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Alkaline Diuresis as Treatment for 2, 4-D Dimethylamine Herbicide Intoxication: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

The use of 2, 4-D dimethylamine is expanding as a result of the rise in glyphosate and paraquat resistance in weeds [1]. Instances of 2,4D dimethylamine intoxication have been on the rise. This herbicide exhibits a range of dose-dependent harmful mechanisms, including cell membrane destruction, disruption of acetyl coenzyme A metabolism, and oxidative phosphorylation uncoupling [2]. The neurological system, heart, lungs, liver, muscles, and endocrine system may all be affected by toxic effects [3]. Results in male cases are fatal [2,4]. The usefulness of alkaline diuresis as a life-saving treatment for 2,4-D dimethylamine poisoning with severe symptoms is discussed in this paper. 2, 4-D dimethylamine intoxication can be fatal if not treated promptly. There is no particular antidote and alkaline diuresis shows good result in the patient who present with severe intoxication of 2, 4-D dimethylamine.

Keywords: Herbicide intoxication; 2, 4-D dimethylamine; alkaline dieresis; chlorphenoxy.

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1. INTRODUCTION

Increasing glyphosate and paraquat resistance has led to an increase in the usage of 2, 4-D dimethylamine [1]. Intoxication from 2, 4-Dimethylamine cases have been on the rise. This herbicide exhibits a range of dose-dependent harmful mechanisms, such as oxidative phosphorylation uncoupling, acetyl coenzyme A metabolism disruption, and cell membrane destruction [2]. The neurological system, heart, lungs, liver, muscles, and endocrine system may all be negatively impacted by toxic effects [3]. The outcomes are deadly when they occur to men [2,4]. Intoxication with 2, 4-D dimethylamine can have severe symptoms, and this paper details show how alkaline diuresis can be a life-saving therapy.

2. CASE PRESENTATION

A 22-year-old male from Western Uttar Pradesh was admitted to Lokpriya Hospital in Meerut (Reg No: RMEE1900995)with vomiting, stomach pain, fatigue, and chest pain. Atropine and stomach lavage was administered right away at a nearby hospital.

The patient was referred to Lokpriya Hospital in Meerut for further treatment after 36 hours of consuming the poison. The patient's background was investigated, and it was found that he had taken 100 ml of ZURA solution, which includes the active ingredient 2, 4-D dimethylamine 58% wsc, on purpose with the goal of killing himself due to family issues.

The patient presented to the emergency room conscious, with a 64 beats per minute pulse, 132/80 mmHg blood pressure, 22 beats per minute respiratory rate, 96% oxygen saturation, and 4 liters per minute oxygenation rate. Regular blood tests indicated leucocytosis and absolute neutrophilia. Haematuria was discovered during a regular urine check, although arterial blood gas readings were normal. Urine's ACR is 1279.4, its R/M volume is 30 ml, its color was clear, its specific gravity is 1.030, its sugar content is zero, and its RBC count is 15-20. The cardiac markers troponin-I 272 and CPK-MB 538 were found.

There was a diagnosis of transaminitis, stress ulcers, herbicide poisoning with sepsis, and attempted suicide in this patient. The patient was treated in the intensive care unit with oxygen therapy, dextrose normal saline intravenous fluids, piperacillin, tazobactam, levofloxacin, pantoprazole, and ondansetron. We performed a stomach lavage upon delivery of 1000 ml of fluid, the liquid changed color from blackish-brown to clear. Pantoprazole loading dosage, followed by a maintenance infusion, and sucralfate were used to treat stress ulcers. Tranexamic acid was also given out. The procedure of alkaline diuresis was started by intravenously administering 100 ml of one mEq/kg sodium bicarbonate in 0.9% normal saline. 500 ml of ordinary saline injected with 25 mEq of potassium and 75 mEq of sodium bicarbonate eight hours later completed the procedure. An intravenous dosage 20 mg of furosemide was given every 12 hours for four days. On the sixth day, the dosage was progressively reduced. Furosemide was then continued orally. Additionally, methylprednisolone is injected intravenously.

