OBSERVATIONS

Diabetic Ketoacidosis: An Uncommon Manifestation of Pesticide Poisoning

Diabetic ketoacidosis in adolescents can be a cause of severe morbidity and mortality, especially if unrecognized at the time of first diagnosis (1). We present a 15-year-old girl who was initially treated for "diabetic ketoacidosis" with further worsening of her general condition. This delayed recovery, coupled with focused investigations, finally led us to a diagnosis and the appropriate management of an intentional overdose with organophosphorous (OP) pesticide, presenting as diabetic ketoacidosis. Our aim is also to raise awareness of the effects of organophosphates, widely used as pesticides, on glucose metabolism, and its potential implications in the epidemic of diabetes worldwide.

A 15-year-old girl was admitted with a 10-h history of giddiness, vomiting, and drowsiness. She was apparently well the evening prior to her admission. Clinical examination revealed a drowsy patient with a Glasgow Coma Scale of 10. Her blood pressure was recorded at 88/60 mmHg. Her pupils were constricted, and there was neither focal neurology nor any meningeal signs. Random glucose was recorded at 18.6 mmol/L (336 mg/dL), and blood pH was 7.2 with a positive urine dipstick for ketones. Routine blood investigations, chest X-ray, and an MRI of the brain revealed no significant abnormalities. A diagnosis of diabetic ketoacidosis was made in view of the above findings, although the history was not convincing. Diabetic ketoacidosis treatment protocol was instituted, but the patient's general condition continued to worsen, and she needed to be ventilated in spite of correcting the metabolic parameters. By day 2, the glucose levels were normal and needed no further insulin therapy, and the acidosis had been resolved but the patient continued to be restless and needed to be persistently ventilated. A literature search for other potential causes for this presentation suggested the possibility of OP poisoning. Cholinesterase levels were low at 326 units (3,700–11,500), supporting the above diagnosis. Upon further detailed questioning, her parents admitted that their daughter had consumed an unspecified amount of insecticide poison on the morning of her admission, attributed to apparently poor results on her board exams. Atropine and pralidoxime therapy were instituted with excellent clinical improvement, followed by extubation in 48 h. She was discharged shortly thereafter with no clinical sequelae and normoglycemia at a 4-week follow-up.

OP compounds are chemicals used in a wide variety of industrial and domestic settings. In recent years, there has been increasing recognition of the effects of OP compounds on glucose homeostasis. In animal models, these compounds seem to affect glucose homeostatic pathways, which eventually lead to hyperglycemia (2). There is, worryingly, a growing body of data linking pesticide exposure and diabetes in humans as well (3,4). OP poisoning may also rarely masquerade as diabetic ketoacidosis (5) with the potential for erroneous treatment.

Traditionally, the risk factors for diabetes have focused on genetics and lifestyle factors. Dramatically increasing pesticide use may be an important missing link, contributing to the epidemic of diabetes worldwide. While pesticide use is well-entrenched in the today's world, urgent research is necessary to further explore this link along with novel approaches to reduce the metabolic effects of such pesticides.

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