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CASE REPORT OF NAPHTHALENE POISONING LEADING TO METHEMOGLOBINEMIA

Emergency Medicine

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ABSTRACT

Naphthalene (C10H8) is a natural component of fossil fuels such as gasoline, diesel and coal. The most common consumer products made from mothballs are moth repellents and household deodorizing blocks in mothball or crystal form. The main toxic effect of naphthalene is due to the precipitation of acute intravascular hemolysis. Very few cases of naphthalene poisoning and its effects have been reported in India. We report a case of accidental naphthalene poisoning, who presented with methemoglobinemia.

KEYWORDS

Methemoglobinemia, Naphthalene, Methylene blue,Emergency Medicine.

INTRODUCTION

Methemogloblobinemia is a potentially life threatening condition which can present with nonspecific clinical features, the lack of specificity increases the probability of misdiagnosis and may cause delays in diagnosis and management. Methemoglobinemia is an increased concentration of methemoglobin in blood which is an altered state of haemoglobin where by the ferrous form of iron is oxidised to ferric state, rendering the heme moiety incapable of carrying oxygen leading to tissue hypoxia[1]

Acquired methemoglobinemia can result from exposure to a wide range of drugs and chemicals including nitrites, and other oxidants. Though most modern blood gas analysers have the ability to measure carboxyhemoglobin and methemoglobin levels without additional resources the lack of such resources especially in India does play an important role in outcome for patients[2]

We report herein a case of acquired methemoglobinemia caused after intentional ingestion of naphthalene balls. This case report highlights the importance of considering the possibility of methaemoglobinemia in all patients with exposure to naphthalene and biological compounds presenting with cyanosis and saturation gap.

Case Report

A 33 years old female with alleged history of ingestion of naphthalene balls (12-15 in numbers) 10-12hrs back. Patient presented complaining of increased mouth secretions, nausea, vomiting and abdominal pain. Patient was initially taken to a outside hospital where gastric lavage was given and was brought to our hospital for further management.After initial 10 second assessment in ED, she was triaged to red area.

Primary Survey

- Airway- patient was able to phonate well.
- Breathing- spontaneous breathing, RR-30/min, SpO2- 75% on room air, air entry bilaterally equal, no abnormal sounds on chest auscultation.
- Circulation- PR-112/min, BP- 110/70 mmHg, peripheral mild cyanosispresent .
- Disability- GCS- E3V4M5 (12/15), GRBS-127mg/dl, pupils bilaterally equal and reactive to light, no focal neurological defects were noted.
- Exposure-body temperature 37.7^{0}C, patient was covered with blankets to prevent hypothermia.

Intervention:-oxygen supplementation with NRBM at 15 litres per minute IV line placed, fluids were initiated, foleys catheter was placed to measure urine output,urinewas dark in color.

Primary Adjuncts

1. ECG- sinus tachycardia.
2. CBC- Hb- 9.1g/dl, WBC-16,000/mm^3.platelets- 195,000/mm^3.
3. ABG- was obtained which showed paO2 -220 mmHg with 95% of Fio2.
4. Portable X-ray chest- clear lung fields.

Provisional Diagnosis– Cyanosis under evaluation.

Initial Management

1. Fluid resuscitation was continued.
2. Oxygen supplementation by NRBM at 15 litres per minute

Secondary Survey

- Signs and symptoms- patient presented complaining of increased mouth secretions, nausea, vomiting and abdominal pain since past 12 hours. Vomiting was non projectile, non-bilious, not blood stained, not foul smelling. There were 8 episodes of vomiting which initially contained food particles. Abdominal pain was diffuse colicky type of pain, intermittent in nature. No history of loose stools, fever, chest pain, excessive sweating, breathlessness, cough, seizures.
- Allergies- no history of any allergies.
- Medications- not on any medications at present.
- Past medical and surgical history- no known comorbidities or any surgery in past
- Last meal- lunch on the previous day.
- Events – patient was initially taken to the outside hospital where gastric lavage was given and was brought to our hospital for further management.

Systemic Examination

Central Nervous System:-

- Higher mental functions-drowsy, disoriented.
- Cranial nerve examination-within normal limits.
- Motor system examination- Power-5/5 in all limbs , Tone-normal Deep tendon reflexes -+inall limbs, Plantars-bilaterallyflexor.
- Sensory system examination-within normal limits
- Cerebellar system examination-within normal limits.

Respiratory system

- Inspection – shape of chest – normal, no dilated veins, scars,

sinuses, swellings or discolorations.
- Percussion-resonant sound heard over all lung field.
- Auscultation –bilaterally air entry equal, no abnormal sounds heard.
- Palpation – trachea central in position, bilaterally chest rise equal.

