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Between 1 January 1980, and 30 September 1989, 93 cases of exposure to herbicides containing glyphosphate and surfactant ('Roundup') were treated at Changhua Christian Hospital. The average amount of the 41% solution of glyphosate herbicide ingested by non-survivors was 184 ± 70 ml (range 85–200 ml), but much larger amounts (500 ml) were reported to have been ingested by some patients and only resulted in mild to moderate symptomatology. Accidental exposure was asymptomatic after dermal contact with spray (six cases), while mild oral discomfort occurred after accidental ingestion (13 cases). Intentional ingestion (80 cases) resulted in erosion of the gastrointestinal tract (66%), seen as sore throat (43%), dysphagia (31%), and gastrointestinal haemorrhage (8%). Other organs were affected less often (non-specific leucocytosis 65%, lung 23%, liver 19%, cardiovascular 18%, kidney 14%, and CNS 12%). There were seven deaths, all of which occurred within hours of ingestion, two before the patient arrived at the hospital. Deaths following ingestion of 'Roundup' alone were due to a syndrome that involved hypotension, unresponsive to intravenous fluids or vasopressor drugs, and sometimes pulmonary oedema, in the presence of normal central venous pressure.

Introduction

Herbicides containing glyphosate (N-phosphonomethyl glycine) are an alternative to paraquat and have recently been used in attempted suicide. Glyphosate acts primarily as a competitive inhibitor of 5-enolpyruvoylshikimic acid-3-phosphatesynthase,[1,2] an enzyme essential to the synthesis of aromatic amino acids in plants. Since this enzyme is absent in animals, glyphosate is considered to have low mammalian toxicity. The acute oral LD_{50} of glyphosate is reported to be approximately 4–6 g kg^{-1} in rat and rabbit.[3,4]

Changhua Christian Hospital is the receiving hospital for a large agricultural area in central Taiwan. During the period 1975–1989, a total of 7035 cases of acute poisoning were seen, of which 80% were due to agricultural chemicals, and 17.6% of the entire series was due to herbicides.[5] This paper reports our experience with cases of poisoning with herbicides containing glyphosate.

Methods

A list of 12 herbicides registered as containing glyphosate was obtained from the Taiwan Agricultural Agents and Toxic Substances Research Institute. The official Monsanto 'Roundup' product was sold under the Chinese names *lan-da, hao-ni-chun,* and *nian-nian-chun*. Roundup is a commercial product containing 41% glyphosate (360 g l^{-1} of free glyphosate) as the isopropylamine salt, 15% surfactant (polyoxyethyleneamine, POEA) and water.[6]

Emergency room records were reviewed for the period from January 1, 1974, to September 30, 1989, for diagnoses related to drug poisoning, poisoning with agricultural agents, poisoning due to unknown agents, 'suicide' with cause not listed and cases of dead-on-arrival (DOA). The charts from this selection were then reviewed and only those recording exposure to glyphosate herbicides were included in the present series. Exposure to

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'glyphosate herbicide' was diagnosed by history and verification of the agent by hospital staff (transcription of the label of the bottle into the chart records). Since other agricultural agents are often involved in poisoning in our area, paraquat was excluded in all cases by the dithionite test for paraquat in urine,[7], and organophosphate poisoning was similarly excluded by monitoring plasma pseudocholinesterase levels by colorimetry (Cholinesterase-NA test, Wako Junyaku Kogyo Coy, Osaka, Japan).

All data were retrospectively extracted from the medical records. Other agents (either agricultural chemicals or alcohol) ingested at the same time were noted, as was the trade-name of the glyphosate product.

The amount ingested was usually given in descriptive terms such as 'a mouthful' or 'a half a bottle'. For statistical purposes, arbitrary volumes were assigned to the phrases that occurred frequently. 'A little' or 'a spoon' was 5 ml, 'a mouthful' was 25 ml, 'a small cup' was 100 ml, and 'a bottle' was taken as 300 ml (bottles available commercially were 150 ml, 300 ml, 600 ml and 1000 ml, but the 300 ml bottle was most commonly used by our patients). Cases in which the amount was not recorded or was not known were excluded from the analyses based on amount ingested.

