Human Plant Exposures Reported to a Regional (Southwestern) Poison Control Center Over 8 Years

Ben Enfield¹ · Daniel E. Brooks^{2,3,4} · Sharyn Welch^{2,3} · Maureen Roland^{2,3} · Jane Klemens^{2,3} · Kim Greenlief^{2,3} · Rachel Olson^{2,3} · Richard D. Gerkin⁵

Received: 24 July 2017 / Revised: 21 November 2017 / Accepted: 28 November 2017 / Published online: 12 January 2018

© American College of Medical Toxicology 2018

Abstract

Background There is little published data about human plant exposures reported to US poison control centers (PCCs).

Methods A retrospective chart review of all reported plant exposures to a single regional PCC between January 1, 2003 and December 31, 2010 was done to understand better the characteristics of plant exposure cases. Specific generic plant codes were used to identify cases. Recorded variables included patient demographics, plant involved, exposure variables, symptoms, management site, treatments, and outcome. Univariate and multivariate regression was used to identify outcome predictors.

Results A total of 6492 charts met inclusion criteria. The average age was 16.6 years (2 months–94 years); 52.4% were male. The most common exposure reason was unintentional (98%), and the majority (92.4%) occurred at the patient's home. Ingestions (58.3%) and dermal exposures (34.3%) accounted for most cases. Cactus (27.5%), oleander (12.5%), *Lantana* (5.7%), and *Bougainvillea* (3.8%) were most commonly involved. Symptoms developed in 47.1% of patients, and were more likely to occur following *Datura* (66.7%), and Morning Glory or Milkweed (25% each) exposures. Almost 94% of patients were managed onsite (home) and only 5.2% involved evaluation in a health care facility (HCF). Only 37 (0.6%) patients required hospital admission, and 2.9% of cases resulted in more than minimal effects. Exposures resulting in more than minimal clinical effects were predicted by several variables: abnormal vital signs (OR = 35.62), abnormal labs (OR = 14.87), and management at a HCF (OR = 7.37). Hospital admissions were increased for patients already at a HCF (OR = 54.01), abnormal vital signs (OR = 23.28), and intentional exposures (OR = 14.7).

Conclusion Plant exposures reported to our poison control center were typically unintentional ingestions occurring at home. Most patients were managed onsite and few developed significant symptoms.

Keywords Poison center · Plants · Toxicity

Electronic supplementary material The online version of this article (<https://doi.org/10.1007/s13181-017-0643-3>) contains supplementary material, which is available to authorized users.

✉ Daniel E. Brooks
daniel.brooks@bannerhealth.com

¹ University of Arizona College of Medicine-Phoenix, Phoenix, AZ, USA

² Banner Poison and Drug Information Center, Phoenix, AZ, USA

³ Center for Toxicology and Pharmacology Education and Research, Phoenix, AZ, USA

⁴ Department of Medical Toxicology, Banner University Medical Center-Phoenix, 1012 East Willetta Road, 2nd Floor, Phoenix, AZ 85006, USA

⁵ Department of Internal Medicine, Banner University Medical Center-Phoenix, Phoenix, AZ, USA

Background

In 2015, there were 46,597 human plant exposures reported to United States (US) poison control centers (PCCs) which represented 1.8% of all exposure calls [1]. This made plants the 21st most common substance category involved in human exposures, and approximately (~) 60% ($n = 28,213$) of these calls involved pediatric patients under the age of 6 years. Unfortunately, there is a common misperception that all plant exposures are benign, despite educational efforts aimed at medical professionals [2–5].

There is, however, little published data about plant calls to PCCs and none for the state of Arizona [6–17]. Although the proportion of plant-related calls to PCCs has decreased over the last 25 years, the continued frequency of these cases

represents a strain on the health care system. A better understanding of outcomes following plant ingestions may improve patient management and prevent unnecessary evaluations in a health care facility (HCF). With this goal, a review of the records from an urban PCC, in the southwestern USA, was performed to better understand the characteristics of human plant exposures.

