the form of muscle fibrillation; exaggeration of reflexes, muscular weakness, rhabdomyolysis and rarely myotonia.[4] Myotonia is reported rarely and may persist for few days to months.[4] Cases of renal failure are reported, often with accompanying electrolyte imbalance which may lead to cardiovascular instability and death.[1,3] The pathophysiological mechanisms of toxicity is dose dependent cell membrane damage, uncoupling of oxidative phosphorylation and disruption of acetyl-CoA metabolism.[1] Our patient presented with local injury and had developed hypertonia and myotonia, confirmed with EMG/NCV findings. However, there was no evidence of any other systemic toxicity. With the best of our knowledge, we could not find any case report of 2, 4 D poisoning having hypertonia and myotonia as the only feature of systemic toxicity. There is no specific antidote available for 2, 4-D poisoning and management is supportive in the form of maintaining hydration, assisting respiration, and preventing arrhythmias and aspiration.[2,4] Since myoglobinuria produces nephrotoxicity and urinary alkalinisation with high flow urine output may enhance its elimination and prevent renal damage.[3-5] Haemodialysis may be helpful but exact mechanism is unclear.[5] Most of the reported cases of human toxicity in past have described the fatal nature of poisoning with multiorgan dysfunction and death of the patient.[1,3] However; cases reported in recent past have described the successful outcome and most of them were treated with

forced alkaline diuresis.[3,4] We treated our patient with forced alkaline diuresis and he improved without any complication.

4. Key Messages

Despite widespread use of 2, 4-D, it is a rare agent used for attempting suicide. Initial manifestations are often non specific. Though it is thought to be fatal poisoning, cases can present with isolated hypertonia and myotonia and can be managed with forced alkaline diuresis and supportive.

5. Conflict of Interest

None Declared

6. References

1. Keller T, Skopp G, Wu M, Aderjan R. Fatal overdose of 2,4-dichlorphenoxyacetic acid (2,4-D). Forensic Sci Int 1994; 65:13-18.
2. Bradberry SM, Watt BE, Proudfoot AT, Vale JA. Mechanisms of toxicity, clinical features, and management of acute chlorophenoxy herbicide poisoning: a review. J Toxicol Clin Toxicol. 2000; 38(2):111-22.
3. Flanagan RJ, Meredith TJ, Ruprah M, et al. Alkaline diuresis for acute poisoning with chlorophenoxy herbicides and ioxynil. *Lancet* 1990; 335:454-8.
4. Oghabian Z, Ghanbarzadeh N, Sharifi MD, Mehrpour O. Treatment of 2, 4-Dichlorophenoxyacetic Acid (2, 4-D) Poisoning; a Case Study. International Journal of Medical Toxicology and Forensic Medicine. 2014; 4(3): 104-107.
5. Nand N, Kumar H. A rare presentation of 2, 4-Dichlorphenoxyacetic acid (2, 4-D) poisoning. JIACM 2013; 14(2): 171-2