Cardiovascular, gastrointestinal system were within normal limits.

Furthur Management
- Probable diagnosis of acquired methemoglobinemia secondary to ingestion of naphthalene balls was considered based on

1) Discrepancy between SpO2 and PaO2 levels
2) Failure of supplemental oxygenation to improve SpO2 and persistent cyanosis
3) Dark Brown/ Chocolate coloured blood

- Diagnosis was confirmed by measurement of methemoglobin levels which were found to be high 30.2%.
- Patient was initiated on methylene blue 1mg /kg iv stat over 10 minutes. Patients spo2 improved to 95% on NRBM . A repeat methemoglobin levels was sent after 6hrs which showed Meth Hb levels of 4.5%. A repeat dose of methylene blue 1mg/kg at 6hr was administered. Along side patient was managed symptomatically. Patient clinically improved and discharged on 5th day

Day of admission	Day	Day	Day	Day	Day
	1	2	3	4	5
Hb(gm/dL)	9.1	8.3	8.9	6.5	9.2
Bloodurea(mg/dL)	41	37	35	22	15
SerumLDH(IU/L)	2,544	3,080	3,719	2,171	1561
Methemoglobin(%)	30.3	4.5			1.2

Final Diagnosis –
Acquired methemoglobinemia secondary to ingestion of naphthalene balls.

DISCUSSION

Even in healthy people, exposure to some oxidizing agents can cause methemoglobinemia. However , in healthy individuals, the increased methemoglobin concentration is lowered to normal levels by means of the cytochrome b5 reductase enzyme found in red blood cells.In some cases, the compensation process does not work properly and the increase in met emoglobin level cannot transport O2, causing haemoglobin oxygen dissociation curve to shift to the left and making it difficult to deliver oxygen to the tissue [1,3].

Methaemoglobinemia should be taken into consideration in the differential diagnosis of patient with normal circulatory and respiratory systemic findings who have persistent cyanosis. Under normal physiological conditions methaemoglobinemia should constitute about less than 2%. Cyanosis is seen if MetHb levels >15% in non anemic patients. Anxiety, headache and dizziness is seen at MetHb>20% with confusion, fatigue and tachypnea between 30% and 50% , followed by acidosis arrhythmias and death at >50%[4].

If MetHb levels are >30% or in case of severe symptoms, the first line of treatment is Intravenous methylene blue. Alternative treatments such as ascorbic acidor N-acetyl cysteine also can be used if methylene blue is unavailable or contraindicated. Exchange blood transfusion is recommended when methylene blue or alternative treatments are ineffective[5].

Methylene blue should not be administered to patients with known glucose 6-phosphate dehydrogenase (G6PD) deficiency, because methylene blue depends upon NADPH generated by G6PD in the reduction process of methemoglobin. As a result, this medication may not only be ineffective but is also potentially dangerous, since methylene blue has an oxidant potential that may induce hemolysis in G6PD deficient subjects[6].

Methemoglobin concentrations should decrease within 30 to 60 minutes after the first dose.

Patients with renal failure should use this medication with caution because both methylene blue and leuco blue are slowly excreted by the kidneys. During treatment, the urine has a bluish tint. The same occurs, in varying degrees, to the skin and mucous membranes, hindering the interpretation of cyanosis after the treatment[7]. Since this patients level of methaemoglobinemia was 30.2% and systemic toxic symptoms were in foreground IV methylene blue was initiated instead of antioxidant therapy.

Additionally, in previous in vitro studies, N-acetyl cysteine has been reported to be an important alternative approach in the treatment of nitric oxide related methemoglobinemia [8,9]. However, N-acetyl cysteine was shown to have no effect on human volunteers in other studies [8,9], effects of treatment with methylene blue, riboflavin, and N-acetyl cysteine were compared in patients with methemoglobinemia but N-acetyl cysteine and low-dose riboflavin were found not to change methemoglobin formation. Therefore, N-acetyl cysteine has not yet found a place.

When encountering hypoxia refractory to 100% oxygen and without identifiable cardiac or pulmonarycauses, methemoglobinemia should be suspected as a potential underlying condition. This suspicion can be initially raised by pulse oximetry findings and subsequently confirmed through co-oximetry analysis.[10]

CONCLUSION

Naphthalene toxicity can cause severe intravascular hemolysis and methemoglobinemia. Since mothballs are widely used in our homes, healthcare professionals need to be aware of the potential harms of mothballs.

Conflicts Of Interest
No conflicts of interest.

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