

A Case of Tablet Poisoning – Lethal Dose of Dapsone

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Abstract

We report a case of Dapsone poisoning, with Tablet Dapsone in a lethal dose with respiratory and cardiovascular effects, due to methemoglobinemia. We have treated this patient with methylene blue in the recommended dose. The patient was asymptomatic at discharge.

Keywords: Leprosy, Dapsone, Tablet Poisoning, methemoglobinemia, Methylene blue

Introduction

Tablets poisoning are usually with drugs such as antihypertensive, sedatives oral hypoglycaemic agents and psychiatric medicines. Dapsone poisoning is a rare entity nowadays. In earlier days cases of dapsone poisoning were more, because of increased prevalence of leprosy.

Case Report

On 9.6.2019, around 10.30 pm a 23yr old female attended the emergency department with an alleged history of ingestion of 20 tablets of dapsone around 9.15 pm. The drug prescribed for her husband was the source of the tablets. The patient vomited twice, half an hour after ingestion of tablets, contents were mainly of food particles. The case was managed by the duty physician and unit CRRI.

Clinical examination

Conscious, oriented, afebrile, had breathing difficulty and tachypnoeic. Heart sound heard tachycardia was present. Apart from some subjective dyspnoea, tachycardia and tachypnoea, there were no lung signs. The examination of the abdomen and nervous system were normal. In acute poisoning of dapsone, nervous system involvement can occur as restlessness, agitation, hallucination, blurred vision, ataxia and

choreiform movements. In our patient, there were no neurological symptoms. In the GI system, nausea, vomiting occurs, our patient vomited twice half an hour after ingestion of tablets. In the cardiovascular system, Tachycardia and hypertension can occur. In our pt BP was normal, Tachycardia was present. In the Respiratory system, the patient will be dyspnoeic. In our patient, the respiratory rate was 47 breaths/min¹

Vitals

- Heart Rate was 134bpm-regular
- Blood Pressure was 110/80 mmHg measured in the right upper limb in the supine position
- Respiratory rate was 47 breaths per minute
- Temperature not elevated
- Capillary blood glucose 111mgdl
- SpO2 87% in room air

The patient was given a stomach wash with saline and was on continuous ryles tube aspiration. On Nil per oral.

Iv fluids crystalloids 60ml per hour.

Methylene blue -sos in the case of methemoglobinemia.

Monitoring of vitals every half an hour.

Investigations

- Complete blood count: Hb: 10.9 g\dl, Tc : 9500, Pcv: 33%, Platelet: 2.4lakhs\cumm.
- Renal function test: normal.
- Liver function test: normal.
- Urine routine and examination does not reveal any abnormality.

Around midnight saturation began to fall and at 7am on the next day morning, saturation was 72%. Suspected methemoglobinemia with Dapsone toxicity.

On comparing normal versus patient's blood on bloating paper the paper showed chocolate brown colour in the patient's sample [Figure 1].

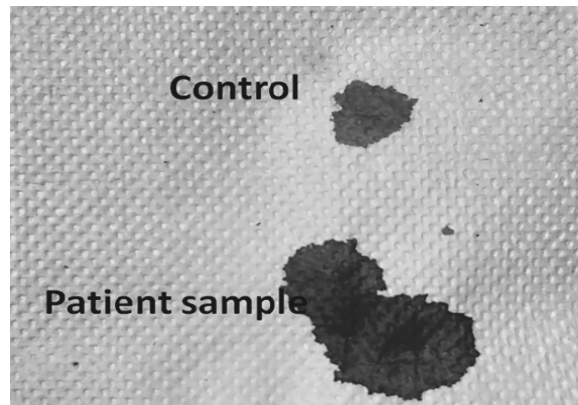


Figure 1

Management

An IV infusion of Methylene Blue 50mg in 200ml of DNS was started immediately. Nearing completion of methylene blue infusion around 8:30AM, the saturation was 90% from 72% before starting methylene blue. The heart rate was 60 per minute. The patient was comfortable and in sleep.

Discussion

Dapsone/diamino diphenyl sulfone(DDS) has indications for more than 20 clinical conditions, among them, indicated primarily for leprosy, other indications are Pneumocystis jiroverci, toxoplasmosis, acne, dermatitis herpetiformis. Dapsone is one of the drugs that undergo enteric recirculation². Activated charcoal was to be considered in case of life-threatening poisoning, as 50g oral (stat) and to be repeated every hour. In our patients, we have not used activated charcoal.

