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Journal

Clinical Toxicology, 52(3)

ISSN

1556-3650

Authors

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Kearney, TE

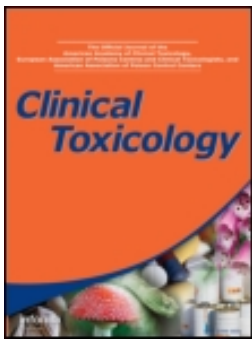
Publication Date

2014-03-01

DOI

10.3109/15563650.2014.885037

Peer reviewed



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To cite this article: P. Armenian & T. E. Kearney (2014) Pediatric ergot alkaloid exposures reported to the California Poison Control System: 1997–2008, *Clinical Toxicology*, 52:3, 214-219, DOI: [10.3109/15563650.2014.885037](https://doi.org/10.3109/15563650.2014.885037)

To link to this article: <http://dx.doi.org/10.3109/15563650.2014.885037>



Published online: 16 Feb 2014.



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POISON CENTRE

Pediatric ergot alkaloid exposures reported to the California Poison Control System: 1997–2008

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Context. The risk of toxicity from exposure to ergot alkaloid-containing medications in children is uncertain. Due to the alarming historical experience with severe toxicity and the syndrome of ergotism from natural and synthetic ergot alkaloids, triage recommendations for pediatric exposures to medicinal agents containing ergot alkaloids may be inappropriate and inconsistent. **Objectives.** The goal of this study was to describe the clinical effects of unintentional ergot alkaloid exposures in children and to identify the need for hospitalization in these cases. **Methods.** This was a retrospective cohort study of all pediatric (<7 years old) ergot alkaloid exposures reported to the California Poison Control System (CPCS) from 1997 to 2008. Case narratives were reviewed and assessed for patient demographics, ergot alkaloid agent and dose, route of and reason for exposure, symptoms, therapy, hospitalization period, and final outcome. **Results.** Of the 374 cases, 353 met the inclusion criteria. The median age was 24 months (Range: 7–72 months) with more than 99% oral route of exposure. The most frequent clinical effect was gastrointestinal distress (16%), followed by lethargy (5%). Two cases with significant vascular and CNS symptoms were identified, both with complete recovery. For symptomatic patients, all symptoms were there at time of initial presentation. The majority, 62%, of all patients were treated in the hospital setting. The median length of hospital stay was 4 h (Range: 1–36 h). Ergot exposures had a similar number of serious outcomes (moderate or worse effects) compared to all other pediatric poisonings reported to the CPCS during the study period (odds ratio [OR], 0.98; 95% confidence interval [CI], 0.25–3.95), but were associated with a disproportionately higher number of hospitalizations (OR, 13.8; 95% CI, 11.1–17.1). **Conclusions.** Pediatric ergot exposures were associated with few transient adverse effects but multiple hospitalizations. Rare cases of significant toxicity associated with methylergonovine exposures were found. Current poison control send-in protocols and emergency department (ED) guidelines should consider home management and short ED stays as opposed to lengthy critical care bed admissions.

Keywords Pediatric poisoning; Ergot alkaloid; Ergotamine, Poison control center

Abbreviations CPCS, California Poison Control System; ED, emergency department; VDL, visual Dot Lab; SPIs, specialists in poison information; AAPCC, American Association of Poison Control Centers; PICU, pediatric intensive care unit; PCC, poison control center

Introduction

Unintentional pediatric ergot alkaloid exposures occur infrequently but incite concern due to historical experience with severe toxicity from natural and synthetic ergot alkaloids in adults. Attributed to cause multiple poisoning epidemics in the Middle Ages from rye bread contaminated with the fungus *Claviceps purpurea*, ergot alkaloids resulted in the syndrome of ergotism, known at the time as St. Anthony's fire.^{1,2} Ergotism was characterized by intense burning of the extremities, vomiting, gangrene, peripheral and central vascular infarctions, agitation, hallucinations, seizures, and

death.^{1,2} Primarily used in the field of obstetrics in the 20th century, ergot alkaloids are now most often used in the treatment of migraine headaches.³

