Clinical Presentations and Prognostic Factors of a Glyphosate–Surfactant Herbicide Intoxication: A Review of 131 Cases

HSIN-LING LEE, MD, KUAN-WEN CHEN, MD, CHIH-HSIEN CHI, MD, JENG-JONG HUANG, MD, LIANG-MIIN TSAI, MD

Abstract. **Objective:** Suicide attempts with agricultural chemicals are common in southern Taiwan. Among them, glyphosate–surfactant herbicide (GlySH) intoxication has been encountered with increasing frequency. Although a number of reports have described the clinical course and outcomes following ingestion, predictors of serious complications and mortality have not been elucidated. The purpose of this study was to define predictors of serious complications and probable mortality. **Methods:** This was a retrospective study of 131 GlySH-intoxicated patients treated at the National Cheng Kung University Hospital from 1988 to 1995. Medical charts were reviewed and clinical and laboratory variables were abstracted, looking for predictors of mortality. **Results:** The most common symptoms included sore throat (79.5%), and nausea with or without vomiting (73.8%). The most common laboratory findings were leukocytosis (68.0%), low serum bicarbonate (48.1%), and acidosis (35.8%). Overall, 11 of 131 patients (8.4%) died; the mean ± SEM time to death was 2.8 ± 0.8 days after presentation. When comparing the clinical and laboratory characteristics among the survivor and fatality groups, significant differences were identified. Respiratory distress, pulmonary edema, respiratory distress necessitating intubation, shock (systolic blood pressure less than 90 mm Hg), altered consciousness, abnormal chest x-ray, renal failure necessitating hemodialysis, larger amount of ingestion (>200 mL), and hyperkalemia were predictors highly associated with poor outcomes and mortality. Using multiple logistic regression, three predictors were identified, which may predict mortality in severely intoxicated patients. **Conclusions:** In managing patients who have larger amount of GlySH ingestion, airway protection, early detection of pulmonary edema, and prevention of further pulmonary damage and renal damage appear to be of critical importance.
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HERBICIDES containing glyphosate, an alternative to paraquat, have recently been used with increasing frequency in suicide attempts throughout Asia.[1–5] Ingestion of a large amount of this chemical may place patients at risk for a toxic syndrome that includes gastrointestinal irritation, hepatic and renal dysfunction, cardiovascular instability, and pulmonary insufficiency.[2–6] Although glyphosate–surfactant herbicide (GlySH) is considered to be only slightly toxic in rats, ingestion of a substantial volume of GlySH has been reported to be associated with toxicity and death in humans.[4,5] A number of reports have described the clinical course and outcomes following ingestion; however, prognostic indicators have not been elucidated. Since June 1988, there appeared to be an increase in the number of patients with GlySH intoxication presenting to our hospital. The purpose of this study was to identify the risk factors and prognostic factors in GlySH intoxication.

METHODS

Study Design. This was a retrospective study of patients with GlySH intoxication presenting to our emergency department (ED) over a seven-year period. This study was reviewed and approved by the institutional review board.

Study Setting and Population. National Cheng Kung University Hospital in Taiwan is a referral center for a large agricultural area of approximately 2 million people in southern Taiwan. The ED's annual census is about 51,000.

All the medical records of patients with GlySH intoxication following oral ingestion who presented to the ED of the National Cheng Kung University

From the Departments of Emergency Medicine (HLL, KWC, CHC, LMT) and Internal Medicine (JJH), National Cheng Kung University Hospital, Tainan, Taiwan.
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Address for correspondence and reprints: Kuan-Wen Chen, MD, Department of Emergency Medicine, National Cheng Kung University Hospital, 138 Sheng-Li Road, Tainan 704, Taiwan. Fax: 886-6-235-9562; e-mail: hllee@mail.ncku.edu.tw

Hospital from June 1988 to December 1995 were reviewed.

