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Case Report

Blue Death: A Case Report

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Accidental Poisoning, Nitrobenzene, Methaemoglobinaemia, Methylene blue, Autopsy findings.

Abstract

Nitrobenzene poisoning very uncommon in India. Very few cases have been reported of Nitrobenzene poisoning. Nitrobenzene is used as a paint solvent in printing industries. Occupational hazards or Accidental exposure is a major cause of premature mortality globally. It is capable of causing serious toxicity, whether ingested or inhaled. When hydrocarbons are aspirated into the lung, they cause chemical pneumonitis, acute respiratory distress syndrome (ARDS). We are reporting a case of ingestion of reducer (Nitrobenzene) by a 21 year old female. She was brought to to Dr. Ram Manohar Lohia Hospital, New Delhi, where she was admitted and expired during the course of treatment. A detail post mortem finding of case was discussed in this case report.

1. Introduction

Nitrobenzene which is of bitter almond odour is a pale yellow or transparent oily liquid. Nitrobenzene also known as Nitrobenzol or oil of Mirbane is used in dyes, paints, printing, lubricating oil and synthetic rubber. Occupational hazards or Accidental exposures are a major cause of premature mortality globally. In India, 20% Nitrobenzene emulsion is widely used as pesticides. and marketed under the brand name Synflower offered by Mandar agrotech.[1-3] The lethal dose is reported to 2-4 gm, ranges from 1 to 10 gm by different studies. Nitrobenzene ingestion leads to formation of methemoglobinemia. The toxic dose resulting in methemoglobinemia was estimated in one case study at 4.3 to 11gm based on urinary p-nitrophenol Level.[4]

2. Case History:

A 21 year old female was brought to Dr. Ram Manohar Lohia Hospital, New Delhi with a history of accidental consumption of transparent liquid reducer (**Fig. No-7B**) lying in the fridge. She was admitted and expired during the course of treatment. Body was shifted to Mortuary of Maulana Azad Medical college to conduct the medicolegal autopsy.

3. Autopsy Findings

The dead body was of an adult female of average built with height of 158 cm. Eyes: both the eyelids were closed and bluish discolouration of both eyes and face present (**Fig. No-1A**), bluish discolouration of both upper and lower lips were present(Photograph No-1A). Conjunctiva and sclera were bluish in colour(**Fig. No-1B**).

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Natural orifices: Bluish discoloration were present on lips, tongue, gums (Fig. No. 1A) and female genitalia (Fig. No. 8). Bluish discolouration of veins of right hand were present (Fig. No-1C).

Fig. No 1: 1A: Bluish face of deceased; 1B: Bluish discolouration of sclera and cornea; 1C: Bluish discolourations of veins of Right hand

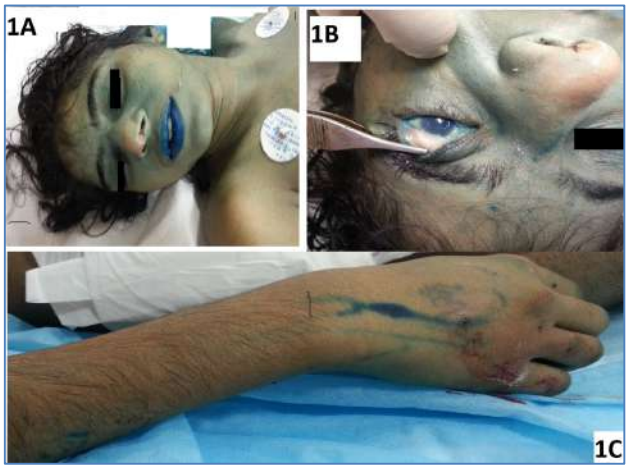


Fig No-2: 2A: Bluish discolouration of scalp; 2B: Scalp hairs were easily removable

Hypostasis could not be appreciated due to diffuse bluish discoloration of the body. Rigor Mortis was developed and could be elicited at all the major joints of body. Bluish discolouration of scalp seen and scalp hairs were easily removable (Fig. No. 2A & 2B). No changes of decomposition were present. On internal examination bluish discoloration was present in the layers of scalp, skull sutures, meninges and brain parenchyma (Fig. No. 3A,3B,3C,6A,6B & 6C).

