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 methemoglobinemia 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. 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. Methemoglobinemia and intravascular hemolysis have been reported with aluminum phosphate poisoning, but microangiopathic hemolytic anemia has not been reported. 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.

In our case, the patient had microangiopathic anemia following ingestion of aluminum phosphate (celphos). Aluminum phosphate is a redox substance; so theoretically, it can cause hemolytic anemia. Aluminum phosphate 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

REFERENCES

Sir,

The presence of blood in the semen is called hematospermia and represents 1% of all andrologic and urologic symptoms.[1] Hematospermia is an uncommon clinical condition that usually follows a painless, benign and self-limited course; but it can be a source of considerable fear and anxiety that is uncommonly associated with significant underlying pathology and is mostly considered to be idiopathic in nature.[2] Hematospermia is classified by its cause, which may be an inflammation or infection, an obstruction or cyst, a vascular abnormality, a systemic disease, a tumor or an idiopathic condition.[3] Management with appropriate clinical evaluation, watchful waiting, and reassurance often suffices without further diagnostic workup or treatment.[2]

A 32-year-old man referred to our hospital in Gorgan, north of Iran; he presented with recurrent painless hematospermia of 18 months' duration. He did not have any history of urinary tract infections; sexually transmitted diseases; prolonged and intense masturbation or sexual intercourse; hypertension; renal, liver or bleeding disorders; abdominal trauma or surgery. He did not report erectile dysfunction (International Index of Erectile Function score, 25). The findings of general physical examination of the patient were normal. No evidence of trauma or self-instrumentation in the urogenital region was found. The prostate was normal in size and consistency, and no abnormal masses were palpable on digital examination. External genitalia, testes, epididymis, cord and penile urethra were normal. Seminal vesicles were not palpable. The urethral meatus was evaluated, and no evidence of trauma, condylomata and phimosis was found. Hematologic studies (PT, PTT, CBC and ESR), urinalysis, urine culture, semen analysis and semen culture were done, and none of these revealed any abnormality. Results of PSA test, mycobacterial cultures, urethral cultures for gonorrhea and chlamydia, transrectal ultrasound of the prostate and abdominopelvic sonography were normal. Empirical treatment with an antibiotic (quinolone) and an anti-inflammatory medication (selective COX-2 inhibitor) was administered, to which there was no response. The patient was evaluated for epididymitis, prostitis, urethritis, HIV, condylomata, urinary stones, liver cirrhosis, arterial hypertension, genitourinary TB and hematologic disease, and he was normal in all these assessments; so the specialists reassured him that this condition was benign and self limited. Finally, he reported ASA consumption due to hemoconcentration 2 years ago. ASA effect as a causation of hematospermia was suspected. We carried out coagulation tests (PT, PTT, BT, and CT), which, again, were normal except for BT, which was slightly prolonged. So ASA was discontinued and hematospermia stopped and was not found to appear during the follow-ups in the following 6 months. For confirmation of the mentioned diagnosis, ASA was restarted and hematospermia reappeared. We can affirm this cause-and-effect relationship to some extent because of the reappearance of