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# Assessment of glufosinate-containing herbicide exposure: A multi-center retrospective study

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ABSTRACT

*Background:* Exposure to glufosinate ammonium, an herbicide used worldwide, can cause CNS and respiratory toxicities. This study aimed to analyze acute human glufosinate ammonium poisoning.
*Materials and methods:* This multicenter retrospective cohort study involved five medical institutes affiliated with the Chang Gung Memorial Hospital system. Patients with glufosinate ammonium exposure visiting the emergency department (ED) between January 2008 and December 2020 were included.
*Results:* In total, 95 patients were enrolled. Compared to exposure via the non-oral route, patients exposed orally ($n = 61$) had lower GCS scores, higher mortality rates, and longer hospital lengths of stay (*P*-value: <0.001, 0.002, and < 0.001, respectively). In the subgroup analysis among oral exposure patients, the survival group had a lower amount of estimated glufosinate ingestion than the non-survival group (10.5 [3.4–27] vs. 40.5 [27–47.3] g, P-value: 0.022), lower rate of substance co-exposure (9 [19.6%] vs. 10 [66.7%] P-value: 0.001), and lower rate of paraquat co-exposure (0 [0%] vs. 7 [46.7%] $P < 0.001$) compared with the mortality group. In the orally-exposed and non-paraquat co-exposure patients ($n = 54$), age > 70 years and GCS score < 9 at triage presented a high sensitivity (100.00%, 95% CI: 63.06–100.00%) and medium specificity (58.70%, 95% CI: 43.23–73.00%) in predicting mortality.
*Conclusion:* Old age, change in consciousness, and paraquat co-exposure were associated with higher mortality in human glufosinate poisoning. Age > 70 years and GCS score < 9 at triage could be predictors of mortality in patients with acute oral glufosinate poisoning.



## 1. Introduction

Glufosinate ammonium is the main herbicide used globally [1], which is predominantly consumed in the regions located in the USA, China, Europe, Northeast Asia and South America [2]. Acute glufosinate poisoning could result in various moderate-to-severe central nervous system (CNS) and respiratory system toxicities [1-3].

Since the 1970s, glufosinate ammonium has been derived from bialaphos, and this herbicide consists of sodium polyoxyethylene alkyl ether sulfate [3]. It acts as a glutamate analogue and causes irreversible inhibition of glutamine synthetase, which hinders the synthesis of glutamine from glutamate and ammonia in plants. Subsequent intracellular accumulation of ammonia causes tissue necrosis that eventually leads to plant death [3]. In humans, the mechanism of glufosinate ammonium poisoning might be related to the inhibition of glutamine synthetase and the toxicity of the surfactant [2].

Gastrointestinal symptoms are the most common indications of glufosinate herbicide intoxication. Moderate-to-severe CNS toxicities include stupor, drowsiness, seizure, coma, agitation, confusion, and retrograde amnesia [4]. In addition, palsy of 6th cranial nerve, prolonged overall cognitive dysfunction and psychosis-like symptoms, and reversible splenial lesion syndrome have been reported in intoxicated patients [5-7]. Cardiovascular effects noted include bradycardia and hypotension, which might be attributed to surfactant poisoning [8-10]. Respiratory symptoms comprised of shortness of breath, hypoxia, and respiratory failure [11,12]. Predictors of mortality and morbidity following acute poisoning have been explored. Elderly people, with large amount of ingestion (>13.9 g), without concomitant alcohol consumption, depressed level of consciousness (Glasgow coma scale (GCS) < 9), severe base deficit (HCO3 < 16.0 mmol/L), hyperammonemia (serum ammonia level > 151 mg/dL), with intervening mechanical ventilator support, and using vasopressors were at a higher risk of mortality

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after exposure [12-14]. Patients who satisfied two systemic inflammatory response syndrome (SIRS) criteria developed severe effects such as respiratory arrest and convulsion following acute glufosinate poisoning [11]. Higher initial level of serum ammonia and serum S100 protein >0.0965 μg/L were predictors of neurologic complications including an altered state of consciousness, seizure, and/or amnesia during hospitalization [15-17]. Relatively higher levels of serum ammonia were observed in patients with severe neurological complications even after 48 h [18]. In one study, the highest incidence of seizure attack was observed in patients with glufosinate poisoning among those intoxicated with herbicides [19].