The risk of toxicity from oral exposure to ergot alkaloid-containing medications in children is uncertain, and few cases have resulted in morbidity or mortality. After ingesting 12 mg of ergotamine and 1.2 gm of caffeine, a 14-month-old child developed cerebral edema and hemorrhagic gastritis and ultimately died.⁴ In another critical case, a 15-day-old girl developed apnea requiring intubation after accidentally receiving 0.04 mg/kg of oral methylergonovine. She was extubated 43 h later and discharged home on hospital day five.⁵

Triage recommendations for pediatric exposures to medicinal agents containing ergot alkaloids are inconsistent and may be inappropriate. Vasoconstriction and ischemia are the primary concerns in the overdose setting and often prompt

Received 1 October 2013; accepted 14 January 2014.

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long observation periods and critical care bed admissions. One study recommended a general 24- to 48-h observation period after all oral and intramuscular methylergonovine exposures.⁵ Other sources have recommended observing asymptomatic patients for 4–6 h and only admitting patients symptomatic at that time.⁶

We propose that the appropriate treatment and observation periods for pediatric patients following ergot alkaloid exposures should be based on and tailored to patient symptoms, the specific substance involved, dosage, and route of exposure. The primary objective of this study is to describe the clinical effects of pediatric ergot alkaloid exposures in our study sample and to determine the need for hospitalization in these cases. A secondary objective is to delineate an evidence-based pediatric ergot alkaloid exposure management guideline.

Materials and methods

Study design and setting

This was a retrospective cohort study of all pediatric (<7 years old) ergot alkaloid exposures reported to the California Poison Control System (CPCS) during the 12-year period between January 1, 1997 and December 31, 2008. The CPCS manages over 330,000 cases annually from the general population, law enforcement and health care professionals, who call CPCS on a voluntary basis. Services are available through a toll-free emergency hotline 24 h a day. All calls are handled by trained specialists in poison information (SPIs). SPIs are pharmacists or nurses working at one of four integrated centers (San Francisco, Sacramento, Fresno/Madera, and San Diego), with medical toxicologists available to assist on complex cases.

SPIs enter each case into a computerized database, Visual Dot Lab (VDL) (WBM Software, Fresno, CA), linked via intranet among the four sites. Once a case is entered into VDL, it is followed at least daily until the final outcome is known, with the exception of cases judged by SPIs to be nontoxic exposures and those lost to follow-up. Initial information is acquired from the caller and subsequent follow-up information is obtained from the treating physician or nurse. Patients followed at home typically received one follow-up call to the parents during that day and no calls on subsequent days. Time-stamped case narratives are entered in a free-text field and clinical symptoms, treatments and outcomes are coded by SPIs using American Association of Poison Control Centers (AAPCC) guidelines. A positive symptom was one that occurred at any point during the hospital stay. Drug effect is coded into the following categories: No effect, minor effect, moderate effect, major effect, and death. AAPCC guidelines define no effect as “no symptoms as a result of the exposure.” Minor effect is defined as “some symptoms as a result of the exposure, but they were minimally bothersome to the patient. The symptoms usually resolve rapidly and usually involve skin or mucous membrane manifestations.” Moderate effect symptoms “are more pronounced, more prolonged or more of a systemic nature than minor symptoms.”

For a major effect, symptoms “are life-threatening or resulted in significant residual disability or disfigurement.”

Study population

The VDL database was queried for all cases less than 7 years of age during the 12-year study period (1997–2008). The AAPCC generic code for all ergot alkaloid products (018000) was used in the search. Since each brand name substance entered by SPIs into VDL automatically generates a generic match, we did not perform individual searches on each brand name drug. Cases outside California and those which were not true ergot exposures were excluded (i.e., intact pills found or other medications identified).