Study Protocol. Data collected included date of admission, age, sex, estimated amount of GlySH ingested, co-ingestants of other agrochemicals, ethanol, or pharmaceuticals, suicide attempts, out-of-hospital interval, initial clinical presentation, initial laboratory data in the ED, and clinical course. Laboratory variables that were reviewed included arterial blood gas, blood urea nitrogen (BUN), creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin, sodium, potassium, calcium, phosphate, white blood cell (WBC) count, hematocrit, platelet, urine analysis, chest x-ray (CXR), and electrocardiogram (ECG). Only the laboratory studies done immediately upon the patients' arrival were taken into consideration. There were some patients who had received first aid and were transferred from other EDs. For these patients, we used the primary data from those EDs. For clinical and statistical consideration, patients whose serum pH values were <7.35 on the arterial blood gas were considered to be "acidotic." Of note, the clinical practice at this hospital was to routinely obtain toxicological screens of other pesticides, such as paraquat and organophosphates, and screens of benzodiazepines. We also performed specific tests according to the history offered by patients themselves, friends, or family members. The amount ingested was usually given in descriptive terms such as "a mouthful," "a small cup," or "half a bottle." For statistical purposes, we assigned a volumetric value to each description: 5 mL for "a little" or "a spoon," 25 mL for "a mouthful," and 100 mL for "a small cup." If the patient said "a bottle," the size was identified as being 150 mL, 300 mL, 500 mL, or 1 liter,[5–7] according to variable brands, empty bottles carried by family members or friends, or the description by family members or friends.

Data Analysis. All analysis was performed using SPSS statistical software version 6.03 (SPSS Inc., Chicago, IL). For univariate analysis, we used Student's t-test for continuous variables, the Wilcoxon test, Fisher's exact test for categorical variables, and odds ratios for each variable. A p-value of less than 0.05 was considered statistically significant. Those variables with odds ratios more than 5 were considered to be prognostic predictors. All prognostic variables initially identified were further quantified by multiple logistic regression analysis (stepwise regression). A patient's probability of survival (Ps) was then predicted using the selected logistic model $Ps = 1/(1 + e^{-b})$ where $b = b_0 + b_1 \times$ risk factor I $+ b_2 \times$ risk factor II $+ b_3 \times$ risk factor III $\cdots\cdots\cdots + b_N \times$ risk factor N.

RESULTS

From June 1988 to December 1995, there were 131 patients who presented to our hospital with GlySH ingestion, 69 of whom were male and 62 female. There were 11 fatalities, for a mortality rate of 8.4%. The most common presentations included sore throat, nausea (with or without vomiting), and fever (Table 1). The most common laboratory abnormalities included leukocytosis (WBC count > $10^4/\mu L$; 85/125, 68%), lowered bicarbonate (HCO_3^- < 22 mEq/L; 39/81, 48.1%), acidosis (serum pH < 7.35, 29/81, 35.8%), elevated AST (>40 U/L; 32/108, 33.6%), hypoxemia (PO_2 < 60 torr while breathing room air; 23/81, 28.4%), and elevated BUN (>21 mg/dL; 21/123, 17.1%).

Eighty-one patients had 12-lead electrocardiograms, 15 of which were abnormal. The most frequent abnormalities were sinus tachycardia and nonspecific ST-T changes. Of 29 patients who had serum pH < 7.35, 13 had metabolic acidosis, one had respiratory acidosis, and 15 had mixed-type acidosis. Of 105 patients who had chest x- rays, 22 revealed abnormal infiltrates or patches. Three of the 131 patients had renal failure that necessitated hemodialysis; these patients all died. Seven of 131 patients had co-ingestants, including sedative drugs (2), hypnotics (3), wine (3), and paraquat (1). The average survival time of the fatalities was 2.8 ± 0.8 days.

Univariate comparisons of clinical variables and laboratory data on arrival comparing survivors with those who died are presented in Tables 1 and 2. The mean ± SEM age of the survivors was 47 ± 2 years, while that of those who died was 60 ± 4 (p = 0.02). No difference was found relating to gender. The estimated amount of GlySH ingested averaged 122 ± 12 mL among the survivors and 330 ± 42 mL among those who died (p < 0.001). The mean out-of-hospital time among the survivors was longer than that for those who died (Table 1), but this difference was not statistically significant.

Using odds ratios analysis, those variables with an odds ratio more than 5 were identified (Table 3). Of the 17 identified variables, eight were highly associated with poor outcome and mortality, including respiratory distress necessitating intubation, respiratory distress, renal dysfunction necessitating hemodialysis, abnormal CXR, shock, larger amount of ingestion (>200 mL), altered consciousness, hyperkalemia, and pulmonary edema. Only the cases with complete data were used for the multiple logistic regression analysis. Multiple logistic regression using these factors identified three variables able to predict the probability of survival, applying a logistic model

TABLE 1. Clinical Variables on Arrival at the Emergency Department among Survivors and Those Who Died*

