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ARTICLE

Common toxidromes of plant poisonings in Taiwan

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Objective. To describe the toxidromes associated with plant poisonings in Taiwan. *Methods*. Retrospective review of acute single-plant exposures with clinical signs and symptoms reported between January 1987 and December 2006 by hospitals to the network of Taiwan Poison Control Centers. Recorded data included demographic data, intent of exposures, exposure routes, clinical findings, and therapeutic strategies. *Results*. There were 389 cases that met the criteria. Each case was placed into one of the expected toxidromes: anticholinergic, mucosal inflammation, gastroenteritis, acute multisystem organ failure, delayed multisystem organ failure, cholinergic, cardiac dysrhythmia, hepatotoxicity, dermatitis, seizures, and dyspnea. Anticholinergic poisoning was the most common toxidrome. *Conclusion*. Plant poisonings can be classified into recognizable toxicologic syndromes. These toxidromes may guide a clinician's evaluation and management before a botanist can confirm the actual plant identity.

Keywords Plant; Epidemiology; Toxidrome; Poison control center

Introduction

Plant exposure is the seventh most commonly reported exposure to poison centers in the United States (1) and the third most frequent reported exposure to poison centers in the Germany (2). In Taiwan from 1985 to 1993, plant exposure was the 13th most common exposure leading to poison center contact (3). Most cases in Taiwan were unintentional and occurred in children under the age of 6 years. Because the concentration of toxin in most plants is low, intentional ingestions in adults, commonly with either suicidal intent or for abuse, are more likely to be associated with the development of signs and symptoms of poisoning.

The 23 million persons living in Taiwan are served by a network of four Poison Control Centers. We were interested in reviewing our plant exposure cases to determine the plants most commonly linked to the development of specific clinical syndromes and how the patients were managed.

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Methods

This study was performed with the support and acknowledgment of the Taiwan Department of Health. Prior to analyzing the data for this study, all patient-identifying data were removed to preserve confidentiality. We reviewed all exposures reported by healthcare providers to the four Poison Control Centers between January 1987 and December 2006. Only those cases with acute exposure to a single, identified plant, in which patients developed clinical signs and symptoms were included. The toxicologists decided whether or not the signs and symptoms were related to the plant based on standard clinical findings and the known toxicologic properties of the plant species. Cases involving consultations for information only, without exposures, were not included in this study. We recorded the name of the reporting hospital and identified the caller as a doctor, a nurse, or a pharmacist. We recorded demographic data, intent of exposure, exposure routes, clinical findings, and therapeutic strategies. The specific plant, quantity, and intent were based on the report of

the treating physician. Intent was either unintentional exposure (inadvertent, occupational, or herbal misuse) or intentional exposure (suicide, abuse, herbal errors, food or herbal adverse reaction). The clinical outcomes are based on the American Association of Poison Control Center National Poison Data System (1,3). We excluded cases of subacute (occurring repeatedly over several weeks or months) and chronic exposure (occurring repeatedly for many months or years), no clinical effect, possible causal effect, confirmed exposure with inadequate clinical information to determine its clinical outcome, unknown outcome, multiple plant exposure, or co-ingestants including alcohol (4).

Data analysis was performed using descriptive statistics. The signs and symptoms were then summarized according to the data recorded and adjudicated by the toxicologists. We classified our cases into one or more of the several recognized toxidromes derived from the plant poisoning literature (5).

