Case Report

Toxic Brain Injury with Nitrobenzene Poisoning

Abstract

Acute methemoglobinemia secondary to nitrobenzene ingestion is a rare but well-known clinical entity. It is extremely important to identify such patients as rapid and effective management with methylene blue and other supportive measures will often save these lives. We present a rare and unfortunate case of a girl who developed acute toxic brain injury following nitrobenzene ingestion and succumbed.

Keywords: *Emergency physician, methemoglobinemia, methylene blue, nitrobenzene poisoning, toxic brain injury*

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Introduction

Several substances cause acquired acute methemoglobinemia (MetHb). The most common are dapsone, aniline dyes, and topical anesthetic agents. It can rarely be caused by accidental or suicidal ingestion of nitrobenzene, which is paint solvent and used in screen painting. Nitrobenzene poisoning is a rare cause of MetHb[1] but reported in the medical literature.[2] Few of such acutely toxic patients were managed promptly and survived. However, we present a case where the patient could not be revived due to severe toxic effects of nitrobenzene on the brain.

Case Report

A 17-year-old girl presented to our emergency department with altered sensorium. On further evaluation, we found out that she had an accidental ingestion of nitrobenzene solution 7 days ago. Her father used nitrobenzene for screen painting at home. She was then taken to a local hospital where she presented with recurrent vomiting, headache, and light-headedness. She had a gastric lavage done and started on supportive treatment with intravenous fluids and intravenous paracetamol. Her blood investigation showed hemoglobin (Hb) 8.6 g/dl, leukocytes 17,300/µl, serum potassium 2.9 mmol/L, and serum sodium 135 mmol/L. Her kidney and renal function tests were within normal limits. She continued to deteriorate in sensorium and was then shifted to a larger hospital in the vicinity. Her father gave a clear history of nitrobenzene ingestion. On examination, she was found to be cyanosed. Her blood investigations now showed Hb 5.4 g/dl, hematocrit 16%, and leukocytes 25,300/µl with predominant neutrophilia. Arterial blood gas (ABG) showed pH 7.57, $PaCO_2$ 24.7 mmHg, PaO_2 106 mmHg, SpO_2 99%, and HCO_3 23.1 mmol/L. Peripheral smear showed moderate anisopoikilocytosis and presence of red cell fragments. Intravenous methylene blue was given in view of high suspicion of acute MetHb. The patient was administered methylene blue and given 30 mg once daily for 3 days that seemed to improve her condition. Four units of packed red cells were transfused for this patient. On the 6th day of illness, her sensorium dipped again and she was sent for a magnetic resonance imaging (MRI) brain. MRI showed hyperintensities in bilateral corticospinal tracts with bilateral hyperintensity in dentate and splenium of corpus callosum. For further management, she was referred to our center. On initial assessment, she was in altered sensorium with a Glasgow Coma Scale of E4V2M5. Her airway was patent, respiratory rate 16/min, SpO_2 94% on 3 L/min oxygen, and lung sounds were normal. Blood pressure was 120/80 mmHg with a pulse rate of 96/min. Her chest X-ray and electrocardiography were unremarkable. Her repeat blood investigation showed hemoglobin 10.3 g/dl, leukocytes 30,000/µl, and platelet count

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317,000/μl. Arterial blood analysis showed pH 7.48, $PaCO_2$ 31 mmHg, PaO_2 141 mmHg, serum bicarbonate 23.1 mmol/L, serum sodium 141 mmol/L, serum potassium 3.5 mmol/L, serum calcium 0.83 mmol/L, and serum lactate 1.6 mmol/L. Low-flow oxygen, intravenous fluids, and antibiotics were used to treat her. The patient was managed in high dependency unit and was on continuous cardiac monitoring. She was intubated and mechanically ventilated in view of her drop in worsening altered sensorium. Ten days after her arrival to our center, she unfortunately passed away secondary to aspiration pneumonitis, sepsis, and toxic brain injury due to nitrobenzene.