	Survivors (n = 120)	Those Who Died (n = 11)	Total (n = 131)	p
Age (years)	47 ± 2	60 ± 4	48 ± 2	0.02
Gender (male/female)	62/58	7/4	69/62	0.47
Out-of-hospital interval (hr)	4.0 ± 0.5	2.2 ± 0.4	3.8 ± 0.4	0.57
Estimated ingested amount (mL)	122 ± 12	330 ± 42	138 ± 12	<0.001
Fever	48/120 (40.0%)	6/11 (54.5%)	54/131 (41.2%)	0.36
Nausea and/or vomiting	88/118 (74.6%)	5/8 (62.5%)	93/126 (73.8%)	0.43
Sore throat	96/118 (81.4%)	5/9 (55.6%)	101/127 (79.5%)	0.08
Diarrhea	25/120 (21.0%)	1/10 (9.1%)	26/131 (19.1%)	0.69
Respiratory distress	19/120 (15.8%)	11/11 (100%)	30/131 (22.9%)	<0.001
Altered consciousness	19/120 (15.8%)	10/11 (90.9%)	29/131 (21.3%)	<0.001
Respiratory distress necessitating intubation	7/120 (5.8%)	11/11 (100%)	18/131 (13.7%)	<0.001
Pulmonary edema	2/119 (4.2%)	4/11 (36.4%)	6/130 (4.6%)	<0.001
Abnormal chest x-ray	15/98 (15.3%)	7/7 (100%)	22/105 (21.0%)	<0.001
Shock	5/119 (4.2%)	8/11 (72.7%)	13/130 (10.0%)	<0.001
Dysrrhythmia	9/71 (12.7%)	6/10 (75.0%)	15/81 (18.5%)	<0.001
Renal dysfunction necessitating hemodialysis	0/120 (0%)	3/11 (27.0%)	3/131 (27.0%)	<0.001
Suicide attempt	105/120 (17.5%)	11/11 (100%)	116/131 (88.5%)	0.36

*Data are expressed as mean ± SEM and $p < 0.05$ is significant.
p-values are comparisons only between survivors and those who died.

$$Ps = 1/(1 + e^{-b})$$

$$b = -216.9295 - 5.0969 \times [\text{acute pulmonary edema}] - 1.8020 \times [K] + 31.2613\,[pH]$$

patients with a Ps = 0, die, and a Ps = 1, survive. We defined that patients with a Ps > 0.25 could be predicted to survive, while those with a Ps < 0.25 were predicted to die. This conditional analysis indicated a sensitivity of 100% and a specificity of 95.7% to predict mortality due to GlySH intoxication. Because pulmonary edema is a binary response, the above formula can be simplified to create the following predictive rules: 1) when pulmonary edema is absent, $31.26 \times [pH] - 1.8 \times [K] < 215.83$ predicts fatality; 2) when pulmonary edema is present, $31.26 \times [pH] - 1.8 \times [K] < 220.93$ predicts fatality.

DISCUSSION

The diagnosis of GlySH poisoning is usually made by history, clinical signs, and chemical analysis. Analysis of GlySH is not readily available except at special research laboratories. Currently, neither the level of serum glyphosate as an indicator of toxicity severity nor a definitive lethal dose in humans has been firmly established. Therefore, to predict outcome from the clinical manifestations at the time of emergency presentation is a considerable challenge to emergency physicians.

When a large quantity of GlySH is ingested, death ensues within 72 hours. Since most fatalities occur soon after ingestion, waiting for serum levels as a means of predicting intoxication severity seems impractical. The aim of this study was to define predictors of serious disease and probable mortality that can be quickly identified.

Clinical presentations of GlySH intoxication show some variation according to different reports.[1–5,8] An analysis of three retrospective reviews of 246 cases[3–5] revealed that patients presented with nausea and/or vomiting (40%), abdominal pain, and diarrhea (12%) initially, followed by sore throat (41–43%), fever (7%), gastrointestinal mucosal damage (7–43%), transient renal (10–14%) and hepatic (19–40%) dysfunction, metabolic acidosis, pulmonary edema (5–13%), shock (9%), and death (10.5–16.7%). In our study, nausea with or without vomiting (73.8%), sore throat (79.5%), and fever (41.2%) were the most common initial manifestations. Leukocytosis (68.0%), low bicarbonate (48.1%), acidosis (35.8%), hepatic dysfunction (33.6%), hypercapnea (30.9%), hypoxemia (28.4%), and renal insufficiency (17.1%) were the most common laboratory abnormalities. These findings were similar to previous reports of severe intoxications, except that our patients showed a higher prevalence of sore throat, nausea and/or vomiting, fever, acidosis, and diarrhea.

The estimated amount of GlySH ingested by the patients who died in our study (330 ± 42 mL) is more than previous reports (Sawada et al., 206 mL; Tominack et al. 263 ± 104 mL).[3,5] In our study, out-of-hospital time was actually longer in the survivor group. This is similar to the reports of Talbot et al. and Tominack et al.[4,5] This might be due to the fact that a lack of significant symptoms following a minor ingestion did not induce a sense of urgency in the patient or family, leading to the delay in seeking hospital help.