Dapsone produces prolonged methemoglobinemia and other adverse effects include Haemolytic anemia, agranulocytosis,

hepatitis, cholestatic jaundice, skin rash, peripheral neuropathy. Dapsone is increasingly implicated as a cause of MetHb because of its frequent usage in patients with AIDS.³

Methemoglobinemia was first described by FELIX HOPPE-SEYLER in 1864. For most patients with mild methemoglobinemia, no therapy is needed other than the withdrawal of offending agents. Abnormalities of vital parameters such as Tachycardia, Tachypnea, and Lactic Acidosis are thought to be caused by tissue hypoxia or functional anemia of methemoglobinemia and should be treated urgently.

Normal methemoglobin fraction is around 1%. At 3 to 15% slight discoloration of the skin occurs. At 15 to 20% causes asymptomatic cyanosis. At 25 to 50% of methemoglobinemia patients may be symptomatic with headache, dyspnoea, light headedness and confusion. More than 50% of methemoglobinemia abnormal cardiac rhythm may follow and mental status may be altered in the form of delirium, seizure, coma and metabolic acidosis. More than 70% of death is imminent. Oxygen saturation with ABG does not help full only spo2 monitoring will show a decrease in saturation. When MetHb concentration approached 20, the pulse oximetry indicated a spo2 of 90%. When MetHb concentration approached 30, the pulse oximetry indicated a decrease in spo2 to 85%. And then leveled off regardless of how much the MetHb levels began. This could be due to earlier spo2 monitoring our case it showed the detrimental value of 72%⁴.

Cases have been described with the occurrence of hemolysis following methemoglobinemia, however most poisons with Oxidant compounds do not manifest both types of toxicity. The combined occurrences are reported with dapsone. In our pt, there was no hemolysis.

Apart from dapsone other drugs that can cause methemoglobinemia are lidocaine,

prilocaine and benzocaine all of which are local anesthetics, antimalarials, metoclopramide, sulfonamides and nitrates. In the late 1930s, methemoglobinemia was recognized as a predictable adverse effect of Sulfanilamide use. Methemoglobinemia with hemolytic anemia was described following excessive Pyridium ingestion⁵

Methylene blue was recommended for methemoglobinemia. Methylene blue in high doses can itself cause methemoglobinemia. Methylene blue increases the rate of reduction of MetHb to Hb. Methylene blue reduced to leucomethylene blue by MetHb reductase in presence of NADPH. Leucomethylene blue then reduces MetHb to Hb⁶. Cimetidine is an inhibitor of cytochrome P50, which reduces MetHb levels at therapeutic doses can be used with Methylene Blue⁷. If methylene blue fails to control MetHb considers Exchange Transfusion or Hyper Baric oxygen.

Despite the rarity of dapsone tablet poisoning with a lethal dose, the patient was saved with timely administration of methylene blue. Our patient was discharged after a week of admission with a few days of vit-c in a stable state, with psychiatric counseling and advice.

Ascorbic Acid is not indicated in the management of acquired MetHb because VIT-C reduces MetHb, is considerably slower than the rate of normal intrinsic Mechanism⁸.



References

1. W.PILLAY. Modern medical toxicology –3rd edition 385 to 386.
2. Drucan Richards. Oxford Handbook of Practical Drug therapy 2nd edition, 698 -699.
3. EleonE, NeuvoenPJ. *Clin Toxicol* 1979; 14:79-85
4. 4. Barker SJ, TremperKK, Hyatt J. Effects of Methemoglobinemia on Pulse Oximetry and mixed venous oximetry. *Anaesthesiology* 1989; 70:112-117.
5. *Clin Paediatrics* 1971; 10:537-540
6. Clifton J, Leiken JB. Methylene Blue. *American Journal of Therapeutics* 2003; 10:289-91.
7. Rhodes LE, TingleMD, Park BK. *Br J Dermatol* 1995; 132:257-262 .
8. Bolyai JZ, SmithRP, GrayCT. Ascorbic acid and chemically induced methemoglobinemia. *Toxicol Appl Pharmacol* 1972; 21:176-185.

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