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A Case of 2, 4-D (Ethyl Ester) Poisoning Presenting Similar to Organophosphorus Poisoning - A Diagnostic Challenge

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Abstract: ***2, 4-Dichlorophenoxyacetic acid (2, 4-D). is a commonly available herbicide seen across North India. Although commonly used as a herbicide, instances of its poisoning and toxicity remain rare and survival rates among reported cases are small. Ingestion of this compound remains a diagnostic challenge due to the overlap of symptoms with Organophosphorus poisoning. Although the presentation might remain similar, treatment modalities and outcomes vary. A search of case reports available revealed the highly toxic nature of the compound with reports indicating pulmonary, renal and neurological side effects [1]. We here report of a 17-year-old female who presented with ingestion of an unknown poison that was initially treated as a case of Organophosphorus poisoning but eventually turned out to be 2, 4-Dichlorophenoxyacetic acid poisoning. The overlap of certain presenting symptoms and lack of awareness of ingested substance led to the initial therapeutic dilemma. This case was managed symptomatically and a trial of forced alkaline diuresis was carried out.***

Keywords: Poisoning, Organophosphorous, 2, 4-D (ethyl ester), Alkaline diuresis

1. Case Report

A 17-year-old previously healthy and intellectually normal female adolescent presented to our centre with alleged history of ingestion of a poisonous substance. Patient had ingested 50ml of the unknown substance around 2 hours prior to presentation. Gastric lavage was already carried out at primary health centre prior to presentation

On examination there was tachycardia with heart rate of 125/min. Saturation was maintained and BP readings were in normotensive range. Systemic examination was uneventful. GCS at time of presentation was 15/15

There was a strong smell from patient suggestive of OP poisoning but in the absence of any other features of poisoning patient was kept NPO and kept on observation. Routine investigations sent did not reveal any abnormality.

At around 4 hour of admission patient developed tachycardia, increased salivation and change in behaviour in the form of anxiety. GCS was 13/15. Keeping possibility of muscarinic symptoms of OP poisoning patient was started on Atropine and Pralidoxime as per protocol. Patient was fully atropinized until secretions were dry and continued on Atropine infusion and PAM infusion.

At 12 hours of admission the patient's father revealed the actual content of the poison which was 2, 4-D Ethyl ester. As the compound had a different mechanism of action from OP, atropine and PAM was discontinued. Patient remained irritable with GCS= 13/15

A search of literature revealed the high lethal nature of the poison and lack of any known antidote. Case reports revealed the usage of forced alkaline diuresis [2]. In the absence of any known antidote patient was started on forced urinary alkalinisation. Forced alkaline diuresis was carried out with the help of IV fluids, soda-bicarbonate and injection furosemide.

Patient gradually improved and within 48 hours was asymptomatic. Renal function tests, liver function tests and coagulation profile done showed normal parameters. Urine output was maintained.

2. Discussion

Poisonings are a common occurrence in our institute especially in the adolescent age group. Identifying the toxin involved is of paramount importance as it decides whether an appropriate antidote is available or not.

In this case the patient was initially managed as a case of OP poisoning owing to the symptoms and signs she presented with. Case reports indicate that 2, 4-D ethyl ester often behaves as an anti-cholinesterase but unlike Organophosphorus compounds no known antidote exists as of yet.

In our patient symptomatic management and forced alkaline diuresis was carried out with good effect and patient was discharged with no long term complications. There was an overlap of smell and symptoms between this poison and those seen with OP poisoning, namely tachycardia, increased salivation and worsening of sensorium.

This case goes to show that poisoning with 2, 4 D Ethyl ester should be considered a differential diagnosis to Organophosphorus poisoning when the exact contents of the poison remain unknown and we only have a clinical picture to work with.

Early identification of poisonous compounds, early and appropriate use of gastric lavage and use of forced alkaline diuresis can be associated with a good outcome in future cases of 2, 4-D (ethyl ester) poisoning. Thus on the basis of our experience we would advice to carry out prompt lavage and alkaline diuresis as possible modes of treatment if incidence of 2, 4-D ethyl ester poisoning is encountered.

References

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