

Methemoglobinemia Due to Pesticide Poisoning: A Case Report

Dr. Geethika Sai Nutakki¹, Dr. Venkata Madhav Makineni², Dr. Madhukiran³

¹Post Graduate Student Of General Medicine, Dr.Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation ,Chinaoutpalli, Krishna District, Andhra Pradesh,India.

²M.D., General Medicine, Associate Professor, Dr.Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpalli, Krishna District, Andhra Pradesh, India.

³M.D., General Medicine, Assistant Professor, Dr.Pinnamaneni Siddhartha Institute Of Medical Sciences And Research Foundation, Chinaoutpalli, Krishna District, Andhra Pradesh, India.

Abstract: Methemoglobinemia is an altered state of hemoglobin resulting in impaired oxygen delivery to the tissues. Deliberate ingestion of certain insecticides and pesticides may result in this condition. We report a case of severe methemoglobinemia after deliberate ingestion of an insecticide marketed to be safe and containing only biological extracts and fillers. Methemoglobinemia should be suspected with low oxygen saturation on pulse oxymetry and the presence of chocolate colored blood. The methemoglobin level of 91% in our patient is the highest level reported among methemoglobinemia survivors.

Keywords: Biological extracts; insecticide; methemoglobinemia

I. Introduction

Methemoglobin (MetHb) is a dyshemoglobin, represents the oxidized form of hemoglobin (Hb), the ferrous (Fe²⁺) state of iron is transformed into ferric (Fe³⁺) state, which makes it incapable of binding to oxygen. Acquired methemoglobinemia is a dyshemoglobinemia which results from exposure to various oxidizing agents, results in impaired oxygen delivery to the tissues and can be potentially fatal if untreated. Deliberate ingestion of certain herbicides, insecticides, and pesticides may produce this condition.

Observations:

Case -1:

A 25-year-old woman presented to emergency with alleged history of poisoning with pesticide within 40 minutes after consumption. She was unresponsive and was intubated and mechanically ventilated. On Examination, She was cyanotic, Blood pressure:130/80mmHg, Pulse rate:90 beats/min, Respiratory rate: 28 breaths/min, CVS :S1 S2 heard, Bilateral breath sounds were clear, Bilateral pin point pupils with sluggish reaction to light. Within 3 hours after admission, she became hemodynamically unstable and developed generalized tonic clonic seizures. Oxygen saturation measured by pulse oximetry (SpO₂) was 67 % at ambient air and 88% with 100% oxygen. Her blood samples appeared chocolate brown.

Investigations:

ABG analysis with CO oximetry (SIEMENS Rapid point 500)

- pH:7.23
- pCO₂:28 mm Hg
- HCO₃:10.2 mEq/L
- paO₂-467.5mmHg
- SaO₂ – 99%
- Lactate:6.5 mmol/l
- CO – oximetry analysis :
- MetHB: 78%

She was treated with 1% methylene blue-2mg/kg,Ascorbic acid, Exchange transfusion, Antiepileptics , Inotropic support, Despite of treatment, she expired on day3.

Case 2 :

A 25 years old man, farmer by occupation with no premorbid illness alleged to have consumed about 100ml of pesticide (biological extract 5%,stabilizers 5%,fillers 90%) with suicidal intention. He was brought to emergency room after 90 minutes of ingestion with complaints of 3 episodes of vomiting. No complaints of abdominal pain, loose stools, blurring of vision, palpitations or fever. On Examination, Patient was conscious and coherent His GCS:15/15 , Blood pressure:110/80 mm Hg , Pulse:120/min , regular, spO₂: 70% at ambient air ; 86% with 4 liters oxygen,CVS :S1 S2 heard,RS : Normal Vesicular Breath Sounds heard,P/A: Soft,Pupils-

Bilateral pinpoint pupils with sluggish reaction to light ,ECG: showed sinus tachycardia. Patient suddenly became tachypneic &drowsy within 15 minutes and became desaturated (spO2-60%),he was intubated & ventilated.

Investigations : ABG analysis showed

- pH - 7.03
- pCO₂ - 29.0 mm Hg
- HCO₃ - 7.6 mmol/L
- paO₂ - 621.9mmHg
- SaO₂ - 99%
- Lactate - 17.18 mmol/L
- Co -oximetric analysis showed:
- Methemoglobin% :- 80.1%

Disparity between paO₂ and SpO₂ and increased level of methemoglobin are suggestive of acquired methemoglobinemia due to pesticide ingestion.

