International Journal of Research in Medical Sciences
Tiwari A et al. Int J Res Med Sci. 2017 Oct;5(10):4652-4654
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20174616

Case Report

Case report- a rare survival of 2,4-D (ethyl ester) ingestions

Arun Tiwari*, V. B. Singh, Deepak Kumar, B. L. Meena

Department of Medicine, Sardar Patel Medical College, Bikaner, Rajasthan, India

Received: 11 August 2017
Accepted: 07 September 2017

***Correspondence:**
Dr. Arun Tiwari,
E-mail: dr.aruntiwari@live.com

ABSTRACT

2,4-Dichlorophenoxyacetic acid (usually called 2,4-D) is a widely used systemic herbicide. Ingestion of 2,4 D (Ethyl Ester) is rarely reported. Ingestion of this substance leads to neurotoxicity, cardiotoxicity, hematotoxicity and nephrotoxicity. Previously reported cases describe toxicities of this agent with very high fatality, only 2 survived case reports are available. In our case the substance was identified readily and treatment started within hours of ingestion with prompt gastric lavage and forced alkaline diuresis. At the time of admission patient had already lost consciousness (i.e. neurotoxicity), patient had developed muscle fibrillations and fasciculations (i.e. myotoxicity) and during the hospital stay he got acute kidney injury (i.e. nephrotoxicity) but all toxicities recovered with treatment. Previously reported fatal cases had late identification of substance and only symptomatic supportive treatment was given whereas we used aggressive approach with forced diuresis and haemodialysis. We are reporting third survived case of 2,4-D (Ethyl Ester) poisoning and emphasizing key points in the management, early identification of substance, early institution of forced alkaline diuresis and use of renal function tests as prognostic marker with timely hemodialysis.

Keywords: 2,4 D (Ethyl Ester) ingestion, Role of alkaline diuresis and haemodialysis, Rare survival

INTRODUCTION

2,4-Dichlorophenoxyacetic acid is one of the oldest and most widely available herbicides in the world. It was one of the contents of Agent Orange used by the USA in Vietnam war. Its chemical formula is $C_8H_6Cl_2O_3$. Ester forms are considered to be highly toxic. It selectively kills broad leaves weeds and spares grass, cereals. Very few cases have been reported with its poisonings and no specific antidote is available.[1,2] Ingestion in humans is not much studied but it is known to cause neurotoxicity, cardiotoxicity, hematotoxicity, nephrotoxicity and eventually death.[3,4] Reported neurological side effects include impaired coordination, confusion to coma. Myotoxic side effects include muscle fibrillation, myotonia, myoglobinuria and muscular dystrophy. Myocardial dystrophy, myocarditis, arrhythmia and pulmonary edema have been reported as myocardial toxicities. Liver necrosis and fatty changes were reported in fatal cases. Whereas, in most patients leading cause of death was Acute Kidney Injury (AKI) i.e. nephrotoxicity.

CASE REPORT

A 40 years old male farmer was brought to us in altered sensorium by his immediate relative with a history of unknown substance ingestion. He was taken to Primary care hospital where gastric lavage was done and intravenous fluids were given, thereafter, referred to tertiary care centre. When we received patient, exact history of ingested substance was not available at that time; patient was alcoholic and opium addict; patient was in altered sensorium having fasciculations in all four limbs, BP 100/70 mm Hg, Pulse 136 per minute, SpO_2 95%, Respiratory rate 21/minute, Random blood sugar 95 mg/dl, bilateral planters' extensor, Pupils equal and

reactive and Glasgow Coma Score was 8 (E2V2M4). Patient was given dextrose, naloxone and thiamine empirically but failed to respond; serum electrolytes: Na+ 139mmol/l, K+3.72mmol/l, Chloride 114.0mmol/l, Ionic Calcium 4.2 mg/dl, Calcium 9.6, Magnesium 2.4mg/dl; WBC 13700, Granulocytes 82%, Lymphocytes 13.9%, Hemoglobin 12.7g/dl, Platelets 3.11/mm cube, Blood urea 38mg/dL and Serum Creatinine 0.9mg/dL.

Table 1: Toxicities caused by 2,4 D (Ethyl Ester) Ingestion.