Results

There were 1,414 cases related to plant poisoning during the 20-year period with 1,185 cases from the hospitals. There were 942 cases of acute exposure, of which 462 cases (49%) presented with signs and symptoms likely related to the exposure. After we excluded the cases with multiple plant exposure, or co-ingestants including alcohol, 389 cases remained. Of these, there were 182 males, 206 females, and 1 of unreported gender. The median age of the patients was 43 (range 0.5–83) years. There were 330 adults (85%), 25 children (<6 years), and 34 adolescents (7–18 years). Unintentional exposures accounted for 189 cases (49%; 147 adults, 42 adolescents and children), whereas intentional exposure accounted for 199 cases (51%; 182 adults, 17 adolescents and children); there was one case of unknown intent. Adults had more intentional exposures (55%), and the children and adolescents had more unintentional exposures (71%). The major route of exposure was oral (375 cases). There were 8 cases exposed by skin contact, 4 through the eyes, and 2 from unknown routes. There were 210 cases of mild toxicity, 153 cases of moderate toxicity, 23 cases of severe toxicity, and 3 deaths. The most common plants to which adults were exposed are mentioned in Table 1 (6–11). The most common plant exposures in children and adolescents are summarized in Table 2 (5–8,10,12,13). The toxidromes, related plants, and patient outcomes are mentioned in Table 3 (5–53). The outcomes of the cases mostly were mild or moderate. Anticholinergic toxidrome was the most common and *Datura* spp. were the most frequently implicated plants. Twenty-nine patients with *Datura* poisoning were treated with physostigmine; no other antidote was administered. The three fatalities were due to *Taxus sumatrana* (Miq.) de Laub. (two cases: hemolysis and multiple organ failure; acute myocardial infarction) and *Areca catechu* L (acute myocardial infarction).

Table 1. Ten most common poisonous plants in adults

Botanical name	Common name	Adults
Datura suaveolens Humb.	Angel's trumpet	62
Datura metel Linn.	Devil's trumpet, metel, downy thorn-apple	54
Alocasis macrorrhiza (L.) Schott and Endl.	Giant Elephant's Ear, Taro	33
Cycas revoluta Thumb.	Cycad, sago palm	19
Dysosma pleiantha (Hance.) Woodson.	Bajiaolian	21
Areca catechu L.	Betel	18
Nerium indicum Mill.	Oleander	9
Melia azedarach L.	Chinaberry, Paradise tree, pride of China, white cedar	9
Erycibe henryi Prain.	Ting Kung Teng	8
Aconitum carmichaeli Debx.	Aconite	7

Table 2. Ten most common poisonous plants in adolescents and children

Botanical name	Common name	Children	adolescents	Total
Alocasis macrorrhiza (L.) Schott and Endl.	Giant Elephant's Ear, Taro	4	7	11
Aleurites fordii Hemsl.	Tung Nut, Tung Oil Tree, Chinawood Oil Tree	0	7	7
Datura suaveolens Humb.	Angel's trumpet	2	4	6
Datura metel Linn.	Devil's trumpet, metel, downy thorn-apple	3	2	5
Cycas revoluta Thumb.	Cycad, sago palm	1	4	5
Dieffenbachia spp.	Dieffenbachia, Dumbcane	3	1	4
Lantana camara L.	Lantana	2	1	3
Hura crepitans L.	White cedar, sandbox tree	0	3	3
Areca catechu L.	Betel	2	0	2

Discussion

It is important to recognize clinical toxidromes associated with plant poisonings because typically the involved plant is not brought to the clinician by the patient (the plant may have been consumed completely or discarded). Often only the common name can be provided by the patient. Because a specific plant often has several common names and because a common name may be applied to several unrelated plants, or be extended across an entire genus, the utility of this information

Table 3. Toxidromes and outcomes of related plants.