Fig No-3: 3A: Bluish disclouration of left temporalis muscle; 3B: Bluish discolouration underscalp & skull vault; 3C: Bluish discolourations of dura matter of brain

Bluish discolouration of all internal organs like Heart, lungs were present (Fig. No-4A & 4B). Pharynx, Larynx and Trachea showed bluish discoloration of mucosa, oesophagus showed bluish discoloration of mucosa along the whole length. Pleural cavity showed diffuse bluish discoloration and contained about 150 ml of bluish coloured fluid was present in plural cavity on both sides. Both lungs were of bluish colour (Photograph No-5A & 5B). Oozing of bluish frothy fluid was seen on compression of the cut surface of both lungs. Pericardial sac contained about 20 ml of bluish coloured fluid and outer surface of heart was bluish coloured (Fig. No-4B). Peritoneum & Peritoneal Cavity showed bluish discoloration of outer surface of all abdominal viscera (Fig. No-4A) and contained about 300 ml of bluish coloured fluid. Kerosene like smell was present on opening of stomach and contained about 100 ml of mucoid material.

Stomach walls were congested and bluish discoloration of mucosa of stomach were present (Fig. No-7A).

Fig No-4: 4A: Bluish discolouration of internal organs; 4B: Bluish discolouration of Heart

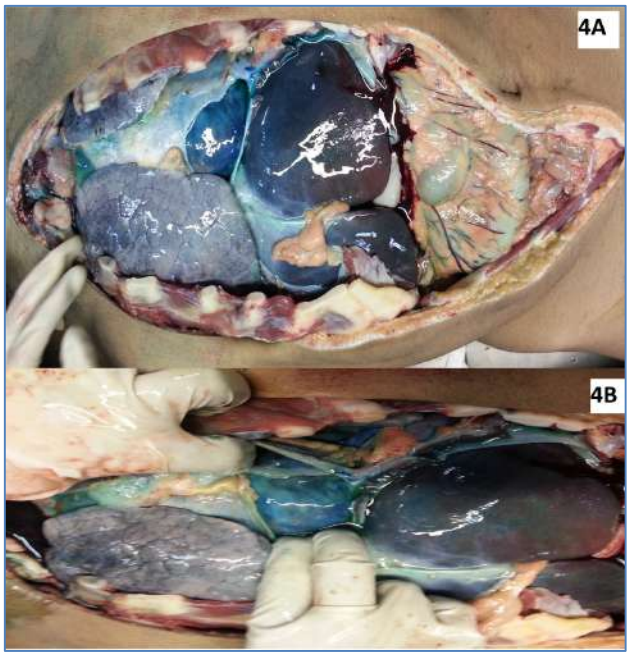


Fig No-5: 5A: Bluish discolouration of internal surface of lung; 5B: Bluish discolouration of external surface of lung

Fig No-6: 6A,6B, 6C: Bluish discolouration of Brain

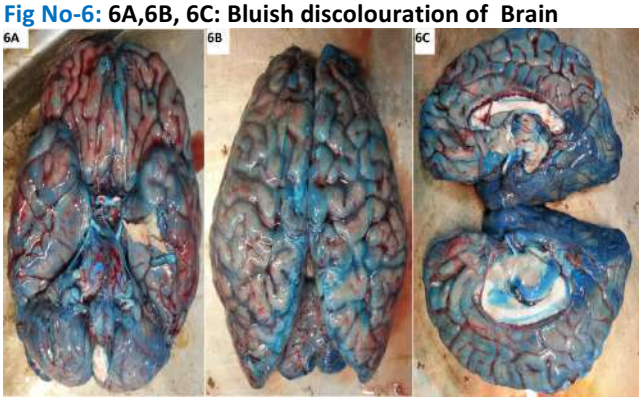
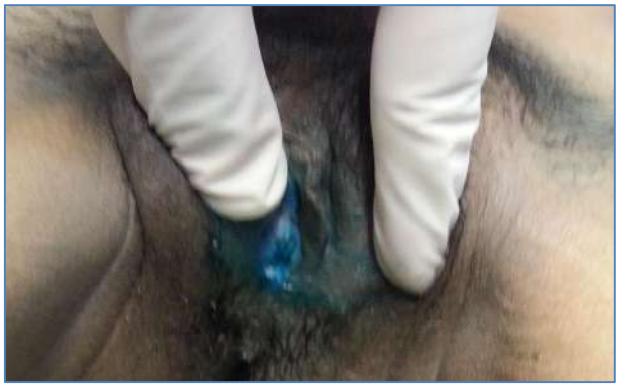


Fig No-7: 7A: Bluish discolouration of stomach mucosa; 7B: Poison bottle consumed by decease

Fig No-8: Bluish discolouration of female genitalia.



