

# **N - ACETYLCYSTEINE TREATMENT FOR CHLOROACETANILIDE AND PROPA NIL HERBICIDE INDUCED METHEMOGLOBINEMIA FOLLOWING ACUTE INGESTION: A CASE REPORT**

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**Introduction:** Propanil and Butachlor toxicity are considered in most references to be of low toxicity. Respiratory and central nervous system effects secondary to tissue hypoxia have been commonly reported. However, management of mixed pesticide ingestion remains to be a challenge in parts of developing countries due to paucity of available antidotes. **The case report:** A 39-year-old female who came in due to non-accidental propanil and butachlor ingestion. Patient initially presented with severe cyanosis, hypoxemia and alteration of metal status leading to acute respiratory failure Type I within 6 hours of ingestion. Supportive management with mechanical ventilation and oral ascorbic acid at 2000 mg daily were initiated due to inavailability of IV methylene blue. There was persistence of hypoxemia (oxygen saturation of 81% with FiO<sub>2</sub> of 100%), and methemoglobinemia of >30% for the succeeding days of admission. Patient also developed cough with minimal secretions. Haemolysis with haematuria, reticulocytosis and hyperbilirubinemia was noted started on the third hospital day. There was no acute liver toxicity or rhabdomyolysis as evidenced by normal transaminases and creatinine kinase were noted unlike in reported cases of butachlor toxicity. Nebulisation with N-Acetylcysteine at 400 mg every 4 hours was started initially as mucolytic was started. After six doses (24 hours) from initiation of N-acetylcysteine, cyanosis and haematuria resolved. Succeeding filter paper test done was also negative for methemoglobinemia. Patient was eventually weaned from mechanical ventilation. Blood transfusion of packed red blood cell was also given for correction of anaemia prior to discharge.

**Discussion and conclusion:** Standard treatment includes the use of methylene blue and exchange transfusion. Methylene blue increases the rate of conversion of methemoglobin to haemoglobin by accepting an electron (in the presence of nicotinamide adenine dinucleotide phosphate [NADPH] and methHb reductase), to form leucomethylene blue, which can then donate this electron to reduce methHb. The use of NAC as a direct reducing agent in treating methaemoglobinaemia have been investigated to be effective in vitro studies (Wright, 1998). However, recent studies showed that NAC failed to show enhanced reduction in human volunteers with nitrite induced methemoglobinemia. In this case report, clinical response to acetylcysteine may be due to as a reducing agent as well as an effective antioxidant.

**Recommendation:** Possible therapeutic effects on herbicide induced methemoglobinemia of acetylcysteine may be investigated as a therapeutic alternative to address the paucity of standard antidotes among developing countries.