Data analysis

De-identified VDL case records were examined and data were extracted into a Microsoft Excel for Mac 2011 spreadsheet by author PA (Microsoft, Redmond, WA). The narratives of each case were reviewed and the details in those free text fields were confirmed with the coded information. Data collected included age, gender, weight (when available), route of exposure (oral, intravenous, and intramuscular), drug type, estimated maximum possible dose, co-ingestants, time to CPCS call after ingestion, location of hospitalization, length of hospital stay, blood pressure, heart rate, symptoms, and abnormalities on physical exam and treatment. The estimated maximum possible dose was determined by examining free text for information about pill counts and observer accounts with confirmation from coded dose estimations. After 1999, coded dose estimations also included certainty estimates (certain, estimate, or unknown dose amount as per SPI input). Doses coded as certain and estimate were included in the analysis. As data were not normally distributed, descriptive statistics such as median and range were determined. Odds ratios with 95% confidence intervals were calculated to determine the risk of serious outcomes and hospitalizations associated with pediatric ergot alkaloid exposures. Serious outcomes were defined as those coded with moderate or major outcomes according to AAPCC guidelines.

This study was reviewed and approved by the Committee for Human Research at the University of California, San Francisco and the CPCS Research Committee.

Results

A total of 374 ergot exposures under the age of 7 were identified upon the initial VDL search. Calls originating from home comprised 64% (239/374) of cases and 36% (135/374) originated from health care facilities. Twenty-one cases were excluded, leaving 353 in the final analysis (Fig. 1). For each case, free-text narrative and coded information were examined and found to be in agreement. General patient and exposure characteristics are presented in Table 1. The overwhelming majority of cases, 99% (351/353) were due to oral route of exposure, with only one case each due to

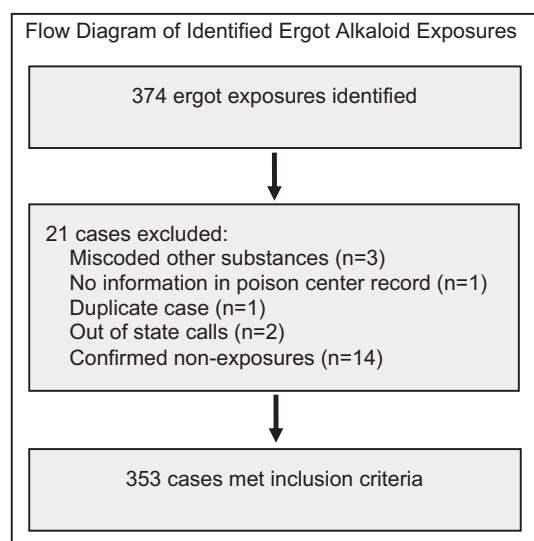


Fig. 1. Flow diagram of identified ergot alkaloid exposures.

inadvertent intramuscular or intravenous administration in neonates. For oral exposures, the median age at presentation was 24 months (range: 7–72 months). 76% Of cases were due to a single ergot alkaloid containing substance. The most common ergot substance involved was methylergonovine (231 cases), followed by ergotamine (56 cases), ergonovine (37 cases) and dihydroergotamine (15 cases). A small number of cases of ergoloid mesylate (6), methysergide (3), and dihydroergotamine (1) exposures were found. In 86 cases at least 1 co-ingestant was present: 1 co-ingestant in 69, 2 co-ingestants in 15, and 3 co-ingestants in 2 cases. The substance most often co-ingested was caffeine in 61 cases, due to caffeine/ergotamine combination pills used in migraine therapy. Doxycycline was the co-ingestant in 13 cases. One hundred and fourteen cases were managed at home and nineteen were lost to follow-up. Of these, 15 were coded as potentially toxic exposures. All 19 lost to follow-up cases were asymptomatic at the initial time of poison control center (PCC) phone call. Of the 220 cases that were managed in a health care facility, the median length of stay was 4 h, ranging from 1 to 36 h. Median time from exposure to CPCS call was 10 min. This information was available in 349/353 (99%) cases.

