Case Report: A rare case of anti-termite solution poisoning presenting as methaemoglobinaemia

S. Venkatesh,¹ V. Satyanarayana,¹ A. Krishna Simha Reddy,¹ M. Madhusudan²

Departments of ¹Emergency Medicine, ²Anaesthesiology and Critical Care, Sri Venkateswara Institute of Medical Sciences, Tirupati

ABSTRACT

Methaemoglobinaemia should be considered in all patients who are cyanotic and not responding to oxygen therapy. We report the case of a 25-year-old male who presented to the emergency room with alleged history of suicidal poisoning with 20 tablets of alprazolam (0.5mg) and anti-termite solution. At presentation he was drowsy, dyspnoeic and cyanosed. He was immediately intubated and intiated on mechanical ventilatory support. Oxygen saturation by pulse oximetry remained at 82% and did not improve even with a fraction of inspired oxygen of 1.0. Arterial blood was chocolate brown in colour. Arterial blood gas analysis revealed presence of significant saturation gap. Co-oxymetric analysis was done which revealed increased methaemoglobin levels following which methylene blue (100 mg) was given intravenously (IV) and symptoms improved. Patient was extubated the following day and was discharged in a haemodynamically stable state. Early diagnosis and antidotal treatment are pivotal in methaemoglobinaemia.

Key words: Anti – termite solution, Methaemoglobinaemia, Methylene blue

Venkatesh S, Satyanarayana V, Krishna Simha Reddy A, Madhusudan M. A rare case of anti-termite solution poisoning presenting as methaemoglobinaemia. J Clin Sci Res 2017;6:179-81. DOI: http://dx.doi.org/10.15380/2277-5706.JCSR.17.03.001.

INTRODUCTION

Methaemoglobinaemia is a rare cause of cyanosis which is a life-threatening and treatable emergency. It is defined as an increase in the methaemoglobin levels in the blood.¹ Methaemoglobinemia is a serious condition which interferes with oxygen transport. It occurs due to oxidation of the ferrous (Fe^{2+}) ion in the haeme molecule of haemoglobin, leading to formation of ferric (Fe³⁺) ion whch has reduced ability to bind oxygen. It reduces the oxygen-carrying capacity of blood and dissociation of oxygen causing tissue hypoxia. It may be congenital or acquired. Acquired methaemoglobinaemia is due to various chemicals containing nitrites, nitrates, anilines and benzene compounds and drugs like sulphonamides, dapsone, phenacetin, primaquine and topical anaesthetic agents like

Received: March 08, 2017, Accepted: September 05, 2017.

Corresponding author: Dr

Satyanarayana, Professor and Head, Department of Emergency Medicine, Sri Venkateswara Institute of Medical Sciences, Tirupati, India. **e-mail:** vulchi2003@yahoo.com

V

benzocaine.² Here we report a patient who presented with anti-termite solution poisoning along with alprazolam tablets who was diagnosed as having acquired methaemoglobinaemia in our tertiary care hospital.

CASE REPORT

A 25-year-old male was found unconscious by his relatives at around 8:00 AM in his room, with an empty strip of tablets (20 tablets of alprazolam 0.5 mg) beside him. He was immediatly taken to a local hospital, where gastric lavage was done and he was referred to our tertiary care hospital for further management.

He was brought to our emergency room (ER) at around 12.30 PM. on examination, patient was stuporous, not obeying verbal commands, responding to deep painful stimulus and his

Online access http://svimstpt.ap.nic.in/jcsr/jul-sep17_files/1cr.17.03.001.pdf DOI: http://dx.doi.org/10.15380/2277-5706.JCSR.17.03.001 Methaemoglobinaemia due to anti-termite solution poisoning

Glasgow Coma Scale (GCS) score was 8/15 (E2V1M5). There was no pallor, icterus, clubbing, oedema or lymphadenopathy. Central cyanosis was present. Patient was afebrile, pulse 75/min, regular, normal volume and character. Blood pressure was 110/70 mm of Hg, respirations 30 min. Pulse oximetry oxygen saturation (SpO_2) was 66% on breathing room air. Paradoxical abdominal wall movements were noticed with respiration. Systemic examination was otherwise unremarkable. Patient was immediately intubated and initiated on mechanical ventilatory (MV) support on synchronized intermittent mechanical ventilation (pressure control ventilation with pressure support) [SIMV(PCV+PSV)] mode. But SpO₂ remained at around 86% even on a fraction of inspired oxygen FiO_2 of 1.0.

