Oral Abstracts

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GLUFOSINATE HERBICIDE POISONING – A REVIEW

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Abstract: Glufosinate (D, L-phosphinothricin) derived from *Streptomyces* spp. with herbicidal activity was registered and marketed since 1984. It is a glutamate analog and is relatively safe for agricultural purposes as compared to other nonselective herbicides such as glyphosate and paraquat based on the oral median lethal dose (LD50) estimation. In rats and mice, the LD50 values were 1510–1660 mg/kg and 436–464 mg/kg, respectively. In humans, the estimated lethal dose is 5.5 ml/kg of oral exposure to 18.5% glufosinate-containing herbicide or serum glufosinate level of ≥ 15 μg/ml. After deliberate ingestion, glufosinate-containing herbicide was rapidly absorbed in the gastrointestinal tract and eliminated by the kidney. The elimination half-life was estimated to be 4.5–4.78 h in rats and 9.59 h in humans with a distribution volume of 0.53 L/kg and 1.44 L/kg, respectively. The common toxic effects were nausea, vomiting, abdominal pain, diarrhea, and mucosal injury within hours after ingestion. The typical toxic effects include delayed neurotoxicity (e.g., consciousness depression, coma, seizure, and amnesia) possibly mediated through the brain *N*-methyl-D-aspartate (NMDA) receptor and respiratory failure that develops 4–44 h post ingestion. Several deaths resulting from hypotension, shock, and cardiovascular collapse, which may have occurred due to the anionic surfactant sodium polyoxyethylene alkyl ether sulfate (AES), have been reported. Both the parent compound and its metabolite have NMDA receptor binding activity that leads to seizure in the studied animals, which could be prevented by pretreatment with NMDA receptor antagonists. Glufosinate herbicide poisoning is diagnosed on the basis of pertinent history or blood and urine glufosinate testing by mass spectrometry (e.g., GC-MS or LC-MSMS) with or without chemical derivation. The current management is supportive, including adequate hydration, seizure control, and artificial ventilation if respiratory failure occurs. Hemopurification, i.e., hemodialysis may not protect patients from seizure or coma; however, its efficacy on AES removal or shock management remains uncertain. Further characterization of the amnesia and evaluation of the possible therapeutic role of NMDA receptor antagonists are recommended.

Learning Objectives:
1. Acute toxicity
2. Mechanism
3. Toxicokinetics
4. Diagnosis and Management
5. Future aims