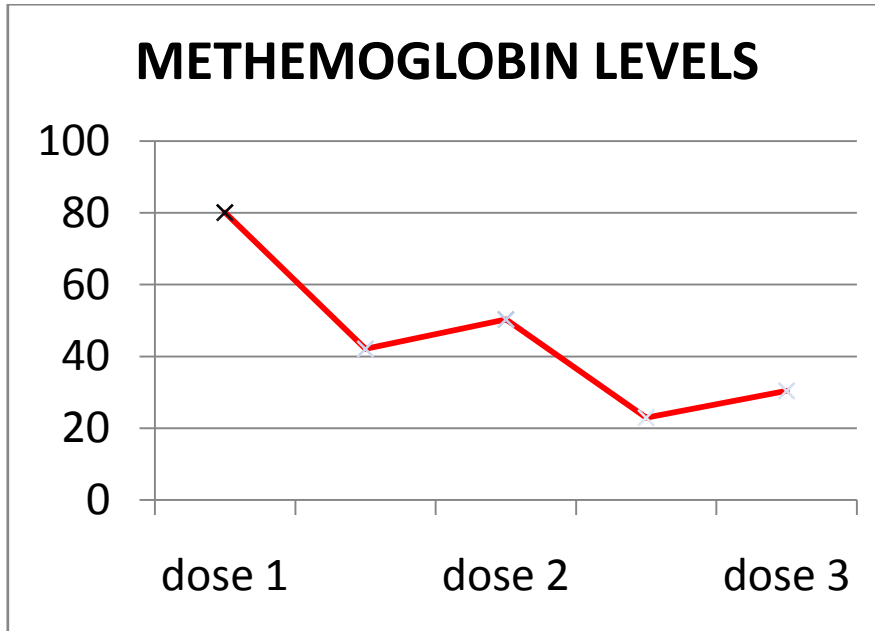
| RAPIDPoint 500 | | |
|----------------------|--------------|----------|
| ARTERIAL SAMPLE | | |
| 27.12.2014 22:59 | | |
| System ID 0500-14721 | | |
| Patient ID 10061422 | | |
| Est Name | | |
| ACID/BASE 37.0 °C | | |
| pH | 7.036 | |
| pCO ₂ | 29.0 | mmHg |
| pO ₂ | 621.9 | mmHg |
| HCO ₃ act | 7.6 | mmol/L |
| HCO ₃ std | 9.2 | mmol/L |
| BE (B) | -22.0 | mmol/L |
| BE (act) | -23.1 | mmol/L |
| ctCO ₂ | 8.5 | mmol/L |
| CO-OXIMETRY | | |
| Hb | 15.97 | g/dl |
| FOHb | 19.97 | % |
| COHb | 0.07 | % |
| %Methb | 80.19 | % |
| FIHb | 0.07 | % |
| hb(t) | 16.77 | g/dl |
| HEMATOLOGIES | | |
| H | 111.2 | mmol/L |
| S | 4.29 | mmol/L |
| Ct | 1.09 | mmol/L |
| Cl | 105 | mmol/L |
| AniGap | 34.9 | mmol/L |
| METABOLITES | | |
| Glucose | 255 | mg/dl |
| Lac | 17.18 | mmol/L |
| BUN | 7.07 | mmol/L |



