LETTERS TO EDITOR

MICROANGIOPATHIC HEMOLYTIC ANEMIA FOLLOWING DISSEMINATED INTRAVASCULAR COAGULATION IN ALUMINUM PHOSPHIDE POISONING

Sir,

Aluminum phosphide is commonly available as an insecticide, which is also a common mode of suicide in India. Intravascular hemolysis and methhemoglobinemia have been reported with aluminum phosphide poisoning, but microangiopathic hemolytic anemia (MAHA) has never been reported. Our case highlights this issue.

A 16-year-old schoolgirl presented to the emergency ward after ingestion of 1 fresh tablet of aluminum phosphide (celphos) mixed with water. According to the attendant, she vomited 8-10 times and had profuse sweating after ingestion of celphos tablet. There was no history of fever, headache, chest pain, breathlessness, convulsion, drug intake or loss of consciousness after ingestion of celphos tablet. Past history was not significant.

At the time of admission, she was drowsy, restless, irritable and not following command with E4M6V3. Extremities were cold; pulse rate was 100/min, low volume; respiratory rate, 18/min; and systolic blood pressure, 70 mm Hg. Results of respiratory and abdominal examinations were normal. Cardiac examination revealed tachycardia with normal heart sounds and rhythm, and there was no murmur. Patient was managed with gastric lavage and airway maintenance, and there was proper management of shock, with regular monitoring. Immediately 16G IV cannula was inserted and 1000 mL of 0.9% saline was given intravenously for 15 minutes. Her BP increased to 110/70 mm Hg. She was put on maintenance IV infusion of 0.9% saline[@] 100 mL/h. Close clinical monitoring was done, particularly for the state of consciousness, urine output and vital signs with continuous electronic monitoring. Her Arterial Blood Gas analysis (ABG) was normal except for hyponatremia with serum sodium of 123 mmol/L. Electrocardiogram was normal at presentation except for tachycardia. The results of her blood investigations, including general blood picture, complete blood count, random blood sugar and routine serum biochemistry, were within normal limits except for hyponatremia. Her hemoglobin was 13.5 g/dL. Four hours after admission, her BP was 112/72 mm Hg without any postural drop, pulse rate was 78/min and respiratory rate was 16 but she was still drowsy.

On day 2, she was conscious, sitting on bed; with evident pallor, which was not present earlier. There was no bleeding spot, epistaxis or history of gastrointestinal bleed. But she developed gum bleed while brushing teeth. Her blood investigations were done again, and reports revealed hemoglobin, 9.1 g/dL; hematocrit, 20.5%; platelet count, 76000/µL; normal total and differential count; serum aspartate aminotransferase (AST) 67 IU/L; serum alanine aminotransferase (ALT) 32 IU/L; total bilirubin, 2.0 mg/dL; indirect bilirubin,

1.5 mg/dL, with normal total protein, alkaline phosphatase, calcium, phosphorus, ABG and renal function test. General blood picture (GBP) showed presence of schistocytes and anisocytosis, normal white blood cells and reduced platelet count, consistent with microangiopathic hemolytic anemia. Corrected reticulocyte count was elevated (4%). Glucose-6-phosphate dehydrogenase (G6PD) level was normal. D-dimer level was elevated (500 ng/mL). Prothrombin time (PT) and activated partial thromboplastin time (APTT) were prolonged, with international normalized ratio (INR) of 1.52. Results of direct and indirect Coombs' tests were negative.

She was given 1 unit of packed cell transfusion on the second day of admission. No other medication was given from the second day onward. On the 4th, 5th and 10th day, hemoglobin level progressively increased, and it was 9.9, 10.2 and 13.8, respectively. On the 10th day, her GBP, liver function test and D-dimer level (40 ng/mL) were normal. Daily ECG was done, and the results of these ECGs indicated normal traces.

Usually there is only a short interval between ingestion of aluminum phosphides and appearance of systemic toxicity. Phosphine frequently causes impairment of myocardial contractility; metabolic acidosis or a combination of metabolic acidosis and respiratory alkalosis; or acute renal failure. Other features include disseminated intravascular coagulation, and hepatic necrosis.[1] Methhemoglobinemia and intravascular hemolysis have been reported with aluminum phosphide poisoning, but microangiopathic hemolytic anemia has not been reported.[2,3] Development of hemolytic anemia usually takes 1 to 3 days following acute insult. Rate of recovery following insult varies from 1 to 4 weeks, depending on concomitant other causes of anemia, like nutritional anemia, and speed of diagnosis and management.[4]

In our case, the patient had microangiopathic anemia following ingestion of aluminum phosphide (celphos). Aluminum phosphide is a redox substance; so theoretically, it can cause hemolytic anemia. Aluminum phosphide could be associated with disseminated intravascular coagulation, as stated above; and disseminated intravascular coagulation (DIC) is a well-recognized cause of MAHA. Mechanism of MAHA is the formation of a fibrin mesh due to increased activation of the coagulation system. The red blood cells are physically cut by these protein networks, and the fragments are identical to the schistocytes seen on light microscopy.

VISHAL KHURANA, I. S. GAMBHIR, DHIRAJ KISHORE
Department of General Medicine, Institute of Medical Sciences (IMS), Banaras Hindu University (BHU), Varanasi, India

Correspondence:
Dr. Vishal Khurana,
Room No. 5, Old PG Doctor`s Hostel,
Banaras Hindu University (BHU)
Varanasi, Uttar Pradesh-221005, India

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DOI: 10.4103/0019-5359.53396