Methods

A retrospective review was performed on all human plant exposure calls to a single, urban PCC between January 1, 2003 and December 31, 2010. All charts within the PCC's electronic medical record system (EMR) were searched using Visual Dot Lab Enterprise® (version 4.6.2; 2017). Plant-specific generic (numerical) codes, established by the American Association of Poison Control Centers' (AAPCC) National Poison Data System (NPDS), were used to identify cases. A total of 16 NPDS codes (e.g., 0088000 for “amygdalin and/or cyanogenic glycosides”) were used to search the EMR for all plant calls during the 8-year study period. Calls unrelated to human exposures were excluded.

All identified cases were manually reviewed by trained researchers who recorded information from the charts into a standardized electronic data abstraction form using Microsoft Excel® (97-2003 worksheet). Recorded variables included patient demographics (gender and age), plant(s) involved, NPDS code, exposure variables (location, route, and reason of exposure), symptoms, management site, treatments, and outcome. Clinical effects and patient outcomes were recorded by using established NPDS coding parameters (NPDS Coding User's Manual© version 3.1; approved May 7, 2014).

Descriptive statistics were used on continuous variables and reported as means with standard deviation and range. Individual analyses of categorical variables are reported with percentages based on the total number of cases with complete data. Univariate and multivariate regression was used to identify outcome predictors. Univariate predictors with $p < 0.10$ were entered into multiple regression using the forward LR method (IBM SPSS for MAC v.24.0; Armonk, NY) for analysis.

A total of 79 (1.2%) cases were coded as “unable to follow” but contained sufficient outcome data to be included in analysis. Individual data points (e.g., gender) were missing for some charts. For missing data, we used case-wise deletion for all variables/outcome analyses.

A random sample of 100 (1.5%) charts was reviewed by a second investigator to ensure accuracy of data abstraction. This re-analysis found a recording accuracy of 98.4% for 1600 reviewed data points. This study received approval from our institution's investigational review board.

Results

The PCC received 823,489 total calls between January 1, 2003 and December 31, 2010; of these, 6492 (0.79%) met inclusion criteria for human plant exposures. The average patient age was 16.6 years (SD 21.7 years; range 2 months–94 years) and most (52.4%) were male. Almost 93% of the exposures occurred at a home, either the patient's (92.4%) or another (0.4%) residence. The locations for all plant exposures are reported in Table 1.

The ten most commonly involved plants are listed in Table 2 and included cactus (27.5%), oleander (12.5%), *Lantana* (5.7%), *Bougainvillea* (3.8%), and *Philodendron* (2.2%); 3.2% of cases involved an unknown plant. A complete list of all reported plant types/species with corresponding frequencies is available in the supplemental file (Appendix 1).

The most common route of exposure was ingestion and accounted for 58.3% of all cases. Dermal (34.3%), ocular (5.7%), inhalational (0.6%), parenteral (0.01%; $n = 1$), and combined routes of exposure were also recorded (Table 3). The most common reason for exposure was unintentional (98%). All exposure reasons are listed in Table 4.

A total of 3168 (48.4%) charts had clinical signs and/or symptoms coded within the EMR, but only 3059 (47.1% of all cases) were attributed to the plant exposure. The most commonly reported (attributable) signs/symptoms were “puncture” ($n = 1489$), “throat” or “tongue irritation” ($n = 753$), “rash/redness” ($n = 634$), “vomiting” ($n = 437$), and “ocular” irritation ($n = 203$).

