International Journal of Medical Science and Health Research

Vol.8, No. 04; 2024

ISSN: 2581-3366

Multi–Organ Damage Due to Glufosinate Ammonium Poisoning

Nguyen Dang Duc[1,2], Nguyen Phuong Sinh[2], Lam Nguyen Hong Anh[2]

[1]Poison Control Center of Bach Mai Hospital, Ha Noi, Viet Nam

[2]Thai Nguyen Medicine and Pharmacy University, Thai Nguyen, Vietnam

Corresponding: Nguyen Phuong Sinh Vice Director of Thai Nguyen University of Medicine and Pharmacy, Thai Nguyen, Viet nam. Tel: +840915965222.

doi: 10.51505/ijmshr.2024.8404 URL: http://dx.doi.org/10.51505/ijmshr.2024.8404

Received: July 20, 2024 Accepted: July 30, 2024 Online Published: Aug 15, 2024

Abstract

Glufosinate Ammonium herbicide poisoning is one of the diseases with a complicated course, a period of 4-60 hours without symptoms followed by sudden appearance of damage to multiple organs such as nerves, breathing, heart. A 67-year-old female patient admitted to our poison control center with symptoms of vomiting and impaired consciousness. Exploiting the patient's information, it was discovered that the patient bought Glufosinate ammonium and Metsulfuron methyl to take it with the intention of committing suicide. The patient was quickly treated with gastric lavage and activated charcoal. After 1 day, the patient lost consciousness, stopped breathing, and had a slow pulse of 45 beats per min, NH3 of blood 93.3 µmol/L, ECG: Sinus bradycardia, no ST elevation. The patient was then prescribed endotracheal tube placement and controlled ventilation. After 7 days of treatment, the patient's condition improved and the patient was extubated. This article aims to talk about multi-organ damage caused by Glufosinate Ammonium poisoning.

Keywords: Multi-organ damage, Glufosinate Ammonium poisoning, herbicide poisoning

1. Overview:

Glufosinate Ammonium is a herbicide that replaced Paraquat after being banned, and has recently become commonly used. Glufosinate Ammonium herbicide poisoning is one of the diseases with complicated developments, a period of 4-60 hours without symptoms, then sudden damage to multiple organs such as nerves, respiratory system, and cardiovascular system [1]. They can be absorbed through the digestive tract with a bioavailability of 30% [2]. Glufosinate Ammonium poisoning by mouth occurs when the patient mistakenly drinks or intentionally drinks it to commit suicide. Glufosinate ammonium is a popular form in Vietnam. In this report, we present a 67-year-old female patient admitted to the hospital for glufosinate Ammonium poisoning 11th hour after ingestion. The patient was brought to the hospital in a state of vomiting, decreased consciousness, along with apnea and bradycardia later.

2. Clinical case report:

67-year-old female patient with the history of depression without any treatment, insomnia for months, coronary artery stenting placed in 2017, hypertension and diabetes. She was admitted to

Vol.8, No. 04; 2024

ISSN: 2581-3366

our poison control center in a state of vomiting and decreased consciousness. It is known that on the afternoon of the same day, the patient was discovered by her family in a state of vomiting with blue fluid, and having a wound on her left wrist. The patient then confessed that she drank 2 bottles (100ml) of Shina herbicide containing glufosinate ammonium and 4 packs of Alyando 200WG herbicide containing metsulfuron methyl 2000g/kg (with little effect on mammals) and slit her left wrist with scissors. Family brought her to the hospital in a state of drowsiness (with the initial Glasgow Coma Scale 13 points), no seizures, no fever.

The patient weighs 50kg, height 153 cm (BMI: 21.3). The inital vital signs were relatively stable (Pulse:65beats per min, Blood pressure: 110/60mmHg, Breathing rate:18 beats per min, Temperature: 36.9 degrees Celcius) . The patient was drowsy, call to inquire and response was slow. Pupils on both sides were 4mm, still reflect light, with no fever. The nape is soft, the meningeal smear was negative. There was no convulsions and the muscle tone was normal, the tendon reflexes were reduced. Other agencies have not detected anything unusual.

The blood count and coagulation tests were within the normal limits. The arterial blood gas test showed pH:7.48, pCO2:31mmHg, pO2:77mmHg, HCO3:23.1 mmol/L, lactate:0.9 mmol/L. The blood sugar was 7.8 mmol/L, and the initla ammonia was 68.7 and increased to 93.3mmol/L 12 hours later. The troponin T was 8.94 ng/L, and the Cholineesterase level was 7661 U/L. The patient received the gastric lavage and one dose of activated charcoal (1g/kg) 4 hours after taking Glufosinate Ammonium at a lower level hospital. The patient's condition got worsened very quickly with a slow pulse of 40 beats per minute, a rapid decrease in consciousness level from 13 points of Glasgow down to 10 points, with a slow, irregular breathing of 8-10 beats per minute. The ECG showed sinus bradycardia as 45 beats per min, and the echocardiogram showed the EF as 68%, no wall motion disorders. The brain magnetic resonance imaging showed several nodules of white matter demyelination over bilateral frontal lobe cortex. The patient was intubated, ventilated, atropine and adrenaline were used to increase the heart rate. After 1 week of treatment, the patient was weaned off the ventilator than the endotracheal tube was removed, and the heart rate became normal (at least 60 beats per).