Toxidrome	Toxic mechanism	Botanical name	Common name	Toxin	Outcome (No. patients)				Mechanism total (no. pt)	Toxidrome total (no. pt)
					Mild	Moderate	Severe	Fatal		
Anticholinergic	Parasympatholytic	*Datura suaveolens* Humb.	Angel's trumpet (6)	Atropine, scopolamine and other anticholinergic alkaloids (4, 13, 14)	15	47	5	0	67	136
		Datura metel Linn.	Devil's trumpet, metel, downy thorn-apple (5)		17	43	0	0	60	
		Datura tatula Linn.	Purple Thorn Apple (15)		0	4	1	0	5	
		Solanum aculeatissimum Jacquin.	Deadly nightshade, potato (6)	Solanine and anticholinergic alkaloids (5, 17)	1	1	0	0	2	
		Solanum nigrum L.	Black nightshade, potato (6)		0	1	0	0	1	
		Solanum verbascifolium L.	Deadly nightshade, potato (6)		0	1	0	0	1	
Mucosal Inflammation	Mucosal irritant	*Alocasia macrorrhiza* (L.) Schott and Endl.	Giant Elephant's Ear, Taro (6, 7)	Calcium oxalate (4, 7, 17)	37	7	0	0	44	55
		Dieffenbachia aculata (Lodd.) Sweet.	Dieffenbachia, Dumbcane (6)		4	1	0	0	5	
		Dieffenbachia amoena cv. 'Bull'.	Dieffenbachia, Dumbcane (6)		4	0	0	0	4	
		Alocasia cucullata Schott & Endl.	No		0	1	0	0	1	
		Caladium x hortulanun Birdsey.	Caladium (6)		1	0	0	0	1	
Gastroenteritis	Gastrointestinal irritant	*Melia azedarach* L.	Chinaberry, Paradise tree, pride of China, white cedar (6)	Tetranortriterpene (4, 18)	6	3	0	0	9	50
		Aleurites fordii Hemsl.	Tung Nut, Tung Oil Tree, Chinawood Oil Tree (4, 11)	Unknown (4, 11)	7	0	0	0	7	
		Narcissus tazetta var.chinensis	Daffodil, paper white narcissus (6)	Lycorine and related phenanthridine (4)	5	1	0	0	6	
		Pachyrrhizus erosus Linn.	No	Rotenone (4)	4	2	0	0	6	
		Derris trifoliata Lour.	No	Rotenone (19)	4	2	0	0	6	

(*Continued*)

Table 3. (*Continued*)

Toxidrome	Toxic mechanism	Botanical name	Common name	Toxin	Outcome (No. patients)				Mechanism total (no. pt)	Toxidrome total (no. pt)
					Mild	Moderate	Severe	Fatal		
		Croton tiglium L.	Croton (9)	Phobol ester (20)	3	1	0	0	4	
		Lantana camara L.	Lantana (6)	Unknown	1	2	0	0	3	
		Hura crepitans L.	White cedar (6), sandbox tree (12)	Hurin (4)	3	0	0	0	3	
		Aloe vera L.var.chinensis ⟨Haw.⟩ Berger	Aloe vera (6)	Barbaloin (4)	2	0	0	0	2	
		Daphne genkwa Sieb. Et Zucc.	Lilac daphne (5)	Mezerein (21)	0	1	0	0	1	
		Ricinus communis Linn.	Castor bean (6)	Ricin (4)	0	1	0	0	1	
		Hippeastrum hybridum Hort.	Amaryllis (5)	Lycorine and related phenanthridine (4, 22)	1	0	0	0	1	
		Phytolacca acinosa Roxb.	No	Phytolaccatoxin (4)	1	0	0	0	1	
Acute multisystem organ failure	Cyanide poisoning	*Cycas revoluta* Thumb.	Cycad, sago palm (5)	Cycasin and neocycasin (4, 23)	23	1	0	0	24	30
		Manihot esculenta Crantz.	Cassava (6), Manihot, tapioca (9)	Linamarin and lotaustralin (4, 24, 25)	4	1	0	0	5	
		Hydrangea macrophylla (Thunb.) Ser.	No	Hydrangin (4)	1	0	0	0	1	
Delayed multisystem organ failure	Mitotic inhibitors	*Dysosma pleiantha* (Hance.) Woodson.	Bajiaolian (8)	Podophyllotoxin (8)	3	6	12	0	21	30
		Catharanthus roseus (L.) G. Doy.	Rose periwinkle (6)	Vinca alkaloids (4)	3	1	0	0	4	
	Cytotoxicity	*Tripterygium wilfordii* Hook. f.	Lei Gong Teng (26)	Unknown	3	2	0	0	5	
Cholinergic	Parasympathomimetic	*Areca catechu* L.	Betel (9)	Arecholine (9)	11	6	2	1	20	28
		Erycibe henryi Prain.	Ting Kung Teng (10)	Tropane alkaloids (10)	4	4	0	0	8	
Cardiac dysrhythmia	cardiac glycoside	*Nerium indicum* Mill.	Oleander (6)	Oleandrin and neriine (4, 27)	6	3	0	0	9	24
		Cerbera manghas L.	pink-eyed cerbera, Sea Mango (22, 28, 29)	Cerberoside (22)	1	2	0	0	3	
		Adenium obesum (Forsk.) Balf. Ex Roem. Et Schult.	No	Convallatoxin (30)	0	1	0	0	1	
		Digitalis purpurea L.	Purple foxglove (9)	Digitoxin (9, 31)	0	1	0	0	1	

