*Case report*

# Encountering dapsone poisoning in a child at the emergency department of a tertiary care hospital in eastern Nepal

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**Abstract**

Dapsone (4, 4'-diaminophenylsulfone), a sulfonamide derivative, was introduced in 1943 as an effective chemotherapeutic agent for leprosy and still is an important drug for the treatment of this disease. Because of its use in various conditions, its toxicity is commonly seen in adults but rare in children. Rapid clinical assessment, measurement of methemoglobin and institution of methylene blue where indicated decreases the morbidity and mortality of dapsone poisoning. We report a case of 2 year old female with accidental ingestion of 20 tablets (20gm) of dapsone.

**Keywords:** dapsone, leprosy, poisoning

## Introduction

Dapsone (4, 4'-diaminophenylsulfone), a sulfonamide derivative, was introduced in 1943 as an effective chemotherapeutic agent for leprosy and still is an important drug for the treatment of this disease.1,2 The indications for dapsone are varied and include the management of *Pneumocystis carinii* pneumonia (PCP) in immunosuppressed patients, dermatitis herpetiformis, acne vulgaris, psoriasis, pemphigus, lupus erythematosus profundus,brown recluse spider bites, as well as prophylaxis and treatment of falciparum malaria.3-7 Because of its use in various conditions, its toxicity is commonly seen in adults. However, dapsone intoxication in children is usually accidental and has been considered invariably fatal.8 Accidental dapsone poisoning is a pediatric emergency in young preschool children and is uncommon. 9 We report a case of dapsone poisoning encountered at the emergency department of B. P. Koirala Institute of Health Sciences (BPKIHS), a tertiary care hospital in eastern part of Nepal.

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## Case report

Two year old female was brought to the emergency department of B. P. Koirala Institute of Health Sciences located in eastern Nepal after 7 hours of accidental ingestion of 20 tablets (2gm) of dapsone. The source of dapsone was a friend whose father was taking it for leprosy. On examination she was centrally cyanosed with pulse rate of 118 beats per minute (regular, good volume), respiratory rate of 36 per minute, blood pressure of 110/70 mm of Hg with normal temperature. Her SPO2 in the emergency varied from 72% to 90% on oxygen. Her mental status varied from irritability to drowsiness.

She looked pale and had no icterus, clubbing or oedema.The bluish discoloration of the lips and tongue soon progressed to face and whole body. Other systemic examination was within normal. The blood investigations revealed normal urine (routine and microscopy), blood glucose, urea, creatinine, serum electrolytes with hemoglobin of 9.6 gm/ dl, hematocrit of 32.1%, total leukocyte count of 12900 cells/cu

mm, differential count of Neutrophils 65 and Lymphocytes 35 and platelet count of 242000 cells/ cu mm. Her arterial blood gas revealed pH=7.456, PCO2=22.8, PO2=105 and HCO3 -=17.8. She was admitted in the Paediatric ward for further management but she left against medical advice (LAMA) on second day of admission. After 6 hours of leaving the Paediatric ward, she was brought to the emergency department again with paleness, weakness, shortness of breath and one episode of abnormal body movements in the form of tightness of body parts with uprolling of eyes. On examination she had respiratory rate of 38 per minute with subcostal and intercostal recession, pulse rate of 144 beats per minute (regular, high volume), blood pressure of 130/80 mm of Hg and temperature of 101 degree F. Her SPO2 was 60-70 % with oxygen via simple face mask at the rate of 8-10 L per minute. She was pale with no jaundice, clubbing, lymphadenopathy and edema. She had normal sized pupils with normal reaction to light. Other systemic examination was normal with no signs of meningism. The blood investigations revealed normal urine (routine and microscopy), blood glucose, urea, creatinine, serum electrolytes with hemoglobin of 8.2 gm/dl, hematocrit of 28.6 %, total leukocyte count of 16900 cells/ cu mm, differential count of Neutrophils 55, Lymphocytes 40 and Monocytes 05 and platelet count of 309000 cells/ cu mm. Her arterial blood gas revealed pH=7.342, PCO2=26.3, PO2=84.8 and HCO3 -=16.1. She was admitted in the Pediatrics ward. Her methaemoglobin level was 35 % at admission. She was given intravenous injection of methylene blue at the dose of 1 mg per Kg of 1.0 % solution with ascorbic acid 500 mg. She was given total four doses of methylene blue. Her methaemoglobin level was 35.2 % on 3rd day of admission and its level decreased to 3 % on 5th day of admission. She had multiple episodes of seizures on the first and the second day of admission which was managed by intravenous infusion of phenytoin. She received three aliquots of blood for her anemia. On 4th day of admission, she developed acute renal failure with urea=256 mg/dl and creatinine=2.4mg/dl. The levels increased to values of urea=321 mg/dl and creatinine=3.8 mg/dl on 5th day. On 6th day of admission in ward, she was transferred to Paediatric Intensive Care Unit (PICU) where she received 83 cycles of peritoneal dialysis with other supportive management under close monitoring. Her renal parameters came to normal levels on 11th day of stay in PICU. After 13 days of treatment in PICU, she was shifted to the Pediatric ward where she was observed for two more days and was discharged. She was followed up after one week with investigations for urea, creatinine and urine (routine, microscopy & culture) all of which were within normal limits.

**Discussion**

Accidental dapsone poisoning in children is uncommon. The poisoning may present with severe clinical symptoms like cyanosis, nausea, vomiting, headache, respiratory difficulty, drowsiness and abnormal body movements. The clinical symptoms vary and depend on the methemoglobin concentration in the blood.10 In our case, there was cyanosis, irritability, paleness, weakness, shortness of breath and abnormal body movements with an initial methaemoglobin level of 35%. Severe poisonings with methemoglobinemia over 70 % usually are fatal. Early administration of the antidote methylene blue usually leads to rapid improvement of the cyanosis. Because of the long half-life of the toxicant, repeated administration of methylene blue may be necessary.11 In our case; four doses of methylene blue was required to decrease the level of methaemoglobin. Methemoglobin has decreased affinity of the unaltered hemoglobin for oxygen, shifting the oxygen dissociation curve to the left thus further impairing oxygen delivery resulting in shortness of breath as seen in our case. This also explains why the delivery of oxygen to patient does not improve the oxygen saturation level. Exchange transfusion has been tried in a case not

responding to methylene blue with successful result.12 However, it was not necessary in our case. It is well known that dapsone is responsible for hemolysis process which explains the low level of hemoglobin in our case.13 In this case, excessive levels of dapsone may have led to corresponding decrease in hemoglobin levels. Renal involvement in dapsone poisoning is not commonly reported. This report tries to emphasize the importance of diagnostic and treatment of dapsone poisoning in children. Theoretically, the majority of patients with leprosy are treated with dapsone in the basic units of health; so many children are at risk to the exposure of dapsone causing its poisoning.

**Conclusion**

Any case presenting to emergency department with cyanosis and breathlessness not responding to oxygen must be suspected as a case of methaemoglobinemia. With increasing use of dapsone in many dermatological conditions along with leprosy, dapsone should be suspected as a possible cause of methemoglobinemia. Rapid clinical assessment, measurement of methemoglbin and institution of methylene blue where indicated decreases the morbidity and mortality of dapsone poisoning in children.

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