Discussion

Nitrobenzene is used as a paint solvent and dye in screen painting. Once ingested, it can rapidly cause MetHb.[1] MetHb is the oxidized form of Hb; the ferrous (Fe^{2}+) iron is present in ferric (Fe^{3}+) form. Normal levels are <1%. The levels of MetHb are maintained by the innate cellular mechanisms of the erythrocytes.[3] Once ingested in toxic levels, the erythrocytes are unable to reduce the MetHb and the patients develop acute symptoms of MetHb.[4] It is important that the MetHb is suspected in patients presenting with cyanosis and normal PaO_2 levels on ABG. When the oxygen level on pulse oximetry is 5% lower than the saturations calculated on ABG it is referred to as 'saturation gap'.[5] The blood may appear dark red, dark brown to chocolate brown. The condition can be confirmed by laboratory tests of co-oximetry and reconfirmed by Evelyn–Malloy method.[6] Pulse oximetry is inaccurate in determining oxygenation levels in toxic patients.[7] In toxic doses, patients will have acute symptoms of nausea, vomiting, and headache followed by dyspnea, tachycardia, arrhythmias, shock, seizures, altered sensorium, and stupor.[8] Methylene blue should be used in all symptomatic patients.[9] Methylene blue can be used at a dose of 1–2 mg/kg up to 50 mg dose in adults diluted as 1% solution over 5 min. Methylene blue may be repeated again in an hour if patients are not showing any signs of improvement. Total cumulative dose should not exceed 7 mg/kg. Ascorbic acid should also be used if symptoms are not improving. It is effective in both oral and intravenous forms.[9] Our patient developed altered sensorium after toxic ingestion of nitrobenzene probably due to MetHb. She seemed to improve clinically after methylene blue but developed symptoms again due to storage of MetHb in various organs of the body and slow release raising the levels of MetHb. She underwent an MRI of the brain on the 6th day of her illness. Fluid-attenuated inversion recovery images showed features of hyperintensity in bilateral corticospinal tracts, dentate, and splenium of corpus callosum, consistent with toxic ingestion [Figures 1 and 2]. Recently, a case of a 67-year-old female patient was reported with similar changes in the brain after massive nitrobenzene ingestion.[10] The MRI in our case did not show changes related to brain hypoxia but direct toxic injury due to nitrobenzene ingestion.

Conclusion

Acute onset MetHb caused by extrinsic agents should be suspected in any patient with acute onset cyanosis and normal PaO_2 on ABG analysis. It remains an important clinical entity for emergency physicians as it may be fatal, and if acted on early and effectively, these patients may survive. Methylene blue is an important antidote and should be used when there is a high suspicion of MetHb. Toxic brain injury is a complication of nitrobenzene poisoning which can be fatal.

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Conflicts of interest

There are no conflicts of interest.

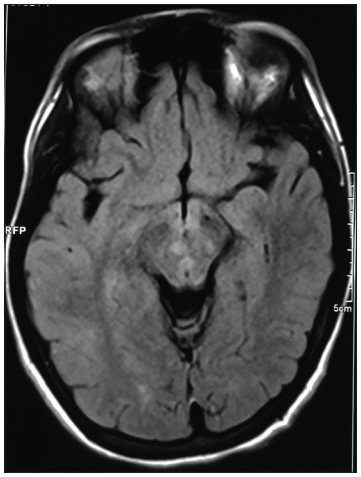

Figure 1: Magnetic resonance imaging–fluid-attenuated inversion recovery image through midbrain showing hyperintensity of the corticospinal tracts and red nucleus

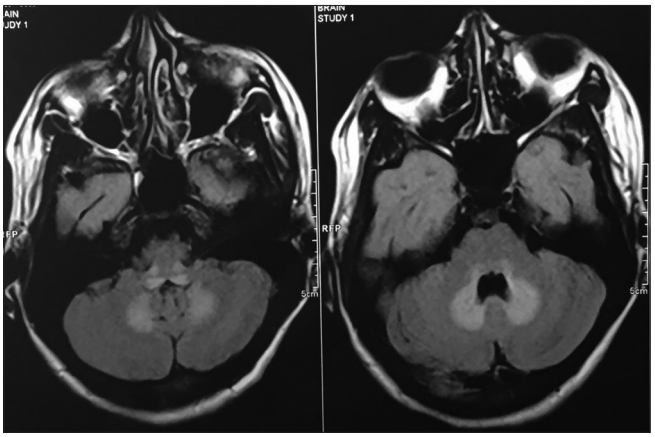

Figure 2: Magnetic resonance imaging–fluid-attenuated inversion recovery images of brain showing hyperintensity in bilateral dentate nucleus and medulla consistent with history of poisoning

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