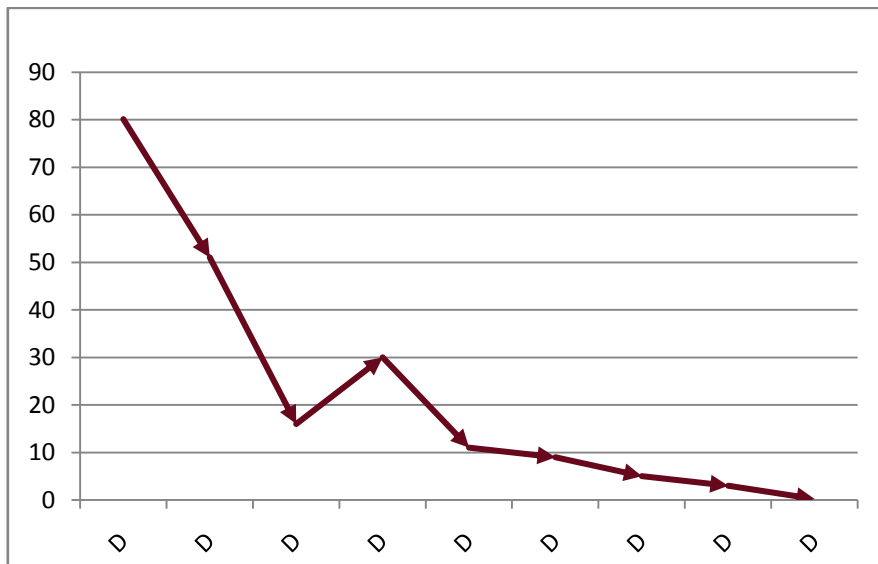
Patient Having Cyanosis



Chocolate Brown Blood



Methemoglobin Levels After Methylene Blue Dose



Methemoglobin levels during the course of hospital stay

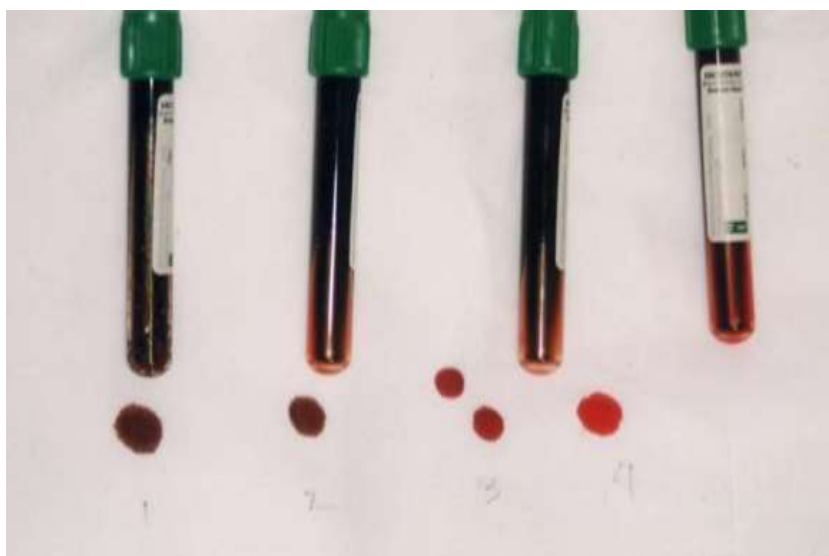


Greenish Blue Urine After Administration Of Methelene Blue

Patient was administered 1% methylene blue. DOSE:1mg/kg in 100ml NS over 5 mins. Initially SpO₂ dropped from 80% to 50% and increased to 86% within 10 -15 seconds. Urine turned greenish blue. ABG was repeated after 1 hour meth hemoglobin was reduced to 50 %. Patient gained consciousness and was oriented to place, person. Two doses were repeated with 12 hours gap with doses of 1mg/kg and 0.5mg/kg respectively and meth hemoglobin was 6.2% after 3rd dose. 500mg vitamin C and Riboflavin in 5%Dextrose IV were given at 12 hours interval. 1 unit of compatible blood transfusion was done.

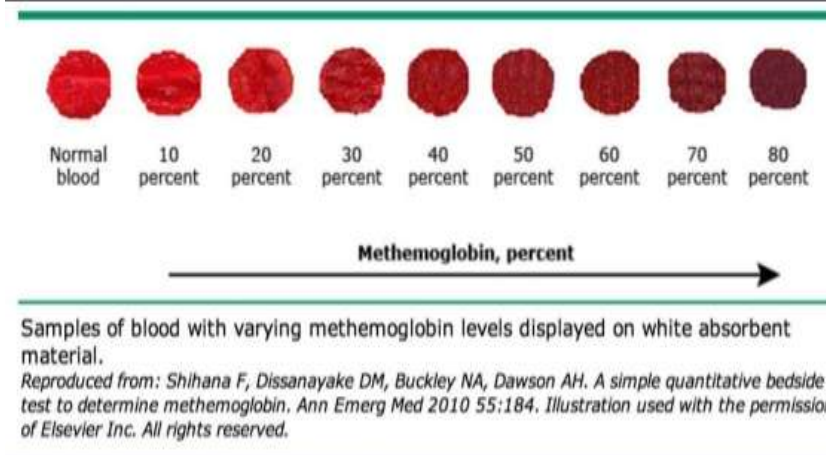
II. Discussion

Diagnostic suspicion of methemoglobinemia is based on clinical findings: generalized cyanosis out of proportion to respiratory status and normal PaO₂, which doesn't improve with administration of O₂. Arterial blood drawn for blood sampling is chocolate brown, blue, or black and fail to change color when exposed to air (unlike deoxyhemoglobin) or when a drop is dried on filter paper. The "oxygen saturation gap" Cooximetry is the diagnostic test of choice .



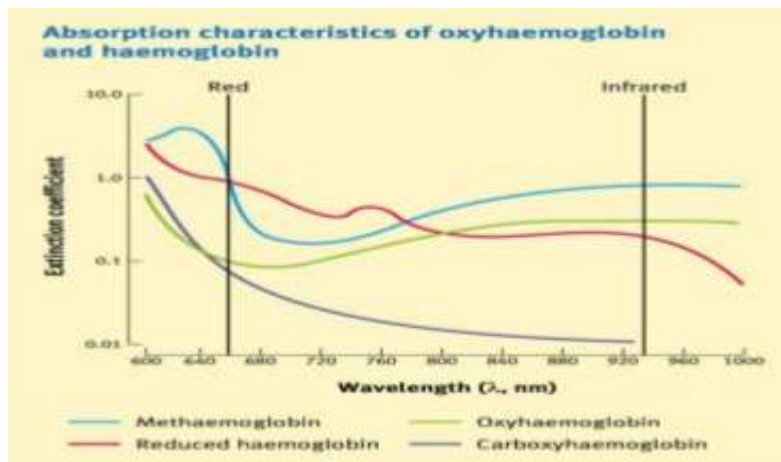
- Chocolate-brown arterial blood, Does not become red with exposure to oxygen
- Filter paper test ---- place drop of blood on filter paper - MHb will not turn red
- Potassium cyanide test----MHb turns red when CN added, sulfhemoglobin does not

