Case Study

Venomous Remedy: A Case of Sodium Thio Sulfate and Methylene Blue Poisoning

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Abstract

Introduction: Deliberate self-poisoning by ingestion of chemicals used for agriculture, insecticides and disinfectants used in houses is a common modality of poisoning across the globe. Poisoning by tablets containing sodium thiosulfate and methylene blue used as water purifying agent (both compounds are used for treatment of cyanide poisoning) is rare. Here we report clinical profile, relevant laboratory investigations and management of a rare case of sodium thiosulfate and methylene blue poisoning (a first case of dual ingestion poisoning with above mentioned chemical and such reports are not availiable in current published literature).

Keywords:

Introduction

Deliberate self-poisoning (DSP) is a significant global health problem and it is estimated that it accounts for approximately 3,70,000 deaths per year in India.[1] Although Indian data regarding prevalence and implicating agents are lacking, it is widely believed that a vast majority of DSP is attributed to organophosphate, carbamate and corrosive poisoning.[1] At times, clinicians may encounter a rare and unknown offending poison, causing therapeutic dilemma. Here we report a rare and unique case of poisoning with sodium thiosulfate (STS) and methylene blue (MB) who presented with early onset altered sensorium, seizures and respiratory distress. Paradoxically the poisoning in our patient involved two substances which by themselves are used as antidotes. While STS which enhances endogenous cyanide detoxification is used to treat mild to moderate cases of cyanide poisoning, MB is a heterogeneous aromatic compound that is approved by the Food and Drug Administration (FDA) for

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methemoglobinemia and as an antidote to cyanide poisoning. In the absence of an antidote for poisoning by these two substances, therapeutic guidance and paucity of literature regarding this, we treated the patient symptomatically to which he responded.

Case report

Following a family dispute, a 20-year-old male consumed 100 tablets of THIO (Fig. 1) (each containing STS 0.0150 gms and MB 0.0001 gms) used for dechlorinating water. He was brought to the hospital two hours later in an agitated and confused state. The initial evaluation showed tachycardia (pulse: 176/minute), hypertension (156/88 mm Hg), tachypnea (respiratory rate: 34/minute) and the oxygen saturation at ambient air was 78%. The Glasgow Coma Score (GCS) was 13/15 (E4, V4, M5) and there was no focal neurological deficit. He was admitted to the intensive care of our hospital, two intravascular accesses were established, gastric lavage was performed followed by instillation of activated charcoal. The arterial blood gases showed metabolic and respiratory acidosis (Fig. 2) (pH: 7.23, pCO_2: 56 and HCO_3: 23.2).

Within an hour of hospitalization he was noted to have worsening sensorium with the GCS dropping to 8/15 and an episode of generalized tonic-clonic seizures. Rapid sequence intubation was concurrently performed to secure the airway. The patient was managed with Nasogastric tube insertion followed by gastric lavage with activated charcoal, benzodiazepine (lorazepam 4 mg sos) and was dilantanized (with 15 mg/kg that is approximately 1 gm sodium phenytoin over 30 mins) for seizure, broad spectrum antibiotics (ceftriaxone & clindamycin) in view of suspicion of aspiration pneumonia (respiratory suppor). Intravenous metoprolol for heart rate control, magnesium sulfate (2gm slow IV) for membrane stabilization and supportive therapy (fluids, proton pump inhibitors, anti-emetics) were also started. Post- intubation, patient was sedated with propofol and fentanyl, but patient continued to throw convulsive seizure. He was administered intravenous levetiracetam 2gm as loading dose following which seizures subsided. His urine output was adequate. Blood investigations revealed leukocytosis (TLC- 21,500/CC), Hemoglobin (Hb) 15 gm%, Hematocrit (PCV) - 44%, platelet – 2,23,000/cc, RBS- 122 mg/dl, urea/ creatinine- 16/1.0 mg/dl, sodium (Na)/potassium (k)- 142/3.6 meq/lit, Bilirubin Total/Direct – 0.4/0.1 mg/dl, SGOT/SGPT-33/43 IU/L, elevated Lactate Dehydrogenase (LDH) -1593 IU/L (Normal range of lab- 240-480 IU/L), C-Reactive Protein (CRP)- 0.5 mg/dl, Calcium – 10.4 mg/dl.