All coded effects (patient outcomes) are reported in Table 5. Only 188 (2.9%) cases resulted in greater than minimal clinical effects: moderate ($n = 179$) and major ($n = 9$). The nine (0.1%) coded major effects including one death (described below) and the following two cases. Case 1: a 32-year-old suffered an anaphylactic reaction to Jimsonweed (*Datura stramonium*) while gardening. The patient was admitted to intensive care (ICU) for airway edema and respiratory distress but made a complete recovery with pharmacotherapy. Case 2: a 43-year-old developed bradycardia and hypotension after a suicidal ingestion of tea brewed from oleander leaves/fruit. The patient had nausea, vomiting, and diarrhea; bradycardia (heart rate in the 40s) and hypotension (systolic pressure in the

Table 1 Exposure location for human plant cases reported to a PCC

Location	Number of calls (percent of total)
Own residence	5995 (92.4%)
School	284 (4.4%)
Public place	164 (2.5%)
Other residence	28 (0.4%)
Workplace	17 (0.3%)

Total calls with coded data, $n = 6488$ (99.94%)

Table 2 Most common plants involved in human exposures reported to a PCC

Rank	Plant/species	Percent of total calls
1	Cactus	27.5
2	<i>Oleander</i> spp.	12.5
3	<i>Lantana</i> spp.	5.7
4	<i>Bougainvillea</i> spp.	3.8
5	Unknown	3.2
6	<i>Philodendron</i> spp.	2.2
7	Pothos (<i>Epipremnum</i> spp.)	1.9
8	Poinsettia	1.7
9	<i>Pyracantha</i> spp.	1.7
10	<i>Ficus</i> spp.	1.6

A complete list of all involved plants and corresponding frequencies is available in Appendix 1

90s); and a (false-positive) serum digoxin concentration (peak 0.9 ng/mL) and normal serum potassium ($K^+ = 4.3$ mEq/L). After an ICU admission, the patient made a complete recovery in 48 h without requiring digoxin-specific antibody.

There was one recorded death during the 8-year study period involving a 34-year-old patient with a witnessed (intentional) yohimbe ingestion. After the ED physician described the patient to PCC staff, a salicylate level (and appropriate treatment) was recommended. The initial salicylate concentration was 115 mg/dL; despite appropriate interventions, the patient died within 4 h of presentation. The exact role that yohimbe played in this case was unknown, but the death was primarily attributed to acute salicylate toxicity.

Symptoms were most likely to occur following *Datura* (66.7%), Morning Glory or Milkweed (25% each), and Pencil cactus (18.2%) exposures. Interestingly, only 2.6% (25 out of 927) of all other (non-pencil) cacti exposures developed symptoms (see Table 6).

Abnormal vital signs (related to the exposure) were coded in only 18 (0.3%) cases. These included tachycardia ($n = 11$),

Table 3 Routes of human plant cases reported to a PCC

Route of exposure	Number of calls (percent of total)
Ingestion	3786 (58.3%)
Dermal	2223 (34.3%)
Ocular	367 (5.7%)
Dermal and other ^a	61 (0.9%)
Inhalation	42 (0.6%)
Inhalational and other ^a	5 (0.07%)
Other ^a	4 (0.06%)
Parenteral	1 (0.01%)

Total calls with coded data, $n = 6489$ (99.95%)

^a Other includes nasal, ear, intra-oral (but not ingested), and mucous membranes

Table 4 Reason for plant exposures reported to a PCC

Reason for exposure	Number of calls (percent of total)
Unintentional (accidental)	6362 (98%)
Misuse/self-harm	75 (1.2%)
Recreational	41 (0.6%)
Unknown	6 (0.1%)

Total calls with coded data, $n = 6484$ (99.87%)

bradycardia ($n = 4$), hypotension ($n = 4$), hyperthermia ($n = 3$), and hypertension or tachypnea ($n = 1$ each). Laboratory abnormalities were coded in 15 (0.2%) of all cases. Lab anomalies, attributable to the plant exposure, included rhabdomyolysis ($n = 6$), measureable blood digoxin concentration ($n = 6$), positive urine drug screen ($n = 3$), acidosis or hyperglycemia ($n = 2$ each), and one abnormal EKG (incomplete right bundle-branch block).

The most common treatments were providing fluids (57.7% of cases), local wound care (31.1%), and ocular irrigation (4.4%). The vast majority (93.8%) of patients were managed (received treatments) onsite, which was usually the patient's home, and only 5.2% involved evaluation in a HCF (Table 7). Only 37 (0.6%) patients required hospital admission. All admitted patients made complete recoveries other than the one death described above.