The most common clinical effect associated with ergot alkaloid exposure was gastrointestinal distress (nausea,

Table 1. Patient and exposure characteristics.

	Cases, n
Median age (months, range)	24 (0–72)
Male gender	195 (55.4%)
Route of exposure: Oral	351 (99.1%)
Route of exposure: IM	1 (0.3%)
Route of exposure: IV	1 (0.3%)
Median time to PCC call post-ingestion (min, range)	10 (1–960)
Single ingestion	267 (75.6%)
More than 1 type of substance ingested	86 (24.4%)
Median length of stay (h, range)*	4 (1–36)

*186 included in analysis; 112 observed at home, 55 unknown Length of Stay.

vomiting, abdominal pain, and/or diarrhea), followed by lethargy, and hypertension (Table 2). Rarely, numbness, and cold extremities were present, although transient and only present on initial physical examination. From the cases referred from home to a health care facility, if no symptoms were present on initial emergency department (ED) evaluation, they did not develop later. A minority of exposures resulted in clinical effects. The clinical effects varied somewhat according to the ergot alkaloid class (Table 3). Amine exposures, the most frequent, resulted in gastrointestinal symptoms 16% of the time. Amino acid exposures had an equivalent number of cases with gastrointestinal and CNS/respiratory symptoms. The highest exposure dosages were in the dehydrogenated amino acids group, with an ergoloid mesylate median dose of 13.5 mg (range: 1.5–30 mg), but clinical effects were present in one of six such cases.

All except two cases were coded as no effect or mild effect. These two cases met moderate effect criteria, and none had major effect or death. The first moderate effect case was a 2-year-old male who ingested an unknown quantity of 0.2 mg of Methergine (methylergonovine) tablets. CPCS was contacted less than 5 min after time of ingestion and the patient's mother was advised to take him to the nearest ED. Upon ED arrival, the child was lethargic with cool and pale extremities and prolonged capillary refill time. His initial vital signs were: Temperature 36.9°C, pulse 90 beats per minute, blood pressure 94/60 mm Hg, respirations 20 per minute, O₂ saturation 82% on 15L nonrebreather mask. Fifteen minutes later, after the placement of a nasogastric tube to administer activated charcoal, an intravenous normal saline bolus and warm blankets, the patient had normal mental status, normal oxygen saturation, and extremities with capillary refill of less than 2 s. The patient was transferred to the nearest pediatric intensive care unit (PICU) and monitored for the next 24 h. He remained asymptomatic without symptom recrudescence.

In the second moderate effect case, a 3.3-kg newborn male was accidentally administered 0.2 mg of intramuscular methergine instead of vitamin K shortly after birth. One hour later, he developed respiratory depression with room air O₂

Table 2. Clinical effects associated with ergot alkaloids.

Symptoms	Cases, n (%)
Gastrointestinal distress*	55 (15.6)
Lethargy	17 (4.8)
Hypertension**	6 (1.8)
Agitation	3 (0.9)
Dizziness	2 (0.6)
Hypoxia	2 (0.6)
Bradycardia***	2 (0.6)
Tachycardia***	2 (0.6)
Numbness	2 (0.6)
Cold extremities	1 (0.3)

*Gastrointestinal distress is defined as nausea, vomiting, abdominal pain and/or diarrhea.

**Hypertension is defined as a single reading of SBP >100 mmHg ages newborn–1yr, SBP >110 mmHg 1–6yr.

***Normal heart rate is defined as HR 120–160 ages 0–1mo, 100–160 1mo–1yr, 90–150 1–3yr, 80–140 3–6yr.

Table 3. Clinical effects and exposed doses of ergot alkaloids.