The degree to which a patient exhibited clinical signs and symptoms was classified into four groups (asymptomatic, mild, moderate, severe) as summarized in Table 1.[8] The severity of intoxication was also quantitated by the APACHE II system using the diagnostic category weight for aspiration/poisoning/toxic to calculate the risk of death.[9]

Organ involvement was defined as positive if laboratory tests of organ function abnormal for our hospital were recorded at least once in the chart. For renal function, this was serum levels greater than 106.8 μmol l^{-1} for plasma creatinine (Cr), 7.12 mmol l^{-1} for blood urea nitrogen (BUN); for liver enzymes, greater than 40 iu l^{-1} for both aspartate aminotransferase (AST) and alanine aminotransferase (ALT). Chest radiographs (CXR) were classified as normal or abnormal, and the degree and type of pulmonary oedema was scored using a standard technique.[10] Bilateral alveolar exudates in the presence of normal central venous pressure were diagnosed as adult respiratory distress syndrome (ARDS). Gastrointestinal tract (GIT) involvement was judged by symptoms (sore throat, oesophagitis, abdominal pain, dysphagia), the presence of melaena or haematemesis, and endoscopy findings when performed. Central nervous system (CNS) involvement was documented if the patient was confused or comatose and other causes had been excluded, but was not differentiated from confusion secondary to hypercapnia or hypoxaemia. Haematological evaluation included a peripheral smear and differential count of leucocytes, with a leucocyte count above 9000 mm^3 defined as 'increased' (leucocytosis). Roger's test[11] was used to test for seasonal trends in incidence.

Table 1 Classification of severity in acute poisoning with Roundup herbicide.

Classification	*Description*
Asymptomatic	no complaints, and no abnormalities on physical or laboratory examination.
Mild	mainly GIT symptoms (nausea, vomiting, diarrhoea, abdominal pain, mouth and throat pain) that resolved within 24 h. Vital signs were stable, and there was no renal, pulmonary or cardiovascular involvement.
Moderate	GIT symptoms lasting longer than 24 h, GIT haemorrhage, endoscopically verified oesophagitis or gastritis, oral ulceration, hypotension responsive to intravenous fluids, pulmonary dysfunction not requiring intubation, acid–base disturbance, evidence of transient hepatic or renal damage, or temporary oliguria.
Severe	pulmonary dysfunction requiring intubation, renal failure requiring dialysis, hypotension requiring treatment with pressor amines, cardiac arrest, coma, repeated seizures, or death.

Results

The final series in which the agent was satisfactorily identified by history and label consisted of 93 patients with seven deaths (Table 2). The first death was seen in 1984.

The different glyphosate products ingested included *nian-nian-chun* 88 cases (including the two with other pesticides); *hao-ni-chun* two cases; *ri-ri-hao* one case; and *jia-lin-sai* two cases. The majority (90 out of 93) were the official Monsanto product.

The number of cases was smallest in the winter months, with an average of two per month for November and December, and showed a peak in the summer months of June (average 10 per month) to September (averge 18 per month). Roger's coefficient of seasonal variation[11] was 8.97 and was significant at the $P < 0.025$ level. This reflected the trend to increased numbers during the summer seen here in poisoning with other agricultural agents.[5]

The age distribution of the patients is shown in Table 3. There is a peak for both sexes in the 20–30-year-old age group; males were more common in the age groups above 30 years. Male patients were twice as frequent as female patients. The average age for the entire series was 37 ± 17 years ($n = 93$). The average age of survivors was 36 ± 16 years ($n = 86$), and for non-survivors the average age was 58 ± 19 years ($n = 7$).