Paraquat has been gradually forbidden in over 60 countries due to high mortality after exposure [20-23]. It was estimated that approximately 200 suicide-related deaths per year in Taiwan would be prevented since paraquat's ban in 2019 [24]; however, after paraquat was banned, the incidence of glufosinate intoxication due to suicide attempts has increased. Therefore, more studies are required to better elucidate the adverse health effects following glufosinate exposure. We conducted a retrospective analysis to better delineate the toxic effects of glufosinate and identify predictors of mortality and morbidity.

## 2. Method

This retrospective study was approved by the Foundation Institutional Review Board (number 202100223B0).

### 2.1. Study setting

We searched the keyword "glufosinate" both in Chinese and English, and extracted the relevant data from the electronic medical records of the medical institutes affiliated with the Chang Gung Memorial Hospital (CGMH). Five medical institutes (Keelung, Linkou, Yunlin, Chiayi, and Kaohsiung branches) located in northern and southern Taiwan were included in this study. After carefully reviewing the patient's prior medical history, 95 patients were enlisted for this study.

### 2.2. Patients

All patients who were exposed to glufosinate and visited the emergency department (ED) between January 2008 and December 2020, were included in this study.

### 2.3. Measurements

The estimated amount of "a sip" in medical records was regarded and defined as 20 mL and 25 mL per sip for males and females, respectively [25]. The estimated amount of "a cup" and "a bottle" in medical records were defined as 200 mL and 1000 mL, respectively. Since the typical marketed formulation of glufosinate in Taiwan is 13.5% (w/v) [14], all glufosinate ingested in our study were assumed to be 13.5% w/v. Impending death discharge was regarded as mortality [23,26,27]. The following demographics were extracted from the CGMH electronic medical records: age, sex, vital signs, symptoms, co-exposure substances, blood ammonia level, and hospital length of stay.

### 2.4. Data analysis

Continuous variables, such as age, vital signs, serum ammonia level, and hospital length of stay were expressed as medians and first quartile to third quartile (Q1–Q3). The distributions of categorical data are presented as numbers and percentages. Continuous variables were analyzed using the Mann-Whitney *U* test, and the categorical data were analyzed using the chi-square test. A one-sample *t*-test was used to analyze the 95% confidence intervals (CIs) for sensitivity and specificity. Results for a 2-tailed test were considered statistically significant if $P < 0.05$. All statistical analyses were performed using SPSS for Windows version 22.0 (released 2013, IBM Corp., Armonk, NY, USA).

## 3. Results

Among the 95 patients enrolled in the study, 61 patients were orally exposed to glufosinate-containing herbicide and 34 patients were exposed via the non-oral route. The demographics of the two groups are shown in Table 1. Patients exposed via the non-oral route had higher GCS scores, lower rates of suicide attempts, lower mortality rates, and shorter hospital stays (*P*-value <0.001, <0.001, 0.002, and <0.001, respectively) compared to orally exposed patients. None of the patients died in the non-oral exposure group. The incidence of glufosinate-containing herbicide poisoning has been increasing annually (Fig. 1). In the subgroup analysis performed for patients exposed orally (Fig. 2), 46 survived, while the remaining 15 patients died (Table 2). A comparison between the survival and non-survival groups revealed that the survival group had a lower amount of estimated glufosinate ingestion (10.5 [3.4–27] vs. 40.5 [27–47.3] g, $P = 0.022$), lower rate of substance co-exposure (9 [19.6%] vs. 10 [66.7%], $P = 0.001$), and lower rate of paraquat co-exposure (0 [0%] vs. 7 [46.7%] $P < 0.001$). To exclude the impact of paraquat toxicity, we further analyzed those without paraquat co-exposure (Table 3). We selected old age (defined as older than 70 years), depressed level of consciousness at triage (defined as GCS < 9), and high ammonia level (defined as >110 μg/dL) as predictor to evaluate the prognosis of glufosinate intoxication [12,13]. Old age and depressed level of consciousness at triage were significantly sensitive in predicting mortality (100.00%, 95% CI: 63.06–100.00%), but with relative low specificity (58.70%, 95% CI: 43.23–73.00%).

