PLANT POISONING IN THAILAND: A 10-YEAR ANALYSIS FROM RAMATHIBODI POISON CENTER

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Abstract. Plant poisoning is not uncommon in Thailand. The objective of this study was to determine the incidence, type, clinical manifestations, severity and outcomes of plant poisoned patients in Thailand over a 10-year period. We retrospectively reviewed data from the Ramathibodi Poison Center Toxic Exposure Surveillance System for 2001-2010. A total of 2,901 poisonous plant exposure cases were identified, comprising 3.1% of the 92,392 poison cases recorded during the study period. This was the fifth most common type of poisoning recorded. Children aged <13 years comprised the largest percent (69.8%) of the cases. The major type of exposure was unintentional ingestion. Ninety-nine types of poisonous plants were recorded as the causative agents among 99.1%of the cases. Gastrointestinal symptoms were reported in 72.0% of cases with *Jatropha curcas* (physic nut) comprising 54.1% of these. Most patients had only minor signs and symptoms. The mortality rate among the total plant poisoning cases was 0.9%, with 26 deaths. Thirteen deaths occurred in children aged <13 years. The greatest number of fatalities were due to ingestion of *Manihot esculenta* (cassava), primarily due to multi-system organ failure. Children aged <13 years are at the greatest risk for plant poisoning in Thailand; mostly unintentional. Most cases were minor and the mortality rate was low. *Jatropha curcas* was the most common cause of poisoning and *Manihot esculenta* was the most common cause of death. Public education is important to minimize these poisonings.

Keywords: plant poisoning, incidence, clinical manifestations, severity, outcomes

INTRODUCTION

Many plants are potentially poisonous to humans. Plant poisoning can cause a variety of signs and symptoms but fatalities are uncommon (Bronstein *et al*, 2011; Lin *et al*, 2009).The toxic effects depend on the species, growth stage, part of the plant, amount, route of exposure, and susceptibility of the victim (Botha and Penrith, 2008). Having information about the toxins and their clinical effects is important in the diagnosis, management and prevention of plant poisonings.

Many plant poisoning studies have been conducted; the types of poisonous plants and the effects vary by regions (Lin

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et al, 2009; Bronstein *et al*, 2011; Fuchs *et al*, 2011). In Thailand and Southeast Asia studies of plant poisoning are limited. Most are case reports (Ruangkanchanasetr *et al*, 1999; Narongchai *et al*, 2005) or small case series (Echeverria *et al*, 1986; Chomchai *et al*, 2011; Sang-A-Gad *et al*, 2011). To better determine the edipemiology of plant poisoning in Thailand, we conducted this retrospective study.

MATERIALS AND METHODS

We conducted a retrospective cohort study of all single agent human exposures to poisonous plants reported to the Ramathibodi Poison Center (RPC), Bangkok, Thailand from January 2001 to December 2010. We excluded exposure to poisonous mushrooms, *Cannabis sativa* (marijuana), *Mitragyna speciosa* (kratom), and industrial processing of plant materials (such as herbal extract pills or health supplement powder). The RPC serves the entire country of Thailand, both healthcare personnel and the general public, 24 hours a day, 7 days a week. The RPC receives more than 15,000 calls per year. The RPC Toxic Exposure Surveillance System database is the primary source of data regarding poisonings in Thailand. Besides providing information for diagnosis and management of patients, follow-up calls are made to provide ongoing management recommendations and to determine the patient's course and outcome. All cases are reviewed by medical toxicologists and recorded in the RPC database. The definitions and terms used in the database have been adopted from the IPCS INTOX Data Management System (IPCS, 2013) and the American Association of Poison Control Centers' National Poison Data System (Bronstein *et al*, 2011).