Aspiration pneumonitis occurred in one case, and the patient survived. In the five fatalities due to glyphosate herbicide alone, the patient was alert and cooperative on admission to hospital, and had not ingested alcohol. Of the 86 survivors, five had taken glyphosate herbicide while drinking alcohol and two had mixed it with fruit juice. All other intentional ingestions were of the undiluted 41% glyphosate herbicide.

The amounts ingested are compared to the clinical symptoms in Table 4. The numbers are suggestive of a dose-response relationship, with increasing amounts resulting in more severe symptoms; however, the patients' estimates of the amount ingested, and the conversion ratio used in this paper may be inaccurate. Two points that can be made are that reported large amounts did not necessarily cause serious symptoms ('moderate' range up to 500 ml), and severe symptoms (death) only resulted from oral ingestion of undiluted herbicide in amounts above 85 ml.

The risk of hospital death compared to clinical symptoms is shown in Table 5. All patients with a favourable outcome had a risk less than 0.2, whereas all eight of those with a risk score above 0.2 were in the severe group and seven of them died.

All cases of accidental exposure (dermal two cases; eating vegetables that had been sprayed four cases) were asymptomatic. Gastrointestinal ingestion by mistake (seven cases) was usually of a small amount that was immediately rejected, and resulted only in minor mouth discomfort.

Atropine and pralidoxime were given to three cases, one of whom was the non-survivor who ingested both glyphosate and parathion.

The times from ingestion to arrival at hospital are shown in Table 6. The average time was

Table 2 Yearly incidence of poisoning with glyphosate herbicides. Numbers in parentheses indicate deaths.

Year	*Male*	*Female*	*Total*
1980	1	0	1
1981	2	1	3
1982	0	2	2
1983	0	1	1
1984	4 (1)	4	8
1985	3	1	4
1986	3	3	6
1987	9	3 (1)	12
1988	14 (2)	12 (1)	26
1989*	23 (1)	7 (1)	30
Total	59	34	93

* The series ends on 30 September

Table 3 Age distribution of patients poisoned with glyphosate herbicides.

Age range	*Female*	*Male*	*Total*
0–10	0	1	1
11–20	4	7	11
21–30	15	10	25
31–40	7	14	21
41–50	3	4	7
51–60	2	11	13
61–70	3	7	10
71–80	1	3	4
81–90	0	1	1

Table 4 Comparison of severity of clinical symptoms with estimated amount of glyphosate herbicide ingested Amount (mean ± s.d.) and range are in ml. Severity groups as described in Table 1.

Severity	*Number*	*Amount*	*Range*
Asymptomatic	13	17 ± 16	5– 50
Mild	38	58 ± 52	5–150
Moderate	22	128 ± 114	20–500
Severe	4	184 ± 70	85–200

Table 5 Risk of hospital death compared to clinical group.

*Risk**	*Asymptomatic*	*Mild*	*Moderate*	*Severe*
0.0–0.1	18	39	19	"
0.1–0.2	"	6	4	"
0.2–0.3	"	"	"	1
0.5–0.6	"	"	"	1
0.6·0.7	"	"	"	2
0.8–0.9	"	"	"	2
0.9–1.0	"	"	"	1
Total (n = 93)	18	45	23	7

* Risk calculated from APACHE II score in first 24 h.[9] Risk levels (0.3–0.5) not represented in this series have been omitted from the table.

Table 6 Time (h) from ingestion of glyphosate herbicide to arrival at the emergency room.