Ergot alkaloid Class (n)	Gastrointestinal n (% class)	Other symp.* n (% class)	Median Dose** (range) (mg)
Amine (271)	42 (15.5)	15 (5.5)	
Ergonovine (37)	4	1	0.4 (0.2–2)
Methylergonovine (231)	37	13	0.4 (0.1–3)
Methysergide (3)	1	1	2 (2)
Amino acid (56)	7 (13)	8 (14)	
Ergotamine	7	8	1 (0.2–11)
Dihydrogenated amino acid (22)	1 (5)	3 (14)	
Dihydroergotamine (15)	1	2	1 (0.25–3)
Ergoloid mesylate (6)	0	1	9 (1.5–30)
Dihydroergotoxine (1)	0	0	1

*Other symptoms include central nervous system (CNS) and respiratory system.

**256 included in analysis; dose unknown for 97 patients.

saturations dropping to 80%. The rest of his vital signs were: Temperature 98.1°C, pulse 120–140 beats per minute, BP 73/43 mm Hg, with reported normal respiratory rate. He was placed under a 30% O₂ hood with O₂ saturations increasing to 97%. For the next 4 h, he was irritable and had a few brief episodes of O₂ desaturations. His extremities remained warm and well perfused. He was breathing well with normal O₂ saturation on room air at 8.5 h after exposure. On hospital day 2, the infant had trace hematuria and hematemesis which spontaneously resolved. His vital signs and abdominal X-ray were normal. He was made NPO and total parenteral nutrition (TPN) feeds were started. By hospital day 7, he was tolerating breastfeeding well and was discharged home on Day 9 without any apparent sequelae from methergine exposure.

Patient disposition and clinical outcomes are presented in Table 4. Symptoms were present in only 21% of all cases. Most patients were observed and discharged from the ED without inpatient hospital admission. Of the admitted patients, most were admitted to unmonitored beds or a PICU. Cases managed at home by CPCS phone consultation manifested symptoms 11% of the time. Table 5 provides a comparison of risks of a serious outcome and hospitalization associated with pediatric ergot alkaloid exposures compared with all substances (drugs, herbal products, chemicals, and plants)

Table 4. Disposition and clinical outcomes.

Disposition	Symptoms present ^a , n (%)	Symptoms absent, n (%)	Total
Home	13 (11.4)	101 (88.6)	114
Outpatient Clinic	0	2 (100)	2
Emergency Department	50 (27.3)	133 (72.7)	183
Unmonitored Inpatient	3 (14.3)	18 (85.7)	21
Telemetry Inpatient	1 (100)	0	1
Newborn Nursery	0	1 (100)	1
Neonatal Intensive Care Unit	1 (100)	0	1
Pediatric Intensive Care Unit	3 (27.3)	8 (72.7)	11
Unknown	n/a	n/a	19
All Patients	74 (21.0)	279 (79.0)	353

^aSymptoms present at time of initial health care facility evaluation. For home calls, all symptoms presented within 1 h of ingestion for all but one case, who developed symptoms 5 h after ingestion and continued to be managed at home.

reported to the CPCS during the study period. Ergot alkaloid exposures had a similar number of serious (moderate, major, or death) outcomes to other pediatric poisonings reported to the CPCS (Odds ratio [OR], 0.98; 95% confidence interval [CI], 0.25–3.95). However, they were associated with a significantly and disproportionately higher number of hospitalizations (OR 13.8; 95% CI, 11.1–17.1).

Discussion

Evaluating the risk of toxicity from ergot alkaloid exposures in pediatric patients is difficult due to the heterogeneity in the pharmacology and pharmacokinetics among these agents. The three classes of ergot alkaloids, amine, amino acid, and dihydrogenated amino acids, differ in peripheral and central mechanisms of action at serotonergic, dopaminergic, and alpha-adrenergic receptors.³ The amino acid group (ergotamine) causes the highest incidence of emesis and along with ergonovine (amine group) produces profound vasoconstriction. The dihydrogenated amino acids such as dihydroergotamine have alpha-adrenergic antagonism properties (alpha 1 and 2), whereas the other ergot alkaloids are partial agonists/antagonists at the alpha-adrenergic receptors, leading to variable vasoconstrictive effects.⁷ Ergot alkaloid action at 5-HT_{1B/1D} receptors potentiating serotonin activity lead to stabilization of cerebrovascular smooth muscle and acute migraine headache relief.³

