Glyphosate-induced Delayed Pyloric Obstruction, Ulcer and Scar Changes

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ABSTRACT
Glyphosate is a widely used herbicide. Acute glyphosate poisoning is not uncommon, mostly caused by a large amount of glyphosate entering the human body in a short time, and the common reason is taking it accidently. The manifestations of acute stage are mainly gastrointestinal reactions. The patient in the present study took glyphosate accidently. In the acute stage, the patient presented with nausea and vomiting, but no gastrointestinal bleeding or perforation was found. Upper gastrointestinal obstruction occurred after two months. Endoscopy revealed no abnormalities in the esophagus, but revealed multiple gastric scar changes, and pyloric obstruction. In addition, active gastric ulcer was also observed. Therefore, the choice of gastric lavage method, avoidance of neutralising acid-base reactions, and the long-term use of acid suppression and mucosal protection therapy may have important therapeutic implications for the repair of gastric mucosa in such cases.

Key Words: Glyphosate, Ulceration, Pyloric obstruction.

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INTRODUCTION
Glyphosate is a herbicide developed by Monsanto in the United States. It is widely used in global agricultural product at present, and is extensively used in countryside of China. Glyphosate mainly inhibits 5-enolpyruvylshikimate-3-phosphate synthase in plants, which inhibits the conversion of shikimic into phenylalanine, tyrosine and tryptophan, interfering with protein synthesis and causing plant death. The toxic mechanism of glyphosate in mammals has been considered to be due to decoupling of oxidative phosphorylation in the energy metabolism pathway. At present, there are many reports of acute glyphosate poisoning. Treatment in acute stage includes gastric lavage, emergency endoscopic hemostasis, oral use of active carbon and egg white to absorb the glyphosate surfactant, and other therapeutic measures. The present study reports a patient who developed pyloric obstruction two months after taking glyphosate accidently.

CASE REPORT
The patient was a 44-year woman, who was admitted to the General Surgery Department of our hospital due to history of accidental intake of glyphosate two months ago, and presenting with intermittent nausea and vomiting for half a month. Two months before admission, the patient took approximately 100 ml of glyphosate accidently. After one hour, the patient presented with nausea and vomiting, and the vomit included a large amount of gastric contents and pesticides. Her family members took the patient to a local hospital. The patient was treated with mechanical gastric lavage in a local hospital; and the gastric lavage fluid used was clear water. After the lavage, the patient had no obvious nausea, vomiting, or other symptoms; and there were no gastrointestinal bleeding symptoms, such as hematemesis and melena. Hence, after 13 days of symptomatic treatment, the patient was discharged, and no discomforts, such as anorexia, nausea, sour regurgitation, heartburn, vomiting, hematemesis, melena, abdominal pain and diarrhea, occurred. At half a month before this admission, the patient presented with nausea and vomiting, without obvious inducement. The vomit contained gastric contents, but no hematemesis and melena was found. The patient did not vomit after taking a small amount of liquid diet. Then, the patient went to the Outpatient Department of our hospital, and was admitted due to upper gastrointestinal obstruction. During the course of the disease, the patient was conscious and oriented, with poor diet and sleep, but had normal urination, and defecated once every 3-4 days. The body weight of the patient decreased by approximately 12 kg in the past two months. The patient previously had good health, and had no history of peptic ulcer or any bad habits.
ment and enhancement of the third group of gastric lymph
fluid can be seen in the gastric body, and irregular scar traction changes
of gastric mucosa. (F) Gastric antral ulcer biopsy showed acute on chronic cycle infection.

Physical examination: body temperature: 36.2°C; pulse: 70
beats per minute (BPM); breathing: 20 times/min; blood pressure: 115/63 mmHg; mental status: lucid; bilateral pupils: 3.0
mm and light reflex: sensitive. No obvious cyanosis on the lips
was found. The neck was soft, and no obvious swelling of superficial
lymph nodes of neck was found. Both-sided lung respiratory
sounds were clear, and there were no obvious dry and moist
rattles in both lungs. Pathological bruits were not heard in all
valvular auscultation areas. The abdomen was flat and soft, and
there was no tenderness and rebound pain in the abdomen. No
obvious mass was felt. Bowel sounds were audible. Shifting dull-
ness was negative. No obvious edema was found in the limbs.