Category	*Number*	*Mean ± s.d.*	*Min*	*Max*
Survived	80	12.2 ± 19.3	0.1	85.3
Dead	6	4.7 ± 3.1	2.5	11.5
Accident	8	26.6 ± 31.8	0.7	85.3
Suicide	79	10.1 ± 16.1	0.1	83.3
Overall	87	11.6 ± 18.7	0.1	85.3

around 12 h, with a range of 10 min to nearly 3 d. The average time in fatal cases was very short (4.7 h); however, death resulted despite medical treatment. Of the seven deaths in this series, two involved ingestion of multiple agricultural agents: one was *nian-nian-chun* with paraquat, and the other was *nian-nian-chun* with parathion. Neither of these patients could be resuscitated in the emergency room. The averge time from ingestion of glyphosate herbicide to death was 26.4 ± 26.4 h (range 3.7–38.5 h): five died within the first 12 h (two were dead on arrival), one died within 24 h, and one died at 38.5 h after ingestion. Death was due to pulmonary oedema or hypotension unresponsive to intravenous fluids or pressor agents. For cultural reasons, autopsies were not performed.

The incidence of signs and symptoms in the 74 symptomatic patients in the series is presented in Table 7. GIT symptoms 66% (49/74) were most common, with upper GIT symptoms similar to ingestion of a corrosive agent (sore throat, mouth ulcer, dysphagia, epigastric pain) being most frequent. Vomiting was poorly recorded because the patient had already been treated by gastric lavage and emetics, or gave a poor history. GIT haemorrhage occurred in 12% (6/49) of patients with GIT symptoms (8% of all patients with symptoms). Paralytic ileus was not seen, and pancreatitis was not evident clinically, although serum amylase was measured in only two cases.

Endoscopy was performed in 23 cases and was abnormal in 22. The oesophagus was involved in 39% (9/23), with ulcers (one case) and superficial (six cases) or corrosive (two cases) oesophagitis. The stomach was involved in 96% (22/23), as gastritis (17 cases) and ulceration (five cases). The duodenum was involved in 26% (6/23) as duodenitis (four cases) and duodenal ulcer (two cases). A lower GIT endoscopy in one case showed colitis.

Pulmonary dysfunction was documented in 23% (17/74) of cases. Asymptomatic mild hypoxaemia (arterial oxygen tension less than 11.3 kPa while breathing room air) was frequent, and chest radiographs were usually normal. There were eight cases of pulmonary oedema diagnosed as ARDS, of whom only one survived. Case 1 is an example.

The cardiovascular system was recorded as abnormal in 18% (17/74) of cases. The arrythmias and abnormal electrocardiograms (ECG) were usually sinus tachycardia or sinus bradycardia. In contrast to Japanese reports,[19] atrio-ventricular block was not seen. Hypotensive shock occurred in all seven of the non-survivors. This hypotension sometimes responded initially to hydration, but then recurred within hours. It was not due to hypovolaemia (as judged by central venous pressure, haemotocrit, and blood urea nitrogen) and did not respond to fluid resuscitation or vasopressor agents (Case 2 is an example).

Renal abnormalities occurred in 14% (10/74) of cases. They all had elevated serum creatinine

Table 7 Incidence of signs and symptoms (n = 74). All – number of patients with data recorded for this organ. No – number of patients with data recorded for this organ who had the sign/symptom.

Organ	*All*	*Sign/Symptom*	*No*
GIT*	49	sore throat	32
		mouth ulcer	32
		dysphagia	23
		epigastric pain	16
		melaena	6
		vomiting	1
Smear	48	leucocytosis	48
		shift-to-left	1
Lung	17	abnormal CXR	9
		aspiration	1
		intubation	7
		respirator	7
Heart	13	shock	7
		arrythmia	4
		abnormal ECG	7
Kidney	10	oliguria	3
		haematuria	3
		Cr increased	9
		BUN increased	0
Liver	14	GOT increased	14
		GPT increased	8
CNS	9	confused	3
		coma	6

* Endoscopy performed in 23 cases. Smear – peripheral smear for leucocytes and differential count. Other abbreviations (GIT, CXR, Cr, BUN, AST, ALT, CNS) as in the text.

(but only above 176.8 μM l^{-1} in three patients), and sometimes oliguria (3/10). Haematuria occurred in three cases. Blood urea nitrogen was normal in all cases.