Post admitting, the patient's condition deteriorated during the next 36 hours, and his blood pressure fell to 80/50 mmHg. Despite prompt intubation, placing the patient on ventilator support (AC-VC mode), and giving 1500 ml of normal saline as fluid resuscitation, blood pressure did not reach the necessary level. The patient's systole goal pressure of >100 mmHg was attained after taking noradrenaline. Breathing acidosis was detected by blood gas analysis.

The patient's liver enzyme levels decreased four days after being admitted to the hospital. He started responding to therapy, became aware, was extubated, and was given BIPAP support.

He was transferred to regular ward on the seventh day after admission, and on the tenth day, he was released in stable condition.

3. DISCUSSION

Anticholinesterase poisons are the most common form of poisoning in India; however,herbicide poisoning is also a suicide method and is associated with high morbidity and death [1]. Paraquat and glyphosate are the two herbicidal toxins that are most regularly used [2].

Despite their accessibility and widespread usage, these herbicides are not often encountered as poisons. These substances seldom cause acute poisoning and the majority of instances end in death [4]. Paraquat and glyphosate were shown to be the two most popular herbicides employed as poisons in demographic research carried out in a tertiary care hospital in India [4].

S.NO	DATE	Pre-Hospitalization DAY-1	Pre-Hospitalization DAY-2	DAY-1	DAY-5	DAY-8
1	HB	13.9	11.7	12.2	13.5	16.2
2	TLC	20.8	15.4	17.4	13.9	18.7
3	NEUTROPHIL	83	82	94	87	92
4	LYMPHOCYTE	10	14	5	10	8
5	PLT	1.57	1.05	1.25	1.3	1.8
6	UREA	59.68	86.83	61	39	69
7	S. CREAT	1.27	1.14	1.22	1.3	1.5
8	S. NA	143	144	148	151	156
9	S.K	3.33	3.13	3.1	3	3.2
10	S.CA	8	8.06	8.8	10.5	9.4
11	T. BIL	0.67	0.63	1.29	0.97	1.23
12	SGOT	68.7	878.75	1131	451	119
13	SGPT	47.63	179.84	158	181	128
14	ALK	235	184.71	59	85	82
15	PRO T	7.12	6.39	6.54	7.47	6.62
16	ALB	4	3.85	3.85	4.1	3.57
17	A/G RATIO	1.28	1.52	1.43	1.22	1.17
18	PT TEST	17.54	14.67	15.5	15.4	-
19	PT INR	1.35	1.13	1.33	1.32	-
20	PT CONTROL	13	13	12	12	-