## 4. Discussion

In total, 95 patients exposed to glufosinate ammonium were included; 61 were orally exposed and the remaining 34 were exposed non-orally (Fig. 1). The presence of disturbed consciousness, seizure attack, respiratory failure, shock, and fever was significantly higher in the orally-exposed group with an incidence of 45.9%, 9.8%, 47.5%, 24.6%, and 23%, respectively (Table 1). The mortality rate was 24.6% in the orally exposed group, who also observed a longer duration of hospital stay. Among the 34 patients with non-oral exposure, only a few were symptomatic, and required further assistance. The main reason for ED visits in

**Table 1**
Demographics of patients exposed to glufosinate-containing herbicide ($N = 95$).

| | Non-oral exposure ($n = 34$) | Oral exposure ($n = 61$) | p-value |
|---|---|---|---|
| Age | 64.5 (53–74) | 61 (48–77) | 0.532 |
| Male sex | 28 (82.4%) | 42 (68.9%) | 0.152 |
| Vital signs during triage | | | |
| Body temperature (°C) | 36.1 (35.8–36.6) | 36.2 (36–36.7) | 0.198 |
| Heart rate (beats/min) | 84 (69–99) | 91 (79–105) | 0.167 |
| Respiratory rate (times/min) | 19 (18–20) | 19 (18–20) | 0.871 |
| MAP (mmHg) | 105.8 (90.3–115.3) | 98.3 (85.7–108.7) | 0.256 |
| SpO2 (%) | 96.5 (95–98) | 96 (94–98) | 0.458 |
| Glasgow coma scale | 15 (15–15) | 15 (11–15) | <0.001 |
| Suicide attempt | 3 (8.8%) | 53 (86.9%) | <0.001 |
| Symptoms | | | |
| Consciousness change | 0 (0%) | 28 (45.9%) | <0.001 |
| Seizure | 0 (0%) | 6 (9.8%) | <0.001 |
| Respiratory failure | 0 (0%) | 29 (47.5%) | <0.001 |
| Shock | 1 (2.9%) | 15 (24.6%) | 0.007 |
| Fever | 2 (5.9%) | 14 (23%) | 0.033 |
| Ammonia (ug/dL) | 53 (53–66) | 80.5 (63–106) | 0.094 |
| Mortality | 0 (0%) | 15 (24.6%) | 0.002 |
| Hospital length of stay (hours) | 4.9 (1.6–21.8) | 64.4 (10.6–192.9) | <0.001 |

Data are presented as numbers (percentages), median (Q1–Q3).
Abbreviations: MAP, mean arterial pressure; SpO2, peripheral oxygen saturation.