To be confirmed as a specific poisonous plant exposure, the plant was identified and the toxic effects determined. Plant identification was made by 1) description given by the patient or a relative, 2) comparison with a sample plant or identification using botany textbooks or other similar resource (Temsiririrkkul, 1992; Silkavute *et al*, 1994; Wuthithammawet, 1997; Medicinal Plant Information Center, 2013; Medicinal Plant Research Institute, 2014; Thai Junior Encyclopedia Project by Royal Command of HM the King, 2014), and 3) in difficult cases, identification by a botanist at the Medicinal Plant Information Center, Faculty of Pharmacy, Mahidol University. The diagnosis of a specific plant poisoning was made only if the clinical effect observed matched the identified plant. Other cases were diagnosed as unknown plant exposures. This verification process was conducted by a team of toxicologists and information scientists.

To simplify the poisoning cases, we grouped them by toxidromes according to the clinical manifestation and mechanism of toxicity (Temsiririrkkul, 1992; Silkavute *et al*, 1994; Nelson *et al*, 2007; Lin *et al*, 2009; Nelson *et al*, 2012; Nelson and Goldfrank, 2015) as shown in Table 1.

The RPC data were obtained and entered into Microsoft Excel, 2010 and then analyzed with SPSS, version 17 (IBM, Armonk, NY). The data recorded included demographic data, age and gender of the patient, identified plant, toxidrome category of the plant, reason for exposure, initial severity, and clinical outcome.

RESULTS

Number and characteristics of poisonous plant exposure in humans

From January 2001 to December 2010, 92,392 poison exposures among humans were reported to the RPC. Pesticides,

medical products and household products were the most common substances involved (Table 2). Of these, 2,901 (3.1%) were single poisonous plant exposures, the fifth most common type of poisoning. After excluding 12 cases with incomplete information, 2,889 cases were included in this study.

The total number of poisoning reported increased yearly during the study period; however, the number of poisonous plant cases peaked during 2008, then remained constant at about 400 cases per year (Fig 1). The plant poisoning cases peaked during June and November (Fig 2).

The majority of the cases were males (60.4%). The median age of plant poisoning victims was 9 years (range 2 months to 93 years); 69.8% were aged <13 years. The most commonly affected age group was school-age (48.3%). Males predominated in the school-age, teenager, and young adult groups, while females predominanted in the older age groups. The overall mortality rate was 0.9%. The older age group had a lower frequency but higher mortality rate (Table 3). Ingestion (99.2%) was the major route of exposure. Only few cases were ocular exposure (0.6%), dermal exposure (0.1%) or combined ocular and dermal exposure (0.1%). Exposure was unintentional in 2,795 cases (96.8%), and accidental exposure accounted for 2,730. Seventy-three cases (2.5%) were intentional and 21 cases (0.7%) were unknown or not recorded.

Specific poisonous plants and toxidromes

Ninety-nine types of plants identified as the causative agents in 2,864 cases (99.1%). Twenty-five cases (0.9%) did not have an identified type of plant. The toxidromes, related plants, numbers of cases and mortality rate are shown in Table 4. Plants with a gastrointestinal toxidrome were the most common toxidrome (72.0%); *Jatropha curcas* (physic nut) was involved in 54.1% of these. A multi-system organ failure toxidrome was the second most common toxidrome (11.1%), followed by neurotoxic (9.7%), anticholinergic (4.2%), cardiovascular (1.3%), cutaneous/mucosal inflammation (0.8%) and nephrotoxic (0.1%) toxidromes.

Clinical severity and outcomes

Table 5 shows the outcomes and initial severity at the time of consultation with the RPC. The majority of case (91.5%) were of minor severity at the time of consultation while 4.0% were of moderate severity and 2.8% had no symptoms. Forty-seven patients (1.6%) were severe at the time of consultation and 1 had died.

When taking into account the entire clinical course, 91.8% of cases were minor, 4.3% were moderate, 1.6% were severe, 0.9% (26 cases) died and 1.9% had no signs or symptoms of problems. None of the surviving patients had residual sequelae after recovery.