Laboratory investigations including chest radiograph revealed no clue to the diagnosis. Arterial blood gas (ABG) analysis was ordered on a FiO₂ of 1.0. Arterial blood was chocolate brown in colour (Figure 1) and showed arterial oxygen tension (PaO₂) 274.6 mm Hg, arterial carbon dioxide tension (PaCO₂) 43.6 mm Hg, pH 7.36, bicarbonate 24 m Eq/L, arterial



Figure 1: Arterial blood colour before (chocolate brown, white arrow) and after (bright red, red arrow) giving methylene blue

oxygen saturation 99.8%. This showed saturation gap. Presence of saturation gap made suspect methaemoglobinaemia or us sulphmethaemoglobinaemia. He was given a loading dose of intravenous (IV) flumazenil, following which his sensorium improved dramatically. On taking careful history from the patient himself, he revealed that he had also consumed anti-termite poison containing nitrobenzene along with tablets of alprazolam. Anti-termite poison containing nitrobenzene compound is known to cause methaemoglobinaemia. Co-oximetric analysis confirmed the presence of elevated methaemoglobin levels in the blood (32%). He was treated with IV methylene blue (50 mg diluted in 50 ml normal saline over 10 min). Pulse oximetry showed improvement in SpO₂ to 99% on FiO₂ of 0.5 over the next 4 hours.Subsequently patient was weaned off from the mechanical ventilatory support and was extubated and discharged on third day in a haemodynamically stable state.

DISCUSSION

When the blood levels of methaemoglobin exceed the normal level of 2% methaemoglobinaemia occurs. It may be due to either congenital cause due to absence of reduced nicotinamide adenine dinucleotide (NADH)-dependent enzyme, Cytochrome B5 reductase, inherited as autosomal recessive disorders or acquired due to consumption of some substances mentioned above.³ Acquired causes also includesepsis and sickle cell crisis. These essentially cause redox imbalances and destroy the normal mechanisms by which the body reduces methaemoglobin. Clinically, the features of methaemoglobinemia includes low oxygen saturation on pulse oximetry, cyanosis, chocolate brown colored arterial blood, with normal partial pressure of oxygen and oxygen saturation on arterial blood gas analysis.⁴ Diagnosis is established by co-oximetric analysis. Pulse oximetry saturation becomes

Methaemoglobinaemia due to anti-termite solution poisoning

unreliable when there is increased levels of methaemoglobin in the blood. Symptoms get worsened with increasing methaemoglobin levels. Symptomatic methaemoglobinaemia and methaemoglobin levels above 20% are treated with methylene blue at a dose of 1-2 mg/kg IV bolus over 5 minutes. This will cause the methaemoglobin levels to fall in 30 to 60 minutes. Thereafter additional doses of 1 mg/ kg bolus can be given after rechecking levels hourly if needed. Total dose of methylene blue injection should not exceed 7 mg/kg due to likelihood of adverse effects like chest pain, dyspnea, hypotension and haemolysis.⁵ In our case we suspected methaemoglobinaemia because of saturation gap and chocolate brown coloured blood. His methaemoglobin levels normalized to 1.8 % after giving IV methylene blue. Anti-termite solution (containing nitrobenzene compound) is a rare cause of acquired methaemoglobinemia. Methylene blue is the first line of treatment in most of the acquired conditions, when the patient is symptomatic or levels of methaemoglobin exceed 20%. In our case, patient presented with decreased consciousness, central cyanosis and his methaemglobin levels were above 20%, so we treated with methylene blue and patient

recovered subsequently. Monitoring of methaemoglobin levels may be required before and after administration of methylene blue. Methaemoglobinemia should always be considered in patients who present with cyanosis which is not responding to oxygen therapy.

REFERENCES

- Honig GR, Hemoglobin disorders. In: Behrman RE, Kleigman RM, Jenson HB, editors. Nelson Textbook of Pediatrics 16th ed. Philadelphia: WB Saunders; 2000: p.1478-88.
- 2. Skold A, Cosco DL, Klein R. Methemoglobinemia: pathogenesis, diagnosis, and management. South Med J 2011;104:757-61.
- 3. Yubisui T, Takeshita M, Yoneyama Y. Reduction of methemoglobin through flavin at the physiological concentration by NADPH-flavin reductase of human erythrocytes. J Biochem 1980;87:1715-20.
- 4. Ashurst J, Wasson M. Methemoglobinemia: a systematic review of the pathophysiology, detection, and treatment. Del Med J 2011;83:203-8.
- Harvey JW, Keitt AS. Studies of the efficacy and potential hazards of methylene blue therapy in aniline-induced methaemoglobinaemia. Br J Haematol 1983;54:29-41.