Organophosphorus Poisoning Presenting as Diabetic Ketoacidosis

A 12 year old male was brought to us with history of fatiguability, vomiting and loose stools for 6 hours. There was no history of fever, convulsion, and altered sensorium. The vitals were stable except some dehydration. Investigations revealed hemoglobin 10.8g/dL, TLC 16.6×10^3/mL, platelet 360×10^3/mL, blood sugar 299g/dL, blood urea 28g/dL, S creatinine 0.9g/dL, Na 139 mEq/L,K 3.3mEq/L, pH 7.38, $PaCO_2$ 32 mmHg, PaO_2 96 mmHg, and Bicarbonate 19 mEq/L. Urine showed sugar 4+ and moderate ketone bodies. In view of dehydration, hyperglycemia, glycosuria, ketonuria, low bicarbonate levels, DKA treatment protocol was started with IV fluids and insulin infusion. The level of consiousness deteriorated by 12 hours and Glasgow Coma Scale was 12. He developed fasiculations and jerky movement of limbs. His respiration was 28/min and shallow, heart rate 64/min, BP 110/70 mm of Hg, oxygen saturation 96%. His pupils were 2mm in size and were reactive. CT scan head was normal. Due to fasiculations and shallow respiration, organophosphorus intoxication was suspected and plasma cholinesterase was done; it was 550 U/L (Normal=2710-11510 U/L). The diagnosis was revised to organophosphorus intoxication and child was managed with atropine and pralidoxime. He responded well and was discharged after 6 days. Retrospectively, boy gave history of ingestion of 4 tomatoes in the field without washing 6 hours prior to admission.

The most common route of exposure to organophosphorus compounds is ingestion of agricultural products [1]. Probably our patient had poisoning from eating tomatoes contaminated with pesticide. Organophosphorus poisoning was not suspected at presentation, as the child presented to us with muscarinic symptoms like vomiting and diarrhea. Low levels of plasma cholinesterase support the diagnosis of OP poisoning [2]. Although plasma acetyl cholinesterase estimation was sufficient to support the diagnosis of organophosphorus poisoning in our case, we were unable to do estimation of RBCs ACE and urinary para-nitrophenol for technical reasons. Hyperglycemia is a known adverse effect of organophosphorus exposure and has been confirmed in animal studies [1,3-5]. The glucose metabolism is affected by several mechanisms, including oxidative stress, inhibition of paroxanase, stimulation of adrenal glands and release of catecholamines, and effect on metabolism of liver tryptophan [4]. In an earlier study glycosuria was observed in 69% of cases with OP poisoning. Organophasphorus intoxication can mimic DKA and its diagnosis may be delayed. Whenever there is discrepancy between clinical features and biochemical features in a suspected, DKA child, we should emphasize the need to look for an alternate diagnosis.

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