Lethal poisonings

Among the 26 deaths, the median age was 16.5 years (range 2-93 years); 19 were male (Table 6) and 13 were aged less than 13 years. Eighteen took the poisonous plant accidentally. Five patients took *Gloriosa superba* or *Strychnosnux-vomica*, wrongly thinking the plants were a folk remedy. Two patients took the plants intentionally and 1 patient misused the plant. There were 11 identified poisonous plants and 2 unidentified poisonous plants ingested in the 26 deaths. The greatest number of deaths were related to the multi-system organ failure toxidrome (15 cases), 8 from the histotoxic toxidrome, 7 from the cellular arrest toxidrome, 7 from the neurotoxic toxidrome, 1 from

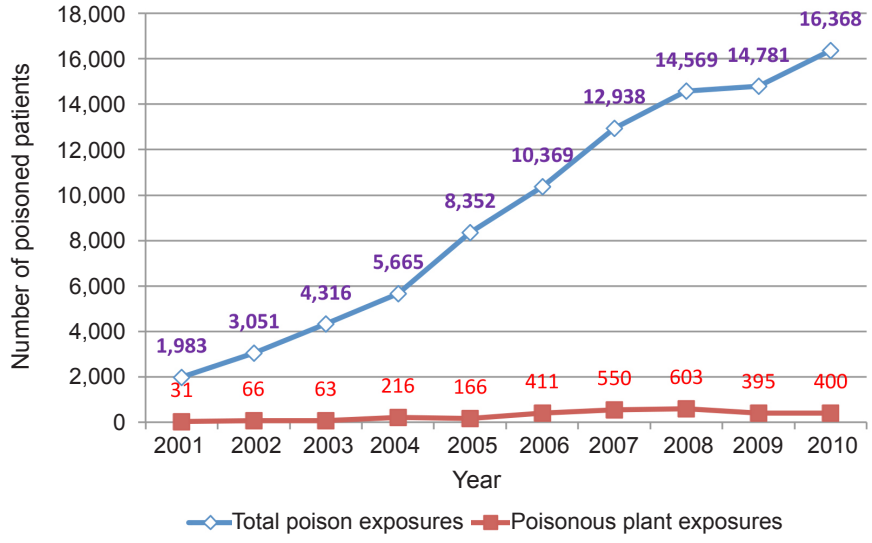

Fig 1–Plant poisoning cases reported by year.

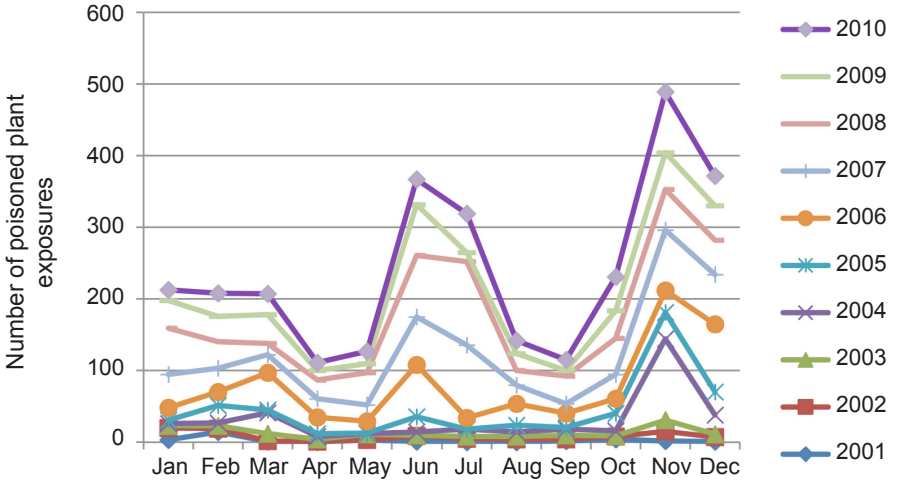

Fig 2–Plant poisoning cases reported by month.

the gastrointestinal toxidrome, 1 from the cardiovascular toxidrome and 2 had an unidentified toxidrome. *Manihot esculenta* (cassava or tapioca plant) was associated with the greatest number of deaths (6). Four deaths presented initially with mild clinical symptoms, 3 with moderate symptoms, 18 with severe symptoms, and 1 was dead on arrival. Among the 4 who initially presented with mild symptoms, the plants ingested were: cassava, rosary pea, glory lily and *Cnestis palala.*

The patient who was dead on arrival was a 93-year-old man who had ingested 10 seedpods of *Pachyrrhizus erosus* with 5 other family members in the evening.

