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PESTICIDE POISONING: DIAGNOSTIC DIFFICULTIES AND FULMINANT HEPATIC FAILURE

Mariam Metreveli[1], Davit Topuria[2], Eka Kurdadze[3]

[1]Clinical Toxicologist, Toxicology Department, TSMU First University Clinic, Tbilisi, Georgia.

[2]MD Dean, International Faculty of Medicine and Dentistry, Tbilisi State Medical University; Associate Professor, Clinical Toxicologist, Tbilisi, Georgia

[3]Assistant Professor, Department of Anesthesiology, Resuscitation and Toxicology, Tbilisi State Medical University (TSMU); Head of the Toxicology Department, TSMU First University Clinic; Clinical Toxicologist, Tbilisi, Georgia

Background. Poisoning incidents involving highly hazardous pesticides often go undetected in low- and middle-income countries (FAO/WHO, 2016), and nonspecific presentations delay recognition.

Case presentation. A 64-year-old hypertensive man had 3–4 days of fever, myalgia, abdominal pain and distension, and a vesicular rash spreading from the face and mouth to the trunk and limbs. Ten days earlier he had sprayed a poultry house and vineyard with unidentified chemicals. On admission he was alert (BP 180/100 mmHg, SpO2 97%, temperature 37.2 °C) with facial vesicles, violaceous chest macules and a distended, diffusely tender abdomen. Tests showed WBC 17.7×10^9/L, CRP 11 mg/L, AST 974 U/L, ALT 833 U/L, bilirubin 12.8 μmol/L, INR 1.03, sodium 123 mmol/L, lactate 4.5 mmol/L and pH 7.51. Contrast CT excluded acute surgical pathology and vascular occlusion.

Management and course. He received antibiotics, corticosteroids, oxygen, albumin, fresh frozen plasma and hepatoprotective therapy. By day 2, AST rose to 3274 U/L and ALT to 2459 U/L, with INR 1.6, fibrinogen 0.8 g/L, LDH 8407 U/L, CK 2687 U/L and creatinine 151.7 μmol/L; periorbital edema, petechiae and purpura developed. Hypoxemia (SpO2 78% on room air) persisted despite CPAP; he was intubated on day 3 and developed hemodynamic collapse requiring vasopressors. Asystole occurred and resuscitation failed; death was declared 44 hours after admission.

Discussion. Without toxicology laboratory capacity, as in many resource-limited settings, diagnosis rested on exposure history and clinical picture, and the agent remained unidentified. Patients often cannot name the product, its source or how it was prepared, and label instructions and protective measures are frequently not followed (FAO/WHO, 2016). Progressive hepatocellular necrosis with lactic acidosis, hypoxemia, rhabdomyolysis, kidney injury and

probable DIC after recent spraying is compatible with severe pesticide toxicity, such as phosphine from aluminium phosphide, a highly toxic fumigant without a specific antidote, although the 10-day interval and vesicular rash are atypical. Rash, leukocytosis and abdominal distension suggested dermatologic, infectious or surgical causes, while INR was normal at presentation and CRP modest.

Conclusion. In patients with unexplained rash, abdominal symptoms and rising transaminases after recent pesticide exposure, toxic hepatitis should be considered early even when INR is normal, and exposure details (product, source, preparation, protective measures) should be sought from the patient and relatives. Serial monitoring of INR, fibrinogen, lactate and organ function and early intensive care are needed to recognize fulminant progression.

Keywords: pesticides; poisoning; fulminant hepatic failure; multiple organ failure; diagnosis.