On the second day, patient remained on ventilator and under sedation but had persistent tachycardia. ABG revealed improvement in mixed acidosis. Lab parameters were suggestive of mild anemia (Hb- 12.5 gm%), rest of the metabolic and biochemical profile being within normal limit. Patient maintained good urine output. The chest radiograph was normal, ECG revealed normal sinus rhythm with sinus tachycardia (Fig. 3). He was continued on two groups of anti-epileptic drugs, antibiotics and supportive measures.

The patient was extubated on third day in view of normal vital parameters, improved sensorium and normal ABG. Patient had one spike of fever, which was managed with anti pyretics. Patient remained restless with emotional outbursts for which psychiatrist opinion was sought. He was started on oral feeding and Foley's catheter was removed. Thereafter he recovered well and revealed that he had consumed tablets with suicidal intention. All legal protocols were completed as per the hospital policy guidelines. He was later diagnosed as a case of schizoaffective disorder by the psychiatrist and was started on antipsychotic drugs.

Discussion

Self-poisoning is one of the oldest methods for committing or attempting suicide. Suicide attempts among young adults, especially in the age group of 21–30 years, may be due to unemployment, break up in the family support system, failure of love relations, an individual's job stress, difficulty in coping with some immediate situation, impulsive behaviors, etc. In any case of poisoning, the mortality and morbidity

relies on a number of factors such as type of poison, amount consumed, availability of medical facilities and the time of interval between intake of poison and arrival at health care facility, etc.[2]

STS is a cyanide poison antidote used along with sodium nitrite and has been conventionally used as an antidote in cyanide poisoning and as a nephron protectant during cisplatin administration.[3-4] It is also used for Calciphylaxis (also called as calcific uremic arteriopathy).[4] STS has known adverse effects gastrointestinal disturbance like nausea and vomiting, headaches, running nose and anion gap acidosis. [5]

MB is an organic chloride salt. It is a commonly used dye, which has medicinal use in view of its antimalarial, anti depressant and cardioprotective properties.[6] The intravenous MB has been approved by the FDA for management of methemoglobinemia in both pediatric and adult patients.[6] MB has off- label use in vasoplegic syndrome (generally defined as an arterial pressure <50 mm Hg, cardiac index >2.5 L /min/m^2 , right atrial pressure <5 mm Hg, left atrial pressure <10 mm Hg and low systemic vascular resistance <800 dyne/sec/cm^5), a type of distributive shock. MB increases systemic vascular resistance in epinephrine refractory cases.[7] It has been proposed that positive outcome of MB usage in vasoplegic shock is due to its blocking effect on nitric oxide synthase and guanylyl cyclase.[8] MB also finds use as an antidote for cyanide poisoning.[9] Adverse effects of MB include central nervous system-related symptoms such as Serotonin syndrome like diaphoresis, tremors, clonus (due to Monoamine oxidase inhibiting property), cardiac arrhythmia, hemolytic anemia, bluish discoloration of urine.[10]

Our patient developed tachycardia, tachypnea, hypoxemia, elevated Blood pressure and seizure. Tachycardia, elevated BP might have been due to anxiety. Tachypnea and hypoxemia occurred most likely because of aspiration pneumonitis. Occurrence of seizure and observed deranged lab parameters (drop in Hb and elevated LDH) could be attributable to interaction of active constituents of poison or direct effect of poison on nervous system and hematopoietic system respectively.

In our case, being a subzonal peripheral hospital, there was lack of facility for emergent drug level testing, and non availability of any definitive antidote for both the drugs, hence our patient was managed symptomatically with complete recovery.

Owing to the non-availability of an effective antidote of above mentioned poison to date, we emphasize early initiation of supportive management (activated charcoal, airway management, circulatory and hemodynamic support), membrane stabilizer like magnesium sulfate, intensive monitoring (laboratory and radiology) and potential role of anti epileptic drugs in decreasing the likelihood of fatal outcome.

Conclusion

Both STS and MB have various clinical use in modern medicine. As the drug used by our patient is being used for water cleaning purpose, caution should be taken with respect to storage of these drugs. It's not clear whether symptoms in our patient are due to drug interaction or adverse effect of individual drug. We should be cautious about the presence of anoxia due to status epilepticus effective sedation.

Statement of ethics: The written informed consent to publish the case was taken from the patient himself

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the manuscript for publication in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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