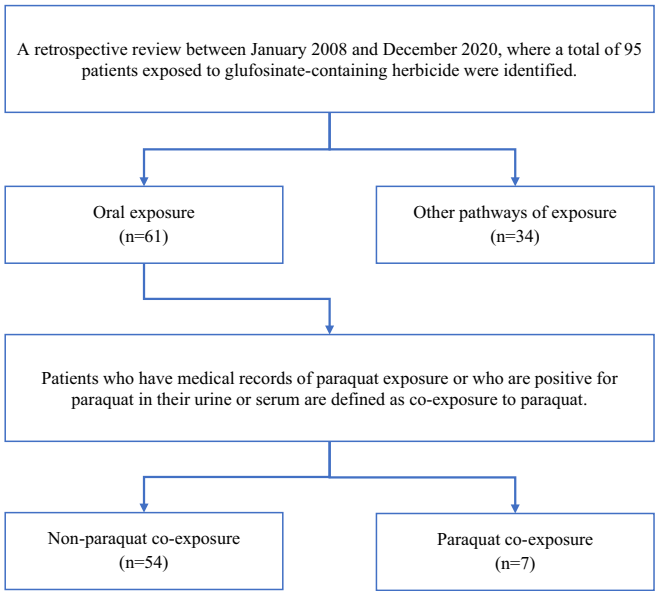


**Fig. 1.** Flowchart of subgroup analyses of patients.

these non-orally exposed patients was that they were worried about glufosinate poisoning, and the most common symptom was mild dizziness. One patient in the non-oral exposed group had hypotension caused by non-ST segment elevation myocardial infarction (NSTEMI), rather than acute glufosinate poisoning itself. Moreover, two patients experienced fever in the non-oral exposure group. The etiology of fever was concomitant with the upper respiratory tract infection, which resolved few days later without complications. None of the patients in the non-oral glufosinate exposure group died. Despite mild symptoms experienced by the non-oral exposure group during ED visit, close observation and delicate evaluation were recommended.

Subgroup analysis was performed to identify the risk of mortality in the oral exposure group (Table 2). Excess estimated glufosinate consumption and a higher rate of substance co-exposure, especially paraquat, were observed in the non-survival group. The occurrence rates of disturbed consciousness, respiratory failure, and shock were significantly higher in the non-survival group. The mortality rate was 100% in those with paraquat co-exposure, highlighting the importance of acquiring a clear medical history.

Based on a previous study, the mortality rate of paraquat toxicity after oral exposure ranged from 45.6% to 75.4% [20-23]. The products of paraquat and glufosinate herbicides available in Taiwan's markets are mostly available as bluish liquid. Owing to this similarity, it is important to seek clarifications on the substance exposed while encountering patients with acute herbicide poisoning. In addition, substance co-exposure should be carefully evaluated, especially for paraquat.

**Table 2**
Clinical characteristics between mortality and survival groups of patients orally exposed to glufosinate-containing herbicide ($n = 61$).

| | Survival ($n = 46$) | Mortality ($n = 15$) | P-value |
|---|---|---|---|
| Age | 55.5 (45–72) | 73 (49–78) | 0.147 |
| Male sex | 32 (69.6%) | 10 (66.7%) | 1.000 |
| Underlying disease | | | |
| Psychoaffective disorder | 12 (26.1%) | 7 (46.7%) | 0.199 |
| Hypertension | 13 (28.3%) | 4 (26.7%) | 1.000 |
| Chronic lung disease | 0 (0%) | 1 (6.7%) | 0.246 |
| Liver cirrhosis | 2 (4.3%) | 3 (20%) | 0.090 |
| Diabetes mellitus | 10 (21.7%) | 5 (33.3%) | 0.491 |
| Chronic kidney disease | 2 (4.3%) | 1 (6.7%) | 1.000 |
| Malignancy | 6 (13%) | 4 (26.7%) | 0.243 |
| Suicide attempt | 39 (84.8%) | 14 (93.3%) | 0.666 |
| Estimated ingested glufosinate (g) | 10.5 (3.4–27) | 40.5 (27–47.3) | 0.022 |
| Ammonia (ug/dL) | 80.5 (63–106) | 80.5 (53.5–455.5) | 0.932 |
| Co-exposure | 9 (19.6%) | 10 (66.7%) | 0.001 |
| BZD | 8 (17.4%) | 0 (0%) | 0.182 |
| Alcohol | 10 (21.7%) | 0 (0%) | 0.055 |
| Glyphosate | 1 (2.2%) | 2 (13.3%) | 0.147 |
| Paraquat | 0 (0%) | 7 (46.7%) | <0.001 |
| Organophosphate | 0 (0%) | 1 (6.7%) | 0.246 |
| Others | 1 (2.2%) | 1 (6.7%) | 0.434 |
| Symptoms | | | |
| Consciousness change | 17 (37%) | 11 (73.3%) | 0.019 |
| Seizure | 4 (8.7%) | 2 (13.3%) | 0.630 |
| Respiratory failure | 14 (30.4%) | 15 (100%) | <0.001 |
| Shock | 6 (13%) | 9 (60%) | 0.001 |
| Fever | 10 (21.7%) | 4 (26.7%) | 0.730 |
| Hospital length of stay (hours) | 88.1 (17.5–223.7) | 15.3 (9.6–88.1) | 0.079 |

