Leukopenia, Hypomagnesemia in 2,4-D Poisoning-A Rare Presentation

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Abstract

2,4-D(2,4-Dichlorophenoxyaceticacid) is a common herbicide in agriculture field. It has moderately toxic effect on mammals.2,4-D is available either in salt or ester formulations for field level use .Although no specific antidote available for this herbicide, forced alkaline diuresis is one of the modalities of treatment to promote early excretion of toxin through kidney and minimize its complications .Other management are essentially supportive measure and prevention of possible complications like aspiration pneumonia .There are a numbers of complications that are reported in varying degree .It affects skeletal muscle ,cardiovascular ,gastrointestinal , renal ,central and peripheral nervous system. Here we report a case who developed unusual complications like leukopenia, hypomagnesemia, proximal muscle weakness after consumption of 2,4-D after suicidal intent.

Keywords:2*,4-D formulations, leukopenia, hypomagnesemia.*

Introduction

2,4-D is a selective broad-leaved weed killer[1]. It is marketed as ester or salt compound in India. The ester formulation is commonly available in North India, whereas in Western part of Odisha state, which is an important agricultural belt of state, both forms are available[2]. Here we report a case who consumed 58% salt formulation of 2,4-D (Weedmar-super58%SL™) for suicidal intent.

Case Report

A 24-year-old lactating mother after quarrel in the family consumed around 30 ml of 2,4-D, methylamine 58% salt and brought to emergency Department of VSS Institute of Medical Sciences and Research, Burla, Odisha. During admission she was anxious, confused and restless. General examination showed pulse rate-88/min; regular, blood pressure (BP) -100/70mm of Hg, respiration rate-22/min, without pallor, icterus, clubbing, cyanosis, and pedal edema. There was no engorgement of neck veins or thyromegaly. Examination of respiratory, cardiovascular, gastro-intestinal, and central nervous system did not show any abnormality. Investigations showed Hb-11.4gm/dl, differential leukocyte count (DLC)-$N_{79}L_{16}M_{5,}$total leukocyte count (TLC)-9100/cu.mm., total platelet count (TPC)-2.24L/ml, blood urea-50mg/dl, serum (s.) creatinine-1.1mg/dl, s. Na^{+}-142meq/l, s. K^{+}-3.7meq/l, s.Ca^{++}-1.0meq/l, s.bilirubin (total)-0.39mg/dl,AST-32IU/L,ALT-23IU/L,ALP-87IU/L. Urine routine and microscopic examination, electrocardiogram were within normal limits. She was treated with gastric lavage, forced alkaline diuresis, iv fluids and inj. ceftriaxone 1 gm IV twice daily, Inj pantoprazole. She was put on nil per oral for next 48 hours. On 4th day she developed proximal muscle weakness. On examination she had lower motor neuron type of quadriparesis. On repeat investigations all investigations mentioned above were within normal limits except s.Mg^{++} which was low (1.3mg/dl; normal1.8-2.3mg/dl) and leukopenia (TLC: 2500/cmm). She was given magnesium 2 gm three times a day orally. After 24 hours, the power was improved to normal. On 8thday there was improvement of muscle power to normal, improvement of leukopenia and s.Mg^{++} to normal range.

Discussion

2,4-D compounds are available either as ester or salt

formulation for commercial use as weedicide for broad leaved weed in agriculture sector. Most case reports done have shown ester compound as culprit. Here in this case a lactating mother consumed salt compound (2,4-Ddimethylamine salt 58%). The lethal dose of 2,4-D lie between 447-826mg/l in plasma[4]. Nausea, vomiting, diarrhea, gastrointestinal hemorrhage are early effects of these compounds[3]. It affects skeletal muscle, kidney, heart, liver, central and peripheral nervous system. Hypokalemia, hypomagnesemia, hyperthermia, leukopenia, thrombocytopenia are rare complications reported[5]. There is no specific antidote available. Supportive measure included alkaline diuresis, assisting respiration, and prevention of aspiration and arrythmia[1 3]. The mechanism of injury by 2,4-D are cell membrane damage, uncoupling of oxidative phosphorylation and disruption of Acetyl coenzyme-A metabolism[6]. Hypomagnesemia usually mimics hypocalcemia, hypokalemia. The present patient developed hypomagnesemia leading to proximal muscle weakness and leukopenia. Although 2,4-D itself can cause myopathy leading to muscle weakness we attribute it to hypomagnesemia as the muscle weakness improved to normal with magnesium supplementation[5]. The transient leukopenia developed in first week (4thday) returned to normal in second week (8th day) prompting to search for such complications in patients of 2,4-D poisoning for proper management. This case highlights that muscular weakness may be due to hypomagnesemia which can be treated with magnesium replacement. Further this observation encourages to search whether the salt and ester formulations have a different clinical profile and whether they have different degree of affection to different systems in humans.

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Ethical Clearance - Taken from virec committee.

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