Small intestine and Large intestines showed bluish discoloration of outer surface. Kidneys showed bluish discoloration was present on outer surface and cut surface of both kidneys. Uterus, Fallopian Tubes and appendages showed bluish discoloration. Opinion about cause of death was kept pending and viscera was preserved. On receipt of viscera report shows presence of Nitrobenzene in **Exhibit 1A** i.e. (Stomach with its contents and loop intestine with its contents) and in **Exhibits 1B, 1C and 1D** no poisons nitrobenzene were detected.

4. Discussion

Nitrobenzene is pale yellow oily liquid, with an odour of bitter almonds is used as an intermediate in the synthesis of solvents, like paint remover. The first report of nitrobenzene poisoning came in 1886[5] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2900745/ - CIT1 and subsequent fatality reports followed[5]https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2900745/ - CIT1[6]. Toxicity of nitrobenzene can be accidental or suicidal. Accidental toxicity are generally seen in those people who are dependent on well water which are having dangerously high

levels of nitrites and nitrates[7,8]. The fatal dose ranges from 1 g to 10 g, by different authors[4,5]. The ill effects after consumption are due to the rapid formation of methaemoglobinaemia, a condition in which the iron within the haemoglobin is oxidized from the ferrous (Fe^{2+}) state to the ferric (Fe^{3+}) state, resulting in the inability to transport oxygen and causes a brownish discolouration of the blood. Once formed, methemoglobin can be reduced enzymatically either via an Adenine dinucleotide (NADH)-dependent reaction, catalysed by cytochrome b5 reductase, or an alternative pathway utilizing the nicotine adenine dinucleotide phosphate (NADPH)-dependent methemoglobin reductase system.[3,7]

Normal level of methemoglobin is 0 to 2% and level of methemoglobin in blood up to 10 to 15% patient remains asymptomatic or sometimes present in hospital with only cyanosis.[3,9] Above 20%, headache, dyspnea, chest pain, tachypnea, and tachycardia develop and beyond 40–50%, confusion, lethargy, and metabolic acidosis occur leading to coma, seizures, bradycardia, ventricular dysrythmia, and hypertension. Fractions around 70% are fatal. G6PD-deficient or anaemic patients developed more severe symptoms[3,6,8] in some patient Leukocytosis has been reported, with relative lymphopenia[10]. Sometime patient develops Heinz body haemolytic anaemia, hepatosplenomegaly and deranged liver functions.[3,6,9] Nitrobenzene metabolites are p-nitrophenol and aminophenol and after five days of ingestion they got excreted in urine up to 65% and in stools up to 15%.

Diagnosis can be established with help of brief history of chemical ingestion, peculiar smell of bitter almonds, and persistent cyanosis even after the continuous hyperbaric oxygen therapy without any pre-existing cardiopulmonary disease, minimal arterial oxygen saturation, and atypical arterial blood gas (ABG) analysis. Blood colour turns to dark brown which fails to turn bright red on shaking, which suggests methaemoglobinaemia and this is supported by the chocolate red colour of dried blood. Presence of nitrobenzene compounds may be confirmed spectrophotometrical methods.[3,9]

Patient can be cured on the principles of decontamination and symptomatic and supportive management. Methylene blue is the antidote of choice in cases of methaemoglobinaemia due to poisoning which accelerates the NADPH-dependant methemoglobin reductase system and is indicated when the blood methemoglobin levels are more than 30%[3,9]. Route of administration is intravenously at 1–2 mg/kg (up to 50 mg dose in adults,) as a 1% solution over five minutes; with a repeat in one hour, if necessary. Methylene blue is an oxidant at levels of more than 7 mg/kg, and therefore, may cause methaemoglobinaemia in susceptible patients. It is contraindicated in patients with G6PD deficiency, because it can lead to severe haemolysis. Ascorbic acid is an antioxidant that may also be administered in patients with methemoglobin levels of more than 30%[3,9]. In recent studies, N-acetylcysteine has been shown to reduce methemoglobin, but it is not yet an approved treatment for methaemoglobinaemia.[11]

Conclusion

The treatment of acute methemoglobinemia due to poisoning is usually associated with high mortality so early prompt management of poisoning should be attempted. Methylene blue and ascorbic acid are the treatment of choice, while RBC exchange transfusion and hyperbaric oxygen therapy are usually reserved for patients who are resistant to standard treatment.

Prevention is always better than cure so be aware of substance around you which can lead to ill effect and also keep these type toxic material out of reach your children and also educate them about the dangers of substances that contain poison. They should not be kept with eatable or store in food container. They should be properly stored and labelled.

Consent: While publishing this case report due care were taken so that identity of deceased was not disclosed at anytime during publication of this case report. Further this case report was sent to only this Journal.

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Conflicts of interests: None.

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