Liver enzymes were mildly elevated (less than twice normal) in 19% (14/74) of cases. In all cases, AST was elevated, and in eight of them ALT was also elevated. The time course of these changes is unknown, as most patients had only one measurement recorded in the chart.

CNS symptoms occurred in 12% (9/74), two of whom followed cardiovascular resuscitation in the emergency room. Glasgow Coma Scale (GCS) scores ranged from 9 to 13, and improved rapidly without treatment.

The peripheral smear showed leucocytosis in 52% (48/93) of cases. The incidence of leucocytosis appeared to be related to the clinical group (asymptomatic 30% (4/12), mild 53% (24/45), moderate 65% (15/23) and severe 71% (5/7)). A shift to the left occurred only in the patient with aspiration pneumonitis.

Case reports

Case 1. A 78-year-old male drank 'a bottle' of *nian-nian-chun* at 11:00 p.m. with suicidal intent. He was treated at our emergency room 45 min later with gastric lavage, ultracarbon, oxygen, and intravenous fluids. Urine was negative for paraquat, and plasma cholinesterase was 8211 U l^{-1} (normal 2816–6971). On arrival, his blood pressure was 21/12 pKa, pulse 102 beats per minute, respiratory rate 28 per minute, and he was dyspnoeic. Lung examination revealed diffuse rhonchi and wheezing. The haemoglobin was 14.5 g 100 ml^{-1}, white cell count 8800 mm^3, platelets 197000 per mm^3, blood urea nitrogen 7.83 mM l^{-1}, plasma creatinine 132.6 μMol l^{-1}, blood glucose 8.03 mM l^{-1}. Electrolytes (sodium, potassium, chlorine, calcium) and enzymes (AST, ALT were normal. Arterial blood gases while breathing room air were pH 7.06, PaO_2 4.62 pKa, $PaCO_2$ 8.76 pKa.

Chest radiographs showed pulmonary oedema progressing to the characteristic picture of ARDS (Figure 1). He was intubated and ventilated with

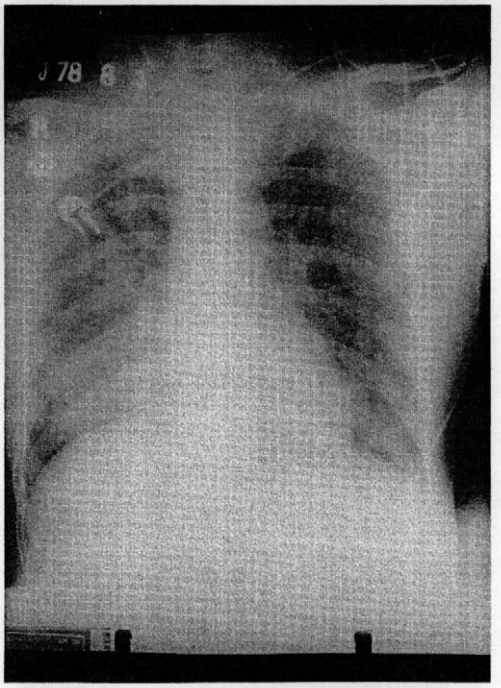

Figure 1 Chest radiograph of patient with glyphosate poisoning (Case 1). The vascular pedicle width is normal as is the heart size, but perihilar irregularities are evident. Patchy involvement of the right lower lobe and peripheral infiltrates in the right upper lobe are prominent. Perviascular and peribronchial cuffing are present.

positive end expiratory pressure (PEEP), but hypoxaemia showed little improvement (PaO_2 5.32–6.65 pKa on 60% inspired oxygen and PEEP 5 cmH_2O), and he developed hypotension that did not respond to intravenous dopamine and fluids. Cardiac arrest occurred 13 h after ingestion.