Glyphosate–surfactant herbicide is a formulated commercial product containing 41% glyphosate as the isopropylamine salt, 15% polyoxyethyleneamine (POEA) surfactant, and water. Its toxicity to plants is dependent on a specific effect on the plants' shikimic acid metabolic pathway.[9] The absence of this pathway in mammals may help to explain the relatively low systemic toxicity of glyphosate [oral median lethal dose (LD_{50}) for rats 4,320 mg/kg, rabbits 3,800 mg/kg].[10] If we apply these figures to a 60-kg man, the estimated LD_{50} would be 226–259.2 g, which is found in 556–632 mL of GlySH. The amount of GlySH ingested in our study population, as in previous reports, is based on estimates and self-reporting, which are unreliable. However, looking at our patients and previous studies,[3–5] humans seem to be less tolerant of GlySH than animals. This suggests that the mechanism of toxicity or level of tolerance in humans differs from that in experimental mammals. The definite mechanism of poisoning is still unclear. Talbot et al.[4] postulated that severe toxicity resulted from the uncoupling of mitochondria oxidative phosphorylation. Sawada and colleagues[1,3] speculated that the POEA surfactant may be responsible for the toxicity. Through the study of intravenous injection of glyphosate, surfactant, and GlySH, Tai et al.[11] concluded that surfactant caused cardiac suppression and increasing pulmonary vascular resistance. Besides, as previous studies also found, we noted that several patients intoxicated with extremely large amounts of concentrated GlySH showed only mild or no clinical symptoms and signs. Thus, the amount of liquid ingested may not be an absolute indicator of severity of GlySH intoxication.

In GlySH intoxication, aspiration pneumonitis and upper respiratory tract irritation are reported findings.[4,15,16] In clinical practice, many GlySH-intoxicated patients presented with mucosal injury of the throat (sore throat) with drooling. Most of the patients were able to overcome these symptoms by conservative treatment; however, some of those severely injured patients developed respiratory distress syndrome, even requiring intubation.

TABLE 2. Initial Laboratory Data in Survivors and Those Who Died*

	Survivors (n = 120)	Those Who Died (n = 11)	p
Complete blood count			
WBC count ($10^4/\mu L$)	13.4 ± 0.5	18.5 ± 2.5	<0.01
Hematocrit (%)	42.0 ± 0.5	45.3 ± 1.5	0.07
Platelet count ($10^3/mm^3$)	265 ± 9	239 ± 30	0.389
Biochemical data			
Urea nitrogen (mg/dL)	16 ± 1	19 ± 3	0.26
Creatinine (mg/dL)	1.0 ± 0.1	1.4 ± 0.2	<0.01
Sodium (mmol/L)	141 ± 1	141 ± 2	0.87
Potassium (mmol/L)	3.8 ± 0.1	4.7 ± 0.4	0.06
Chloride (mmol/L)	105 ± 1	103 ± 4	0.74
Total calcium (mg/dL)	9.1 ± 0.1	9.0 ± 0.2	0.79
Phosphate (mg/dL)	3.4 ± 0.1	3.9 ± 0.9	0.56
Total bilirubin (mg/dL)	1.0 ± 0.1	1.2 ± 0.4	0.99
ALT (U/L)	35 ± 3	64 ± 21	0.20
AST (U/L)	37 ± 3	110 ± 44	0.13
Arterial blood gases			
pH	7.39 ± 0.01	7.17 ± 0.05	<0.001
PO_2 (torr)	75.3 ± 2.6	48.2 ± 7.2	<0.001
PCO_2 (torr)	36.8 ± 0.8	41.8 ± 4.5	0.65
HCO_3^- (mEq/L)	22 ± 1	15 ± 2	<0.001

*Data expressed as means ± SEM, and $p < 0.05$ is significant. WBC = white blood cell; ALT = alanine aminotransferase; AST = asparate aminotransferase.