Data are presented as number (percentage) or median (Q1–Q3).

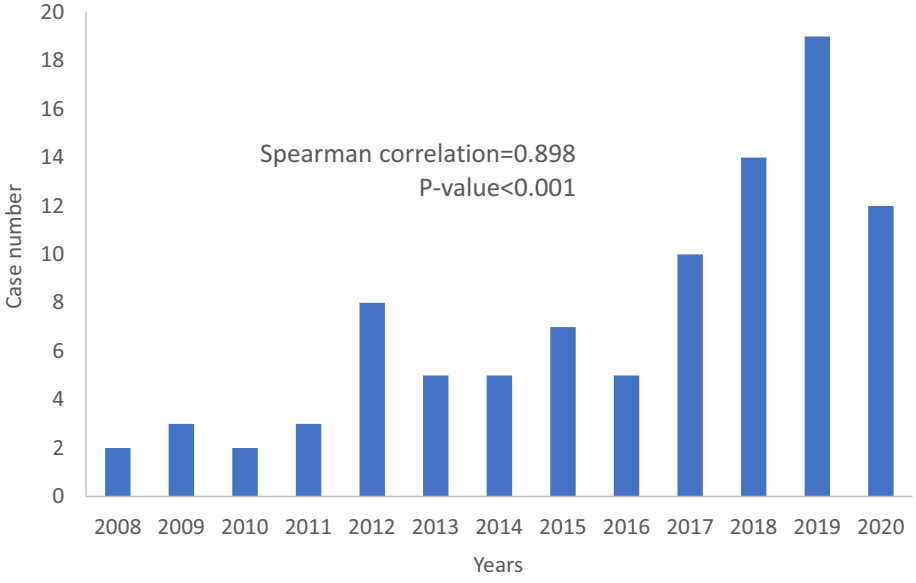


**Fig. 2.** Case numbers of patients with glufosinate-herbicide exposure per year.

**Table 3**
Sensitivity and specificity of assessment in non-paraquat co-exposure patients with oral glufosinate poisoning ($n = 54$).

| Assessment using | Age > 70 | | GCS < 9 | | Ammonia > 110 | | Age > 70 or GCS < 9 | |
|---|---|---|---|---|---|---|---|---|
| | Mortality | Survival | Mortality | Survival | Mortality | Survival | Mortality | Survival |
| No. of positive results | 7 | 14 | 2 | 8 | 1 | 4 | 8 | 19 |
| No. of negative results | 1 | 32 | 6 | 38 | 2 | 14 | 0 | 27 |
| Statistic | Value | 95% CI | Value | 95% CI | Value | 95% CI | Value | 95% CI |
| Sensitivity | 87.50% | 47.35% to 99.68% | 25.00% | 3.19% to 65.09% | 33.33% | 0.84% to 90.57% | 100.00% | 63.06% to 100.00% |
| Specificity | 69.57% | 54.25% to 82.26% | 82.61% | 68.58% to 92.18% | 77.78% | 52.36% to 93.59% | 58.70% | 43.23% to 73.00% |

In Korea, paraquat was banned to purchase since 2012, and pesticide associated mortality was significant decreased since that time; however, the suicide rate from other poisoning increased after prohibition of paraquat [28,29]. In the current study, the number of patients with acute glufosinate toxicity have been increasing in recent years ($P < 0.001$, Fig. 2). This might be related to the stepwise paraquat prohibition policy executed by Taiwan's government from 2017 to 2019.

