CASE REPORT

Death Due To 2, 4-DI-Ethyl Ester Poisoning – A Rarely Documented Compound in Clinical and Forensic Practice

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Abstract:

Poisoning with Herbicides, Pesticides and Rodenticides etc. is very common nowadays especially in the northern parts of India due to their easy availability from market. Such compounds are available in the houses of farmers, persons dealing in agriculture sectors. Most of the time the cases of poisoning which are brought to our hospitals or are brought for autopsy in the mortuaries are of Organo phosphates, Organo chloro compounds or Aluminum Phosphide poisoning. In this case, we will discuss about the poisoning with a rarely documented compound 2, 4 Di Ethyl Ester which is a compound of Phenoxy acetic group. 2,4 Dichlorophenoxyacetic acid (2,4-D) is a selective herbicide used to kill broad leafed plants. The signs and symptoms of this compound mimics with anti-cholinesterase compounds which might be the reason for under reporting of this compound as a potential cause of poisoning. The role of preserving blood sample during post-mortem to look for myoglobin and creatine phyphokinase levels may be of help in giving opinion regarding cause of death in case of negative FSL's reports.

Keywords: 2, 4 Di Ethyl Ester; Herbicide; Phenoxy acetic group.

Introduction:

There are many pesticides and herbicides available for farming purpose. A herbicide, also commonly known as weed killer is a substance used to kill unwanted plants.¹ 2, 4-Di Ethyl ester is an herbicide of Phenoxy acetic group widely used in Northern India against broad-leafed weeds in cereal crops, lawns and parks. Ingestion, skin contact and inhalation are three main routes of human exposure to 2, 4-D herbicides. Very few documented poisoning cases with this compound have been reported. Chances of misdiagnosis are high as initial symptoms may mimic as that of anti cholinesterase poisoning.^{2,3} 2, 4-Di-Ethyl Ester (2, 4-D) is an herbicide with highly toxic effects. Complications occur due to difficulty in diagnosis. Management is only supportive with alkaline diuresis and treatment of complications as there is no specific antidote to this compound.^{4,5}

This poison has severe pulmonary, renal, neurological, gastrointestinal and myotoxic effects leading to multiorgan dysfunction and rapid death. Muscle fibrillations, myotonia, myoglobinuria and muscular weakness are the myotoxic effects leading to acute kidney injury.⁶

Case Description:

A 36 yrs. old male found unconscious on roadside during morning hours in August, 2020. A bottle of HEERA 44 (half

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Article History DOR: 24.11.2022 DOA: 01.07.2023 empty) was present besides the body. Patient was brought to the casualty of DRPGMC, Tanda by relatives with the history of consumption of about 150 ml of HEERA 44. Police was informed by the doctor posted in casualty and medico legal report was issued, gastric lavage and blood sample were preserved for chemical analysis.

As per hospital record, patient was admitted and treated symptomatically for 2, 4-Di-Ethyl Ester (HEERA 44) poisoning. Sodium bicarbonate 60 mg equivalent slow bolus intravenous stat was given after checking serum Potassium levels. The patient died next day and the body was handed over to police for further necessary action. Medico legal autopsy was conducted in the mortuary of Dr. Rajendra Prasad Govt. Medical College, Kangra at Tanda.

Post-mortem findings:

It was the dead body of a male, 162 cm in length, moderate built and moderately nourished. Rigor mortis was present over the upper part of the body. Post-mortem staining, purplish in colour was present over the back of neck, trunk and lower limbs except on the areas of contact flattening and was fixed. No injury was appreciable over the body externally except therapeutic puncture marks present over bilateral cubital regions.

Both lungs were intact and showed pulmonary oedema. Lower lobes of both the lungs were of liver like consistency. Stomach contained about 50 ml of brownish coloured fluid. Mucosa of stomach was congested and sub-mucosal haemorrhages were present. Abnormal smell was present from its contents. All other organs were congested. Urinary bladder was empty.

Samples sent:

Viscera and blood were preserved and sent for chemical analysis

to Regional Forensic Science Laboratory, Dharamshala through investigating officer of police. Blood sample was also preserved in plain vial to check Creatinine Kinase, Myoglobin levels and was sent to diagnostic lab in DRPGMC, Tanda.

Reports: 1. No poison or ethyl alcohol was detected in the Chemical analysis report issued from RFSL, Dharamshala.

- 2. Creatinine Kinase in serum of deceased: 5359 U/L(Normal range is less than or equal to 171U/L).
- 3. Myoglobin :>30000ng/ml (Normal range is 28.0-72.0ng/ml).

Discussion:

As 2, 4 Di Ethyl ester has been available as a herbicide for many years but there are only few reported cases of occupational exposure and its used as a suicidal agent. This can be due to the inability to distinguish clinically from the Organophosphorus compounds as the initial symptoms mimics with these compounds. Secondly, as our chemical analysis centres i.e. FSL's are not well equipped so that the wide range of compounds available in the market and consumed by the public by mistake or with suicidal intent could be detected on chemical analysis.



Figure.1 Shows congested mucosa of stomach along with sub-mucosal haemorrhages.

2,4-D is cardiotoxic, myotoxic, neurotoxic, hepatotoxic, nephrotoxic and also produces haematological disturbances. Since there is no specific antidote available, management is supportive in the form of maintaining hydration, supporting respiration, preventing aspiration. Since myoglobinuria produces nephrotoxicity, alkaline diuresis may be helpful in preventing renal damage. Urine alkalinisation with high-flow urine output may possibly increase the herbicide elimination and should be considered in all seriously poisoned patients. In addition haemodialysis can play a vital role in the management as myotoxic effect is the main feature of this compound.

No poison was detected in the viscera and blood of the deceased as per the chemical analysis report but the values of creatinine kinase and myoglobin were much higher than the normal serum value in the blood which was preserved and sent to diagnostic lab of DRPGMC Tanda. It shows that this compound is highly myotoxic.

Conclusions:

In cases of poisoning brought for treatment, where the diagnosis of organo phosphates and organo-chloro compounds is not clear to the clinician, the poisoning with 2, 4-dichlorophenoxyacetic acid compounds should also be kept in differential diagnosis. Alkaline diuresis may increase the elimination of such poisons and treatment for rhabdomyolysis may also be done in such cases. Even during post mortem examination, blood may be preserved to check Creatine kinase and Myoglobin levels in cases where the diagnosis of Organophosphates or Organo chloro compounds was not made by the clinician; as per treatment record. This poison may also be called as "Misdiagnostic poison." Besides stringent laws and their implementation at the ground level to regulate the sale and possession of herbicides, pesticides etc. are also the need of the hour.

FSL's should be well equipped so that the wide range of compounds available in the market and consumed by the public by mistake or with suicidal intent could be detected on chemical analysis. The number of groups/compounds which are checked by scientists at FSL's in routine should also be increased. The casualty/emergency department of every Medical College or even District hospital should have a poison information centre where the facility of detection of poisons should also be made available.

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