Table 1
Toxidromes and example of poisonous plants.

Common name	Scientific name	Toxic ingredients	Clinical manifestations
Group 1. Gastrointestinal toxidrome			
Mechanism: gastrointestinal irritant			
Elephant's ear[a]	*Alocasia cucullata*	Calcium oxalate	Nausea, vomiting, burning sensation, perioral numbness, salivary drooling, dysphagia, diarrhea, abdominal pain.
Dumb cane[a]	*Dieffenbachia* spp		
Japanese taro	*Colocasia esculenta*		
Indian spurge tree[a]	*Euphorbia tirucalli*	Diterpene ester	
Physic nut	*Jatropha curcas*	Curcin (toxalbumin)	
Coral plant	*Jatropha multifida*	Hurin and crepitin	
Sandbox tree	*Hura crepitans*	(toxalbumin)	
Group 2. Neurotoxic toxidrome			
2.1 Neurotoxic toxidrome: "spinal seizures"			
Mechanism: competitively blocks the binding of glycine			
Nux vomica, poison nut	*Strychnos nux-vomica*	Strychnine	Muscular cramps, muscle spasms, muscle pain, patient remains awake and lucid during seizure.
2.2 Neurotoxic toxidrome: seizures; encephalopathy			
Mechanism: unknown			
Asiatic bitter yam, Wild yam	*Dioscorea hispida*	Dioscorine	Dizziness, alteration of conscious, muscle paralysis.
None	*Cnestis palala*	L-methionine sulfoximine	Seizure, dyspnea, respiratory failure.
None	*Urobotrya siamensis*	Unknown	Dizziness, alteration of conscious, metabolic acidosis, coma.
Group 3. Anticholinergic toxidrome			
Mechanism: competitive antagonism of acetylcholine			
Jimson weed, Devil's apple, Thorn apple	*Datura metel*, *Datura* spp	Scopolamine, atropine and other anticholinergic alkaloids	Flushing, tachycardia, dry skin and oral mucosa, dilated pupils, restlessness, agitation, hallucinations.
Group 4. Cardiovascular toxic toxidrome			
Mechanism: digitalis-like effect			
Foxglove	*Digitalis purpurea*	Digitoxin	Bradycardia, hypotension, cardiac dysrhythmias.
Oleander	*Nerium indicum*	Oleandrin and neriine	
Saat, Phansaat	*Erythrophleum succirubrum*	Cassaine, cassaidine, erythropheline	
Yellow Oleander	*Thevetia peruviana*	Thevetin, thevetoxin	

Table 1 (Continued).

Common name	Scientific name	Toxic ingredients	Clinical manifestations
Group 5. Multi-system organ failure toxidrome			
5.1 Multi-system organ failure toxidrome: histotoxic			
Mechanism: histotoxic			
Cassava, Tapioca plant	*Manihot esculenta*	Linamarin	Altered mental status, seizures, cardiovascular collapse, multisystem organ failure.
Yam bean, Jicama	*Pachyrhizus erosus*	Rotenone and rotenoid compounds	
5.2 Multi-system organ failure toxidrome: cellular arrest			
Mechanism: cellular arrest			
Rosary pea, Jequirity bean	*Abrus precatorius*	Abrin (toxalbumin)	Nausea, vomiting diarrhea, renal failure, liver failure, and multisystem organ failure.
Castor bean	*Ricinus communis*	Ricin (toxalbumin)	
Climbing lily, Glory lily	*Gloriosa superba*	Colchicine	Severe gastroenteritis, shock, leukocytosis, rhabdomyolysis, acute renal failure, bone marrow failure, multisystem organ failure.
Group 6. Cutaneous/mucosal inflammation toxidrome			
Mechanism: cutaneous/mucosal irritant			
Elephant ear[a]	*Alocasia cucullata*	Calcium oxalate	Dermal exposure: irritation, redness, burning sensation. Eye exposure: burning pain, blurred vision, keratoconjunctivitis.
Dumb cane[a]	*Dieffenbachia* spp		
Indian spurge tree[a]	*Euphorbia tirucalli*	Diterpene ester	
Group 7. Nephrotoxic toxidrome			
Mechanism: acute kidney injury			
Djenkol bean	*Archidendron pauciflorum*	Djenkolic acid crystals	Abdominal/flank pain, dysuria, gross hematuria, urinary obstruction, acute kidney injury (djenkolism).