3. Discussion:

Glufosinate is one of the toxins that causes damage to multiple organs, especially the central nervous system [3]. Glufosinate is commonly known in the herbicide form glufosinateammonium. The mechanism of toxicity is due to glufosinate irreversibly inhibiting the glutamine synthase enzyme in the brain, cutting off the glutamate-glutamine cycle and then causing an increase in extracellular glutamate concentration, leading to irritation, damage and edemaous change of nerve cells [4]. In mammals, there are multiple pathways for ammonia metabolism, so brain damage due to increased ammonium has not been demonstrated [5]. Glufosinate has a similar structure to glutamate, so it can bind to the N-Methyl-D-Aspartate receptor of glutamate, causing the depolarization of nerve cells, leading to the neurological complications such as coma, seizures, and memory loss. [6]. Neurological symptoms often appear late but suddenly, difficult to predict and usually appearing 4-60 hours after ingestion [7]. Mild levels may cause drowsiness, headache, dizziness, vertigo, and irritation. Severe levels can

cause coma, convulsions, memory loss, and respiratory depression. Clinical symptoms of glufosinate poisoning often appear late, so predictive factors are very important during treatment. Quantifying the blood ammonia level helps monitor and predict the clinical course. Increased blood NH3 more than 90mcg/dL is a valuable factor predicting the occurrence of late neurological complications [8]. The toxicity of glufosinate is also due to the surfactants in the anionic formula and polyxyethylenealkylether sulfate causing increased vascular permeability, reduces vascular resistance, and inhibits myocardial contractility. High doses greater than 30mg per kg cause cardiac depression, bradycardia, and hypotension [9]. Our patient, intubated and mechanically ventilated, received atropine and adrenaline to increase heart rate.

4. Conclusion:

In our patient, the patient's signs and symptoms manifested in organs sensitive to glufosinate including the brain, heart and respiratory system. Identifying patients with glufosinate poisoning is through medical inquiries and toxicology tests. Once again we want to emphasize that glufosinate poisoning is one of the severe poisonings with high mortality and sequelae. Early identification of toxins and close monitoring of the patient's clinical and paraclinical condition can help the patient have a higher chance of being saved.

References

Mao YC et al.(2012) Acute human glufosinate-containing herbicide poisoning *Clin Toxicol (Phila).*;50(5):396-402. doi: 10.3109/15563650.2012.676646

Hirose Y, Kobayashi M, Koyama K, et al.(1999) A toxic kinetic analysis in a patient with acute glufosinate poisoning. *Hum Exp Toxicol.*;18(5):305-308. doi:10.1191/096032799678840110

Toshiaki Watanabe and Takuya Sano,(1998) Neurological effects of glufosinate poisoning with a brief review.*Hum Exp Toxicol.*;17(1):35-39.doi:10.1177/096032719801700106

Jeong TO et al.(2015) RESLES Following Glufusinate Ammonium Poisoning. *J Neuroimaging.*;25(6):1050-1052. Doi:10.1111/jon.12216

Hack R, Ebert E et al. (1994) Glufosinate ammonium-some aspects of its mode of action in mammals. *Food Chem Toxicol.*;32(5): 461-470. doi:10.1016/0278-6915(94)90043-4

Lantz SR, Mark CM et al.(2014) Glufosinate binds N-methyl-D-aspartate receptors and increases neuronal network activity in vitro. Neurotoxicology.;45:38-4. doi: 10.1016/j.neuro.2014.09.003

Nguyễn Thị Ngọc, (2022) Đặcđiểmlâmsàng, cậnlâmsàngvàkếtquảđiềutrịbệnhnhânngộđộccấphóachấtdiệtcỏGlufosinate. *Luận vănthạcsĩ y học*. Trường Đạihọc Y Hà nội

Cha YS et al. (2018) The relationship between serum ammonia level and neurologic complications in patients with acute glufosinate ammonium poisoningL A prospective observational study. *Hum Exp Toxicol.*;37(6): 571-579. doi:10.1177/0960327117715902

Koyam K, Goto K. (1997). Cardiovascular effects of a herbicide containing glufosinate and a surfactatnt: in vitro and in vivo analyses in rats. *Toxicol Appl Pharmacol.*; 145(2):409-414. doi:10.1006/taap.1997.8196