Case 2. A 60-year-old male drank 'a bottle' of *nian-nian-chun* at 8:30 a.m. with suicidal intent. Vomiting occurred immediately. He was treated by a local doctor with an 'injection' (probably atropine), and was then transferred to the emergency room at 2:00 p.m. On arrival, he was confused (GCS of 11) but 30 min later was fully conscious (GCS 15) with normal pupillary reflexes. Blood pressure was 16/11 pKa, pulse 90 beats per minute, respiratory rate 24 per minute, and temperature 36.5 C. The haemoglobin was 13.9 g 100 ml^{-1}, with a white blood cell count of 17,400 (band 6%, segmented 87%, lymphocytes 10%). Electrolytes (sodium, potassium), plasma creatinine, blood urea nitrogen, and enzymes (AST, ALT) were within normal limits. Blood glucose was 3.74 mM l^{-1}. Urine paraquat was negative and plasma cholinesterase was normal.

He was treated with gastric lavage and ultracarbon. A toxicology screen for barbiturates, phenytoin, carbamazepine, and alcohol was negative. Arterial blood gases on 3 l min^{-1} oxygen by face mask were pH 7.24, PaO_2 15.4 pKa, $PaCO_2$ 4, 23 pKa. A chest radiograph was normal. The resting electrocardiogram showed no abnormalities.

He was sent to the ward, but 2 h later, blood pressure was 9/5 pKa and respiration was regular and shallow. The patient was transferred to the intensive care unit, but the hypotension failed to respond to intravenous fluids and vasopressor agents. Lung fields remained clear, and the bedside ECG showed sinus tachycardia (130–160 beats per minute). Cardiac arrest occurred 14 h after ingestion.

Discussion

There are few reports of the effects of glyphosate or Roundup on man.[12,13] The annual report of the American Association of Poison Control Centers does not specifically refer to it.[14] The US Environmental Protection Agency has an unedited series of 109 pesticide incidents involving glyphosate[15] reported during the period 1966–1980. Of them, 94 involved glyphosate alone and 15 cited glyphosate in combination with other ingredients. In the 91 incidents involving humans and glyphosate alone, four persons were hospitalized, 80 received medical attention, and nine were affected but did not receive medical attention. There were no deaths.

Reports discussing post-mortem measurement of glyphosate refer to three deaths but not in clinical detail.[16,17]

In Japan, glyphosate is sold under the tradename 'Roundup' (Monsanto Co, St Louis, MO, USA). The Japanese experience was summarized by Sawada[18,19] in a series of 56 cases with nine deaths. The Monsanto Company commented that it was comparable in safety to many toiletries and household products containing surface-active ingredients.[20] The Sawada series is the one referred to in most reviews.[21–23] Details of one death[16] and three survivors[24,25] have been reported, but other case reports (Table 8) have appeared only as abstracts,[26–31] and it is not clear how many of them are duplicated in the Sawada series.

The first case in the series was seen in 1980, one year after the introduction of Roundup to Taiwan, and there has been a dramatic increase in the number of cases since 1987. The results for the amount ingested (Table 4) suggest a dose-response relationship, but this needs to be investigated with more accurate identification of the dose by, for example, measurement of the plasma level of herbicide. Glyphosate and its

Table 8 Summary of reports from Japan. Amount – amount of Roundup ingested (ml); nr – amount ingested not recorded; HP – charcoal haemoperfusion; HD – haemodialysis; Ref – reference number. Where reports refer to the same patient, multiple references are shown.

Age	*Sex*	*Amount*	*Outcome*	*Ref.*	*Comment*
60	**F**	200	Dead	26, 27	HP, shock, coma, acidosis
73	**M**	100	Survived	23, 25, 28	endotracheal intubation
27	**M**	10	Survived	23, 25, 28	endotracheal intubation
36	**M**	100	Dead	29	hypotenstion, pulmonary oedema
52	**F**	280	Survived	30	HP pancreatitis, pulmonary oedema
64	**F**	nr	Survived	31	HD acidosis, hypotension
48	**F**	nr	Dead	31	acidosis, hypotension
31	**M**	50	Survived	31	sore throat, headache
72	**F**	30	Survived	31	diarrhoea, renal dysfunction
37	F	50	Survived	24	acidosis, creatinine increased

major plant metabolite, aminomethyl phosphonic acid (AMPA), have been measured by gas chromatography,[32] high-performance liquid chromatography,[33,34] gas chromatography/mass spectrometry,[16] and nuclear magnetic resonance spectroscopy,[17] but none of those techniques are likely to be available in referring hospitals where most of the cases occur.