[a]Plant with more than one toxidrome.

Table 2
Reported causes of poisoning.

Category	Number of cases (%)
Pesticides	33,018 (35.7)
Medical products	18,662 (20.2)
Household products	18,246 (19.8)
Technical and occupational products	10,503 (11.4)
Poisonous plants	2,901 (3.1)
Poisonous and venomous animals	2,248 (2.4)
Combined chemicals	1,961 (2.1)
Mushrooms	1,252 (1.4)
Recreational and abused agents	959 (1.0)
Thai/Chinese herbs	721 (0.8)
Food toxins and contaminants	452 (0.5)
Miscellaneous	684 (0.7)
Unknown	785 (0.9)
Total	92,392 (100.0)

Table 3
Poisoning cases by age group and gender.

Age (years)	Both genders		Male	Female
	Number (% of total)	Mortality, Number (% per age group)	Number, (% per age group)	Number, (% per age group)
0-5	623 (21.6)	9 (1.4)	317 (50.9)	306 (49.1)
6-12	1,394 (48.2)	4 (0.3)	904 (64.9)	490 (35.2)
13-19	260 (9.0)	0	204 (78.5)	56 (21.5)
20-39	300 (10.4)	4 (1.3)	187 (62.3)	113 (37.7)
40-59	207 (7.2)	2 (1.0)	94 (45.4)	113 (54.6)
≥60	105 (3.6)	7 (6.7)	39 (37.1)	66 (62.9)
Total	2,889 (100.0)	26 (0.9)	1,745 (60.4)	1,144 (39.6)

Four hours after ingestion, all of them developed nausea, vomiting and diarrhea. Since the man had taken a large amount of the plant, he persisted in having nausea and vomiting and was found dead the next morning.

The gastrointestinal toxidrome is usually mild and self-limited. In our study one of the deaths was a 61-year-old man who died after ingesting the milky latex of *Euphorbia antiquorumis*. The patient mistook it as an herb for treating asthma. He developed severe hemorrhagic gastroenteritis and then developed shock. Emergency surgery revealed gangrene of the small and large bowel.

Of the 2 deaths related to unidentified plants, the first was a 62-year-old-man with underlying kidney disease who had taken the leaves of plant to enhance his

Table 4
Number and death rate by type of plant poisoning.