In routine blood tests, leukocyte count was 3.24×10^9/L, neutrophil percent was 0.49, and lymphocyte, 0.43. In routine
stool test, the occult blood test was weakly positive. In routine
urine test, occult blood was +1, and urinary cholecystostogen was
131 μmol/L. In biochemistry tests, potassium was 3.00 mmol/L, albumin was 33.4 g/L, globulin was 38.8 g/L, and the albumin-to--
globulin ratio was 0.86. In disseminated intravascular coagulation
(DIC) test, D-dimer was 1.96 mg/L, and the fibrinogen degra-
dation product (FDP) was 6.3 μg/ml.

On gastroscopy, no abnormality in the esophagus was found
(Figure 1A&B). Multiple ulcer scars could be observed in the
stomach (Figure 1C). The gastric cavity was deformed and
contracted by mucosal traction. Longitudinal ulcers were
observed in the greater curvature of the stomach body. The
normal shapes of gastric antrum and pylorus were distorted,
and a contracted outlet was observed (1D). The patient was
diagnosed with pyloric obstruction (Figure 1E). In addition,
biopsy was performed from the edge of the ulcer. The patholog-
ical examination showed lymphocytes, plasma cells and
neutrophil infiltrations in the lamina propria. The histology
results showed acute on chronic inflammation of gastric body
(1F). The computed tomography (CT) abdomen revealed poor
gastric filling, diffuse thickening of the gastric wall, and enlarge-
ment and enhancement of the third group of gastric lymph
nodes. The patient underwent distal gastrectomy with gastroje-
junostomy. The postoperative pathological examination showed mucosal surface erosions, glandular hyperplasia, and focal ulcer formation in some areas.

DISCUSSION

Although glyphosate is generally considered to be of low toxicity
to humans, in 2015, the International Agency for Research on
Cancer concluded it as carcinogenic. Long-term exposures to
glyphosate may lead to kidney cell necrosis and leukemia, and
may also lead to the occurrence of rare bacteremia. CNS depres-
sion, acute kidney injury, hepatic dysfunction, gastrointestinal
mucosal damage, pancreatitis, electrolyte abnormalities, acidosis, acute respiratory distress syndrome (ARDS), hypoten-
sion, cardiovascular collapse, and death are all described. In
the epidemiology of glyphosate poisoning in Taiwan from 1986
to 2007, cardiovascular collapse and respiratory failure were
found to be the two major causes of fatality. Intractable shock,
especially hypovolemic shock, has been proposed to be the most
important cause of fatality. In addition, noncardiogenic pulmo-

ary edema or ARDS has been described as an important cause
contributing to both respiratory failure and death. At present,
herbicides on the market are mostly mixed preparations of
glyphosate and other chemicals, making its toxicity higher, and
its mechanism of poisoning more complicated.

At present, treatments for acute glyphosate poisoning mainly
include gastric lavage, inhibition of gastric acid, protection of
mucosa, and other symptomatic treatments. Glyphosate is
acidic diesel oil or laundry powder can enhance its medicinal
properties, and its activity can be lost after complexing with Ca,
Mg, and Al. In cases of severe glyphosate poisoning, such as
development of ARDS, cardiovascular and respiratory compro-
mise, extracorporeal membrane pulmonary oxygenation
(ECMO) may be effective in the treatment.

Occasional cases of esophageal perforation and death caused
by glyphosate poisoning are reported. In the present case, no
obvious abnormality was found in the esophagus during the
whole course of treatment. The endoscopic images revealed that
the fornix of the fundus of the stomach showed mostly round and
concave scar marks and a longitudinal ulcer (0.8 × 3.5 cm), and
multiple longitudinal ulcer scars could be observed in the greater
curvature of the stomach body. It was hypothesized that the site
and shape of ulcer formation correlated to the site of accumula-
tion of glyphosate in the gastric cavity.

The choice of gastric lavage method, avoidance of neutralising
acid-base reactions with drugs such as sodium bicarbonate, and
the long-term use of acid suppression and mucosal protection
therapy may have important therapeutic implications for the
repair of gastric mucosa. However, further observations are
needed to determine whether the occurrence of complications
caused by ulcer scars, such as gastric cavity deformation and
pyloric obstruction, can be reduced by prolonging the time of
acid suppression therapy, and increasing the dosage of acid
suppression and mucosal protection drugs.
PATIENTS’ CONSENT:
Consent for publication was obtained from all individuals whose data are included in this manuscript.

CONFLICT OF INTEREST:
All authors have contributed significantly to the manuscript and declare that the work is original and has not been submitted or published elsewhere. None of the authors have any financial disclosure or conflict of interest.

AUTHORS’ CONTRIBUTION:
JYH: Collected data and written manuscript.
TJL: Collected data and involved in written.
XJH: Edited and critically reviewed the manuscript.

REFERENCES