TABLE 3. Predictors Associated with Poor Patient Outcome (Odds Ratios > 5)

Predictors	Those Who Died (n = 11)	Survivors (n = 120)	Total (n = 131)	Odds Ratio (95% CI)
Respiratory distress necessitating intubation	11/11	7/120	18/131 (13.7%)	348.1 (98.8, ∞)*
Respiratory distress	11/11	19/120	30/131 (22.9%)	119.7 (29.6, 484.6)*
Renal failure necessitating hemodialysis	3/11	0/120	3/131 (2.3%)	99.2 (26.4, 372.4)*
Abnormal chest x-ray	7/7	15/98	22/105 (21.0%)	80.8 (18.2, 359.0)*
Shock (SBP < 90 mmHg)	8/11	5/119	13/130 (10.0%)	60.8 (10.1, 435.8)†
Larger amount of ingestion (>200 mL)	9/10	17/101	26/128 (20.3%)	53.5 (13.6, 210.9)†
Altered consciousness	10/11	19/120	29/131 (22.1%)	53.2 (13.6, 207.5)*
Hyperkalemia (potassium > 5.5 mmol/L)	4/10	2/118	6/128 (4.7%)	38.7 (4.6, 398.6)†
Pulmonary edema	4/11	2/119	6/130 (4.6%)	33.4 (4.1, 330.7)†
Elevated creatinine (>1.5 mg/dL)	4/11	4/116	8/127 (6.3%)	16.0 (2.6, 103.3)†
Lowered bicarbonate (HCO_3^- < 22 mEq/L)	10/11	29/70	39/81 (48.1%)	14.1 (1.7, 311.2)†
Acidosis (pH < 7.35)	9/11	20/70	29/81 (35.8%)	11.25 (1.98, 83.3)†
Dysrrhythmia	6/10	9/71	15/81 (18.5%)	10.3 (2.0, 56.5)†
Hyperphosphatemia (phosphate > 5.0 mg/dL)	2/10	3/95	5/105 (4.8%)	7.7 (6.8, 71.4)†
Elevated AST (>40 U/L)	8/11	32/108	40/119 (33.6%)	6.33 (1.4, 32.5)†
Hypoxemia (PO_2 < 60 torr)	7/11	16/70	23/81 (28.4%)	5.9 (1.3, 28.2)†
Leukocytosis (WBC > $10^4/\mu L$)	10/11	75/114	85/125 (68%)	5.2 (0.6, 112.5)†

*Test-based 95% confidence interval for odds ratios.
†Cornfield's 95% confidence interval for odds ratios.

In our study, 21.5% of the patients presented with various respiratory distress symptoms and 60% of these (18/30) needed intubation during their hospitalizations. Hung et al.[16] reported that severe laryngeal injury is strongly suspected to be the primary mechanism of respiratory aspiration and the leading cause of morbidity and/or mortality following glyphosate intoxication. Besides, we noted that rapid ingestion of GlySH frequently induced airway stimulation and aspiration. We propose that direct damage to the airway passage and the respiratory tree may also play an important role in the effects of GlySH intoxication. If the damage to the pulmonary system is severe enough to lead to severe hypoxemia, it can further impact on other major body systems such as the cardiovascular system, with other critical factors such as metabolic acidosis and hyperkalemia reflecting the severity of major organ dysfunction. Although a reduced mortality rate has been reported,[17] the increasing number of cases of GlySH intoxication should serve to highlight the importance of detection and appropriate management of pulmonary dysfunction in such a patient.

Regardless of whether the major toxic component is GlySH or POEA surfactant, pulmonary edema has also been reported previously in fatal cases.[3–5,12] The onset of noncardiogenic pulmonary edema may range from several hours to 72 hours after exposure. Other critical factors in fatalities include shock, acidosis, and hyperkalemia.[3–5,13,14] By using multiple logistic regression analysis, we identified three factors (pulmonary edema, acidosis, and hyperkalemia) that could help to predict outcome more precisely. Modification of the formula has been validated on a few living patients and one fatality; further prospective validation is needed. With the predictors of poor outcomes, our emergency physicians who attend those intoxicated patients first can quickly detect and dispatch the patients who need intensive care.

Limitations and Future Questions

There are some limitations to our study. Almost all of the ingested amount of GlySH in our study, as in previous reports, was based on estimates and self-reporting. Estimates are unreliable and therefore may result in inaccuracies in the evaluation of the amount ingested by patients. Although we had only 11 fatalities in our case series, we used three different predictor variables in our equation. By convention, the number of predictor variables is no more than 1 for every 5–10 outcome observations of interest when using regression techniques; our model, therefore, might be unstable. Further evaluation of additional cases is mandatory to obtain a more precise equation in predicting outcome.

Conclusions

We recommend that all the patients who are reported to have ingested large amounts of GlySH be carefully observed, especially those who present in respiratory distress. Some prognostic variables, such as severe respiratory distress, requiring hemodialysis, and pulmonary edema, can help to identify those patients expected to deteriorate or die. The risk of immediate death is much less likely if the patient has no risk factors on presentation.

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