Common name	Scientific name	No. of cases (%)	No. of deaths (%)
Group 1. Gastrointestinal toxidrome		**2,079 (72.0)**	**1 (0.05)**
Physic nut	*Jatropha curcas*	1,563 (54.1)	0
Sandbox tree	*Hura crepitans*	118 (4.1)	0
Coral plant	*Jatropha multifida*	81 (2.8)	0
Japanese taro	*Colocasia esculenta*	62 (2.1)	0
Dumb cane	*Dieffenbachia* spp	35 (1.2)	0
Chinese taro	*Alocasia cucullata*	32 (1.1)	0
Other 70 plants		189 (6.5)	1 (0.5)
Group 2. Neurotoxic toxidrome		**280 (9.7)**	**7 (2.5)**
2.1 Spinal seizue		**11 (0.4)**	**1 (9.1)**
Nux vomica	*Strychnos nux-vomica*	11 (0.4)	1 (9.1)
2.2 Seizures, encephalopathy		**269 (9.3)**	**6 (2.2)**
Wild yam	*Dioscorea hispida*	252 (8.7)	3 (1.2)
None	*Cnestis palala*	13 (0.5)	0
None	*Urobotrya siamensis*	2 (0.07)	2 (100)
Graceful gelsemium	*Gelsemium elegans*	1 (0.03)	0
None	Unknown species	1 (0.03)	1 (100)
Group 3. Anticholinergic toxidrome		**120 (4.2)**	**0**
Jimson weed	*Datura* spp	120 (4.2)	0
Group 4. Cardiovascular toxidrome		**37 (1.3)**	**1 (2.7)**
Saat, Phansaat	*Erythrophleum succirubrum*	32 (1.1)	1 (3.1)
Oleander	*Nerium oleander*	3 (0.1)	0
Desert Rose	*Adenium obesum*	2 (0.07)	0
Group 5. Multi-system organ failure toxidrome		**321 (11.1)**	**15 (4.7)**
5.1 Histotoxic		**206 (7.1)**	**8 (3.5)**
Cassava	*Manihot esculenta*	171 (5.9)	6 (3.5)
Yam bean	*Pachyrhizus erosus*	22 (0.7)	2 (9.1)
Derris	*Derris elliptica*	13 (0.5)	0
5.2 Cellular arrest		**115 (4.0)**	**7 (6.1)**
Rosary pea	*Abrus precatorius*	71 (2.5)	4 (5.6)
Castor bean	*Ricinus communis*	40 (1.4)	0
Climbing lily	*Gloriosa superba*	4 (0.1)	3 (75.0)
Group 6. Cutaneous/mucosal inflammation toxidrome		**24 (0.8)**	**0**
Indian spurge tree	*Euphorbia tirucalli*	9 (0.3)	0
Crown flower	*Calotropis gigantea*	6 (0.2)	0
Elephant ear	*Alocasia cucullata*	1 (0.03)	0
Other 6 plants		8 (0.3)	0
Group 7. Nephrotoxic toxidrome		**3 (0.1)**	**0**
Djenkol bean	*Archidendron pauciflorum*	3 (0.1)	0
Unidentified plants		**25 (0.9)**	**2 (8.0)**
Total cases		**2,889 (100.0)**	**26 (0.9)**

Table 5
Outcomes by severity of initial signs and symptoms.

Initial severity	Medical outcome No. of cases (%)					Total
	No effects	Minor	Moderate	Major	Death	
None	56 (68.3)	24 (29.3)	2 (2.4)	-	-	82 (2.8)
Minor	-	2,624 (99.3)	14 (0.5)	1 (0.04)	4 (0.2)	2,643 (91.5)
Moderate	-	5 (4.3)	106 (91.4)	1 (0.9)	4 (3.5)	116 (4.0)
Severe	-	-	2 (4.3)	28 (59.6)	17 (36.2)	47 (1.6)
Death	-	-	-	-	1 (100.0)	1 (0.03)
Total	56 (1.9)	2,653 (91.8)	124 (4.3)	30 (1.0)	26 (0.9)	2,889

renal function. He initially developed alteration in consciousness, jaundice and subsequently died from hepatotoxicity and encephalopathy. The other death from an unknown plant was a 54-year-old- man who ingested the roots of a wild plant. He developed shock with bloody stool, quickly deteriorated and died.

DISCUSSION

We report here poisonous plant exposures in Thailand reported to the RPC. The majority of exposures were unintentional exposures in children. This finding is consistent with previous studies from other regions (Vichova and Jahodar, 2003; Pietsch *et al*, 2008; Fuchs *et al*, 2011; Krenzelok and Mrvos, 2011; Plenert *et al*, 2012). Lack of knowledge and inability to differentiate between edible and poisonous plants are common factors leading to unintentional exposure (Krenzelok, 1995; Krenzelok and Mrvos, 2011). This probably explains why children especially in school age, were the most common victims. Education, supervision and providing a safe environment are all useful preventive measures.