Toxicities	Manifestations
Neurotoxicity	Impaired coordination, confusion, coma
Myotoxicity	Myotonia, muscle fibrillations, fasciculations, muscular dystrophy
Cardiotoxicity	Myocarditis, pulmonary edema, arrhythmia
Nephrotoxicity	Acute kidney injury
Hepatotoxicity	Liver cell necrosis

Meanwhile, Box of the poison "Veerkill" 2,4-D (Ethyl Ester) was retrieved. This is a very rare poisoning to be reported. Correalting case, patient was unconscious and was having muscle fasciculations, hence patient had already developed neurotoxicity and myotoxicity at presentation. ECG, electrolytes, liver function tests and renal function tests were normal suggesting no toxicities of these organs at the time of presentation. According to the literature available, this poison leads to multi organ dysfunction syndrome followed by renal shut down and cardiac arrest. After studying literature thoroughly, we came know that there is a role of forced alkaline diuresis hence patient was given injection sodium bicarbonate with intra venous fluids and diuretics. Over 12 hours patient regained consciousness Glasgow Coma score 14 (E4V4M6).

On second day patient developed acute kidney injury had blood urea 98mg/dL and serum creatinine 5.0mg/dL; patient underwent 2 seasons of hemodialysis over next 2 days. On 4th day patient became fully conscious and renal function tests were improving blood urea 68 and serum creatinine 2.0. Patient became fully oriented to surroundings on day 4 and gave the retrospective history of consumption of same poison. During the hospital stay, the patient developed hypokalemia (K+2.7mMol/dL) which was managed with intra venous and oral supplementation of potassium. Patient's renal function tests and urine output also became normal and the patient discharged after 1 week of hospital stay with full recovery. In follow, up for 3 months no residual abnormality or carcinogenesis reported.

DISCUSSION

2,4-Dichlorophenoxyacetic acid compounds in Ester forms are very commonly used herbicides in agriculture of cereals and forestry. Very few cases have been reported with poisonings of 2, 4-D (Ethyl Ester). Poisoning is known to cause neurotoxicity, myotoxicity, hematotoxicity, cardiotoxicity, nephrotoxicity and toxic hepatitis.[5-7] Clinically unconsciousness, coma, muscle fibrillations, myotonia, arrhythmias, jaundice, decreased urine output, anasarca can be seen.

In investigations, there can be derangement in Liver Function tests e.g. SGOT, SGPT and alkaline phosphatase; acute kidney injury e.g. increased blood urea and serum creatinine. In our patient, we witnessed neurotoxic, myotoxic and nephrotoxic manifestations. No specific antidote is available till date. Forced alkaline diuresis and hemodialysis have been used in few cases.[8-10] Our patient improved with forced alkaline diuresis and hemodialysis. Acute kidney Injury being most common cause of death and renal function tests can be used as prognostic marker.

CONCLUSION

In cases of 2,4-D (Ethyl Ester) poisoning early gastric lavage and early institution of forced alkaline diuresis and hemodialysis can improve outcomes. Renal function tests can be used as prognostic marker in this poisoning as nephrotoxicity is most common cause of death.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Keller T, Skopp G, Wu M, Aderjan R. Fatal overdose of 2,4dichlorphenoxyacetic acid (2,4-D). Forensic Sci Int. 1994;65:1318.
2. Jorens PG, Heytens L, De Paep RJ, Bossaert L, Selala MI, Schepens PJ. A 2,4-dichlorophenoxyacetic acid induced fatality. Eur J Emerg Med. 1995;2:52-5.
3. Singh S, Yadav S, Sharma N, Malhotra P, Bambery P. Fatal 2,4-D (ethyl ester) ingestion. J Assoc Physicians India. 2003;51:609-10.
4. 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.
5. Nand N, Kumar H. A rare presentation of 2, 4-Dichlorphenoxyacetic acid (2, 4-D) poisoning. J Indian Academy Clin Med. 2013;14(2):171-2.
6. Singh S, Yadav S, Sharma N, Malhotra P, Bambery P. Fatal 2,4-D (Ethyl Ester) Ingestion. JAPI. 2003;51:609-10.
7. Nisse P, Cezard C, Peucelle D, Durocher A, Mathieu-Nolf M. [Fatal poisoning caused by the ingestion of a concentrated solution of 2,4-D and MCPP]. Acta Clin Belg. 2006;61(1):68-70.
8. Flanagan RJ, Meredith TJ, Ruprah M, Onyon LJ, Liddle A. Alkaline diuresis for acute poisoning with

chlorphenoxy herbicides and ioxynil. Lancet. 1990;335;454-8.
9. Durakovic Z, Durakovic A, Durakovic S, Ivanovic D. Poisoning with 2,4-dichlorphenoxyacetic acid treated with haemodialysis. Arch Toxicol. 1992;66:518-21.
10. Jearth V, Negi R, Chauhan V, Sharma K. A rare survival after 2, 4-D (ethyl ester) poisoning: Role of forced alkaline diuresis. Indian J Critical Care Med. 2015 Jan 1;19(1):57-8.

Cite this article as: Tiwari A, Singh VB, Kumar D, Meena BL. Case report- a rare survival of 2,4-D (ethyl ester) ingestions. Int J Res Med Sci 2017;5:4652-4.