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Case Study

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CASE OF PLETHORA POISONING

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ABSTRACT

We describe the management of an interesting case of poisoning of Plethora which is an oxadiazine insecticide

KEYWORDS: Plethora, methemoglobinemia.

INTRODUCTION

Plethora is broad-spectrum insecticide which has phytotonic [the additional positive advantages in growth and development] effect on the crop. It mainly acts as a chitin synthesis inhibitor and affects the insect nervous system by inhibiting entry of sodium ions into nerve cells, thereby hampering the insect's moulting & also paralysing the insect.[1] In our case we discuss about a patient who had consumed plethora along with alcohol and presented to our casualty.

Clinical case

A 65 year old male who is a known alcoholic was bought to casualty in a state of altered sensorium by his attenders with alleged history of consumption of plethora insecticide mixed with alcohol at his residence followed by which he vomitted and became unresponsive due to which he treated in a local hospital before bringing them to MVJ. He was evaluated clinically where he was found to have low GCS with Pulse Rate 88 with BP of 170/90mm of Hg with saturation at 84% at room air and 93% with 15 L of O2. He was immediately shifted to ICU.

In ICU, because of low GCS, decision was taken for Endotracheal Intubation And Ventilator Support. After taking the consent from attenders, he was put on Mechanical ventilator with Fi02 of 100% despite which his saturation was only 88%. Arterial blood gas analysis revealed a PaO2 of 298 mmHg (on a FiO2 of 100% and SpO2 89%).

So Methemoglobinemia was considered secondary to ingestion of Plethora component- Indoxacarb. Blood was sent for methemoglobin levels. Methemoglobin level was found to be 34% and Glucose 6 phosphate dehydrogenase (G6PD) level was normal, so Inj. Methylene Blue was administered intravenously at a rate of 1mg /Kg With Of 100 Ml Normal Saline And Inj.Vitamin-C 1g IV OD was started, followed by Inj. Methylene blue 60 mg IV BD for 2 days and Inj. Vitamin-C 1g IV continued. Other routine investigations were sent, which showed Hb of 10, TLC 14000, Creatinine 1.0, LFT showed SGOT 51, SGPT 22, Na 143, K 3.3. The patient gradually started showing signs of improvement and his SpO2 gradually improved to 92-94% on day three. On Day 3 he was extubated, and was maintaining Spo2 of 93% with 8 L of O2. On day 4, he was shifted to ward with O2 support. On fifth day, repeat methemoglobin level was done which was 2%. After 2 days of ward stay he was weaned off from O2 and was discharged at request in a hemodynamically stable state.

DISCUSSION

Methemoglobinemia most commonly results due to exposure to an oxidizing agents, but may also arise due to genetic, dietary, or even idiopathic etiologies. Common insecticides causing methemoglobinemia are – Paraquat, Indoxacarb and Aluminum Phosphide. In such cases, patient usually presents with complaints of headache, nausea, vomiting, cyanosis, altered mental state eventually progressing to drowsiness, coma, and death.[2]

Plethora is a lepidopteran insecticide containing (a) Novaluron, which inhibits chitin formation, targeting specifically larval insect stages that actively synthesize

chitin, (b) Indoxacarb which block voltage-dependent sodium channels, leading to severe dehydration and death. Our patient had consumed this compound along with alcohol as a suicide attempt, after which he was assessed and diagnosed to have meth hemoglobinemia after seeing the nail changes and dark brown colored urine.

Usually oxidation of the heme iron moieties to ferric state causes generation of Methemoglobin, leading to bluish-brown muddy color resembling cyanosis.

Hemoglobin carries iron in the ferrous (Fe2+) state, which is the reduced state of iron. Methemoglobin carries iron in the oxidized form, which is Fe3+. Oxidized iron (Fe3+) in meth-hemoglobin has high affinity to oxygen in blood, due to which the oxygen is not delivered to the tissues (oxygen dissociation curve shifted to the left) leading to hypoxia and tissue damage.[3]

Gold standard diagnosis is by co-oximetry blood gas analysis showing chocolate color blood, high or normal partial pressure of oxygen, and high methemoglobin level. The discrepancy between pulse oximetry and partial pressure of oxygen is due to the presence of methemoglobin in blood.

Treatment includes ceasing the offending agent, correcting the metabolic abnormalities, administering Methylene Blue at a dose of 1-2 mg/kg loading dose q 30-60 min, to a maximum of 7 mg/kg, followed by 50-100 mg twice or thrice daily along with other medications for symptomatic management. The principle of administering methylene blue is that, it gets reduced to leucomethylene blue which in turn reduces methemoglobin by NADPH reductase.[4] .If MHb level is high > 50% or if the clinical condition worsens then higher dose of methylene blue 2mg/Kg initially can be given. The dose can be repeated if symptoms fail to improve or high MHb levels persist. Much higher level of MHb > 70% may require transfusion or dialysis. Recognition is critical, since delay in treatment can lead to cardiopulmonary compromise, neurological sequelae, and even death of patient.[5] Methylene blue is contraindicated in G6PD positive cases it is also responsible for causing serotonin syndrome in patients using Selective Serotonin Receptor Uptake Inhibitors. As it inhibits the activity of MAO- A.[6]

Methylene blue reduces half-life of methemoglobin from 15-20 hours to 40-90 minutes. We also administered vitamin C 1gm/day and dextrose containing fluids for supplementing NADH/NADPH which is needed for reduction of methemoglobin by NADPH reductase enzyme. Test to check for methemoglobin levels were asked, but attenders were non compliant for personal reasons. However the condition of patient started improving so he was shifted and managed in wards. He was advised to continue 02 inhalation with other medications at the time of discharge.

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