Plant poisoning was the fifth most common type of poisoning reported to the RPC. The peak number of reported cases occurred in 2008 (600 cases) but the average after that was about 400 cases per year. This high number may be explained by the increasing planting of *Jatropha curcas* (physic nut) for biodiesel fuel since 2001 which was the most common poisonous plant reported in our study (Wood, 2006; Prueksakorna and Gheewalaa, 2008; Prueksakorna *et al*, 2010). Biofuels production has been promoted by the Thai government as renewable energy source to replace petroleum-based diesel. Even though palm oil is the major source for biodiesel fuel in Thailand, the physic nut has been recommended due to the ability to be grown in dry and marginal lands (Prueksakorna and Gheewalaa, 2008). The physic nut is planted just before the rainy season (April-May) and 6 to 10 months later it produces a fruit that can be harvested (Center of Excellence for the Jatropha, 2015). The planting and growing season of the physic nut is consistent with the peaks in poisoning cases found during June and November (Fig 2). This plant is cultivated near homes and schools (Chomchai *et al*, 2011). Its seedpods are attractive to curious children. Many people,

Table 6
Details about fatal cases.

Case No.	Gender	Age (Year)	Exposure type	Plant name	Toxidrome	Initial severity	Remarks
1	F	4	Accidental	*Manihot esculenta*	Multi-system organ failure	Minor	Initial symptoms were nausea and vomiting.
2	M	8	Accidental	*Manihot esculenta*	Multi-system organ failure	Moderate	
3	F	4	Accidental	*Manihot esculenta*	Multi-system organ failure	Severe	
4	M	3	Accidental	*Manihot esculenta*	Multi-system organ failure	Severe	
5	M	5	Accidental	*Manihot esculenta*	Multi-system organ failure	Severe	
6	M	4	Accidental	*Manihot esculenta*	Multi-system organ failure	Severe	
7	M	93	Accidental	*Pachyrhizus erosus*	Multi-system organ failure	Death	
8	M	2	Accidental	*Pachyrhizus erosus*	Multi-system organ failure	Severe	
9	M	5	Accidental	*Abrus precatorius*	Multi-system organ failure	Moderate	
10	M	4	Accidental	*Abrus precatorius*	Multi-system organ failure	Moderate	
11	M	5	Accidental	*Abrus precatorius*	Multi-system organ failure	Minor	Initial symptoms were nausea, vomiting and diarrhea.
12	M	6	Accidental	*Abrus precatorius*	Multi-system organ failure	Severe	
13	M	47	Folk remedy	*Gloriosa superba*	Multi-system organ failure	Severe	
14	M	30	Folk remedy	*Gloriosa superba*	Multi-system organ failure	Minor	Initial symptoms were nausea, vomiting and diarrhea.
15	M	25	Folk remedy	*Gloriosa superba*	Multi-system organ failure	Severe	
16	M	65	Folk remedy	*Strychnos nux-vomica*	Neurotoxicity	Severe	
17	F	60	Accidental	*Dioscorea hispida*	Neurotoxicity	Severe	
18	F	61	Accidental	*Dioscorea hispida*	Neurotoxicity	Severe	
19	F	66	Accidental	*Dioscorea hispida*	Neurotoxicity	Severe	
20	M	25	Accidental	*Urobotrya siamensis*	Neurotoxicity	Severe	Initial symptoms were nausea, vomiting and dizziness. The patient rapidly developed mental status changes, cardiovascular collapse and died 2-3 days after ingestion.

Table 6 (Continued).

