Organophosphorus Poisoning Presenting as Diabetic Ketoacidosis: A Real Challenge for the Endocrinologist

Sir,

Diabetic Ketoacidosis (DKA) is characterized by hyperglycemia, metabolic acidosis and hyperketonaemia.1 Toxic exposure to Organophosphorus (OP) poisoning is a major public health problem globally with more than three million poisonings and 200,000 reported deaths per year.2 Presentation of OP poisoning as DKA is very rare. The cause of hyperglycemia in OP poisoning may be due to body glucose homeostasis by physiological, oxidative and nitrosative stresses, disturbance in liver tryptophan metabolism, stimulation of adrenal gland, inhibition of cholinesterase, pancreatitis and inhibition of paraoxonase.3 The mode of exposure to OP poisoning is not obvious in some patients; this can lead to higher morbidity and mortality.

We are reporting the case of a 17-year-old male, student of Grade 10, who was presented in Emergency Room (ER) with a 2 days history of vomitings and drowsiness since two hours. There was no history of fever or convulsions. His past medical history was unremarkable. When he arrived in ER, the patient was drowsy and dehydrated with Glasgow Coma Scale of 7. He suddenly went into asystole cardiac arrest. ER team followed Advanced Cardiac Life Support (ACLS) protocol and intubated the patient.

Post-Cardio Pulmonary Resuscitation (CPR) vitals were blood pressure was 122/88 mmHg, pulse was 60/minute, temperature was 37°C, oxygen saturation was 100% on ventilator support. There were right basal crackles on auscultation. Cardio vascular and abdominal examinations were normal. In cerebrovascular examination, patient was intubated and ventilated, pupils were 3 mm in size, reactive to light and planters’ reflex was down going bilaterally.

Laboratory investigations revealed hemoglobin 13.6 g/dL, total leucocyte count 26 x 10^9/mL, platelets 189 x 10^9/mL, blood urea 32 g/dL, serum creatinine 1.23 g/dL, serum sodium 144 mEq/L, chloride 106 mEq/L, potassium 4.0 mEq/L, bicarbonate 23 mEq/L, anion gap-15, blood glucose level 462 g/dl. Arterial blood gases showed pH 6.9, PaCO2 62.3 mmHg, PaO2 59.4 mmHg and bicarbonate 12.8 mEq/L. Urine detailed report revealed glucose of 300 mg/dl and ketones of 150 mg/dl. Chest radiograph showed right lower lobe pneumonia. In view of hyperglycemia, metabolic acidosis and ketonuria, diagnosis of DKA was made in the ER.

Treatment for DKA was started immediately and patient was later shifted to intensive care unit. With the management of DKA, the blood glucose levels improved within four hours and needed no further insulin therapy. However, the condition of the patient and the pH in the arterial blood gases did not improve. Due to increased lacrimation, profuse sweating and treacheal secretions, heart rate of 56/minute and pinpoint pupils, there was suspicion of OP intoxication, therefore, plasma cholinesterase level was sent. Patient was managed in lines of OP poisoning after five hours of coming to the hospital with intravenous atropine and pralidoxime. He responded well to treatment, extubated after 48 hours and discharged after 96 hours of extubation. Plasma cholinesterase result was 392 U/L (normal = 2710 - 11510 U/L) and HbA1C was 5.5%. There were no clinical sequelae and later patient had normoglycemia with a two weeks follow-up. Retrospectively, he gave history of ingestion of OP poisoning due to failure in his previous examinations.

Most common route of exposure of OP poisoning is through ingestion of agricultural products. A 5-year-old girl presented with DKA after using locally made anti-lice shampoo containing OP.4 This patient ingested OP for deliberate self-harm. The patient classically presented in the ER with features of DKA, therefore, OP poisoning was not suspected at presentation. It is important to take detailed history when the mode of poisoning is not obvious. Clinical muscarinic symptoms, low cholinesterase levels, and improvement with atropine and pralidoxime supported the diagnosis of OP poisoning. Previous few cases of DKA with OP poisoning were also initially managed in lines of DKA and later treated for OP poisoning because of clinical symptoms and low cholinesterase levels.5 There are various signs and symptoms of OP poisoning and it is very difficult to diagnose this problem especially in children. It becomes even more difficult if we do not have reliable history while initially managing adult patients in the ER. Whenever, the patient of DKA is not improving with the standard management, we emphasize that an alternate diagnosis should be explored. OP poisoning can present as DKA in rare cases for which clinical symptoms and cholinesterase level is required to establish the diagnosis.

REFERENCES


