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RESEARCH ARTICLE

A CASE OF ACUTE DAPSONE POISONING

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ABSTRACT

2 year old child presented with acute onset respiratory distress after ingestion of multiple Dapsone tablets accidentally. Relevant blood investigations revealed methemoglobinemia. Child responded with freshly prepared Inj. Methylene Blue and discharged after 7 days.

Key Words:

Dapsone poisoning,
Methemoglobinemia,
Methylene Blue.

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INTRODUCTION

A 2-year-old male child presented with acute onset respiratory distress along with bluish discoloration of lips, tongue, fingers, nails and general body surface. On enquiry the attendant's give history of accidental intake of multiple tablets by the child. The medication was for the child's uncle who is suffering from Leprosy. There is no history of fever, cough, cold, swelling of lower limbs. The child was absolutely normal before the incident. On examination the child is irritable, tachypneic. Vitals: RR -78/min, PR - 114/min, central and peripheral cyanosis present, chest-bilateral vesicular breath sound present, CVS -S1,S2 normal, Spo2 with O2 -72%. The child was given moist O₂ inhalation and investigations were sent. The peripheral blood was muddy coloured and did not change its colour when free flow oxygen was given through it. His lab reports were Hb of 7.9 gm%, retic count 2.2 % of RBC, G6PD - Normal, PBS for cell morphology -RBC series - A mixed population of normocytes with microcytes, moderate hypochromia with mild anisocytosis. Platelets and WBC series are normal. ESR -6, Urine R/E-WNL, Screening test for Methemoglobinemia -Positive. Urea -23.11, Creatinine - 0.30, Sodium 5, Potassium 5.3, Calcium 10.7. Arterial Blood Gas analysis was within normal limit. CXR and ECG were normal.

Based on the history, clinical examination and investigation a provisional diagnosis of Acute Dapsone Poisoning was made and the child was given gastric lavage with activated charcoal. Freshly prepared Inj. Methylene Blue (1%) solution @2mg/kg was given. The child improved and was discharged after 7 days. Before giving discharge, Blood was screened for Methemoglobinemia and it was negative.

DISCUSSION

Accidental dapsone poisoning is a pediatric emergency in young preschool children (Nair, 1984). With the increasing use of dapsone for diseases other than leprosy and dermatitis herpetiformis, such as acne vulgaris, psoriasis, and pneumocystis carinii pneumonia infection in acquired immunodeficiency syndrome, clinicians should be aware of its toxic potential (Trillo, 1992; CDC, 1991). Dapsone, (4,4-diaminodiphenylsulfone) a sulfone' is well absorbed on oral ingestion, with peak levels after two to six hours. The drug can be detected in tissues up to three weeks after ingestion. The half-life normally varies from 9-45 hrs (mean 30 hrs.) but in toxic doses may be prolonged to two to four days. It causes methemoglobinemia resulting in cyanosis. The clinical symptoms vary and depend on the methemoglobin concentration in the blood. Methemoglobin is incapable of binding oxygen and also increases the affinity of the unaltered hemoglobin for oxygen, shifting the oxygen dissociation curve to the left thus further impairing oxygen delivery resulting in dyspnoea. The CNS manifestations occurring in children with dapsone poisoning have been described earlier (Schvartsman, 1964; Khan, 1981; Davies, 1950).

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| Conc. of methemoglobin | Clinical Symptoms |
|------------------------|--|
| <15% | Mostly no symptoms |
| 15-20% | Cyanosis, headache, drowsiness |
| 20-45% | Marked cyanosis, nausea |
| 45-70% | Severe cyanosis, vomiting, convulsions |
| >70% | Death |

The manifestations observed in these children were irritability, hypotonia, truncal ataxia, choreiform movements and dysarthritic speech. Direct effects of the drug on CNS and cerebral anoxia due to methemoglobinemia have been attributed as the main causes. In acute dapsone toxicity, initial attempts should be directed towards gut decontamination (gastric lavage, activated charcoal orally) and improvement of oxygen delivery. To improve oxygen delivery, the main emphasis is on the administration of reducing agents such as methylene blue and ascorbic acid. Methylene blue given intravenously is rapidly reduced to leukomethylene blue in the presence of NADPH and methemoglobin reductase, leukomethylene blue then becomes available to reduce methemoglobin to hemoglobin. It is the mainstay of treatment in severe methemoglobinemia. In less severe cases, ascorbic acid 200-500 mg can be given intravenously (Kumar, 1988). It is suggested that cases previously perfectly normal and presenting with unexplained central cyanosis with history of ingestion of dapsone be considered as having methemoglobinemia.

Dapsone, a commonly used drug in the treatment of leprosy should be kept out of reach of children to prevent significant morbidity and mortality.

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