There was only one case of aspiration pneumonitis, and the outcome was favourable. Oral ingestion was not associated with 'aspiration pneumonia after suppression of the protective reflexes by alcohol consumption',[20] and the reference to unpublished records of 'many cases of survival after ingestion of quantities of roundup estimated to be several hundred millilitres'[20] is not supported by our findings.

The pharmacokinetics of glyphosate in man are unknown. Plasma glyphosate about 7 h after ingestion was 69.2 ppm in one case, but was undetectable (less than 10 ppm) by the second day and over the next 4 days.[24] In another case, a spot urine on arrival at the hospital 30 min after ingestion contained a high concentration of glyphosate (15,100 $\mu g\ ml^{-1}$), which had become undetectable (less than 2.5 $\mu g\ ml^{-1}$) by day 3.[25] In rats, a single oral dose was 15% (males) to 35–40% (females) absorbed, with a small amount of biliary excretion and enterohepatic circulation.[4]

Symptomatology in the present series agreed with the series from Japan,[18,19] although the incidence of individual effects varied. The lower incidence of GIT symptoms in our series may be due to under-reporting in the chart, as, until recently, the cases were considered very safe and received little attention after admission to hospital. Vomiting and diarrhoea were obscured by medical treatment, which included gastric lavage, emetics, and bowel washout in some cases. We have seen no cases of paralytic ileus. The mucosal ulceration documented by endoscopy, resembles that of other corrosive agents. Studies using the gastric mucosa of dogs showed that pure glyphosate, the surfactant contained in Roundup, and the Roundup formulation, all caused injuries similar to those made by 0.25 N HCl.[35–37]

The toxicity of the commercially available herbicide may be due to constituents other than glyphosate.[38,39] Sawada has proposed that the syndrome in fatal cases resembles that of toxicity due to the surface-active agent,[18,19,40] although other commercially available products containing surfactants are usually benign.[14] In beagle dogs using concentrations equivalent to those in Roundup, pure glyphosate caused increases in heart rate, cardiac output, and blood pressure.[41] The surfactant from Roundup, and the commercial Roundup preparation, both caused decreases in these parameters. All three agents caused increases in mean pulmonary artery pressure and both systemic and pulmonary vascular resistance. Sawada[19] suggested that the cardiovascular shock was due to hypovolaemia. In our series, haematocrit, blood urea nitrogen, and central venous pressure did not support hypovolaemia as the cause of the shock that developed after rehydration.

Uncoupling of mitochondrial oxidative phosphorylation has been suggested as one lesion in glyphosate poisoning,[42,43] but technical difficulties make the results difficult to interpret. There is variability in the mitochondrial State 4 respiration rates displayed, and they were then used to calculate respiratory control ratios. Moreover, the mitochondria recover after a single addition of ADP, in contrast to the result with dinitrophenol, which causes true uncoupling. In rats given glyphosate intragastrically for 2 weeks, glyphosate decreased the hepatic level of cytochrome P450 and monooxygenase activities, and the intestinal activity of aryl hydrocarbon hydroxylase.[44] The clinical significance of these biochemical abnormalities is unclear.

In conclusion, the toxic syndrome resulting from the massive ingestion of Roundup herbicide consists of mucosal and gastointestinal irritation, hypotension, and pulmonary insufficiency. Effects on organs other than the gastrointestional tract tend to occur with increasing amounts ingested. The data suggest that those over 40 years of age, who ingest more than 100 ml, are at the highest risk of a fatal outcome.

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