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## CASE REPORT

# ACUTE METHAEMOGLOBINAEMIA DUE TO INGESTION OF PLANT YIELD BOOSTER (NITROBENZENE): A CASE REPORT

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**ABSTRACT**

In India insecticidal poisoning containing organophosphorous is most commonly seen. Poisoning due to plant yield booster containing nitrobenzene is also on the rise. A 35 year old male patient was admitted with ingestion of plant yield booster (Boom Flower) showed the symptoms and signs of nitrobenzene poisoning with methaemoglobinemia. Timely use of mechanical ventilation and repeated parenteral methylene blue help to save a life.



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## INTRODUCTION

Intentional exposure is a major cause of premature mortality globally and 113914 suicides are recorded annually from India for which a variety of chemicals have been used. Nitrobenzene also known as nitrobenzol or oil of mirbane is used in dyes, paints, printing, lubricating oil and synthetic rubber. In India, 20% nitrobenzene emulsion is widely used as pesticide and marketed under the brand name Bloom flower (Hisham *et al.,* 2012). The lethal dose is reported to range from 1 g to 10 g, by diffrent authors (Virendra Goyal *et al.,* 2014). Acute poisoning with nitrobenzene causing methaemoglobinemia is uncommon but life threatening emergency early aggressive management of severe poisoning strongly suspected on clinical ground may change the outcome of a patient (Saxena *et al.,* 2010).

### Case report

A 40 year old male patient presented to emergency department of our hospital with alleged history of consumption of plant yield booster under influence of alcohol at about 4:30 pm. Patient was conscious disoriented with pulse rate 100/min, blood Pressure 130/70 mmhg, respiration rate 20/min, $spo_2$ 92%, no fasciculations, pupils were normal sized reacting to light. He was given whole body wash and gastric lavage was started, after one hour patient developed cyanosis (tongue and nails), laboured respiration with respiratory rate 26/min, pulse rate 150/min, $spo_2$ 60% his chest was clear. Immediate intubation and ventilation with 100% oxygen using assist control mode was done which improved his $spo_2$ to 80%. Due to presence of cyanosis and $spo_2$ less than 90% on ventilation, Methaemoglobinemia was suspected. Blood sample was drawn from the radial artery for ABG which showed chocolate brown colour, Report showed methaemoglobin value 87.5%. CBC, creat, electrolytes, liver enzymes were within limits. X ray chest and ECG were within normal limits. A clinical diagnosis of severe methaemoglobinemia was confirmed. Injection Methylene Blue 60mg (1mg/kg) IV was given, antibiotics and IV fluids were started. The initial methaemoglobin level was 87.5 % which decreased to 23.4% after giving methylene blue, the second dose of methylene blue (50mg IV) was given after

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6 hours. Inravenous vit. K and oral vit.C tablets were given after six hours.


**Fig. 1. Bloom Flower – n Nitro Benzene 20% v/v**

ABG was repeated after 12 hours which showed MetHb level down to 7.7%. Patient became conscious, obeying commands with pulse rate 100/min, BP 110/70 mmhg, $spo_2$ 80%. ABG showing saturation 98%. Patient was given supportive management and extubated after 48 hours. Patient was shifted to general ward and observed for seven days his CBC, Renal Fuction Test, Liver Function Test were normal and $spo_2$ of 95% on room air. Patient was discharged on seventh day with discharge medications Tab Vit C, Oral Iron and folic acid.

| | pH | $PCO_2$ | $PO_2$ | $HCO_3$ | $SO_2$ | MetHb |
|---|---|---|---|---|---|---|
| At Admission | 7.316 | 31.5 | 80 | 15.7 | 90 | 87.5 |
| 6 hours | 7.38 | 36.9 | 86 | 21.6 | 95 | 23.4 |
| 12 hours | 7.37 | 37.1 | 159.2 | 21 | 98.5 | 7.7 |
| 48 hours | 7.41 | 36.2 | 90 | 22.1 | 99 | 1.5 |