Colorimetric Representation Of Methemoglobin Levels



Diagnosis

- Diagnostic suspicion of methemoglobinemia is based on clinical findings: generalized cyanosis out of proportion to respiratory status and normal PaO₂, which doesn't improve with administration of O₂. Arterial blood drawn for blood sampling is chocolate brown, blue, or black and fail to change color when exposed to air (unlike deoxyhemoglobin) or when a drop is dried on filter paper. The "oxygen saturation gap" Cooximetry is the diagnostic test of choice.
- The Oxygen Saturation Gap : Refers to the difference between the high O₂ sat *calculated* from routine ABG analysis and the low O₂ sat *measured* by pulse oximetry Methemoglobinemia should be suspected when O₂ Sat (ABG) > O₂ Sat (pulse OX)
- Multiple Wavelength Spectrophotometry (COoximetry) - Is the diagnostic test of choice for methemoglobinemia. It is based upon analysis of methemoglobin absorption spectra which has peak absorbance at 631 nm. A fresh specimen should always be obtained as the methemoglobin levels increase with storage.
-



Management :

- SUPPORTIVE THERAPY
- Methyl thioninium chloride aka **Methylene Blue**
- Hyperbaric oxygen
- Blood transfusion or exchange transfusion
- Ascorbic acid

III. Conclusion

The triad for suspicion of methemoglobinemia is :a) clinical cyanosis b) normal paO₂ and c) chocolate brown colored arterial blood. The saturation gap should alert the physician, and the diagnosis should be confirmed by co-oximetry and absorption spectro-photometry. All the physicians should be cautious in case of poisoning with unknown chemical composition as these compounds can cause fatal complications like

methemoglobinemia. Methemoglobinemia should be considered in the differential diagnosis of all cases of unexplained cyanosis.

Bibliography

- [1]. Dewan A, Patel AB, Saiyed HN. Acute methemoglobinemia- A common occupational hazard in an industrial city in western India. *Journal of Occupational Health*. 2001; 43(3): 168-171.
- [2]. Donovan JW: Nitrates, nitrites and other sources of methemoglobinemia. In *Clinical management of poisoning and drug overdose*. Edited by Haddad LM, Winchester JF. W.B. Saunders Company, Philadelphia; 1990:1419-1431.
- [3]. Ash-Bernal R, Wise R, Wright SM. Acquired methemoglobinemia: a retrospective series of 138 cases at 2 teaching hospitals. *Medicine*. 2004 Sep; 83(5):265-73.
- [4]. George T, Shaikh AI, Thomas L, Kundavaram AP. Severe methemoglobinemia due to insecticide poisoning. *Indian J Crit Care Med* 2014; 18:113-4
- [5]. Abu-Laban RB, Zed PJ, Pursell RA, Evans KG. Severe methemoglobinemia from topical anesthetic spray: Case report, discussion and qualitative systematic review. *CJEM* 2001; 3:51-6.
- [6]. Gharahbaghian L, Massoudian B, Dimassa G. Methemoglobinemia and sulfhemoglobinemia in two pediatric patients after ingestion of hydroxylamine sulfate. *West J Emerg Med* 2009; 10:197-201.
- [7]. Chongtham DS, Phurailatpam J, Singh MM, Singh TR: Methaemoglobinemia in nitrobenzene poisoning. *J Postgrad Med* 1997, 43(3):73-4.
- [8]. Rees SM, Nelson LS: Dyshemoglobinemias. In *Emergency medicine - a comprehensive study guide*. 6th edition. Edited by Tintinalli JE, Kelen GD, Stapczynski JS. McGraw-Hill, New York; 2004:1169-1171.
- [9]. do Nascimento TS, Pereira RO, de Mello HL, Costa J. Methemoglobinemia: From diagnosis to treatment. *Rev Bras Anestesiol* 2008; 58:651-64
- [10]. Perera M, Shihana F, Kularathne K, Dissanayake D, Dawson A. Acute methaemoglobinaemia after massive nitrobenzene ingestion, *BMJ Case Rep* 2009; 2009.