	Sodium channel agonists	*Aconitum carmichaeli* Debx.	Aconite (6)	Aconitine and related alkaloids (4, 32)	3	2	2	0	7	
		Rhododendron spp.	Azalea (6)	Grayanotoxins (4, 33)	0	1	0	0	1	
	Sodium and calcium channel blocker	*Taxus sumatrana* (Miq.) de Laub.	Yew (6)	Taxine alkaloids (4, 34–37)	0	0	0	2	2	
Hepatotoxicity	Hepatotoxicity	*Lycopodium serratum* Thunb. var. longipetiolatum*(38)	No	Unknown	6	1	0	0	7	15
		Breynia officinalis Hemsl.	Chi R Yun (39, 40)	Unknown (39, 40)	2	1	1	0	4	
		Crinum asiaticum L. (41)	No	Unknown	3	0	0	0	3	
		Polygonum multiflorum Thunb.*(42)	He Shou Wu (42)	Unknown	1	0	0	0	1	
Dermatitis	Irritation or Allergy	*Euphorbia tircucalli* L.	Pencil tree, Milkbush, Finger tree, Rubber euphorbia (5)	Diterpene esters (4)	6	0	0	0	6	14
		Euphorbia pulcherrima Willd. et. Klotz.	Poinsettia, Christmas flower (5)	Diterpene esters (4, 43)	2	0	0	0	2	
		Urtica thunbergiana sieb. (44)	Nettle (44)	Histamine, serotonin, substance P, oxalic acid and tartaric acid (44, 45)	2	0	0	0	2	
		Rhus verniciflua Stokes.	No	Unknown (4, 46)	2	0	0	0	2	
		Hedera helix L.	Oakleaf ivy, Common ivy (4, 6, 9, 49)	Hederin, falcarinol (4, 47)	1	0	0	0	1	
		Euphorbia milii Ch. des Moulins.	Crown of thorns (6)	Diterpene esters (4)	1	0	0	0	1	
Convulsion/Seizure	Block the inhibitory effects of GABA	*Strychnos nuxvomica* L.§	Strychnine (6)	Strychnine (4, 48)	3	0	0	0	3	5
	An antagonist of pyridoxine and gastrointestinal irritant	*Ginkgo biloba* Linn. §	Ginko (6)	4-O-methylpyridoxne (4, 49, 50)	2	0	0	0	2	
Dyspnea	Unknown	*Sauropus Androgynus* (L.) Merr.	Sauropus albicansAsin-Asin Chekor Manis (51)	Unknown (51)	1	1	0	0	2	
Total numbers of patients					210	153	23	3	389	389

*Only mild signs and symptoms of gastroenteritis in our case series without hepatotoxicity recorded.
§Only mild signs and symptoms of gastroenteritis in our case series without seizures recorded.

is limited and possibly misleading. Healthcare providers seldom were able to identify the plants without the help of the Poison Control Centers or botanists.

In many situations, the emergency physician must manage a clinically ill patient before definitive identification of the plant is available. Typically, these patients are managed based on the clinical condition rather than on the knowledge of an exposure or suspicion of a toxin (5). We classified our clinically ill patients into one of the commonly described plant-related toxidromes. Understanding this classification is particularly useful when the toxidrome manifested by the patient is not compatible with the common name of the plant described by the patient. Categorizing a patient's clinical findings into one of these plant-related toxidromes may allow proper therapy to be administered despite specific insight into the exposure.

Anticholinergic syndrome was the most common encountered plant poisoning in hospitals, accounting for 136 cases. Many plants may cause this syndrome, including plants of the genus *Atropa*, *Brugmansia*, *Datura*, *Hyoscyamus*, *Solandra*, and *Solanum* (5). In our adult patients, *Datura suaveolens* Humb. was the most commonly encountered and shown in Table 1.