Since the 1980s, paraquat has been banned in several countries worldwide because of its high mortality rate after exposure. Owing to the increasing trend of acute glufosinate poisoning and the ban on paraquat, clinicians are urged to identify high-risk patients who are exposed to glufosinate only via the oral route. Limited evidences focused on the prognostic factors of glufosinate intoxication. Lee et al. found that initial ammonia level was a predictor for neurologic complication for glufosinate poisoning [16], and another study also revealed that initial serum ammonia level was a predictor for in-hospital mortality [13]. In the present study, we did not observe statistically significant difference of ammonia level between mortality and survival groups. In fact, ammonia level was checked in only 25 patients in our database. Due to the limited number of cases, it might not truly reflect the impact of ammonia level on the prognosis. On the other hand, Mao et al. demonstrated that large amount of ingested glufosinate (>13.9 g) was also a predictor for poor prognostic [14]. The present study also found that the estimated ingested glufosinate was statistically higher in mortality group. However, only 37 patients had records of possible amount of intake. Among these 37 patients, 10 patients ingested other pesticides or drugs at the same time. Therefore, we did not choose estimated ingested glufosinate in Table 3. Older age (defined as age > 70 years) in combination with depressed levels of consciousness (defined as GCS score < 9) at triage seems to be a predictor of mortality for acute glufosinate poisoning in patients solely exposed to glufosinate orally (Table 3). The sensitivity of this assessment tool was 100% (95% CI: 63.5–100.00%) and the specificity was more than 50%. Owing to its high sensitivity, it might be considered as a screening tool for predicting severe effects.

## 5. Limitations

This study was a retrospective analysis, which inherited the limitations of all observational studies. Second, the amount of oral ingestion was not precisely recorded. The estimated amount of "a sip" ingestion in medical records was defined as 20 mL and 25 mL per sip for men and women respectively, in our study. The estimated amount of "a cup" and "a bottle" were defined as 200 mL and 1000 mL, respectively [25]. However, the actual amount of oral ingestion might be under or overestimated, since the description in the medical records were obscure, such as drinking "some" glufosinate. Third, the time interval from glufosinate exposure to ED arrival was not clearly documented, which made further analysis difficult. Fourth, the number of patients enrolled in this study was relatively small, which made it difficult to identify the potential predictors of mortality. The data were collected from five main medical institutes affiliated with Chang Gung Memorial Hospital (CGMH) located in northern to southern Taiwan; hence, the patients studied might represent the main population in Taiwan.

## 6. Conclusion

Patients with acute glufosinate poisoning and concomitant exposure to paraquat were at a high risk of mortality (100%); thus, acquiring a clear medical history is important. Old age (defined as age > 70 years) in combination with depressed level of consciousness (defined as GCS score < 9) at triage might be a predictor of mortality in patients solely exposed to glufosinate via the oral-route.

## Geolocation information

The five medical institutions participating in this study are situated at Keelung, Linkou, Chiayi, Yunlin, and Kaohsiung branches, which are located from northern to southern Taiwan.

## Author contributions

FJC and PCC designed the study. PCC supervised the conduct of the trial and data collection. PCC and CTK performed the data analysis and takes responsibility for the integrity of the data and the accuracy of the data analysis. CTK drafted the article, and all authors contributed substantially to its revision. JTH takes responsibility for the paper as a whole.

## Prior presentations

None.

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## Declaration of Competing Interest

None.

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