Fig. 1. Investigations

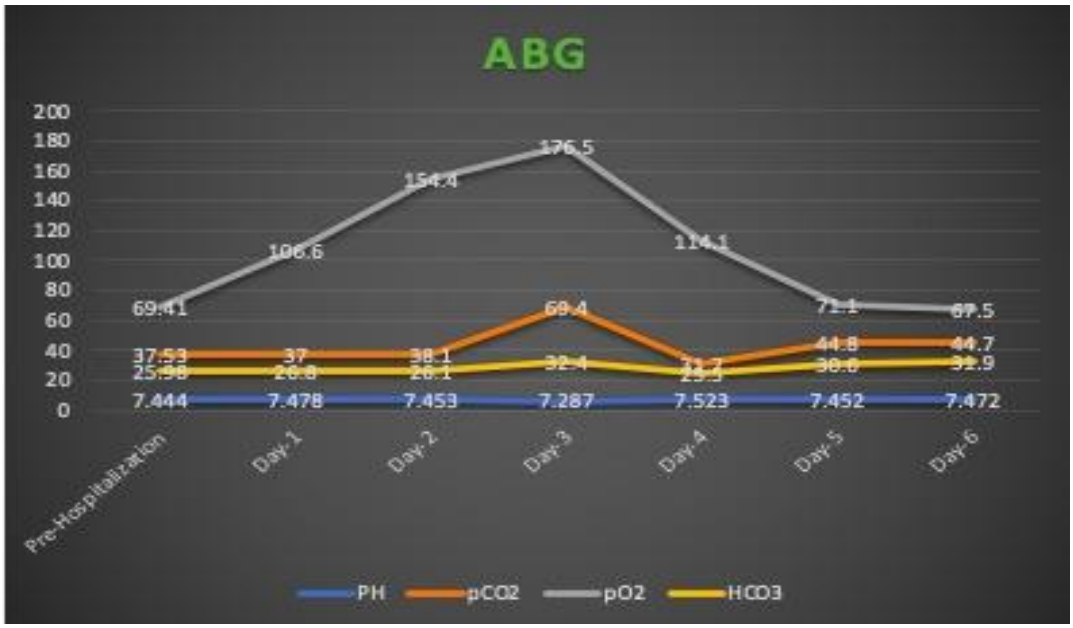

Fig. 2. ABG

The toxicity associated with ingestion, whether accidental or purposeful, is made worse by the high concentration of the active ingredient as well as the stabilizers, emulsifiers, and solvents [5].

The patient received an immediate diagnosis based on her medical history; prompt identification of the causative agent is essential for successful treatment. There was a significant amount of herbicide (100 ml) consumed since this was an attempt at suicide. The neurological, cardiac, gastrointestinal, and respiratory systems all showed toxic consequences in this situation. Additionally, septic shock was identified as a result of symptoms, a physical examination, and laboratory results. A direct CNS depressive activity or patient metabolic abnormalities may cause patients to become unconscious or go into a coma. During the progression of systemic poisoning, hypertonia, hyperreflexia, ataxia, nystagmus, miosis, hallucinations, convulsions, fasciculation, and paralysis may manifest at varying intervals [6].

In our situation, urine alkalinization produced a positive result. One method of improving elimination is urine alkalinization, which may be helpful in cases of poisoning from substances like phenobarbital, chlorpropamide, salicylate, and chlorophenoxy herbicides, particularly 2, 4-dichlorophenoxy acetic acid and mecoprop [4].

A particular remedy for 2, 4-D dimethylamine poisoning has not yet been discovered. The same form of chlorophenoxy, a weak acid (pKa 2.6 for 2, 4-D), is eliminated in the urine. The action of sodium bicarbonate intravenously is to raise urine pH. When urine is alkaline (63 ml/min at pH 8.3), renal excretion is more than when urine is acidic (0.14 ml/min at pH 5.1) [2]. The predicted increase in 2, 4-D clearance by the kidney for every unit rise in urine pH is over five-fold [3]. In order to enhance the excretion of 2, 4-D dimethylamine, sodium bicarbonate should be administered with a goal urine flow of 4-6 ml/minute. This patient's renal clearance and 2, 4-D half-life could not be calculated since the urine pH following alkaline diuresis was not assessed in this case. Hypokalaemia may develop during alkaline diuresis; hence potassium should also be administered together with sodium bicarbonate, per the literature [2].

Supportive care may be sufficient in moderate cases of intoxication, while alkaline diuresis may be used in severe cases [4]. To choose the best therapy, there is no severity rating to use as a guide. In severe 2, 4-D poisoning, alkaline diuresis, in particular, may be lifesaving [7].

Ion trapping in the plasma may also serve to restrict the spread of phenoxy chemicals from the central circulation [7,8].

Intoxication with 2, 4-D dimethylamine is not likely to end well if shock and unconsciousness occur. Alkaline diuresis administration when necessary, nevertheless, can be life-saving. Despite the initial acute toxicity, recovery may range from a few weeks to months [2,4].

4. CONCLUSION

In Western Uttar Pradesh (the sugarcane area), 2, 4-D dimethylamine poisoning is uncommon and has no known cure. It has a significant morbidity and death rate. The patient could be saved by an early diagnosis and urine alkalization with high flow pee. Our patient, who had a terrible prognosis before forced alkaline diuresis, was saved. Alkaline diuresis must be used in conjunction with several treatments, such as early emergency resuscitation, gastrointestinal system purification, and supportive care.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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