Mucosal inflammation following plant exposure is caused commonly by the local irritation of calcium oxalate crystals that are released in proximity to the mucosa on chewing. In our study, 55 such cases were identified in Table 3 (5,8,18). *Alocasia macrorrhiza* (L.) Schott and Endl. was the leading plant poisoning in adolescents and children as shown in Table 2. Our patient who consumed the root of this plant presented with signs and symptoms of calcium oxalate poisoning with mucosal inflammation.

Gastrointestinal irritation (nausea, vomiting, diarrhea, or abdominal pain) occurred in 50 cases in our series due to 13 distinct species of plants noted in Table 3.

There were 30 cases of acute multisystem organ failure, caused mainly by plants that contain cyanogenic poisons.

Delayed multisystem organ failure occurred in 30 cases. Plants that contain colchicine-like mitotic inhibitors may induce gastrointestinal symptoms, abdominal pain, diarrhea, peripheral neuropathy, bone marrow suppression, and cardiovascular collapse. *Tripterygium wilfordii* Hook. F., containing an unidentified toxin, causes profuse vomiting and diarrhea, leukopenia, renal failure, profound hypotension, shock, and cardiac toxicity (54).

Cholinergic symptoms may also develop in patients with plant poisoning. *Erycibe henryi* Prain. produces muscarinic clinical effects. Nicotinic clinical findings may be produced by betel nut, *Areca catechu* L. (10,11,55). There were 20 cases of poisoning by *Erycibe henryi* Prain. and 8 cases of *Areca catechu* L. poisonings in our series (one of whom died). Alkaloids in betel nut, such as arecoline, may play a contributing role in coronary artery vasoconstriction due to sympathomimetic effects on vessels with abnormal endothelium in a manner analogous to nicotine (55).

Cardiac toxicity may be due to effect of cardiac glycoside, sodium channel agonists, and sodium and calcium channel blocker. In our series, there were 24 cases of plant poisonings with cardiac toxicity. There were 8 cases of *Aconitum carmichaeli* Debx. and *Rhododendron* spp. associated with sodium channel agonists. These patients developed cardiac dysrhythmias in addition to burning in the mouth, salivation, vomiting, diarrhea, and a tingling sensation in the skin (5,33). There were two deaths caused by *Taxus sumatrana* (Miq.) de Laub., which results in sinus bradycardia, premature ventricular contractions, atrioventricular conduction defects, or ventricular tachydysrhythmias (5,35–38). In the two *Taxus* fatalities, one died from hemolysis and multiple organ failure and the other from acute myocardial infarction.

Breynia officinalis Hemsl., *Lycopodium serratum* Thunb. var. longipetiolatum, *Polygonum multiflorum* Thunb., and *Crinum asiaticum* L. are reported to cause hepatotoxicity (39–43). *Breynia officinalis* Hemsl. resulted in hepatotoxicity in four cases; the other plants caused mild gastrointestinal irritation.

Dermatitis (allergic or irritant) is common after contact with certain plants. Ingestion of these same plants can often result in gastroenteritis. There were 14 cases manifesting dermatitis. *Hedera helix* L. usually causes allergic contact dermatitis (47). Both *Urtica thunbergiana* sieb. and *Rhus verniciflua* Stokes. produced itching and contact dermatitis, similar to other more widely reported species of the same genus (5,45–47).

In our case series, we had five patients poisoned by the *Strychnos nux-vomica* L. and *Ginkgo biloba* Linn. Strychnine interferes with the negative feedback function of glycine, resulting in excessive motor neuron activity (56,57). The toxicity of *Ginkgo biloba* Linn. comes from 4-O-methylpyridoxine, a competitive antagonist of pyridoxine, which is needed for GABA synthesis (50,51). However, our cases had only mild gastrointestinal irritation without seizures.

Chronic use of *Sauropus Androgynus* (L.) Merr. has caused constrictive bronchiolitis obliterans (52). Our three cases were single exposures and none developed clinical toxicity.

Conclusions

Anticholinergic plants are the primary cause of clinical poisoning from plants in adults, and mucosal irritants are primarily responsible for clinical effects in children following plant exposure. Plants produce predictable syndromes that can help clinicians in the absence of details about the specific exposure. If the plant implicated in an exposure cannot be specifically identified in a rapid fashion, the poisoning syndromes may help the clinician evaluate and manage the poisoned patients.

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