Multiple logistic regressions were used to identify independent predictors for two outcomes [1]: development of moderate/major clinical effects (compared to minimal or no effects) and [2] need for hospital admission. The odds ratios (ORs) for these variables were determined based on unadjusted, univariate analysis for each outcome (Tables 8 and 9, respectively) and reported with 95% confidence intervals (CIs).

The development of more severe (moderate or major) clinical effects, compared to minor effects, was associated with several variables. These included abnormal vital signs, OR = 35.62 (95% CI 7.69–165.1); abnormal laboratory data, OR = 14.87

Table 5 Outcomes (clinical effects) for plant exposures reported to a PCC

Patient outcome (clinical effect)	Number of calls (percent of total)
Non-toxic exposure	741 (11.4%)
No effect	1246 (19.2%)
Minor	360 (5.5%)
Moderate	179 (2.8%)
Major	9 (0.1%)
Death (judged as unrelated to plant exposure)	1
Unrelated (effects not related to exposure)	194 (3.0%)
Unable to follow (lost to follow-up)	79 (1.2%)

Total calls with coded data, $n = 6489$ (99.95%)

Table 6 Plant exposures resulting in moderate or major symptoms

Plant type/species	Patients with moderate/major symptoms (case/total number of calls)	Percentages
<i>Datura</i> spp.	14/21	66.7%
Morning Glory	2/8	25%
Milkweed	2/8	25%
Pencil Cactus	48/263	18.2%
Aloe	4/39	9.3%
Mexican Bird of Paradise	4/46	8%
Palm	4/89	4.3%
Poison Ivy	2/46	4.2%
Oleander	29/762	3.7%
Other cactus (NOS)	25/927	2.6%

(95% CI 3.42–64.7); management site (at a HCF prior to PCC contacted), OR = 7.37 (95% CI 5.08–10.7); and age, with an increased OR = 1.02 for each increased year of age (95% CI 1.01–1.03). Interestingly, patients with inhalation or ingestion exposures were less likely to develop >minor effects (OR = 0.56; 95% CI 0.37–0.87) compared to all other exposure routes.

Hospital admission was increased for several variables, including management site (HCF compared to a non-HCF), OR = 54.01 (95% CI 17.3–168.2); abnormal vital signs, OR = 23.28 (95% CI 4.94–109.6); and intentional or recreational exposures, OR = 14.7 (95% CI 5.97–36.2).

Discussion

All PCCs answer calls 24 hours each day from the public and medical professionals concerning exposures to (or questions about) medications, chemicals, and environmental agents (e.g., plants and venomous creatures). Poison centers are staffed by specialists in poisoning information (nurses or pharmacists) and poison information providers with specialized training (and sometimes national certification) in poisoning emergencies. Since staff make triage decisions based on telephonic (unverifiable and often limited) information, a better understanding of epidemiological outcomes for specific call types may assist with optimal treatment recommendations.

Table 7 Patient management site following plant exposures

Management site	Number of calls (percent of total)
Onsite (site of exposure, e.g., home)	6088 (93.8%)
Health care facility	336 (5.2%)
Other (e.g., school, work, and public place)	37 (0.6%)

Total calls with coded data, $n = 6489$ (99.95%)

Table 8 Multiple regression: developing > minor clinical effects

Variable	Odds ratio (95% CI)
Abnormal vital signs (attributed to the exposure)	35.62 (7.69–165.1)
Abnormal laboratory data (attributed to the exposure)	14.87 (3.42–64.7)
Management site (in a HCF compared to non-HCF sites)	7.37 (5.08–10.7)
Age (increase/year)	1.02 (1.01–1.03)
Route of exposure (inhalation/ingestion vs. all other routes)	0.56 (0.37–0.87)

