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SCIENTIFIC LETTER

Acute Kidney Injury and Encephalopathy in a Child: Diethylene Glycol Poisoning

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To the Editor: Diethylene glycol (DEG), a clear, odorless solvent is sometimes present as a toxic adulterant in propylene glycol, a component of common medications like paracetamol and cough syrups [1]. It's ingestion can result in multiorgan failure with lethal consequences. Several such incidents of mass poisonings, causing significant morbidity and mortality have been reported [2–4].

A 5-y-old boy was brought with complaints of fever, cough and cold for 3 days, vomiting and anuria for one day. He had taken some oral medications for fever and cough. He was febrile (100° F) and tachypneic. Laboratory studies showed high anion gap metabolic acidosis (pH- 7.298, bicarbonate 10.8/mEq/L, lactate 0.6 mmol/L), uremia (urea 195 mg/dl, creatinine 5.8 mg/dl) and raised transaminases (Aspartate transaminase 403 IU/L, Alanine transaminase 408 IU/L). Renal ultrasonography showed bilateral raised cortical echogenicity. The child was started on antimicrobials, hemodialysis and supportive therapy. He developed hypertension, encephalopathy with absent brainstem reflexes, flaccid quadriparesis and respiratory failure; mechanical ventilation was initiated. Dengue antigen test, scrub typhus and Leptospira serology and markers for acute viral hepatitis were negative. Anti-streptolysin-O antibody and C3-complement levels were normal, antinuclear antibody and anti-ds DNA antibody were negative. Urinalysis could not be done as the child had complete anuria. Media reports of deaths of 9 children in Jammu region attributed to DEG poisoning from contaminated cough syrup, the same brand and batch which our index case had consumed alerted us to the possibility of DEG poisoning [5]. Analysis of medicine revealed high concentration of DEG. With supportive measures, over next two weeks, urine output and renal functions improved, brainstem reflexes and spontaneous respiratory efforts reappeared. The child was weaned from ventilator after 2 months and was discharged with normal renal functions but severe neurological sequalae and quadriparesis.

DEG, because of its physical properties and low cost, is substituted in some pharmaceutical preparations, which if consumed in toxic doses (~1 ml/kg, 14%) leads to lethal outcomes [1]. It causes acute kidney injury, hepatotoxicity and neurological complications. Poisoning can be suspected in a patient with typical clinical manifestations with high anion gap metabolic acidosis along with unexpected clustering of similar cases. Definitive diagnosis requires DEG serum levels. Presumptive diagnosis can be made by testing suspected medication for presence of DEG. Management centres around acid-base and fluid status, early hemodialysis and timely administration of antidote (fomepizole, ethanol) [1].

It is important to generate awareness amongst physicians about this entity as only a high index of suspicion can lead to timely withdrawal of offending agent. There is an urgent need to impose strict regulations on pharmaceutical companies and regular checks on the production as well as sale and purchase of drugs.

Compliance with Ethical Standards

Conflict of Interest None.

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