## DISCUSSION

Intentional exposure is a major cause of premature mortality globally and 113914 suicides are recorded annually from India for which a variety of chemicals have been used. In India, 20% nitrobenzene emulsion is widely used as pesticide (Hisham *et al.,* 2012). Acute ingestion of nitrobenzene leads to rapid development of methaemoglobinaemia (Chongthm *et al.,* 1997). Nitrobenzene is metabolized to p-nitrophenol and aminophenol and excreted in urine upto 65% and in stools upto 15% after 5 days of ingestion (Saxena *et al.,* 2010). Liver, stomach, blood and brain may act as a stores and release nitrobenzene gradually (Saxena *et al.,* 2010). Acute intoxication is asymptomatic upto the level of 10-15% of Methhaemoglobin showing only cyanosis, Beyond 20% headache, dyspnea, chest pain, tachypnea and tachycardia develop. At 40-50 % confusion lethargy and metabolic acidosis occur leading to coma, seizures, bradycardia, ventricular dysarrhythmia and HTN (Saxena *et al.,* 2010). Levels greater than 70% cause cardiovascular collapse and have a high degree of mortality if left untreated (Chongthm *et al.,* 1997). Other effects include hepatosleenomegaly, altered liver functions and Heinz body haemolytic anemia (Saxena *et al.,* 2010). Methaemoglobinaemia is a condition in which the ferrous ($fe^{+2}$) state of iron within Haemoglobin gets oxidized to ferric ($fe^{+3}$) state which results in the incompatibility in oxygen sy Normal level of methemoglobin is 0 to 2% (Hisham *et al.,* 2012). Two different mechanisms define the low level of Methhaemoglobin the first happens during hexose monophosphate shunt pathway in the erythrocyte which reduces oxidizing agents by glutathione, The other mechanism which is contrary to methhaemoglobin formation utilizes diaphorase-I and diaphorase-II eenzyme systems which necessitate NADH and NADPH enzymes respectively. Reducing methaemoglobin to its ferrous state. The cytochrome b5 reductase enzyme catalyzes the NADH dependant reaction (Hisham *et al.,* 2012). Clues for diagnosis include a history of chemical ingestion the characteristic smell of bitter almonds, persisting cyanosis on oxygen therapy without severe cardiopulmonary disease, low aterial oxygen saturation with normal ABG (calculated) oxygen saturation (Virendra Goyal *et al.,* 2014). Dark brown blood that fails to turn bright red on shaking which suggest Methaemoglobinaemia and this is supported by chocolate red colour of dried blood. Presence of nitrobenzene compound may be confirmed spectrophoto metrically and estimated by the butanone test of schrenk methhaemoglobin level in blood and presence of p-nitrophenol and p-aminophenol (Virendra Goyal *et al.,* 2014). Methylene blue is the antidote of choice for acquired (toxic) Methaemoglobinaemia when the methaemoglobin level is greater than 35-40% and the patient has cardiorespiratory symptoms (Chongthm *et al.,* 1997). It acts as an exogeneous cofactor which greatly accelerate the NADPH dependant methaemoglobin reductase system (Chongthm *et al.,* 1997). Methylene blue 1-2 mg/kg is administered as a 1% solution undiluted as direct IV over 3-5 min repeated as 1mg/kg in 1hour as necessary to control fluctuating symptoms (Hisham *et al.,* 2012).

In higher doses methylene blue itsekf is an oxidizing agent as little as 5mg/kg has caused asymptomatic mathaemoglobinaemia. Cumulative doses greater than 7 mg/kg have an increased risk of methaemoglobin induction and can cause chest pain, nausea, vomiting, dizziness, hypertension, confusion, diaphoresis, tremor, dyspnoea and cyanosis (Chongthm *et al.,* 1997). If methylene blue is contraindicated or ineffective, ascorbic acid is often mentioned as an alternative therapy, but its reducing effect is probably too slow to have significant benefit, Exchange transfusion is indicated in severe cases, when both fail. Exchange transfusions equal to or less than the total volume and up to / greater then twice the volume have been used (Chongthm *et al.,* 1997). The present index case represents an uncommon poisoning with nitrobenzene which was managed successfully with injectible methylene blue and ascorbic acid with intensive haemodynamic and cardiopulmonary support.

### Conclusion

The treatment of poisoning caused by an unknown compound is a challenge. Acute Methaemoglobinaemia is associated with high mortality. Hence, aggressive management of nitrobenzene poisoning should be attempted. The authors wish to point that markedly increased level of methaemoglobin can be managed efficaciously using intravenous Methylene blue and Ascorbic acid provided that the diagnosis of the condition is confirmed.

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