All values are statistically significant, $p < 0.001$

Several previous studies have reviewed PCC databases to describe patient outcomes following plant exposures. These data showed that most exposures result in minimal effects, and that children are more likely to be involved in serious clinical effects, including death (e.g., a pokeweed-related death in 2008) [7, 14, 15]. A Czech Republic study found that 50% of plant exposures resulted in symptoms, most commonly gastrointestinal (nausea, emesis, or diarrhea) or neurologic (non-specific; up to 33%) [13]. While serious adverse effects may be rare, it is important to educate the public about potentially toxic plants (based on geographic area) in hopes of preventing exposures and adverse effects. Likewise, health care providers also require knowledge about the implications of plant exposures.

Previous studies have shown a lack of plant knowledge among emergency department (ED) physicians [18, 19]. For example, 56 physicians were asked to identify 12 common household plants, but only 17% of plants were identified by name, and fewer (13%) were correctly identified based on toxicity [18].

Arizona's desert climate and increased access to irrigation water has prompted the growth of both endemic (e.g., cactus) and transplanted (e.g., oleander) species, including potentially poisonous varieties. And many toxic species (e.g., oleander and Jimsonweed) are more commonly found in Arizona than in other areas of the USA. This fact, combined with a rapidly growing urban population, may explain the differences

Table 9 Multiple regression: hospital admission

Variable	Odds ratio (95% CI)
Management site (in a HCF compared to non-HCF sites)	54.01 (17.3–168.2)
Abnormal vital signs (attributed to the exposure)	23.28 (4.94–109.6)
Reason for exposure [intentional (recreational/self-harm) compared to unintentional and all other exposures]	14.7 (5.97–36.2)

All values are statistically significant, $p < 0.001$

between our (regional) findings and national results. On a national level, unknown (plant/botanical name), *Phytolacca*, toxicodendrol-containing (skin irritants), and Cherry (cyanogenic) *species* were the most common categories of plants involved in human exposures [1]. The larger percentage of unknowns among the national data is probably due to increased plant diversity but may also have been affected by differences in coding between PCCs.

Knowing the most common plant exposures for the southwestern region of the USA may provide opportunities for preventing unintentional exposures. Since PCCs and toxicologist are tasked with providing appropriate medical advice, while preventing unnecessary medical evaluations, it is important to understand the potential outcomes of specific plant exposures. This knowledge may also affect how medical toxicologists assist with patient disposition following ED presentation. These data suggest that several variables might be monitored when attempting to predict the development of significant clinical effects (i.e., abnormal vital signs and laboratory results) or need for hospital admission (i.e., intentional exposures, abnormal vital signs, and need for HCF management). Several of the reported ORs were limited by wide confidence intervals (due to small sample sizes). Finally, the significant ORs indicate association, and not necessarily causation.

There are limitations with all retrospective PCC-based studies, including the potential for reporting bias (e.g., only being called about symptomatic patients) and accuracy of reported and coded data. Unfortunately, the information coded in our charts could not be independently verified. Also, abstractors were not blinded to the purpose of this work and may have interpreted data in a biased manner. Some calls were excluded due to callers refusing our recommendations (not going to an ED) or follow-up attempts; other calls were simply lost to follow-up. The outcomes of these cases were unknown and may have affected findings. Inevitably, some plant exposures were not reported to our PCC, including some that involved evaluation at a HCF, and thus our findings underestimate the real number of plant exposures managed at home or by healthcare providers.

Conclusion

Human plant exposures reported to our southwestern US poison center were typically unintentional ingestions occurring at home. The majority of patients were managed onsite and few developed more than minor symptoms. The development of significant clinical effects and need for hospital admission were each associated with several variables.

Compliance with Ethical Standards

Conflicts of Interest None.

Sources of Funding There was no direct grant support for this work.