Case No.	Gender	Age (Year)	Exposure type	Plant name	Toxidrome	Initial severity	Remarks
21	F	28	Accidental	*Urobotrya siamensis*	Neurotoxicity	Severe	
22	F	8	Accidental	*Cnestis palala*	Neurotoxicity	Minor	The patient presented with nausea, vomiting and diarrhea; then developed, seizures, respiratory failure and death.
23	M	8	Intention unknown	*Erythrophleum succirubrum*	Cardiovascular	Severe	
24	M	61	Misuse	*Euphorbia antiquorum*	Gastrointestinal	Severe	
25	M	54	Intention unknown	Unidentified	Unknown	Severe	Patient presented with hematochezia and shock.
26	M	62	Folk remedy	Unidentified	Unknown	Severe	Initial symptoms were altered conscious and jaundice.

especially children, are not aware of its toxicity and wrongly recognize it as edible seed (Devappa *et al*, 2010). Although the physic nut was the most common poisonous plant reported in this study, none of the reported cases were fatal. It contains curcin, which causes gastrointestinal symptoms of nausea, vomiting, abdominal pain and diarrhea (Chomchai *et al*, 2011).The toxidrome is mostly self-limited and treated symptomatically.

Our findings are similar to previous studies (Lin *et al*, 2009; Su and Lai, 2010).The toxidrome is useful for understanding and identifying the cause of poisoning. For example patients who present with alteration in consciousness, hypotension, severe metabolic acidosis and a normal arterial blood gas after taking an unknown plant. The histotoxic toxidrome, caused by plants such as *Manihot esculenta*, *Pachyrhizus erosus* and *Derris elliptica* should be suspected (Table 4). A detailed history and proper laboratory testing should be obtained for supporting the provisional diagnosis. Proper management can be implemented. These methods helped to make the diagnosis in 99.1% of reported plant poisonings in our study.

Most of the plant poisonings reported to the RPC had no to minor signs and symptoms (93.7%) with a low mortality rate (0.9%). This is consistent with previous studies from other regions which had low mortality rates ranging from 0% to 0.03% (Pietsch *et al*, 2008; Fuchs *et al*, 2011; Krenzelok and Mrvos, 2011; Plenert *et al*, 2012). The majority of poisonings in our study presented

with a gastrointestinal toxidrome (72.0%). Most of the deaths in our study had histotoxic, cellular arrest or neurotoxic toxidromes. Four deaths initially had mild clinical signs and symptoms. Plants with these toxidromes may have a delayed onset in signs and symptoms and need longer observation periods. Some may need specific treatment, such as using a cyanide antidote kit for cyanide toxicity from cassava ingestion, providing promt airway protection, sedation and neuromuscular blockade with strychnine poisoning and being aware of ventricular arrhythmias caused by digitalis-like plants poisoning. Many of the fatalities in this study were uncommon types of poisoning. Healthcare providers may not recognize the clinical picture or potential harm of these plants may not know how to take care of these cases. A poison center is helpful for providing useful information and assisting healthcare providers in making the diagnosis and taking care the poisoned patients. The poison center can provide education to healthcare providers and the general population about prevention, diagnosis and management of plant poisoning (Spiller and Griffith, 2009).

As a retrospective poison center data study, this study has limitations. First, the diagnosis was based on history of exposure, plant specimens, and clinical symptoms. There was no laboratory confirmation. Some of the identified plants and diagnoses may be incorrect. Second, the retrospective design may have incomplete or missing data. Third, the poison center data is from a voluntary reporting system. Some poisonous plant exposures are not reported to the poison center, especially cases which are misdiagnosed as being other poisonous or toxicological cases who present with mild clinical effects.

In conclusion, the majority of poisonous plant exposures reported to the RPC involved unintentional exposures among children and had a gastrointestinal toxidrome. The most common poisonous plant reported was *Jatropha curcas* (physic nut). Deaths were rare and commonly involved plants with histotoxic, neurotoxic or cellular arrest toxidromes. *Manihot esculenta* caused the greatest number of deaths. Knowing the toxidrome and accessing a poison center are useful for clinicians caring for these cases. The public and clinicians need to be educated about plant poisoning to improve prevention and treatment of plant poisoning.

ACKNOWLEDGEMENTS

The authors thank the Medicinal Plant Information Center, Faculty of Pharmacy, Mahidol University for the information and identification of the suspected plant samples. The authors declare no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

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