References

1. Mowry JB, Spyker D, Brooks DE, et al. 2015 annual report of the American Association of Poison Control Centers National Poison Data System (NPDS): 33rd annual report. *Clin Toxicol.* 2016;54(10):924–1109. <https://doi.org/10.1080/15563650.2016.1245421>.
2. Lawrence RA. Poisonous plants: when they are a threat to children. *Pediatr Rev.* 1997;18(5):162–8. <https://doi.org/10.1542/pir.18-5-162>.
3. Kristanc L, Kreft S. European medicinal and edible plants associated with subacute and chronic toxicity part I: plants with carcinogenic, teratogenic and endocrine-disrupting effects. *Food Chem Toxicol.* 2016;92:150–64. <https://doi.org/10.1016/j.fct.2016.04.007>.
4. Kristanc L, Kreft S. European medicinal and edible plants associated with subacute and chronic toxicity part II: plants with hepato-, neuro-, nephron- and immunotoxic effects. *Food Chem Toxicol.* 2016;92:38–49. <https://doi.org/10.1016/j.fct.2016.03.014>.
5. Boukandou Mounanga M, Mewono L, Aboighe AS. Toxicity studies of medicinal plants used in sub-Saharan Africa. *J Ethnopharmacol.* 2015;174:618–27.
6. Thornton SL, Darracq M, Lo J, Cantrell FL. Castor bean seed ingestions: a state-wide poison control system's experience. *Clin Toxicol.* 2014;52(4):265–8. <https://doi.org/10.3109/15563650.2014.892124>.
7. Petersen DD. Common plant toxicology: a comparison of national and southwest Ohio data trends on plant poisonings in the 21st century. *Toxicol Appl Pharmacol.* 2011;254(2):148–53. <https://doi.org/10.1016/j.taap.2010.10.022>.
8. Mrvos R, Krenzelok EP, Jacobsen TD. Toxidromes associated with the most common plant ingestions. *Vet Hum Toxicol.* 2001;43(6):366–9.
9. Afshari R, Majdzadeh R, Balali-Mood M. Pattern of acute poisonings in Mashhad, Iran 1993–2000. *Clin Toxicol.* 2004;42(7):965–75.
10. Bentur Y, Lurie Y, Cahana A, Lavon O, Bloom-Krasik A, Kovler N, et al. Poisoning in Israel: annual report of the Israel poison information center. *Isr Med Assoc J.* 2008;10(11):749–56.
11. Bernstein J. Common plant ingestions. *J Fla Med Assoc.* 1994;81(11):745–6.
12. Krenzelok E, Jacobsen T, Aronis J. Plant exposures: a state profile of the most common species. *Vet Hum Toxicol.* 1996;38(4):289–98.
13. Vichova P, Jahodar L. Plant poisonings in children in the Czech republic, 1996–2001. *Hum Exp Toxicol.* 2003;22(9):467–72. <https://doi.org/10.1191/0960327103ht387oa>.
14. Krenzelok EP, Mrvos R. Friends and foes in the plant world: a profile of plant ingestions and fatalities. *Clin Toxicol.* 2011;49(3):142–9. <https://doi.org/10.3109/15563650.2011.568945>.
15. Fuchs J, Rauber-Luthy C, Kupferschmidt H, et al. Acute plant poisoning: analysis of clinical features and circumstances of exposure. *Clin Toxicol.* 2011;49(7):671–80. <https://doi.org/10.3109/15563650.2011.597034>.
16. Sriapha C, Tongpoo A, Wongvisavakorn S, et al. Plant poisoning in Thailand: a 10-year analysis from Ramathibodi poison center. *Southeast Asian J Trop Med Pub Health.* 2015;46(6):1063–76.
17. Mrvos R, Dean B, Krenzelok E. Philodendron/dieffenbachia ingestions: are they a problem? *Clin Toxicol.* 1991;29(4):485–91.
18. Harchelroad F, Scalise J, Dean B, Krenzelok E. Identification of common houseplants in the emergent care setting. *Vet Hum Toxicol.* 1988;30(2):161–3.
19. Scalise J, Harchelroad F, Dean B, Krenzelok E. Berry identification by emergency health care providers. *Vet Hum Toxicol.* 1988;30(5):426–8.