Most ergot alkaloids have an elimination half-life of about 2 h and peak plasma concentrations occur within

Table 5. Odds ratios for serious outcomes and hospitalizations associated with pediatric ergot alkaloid exposure compared with all substance exposure.^a

Variable	Ergot products, n (%)	All substances, ^b n (%)	Odds ratio (95% CI)
Serious outcome ^c	2 (0.6)	7,872 (0.6)	0.98 (0.25–3.95)
Hospitalization	218 (61.8)	143,199 (10.5)	13.8 (11.1–17.1)

^aChildren (age, 0–5); data from 353 cases reported to the California Poison Control System (1997–2008). ^bIncludes drugs, herbal products, chemicals and plants; *n* = 1,365,541.

^cSerious outcome defined as moderate, severe, or death, per American Association of Poison Control Centers' criteria.

60 min of ingestion.⁸ However, bioavailability varies widely due to extensive hepatic first-pass metabolism and poor oral absorption. The notable exception is methylergonovine (amine alkaloid), which is rapidly absorbed and has high bioavailability after ingestion.³ Ergot alkaloid poisoning manifestations are an enhancement of its therapeutic effects. Vasoconstriction has been known to cause lower extremity, cerebral, mesenteric, coronary, and renal vascular ischemia in adult patient exposures.^{9,10}

The historical accounts of epidemic and catastrophic ergot-induced vasospastic disease as well as adult poisoning reports may have overtly alarmed clinicians about management and triage strategies for ergot alkaloid exposures. It is noteworthy that only 2 of 353 cases in this study manifested serious toxicity and both involved methylergonovine. Our case involving a newborn who inadvertently received an intramuscular dose of methylergonovine and developed respiratory depression is consistent with previous case reports of newborn poisonings from intramuscular administration of the amine class of ergot alkaloids.^{5,11–15} These cases were also iatrogenic and involved administration of the wrong medication that was mistaken for vitamin K.

The other case with serious toxicity in our study involved the ingestion of an unknown amount of a methylergonovine-containing product in a 2-year old. The symptoms were rapid in onset and predominately neurological (CNS depression), respiratory (hypoxia), and there was clinical evidence of peripheral vasospasm (prolonged capillary refill time). However, the symptoms rapidly resolved with supportive care and there was no evidence of recrudescence or prolonged toxic effects. Methylergonovine has good bioavailability when ingested and oral exposure in pediatric patients has rarely been associated with severe toxicity.⁵

Another ergot substance in this class, ergonovine, was involved in a death of a 14-month old who ingested 12 mg.¹⁶ It is also noted that unintentional ingestions of up to 10 tablets (totaling 2 mg) of methylergonovine have resulted in no ill effects in children aged 1–3 years.¹⁷ The dosage range in our series was 0.5–3 mg with a median dose of 0.4 mg of methylergonovine. Unintentional dihydroergotamine ingestions represented only 15 cases in our series with very few ill effects. There are no other dihydroergotamine exposures reported in the pediatric literature.

None of the cases involving ergotamine ingestions were associated with serious toxicity. Only 13% developed gastrointestinal and 14% had other symptoms (CNS or respiratory). One case, as mentioned above, had evidence of peripheral vasospasms as documented in the poison center record. Ergotamine has poor bioavailability and is formulated as a single agent sublingual tablet. This, coupled with its propensity to cause gastrointestinal symptoms with nausea and vomiting, may limit the risk of systemic toxicity after ingestion. There is a case report of a 14-month old who died after ingestion of 12 Cafergot[®] tablets (totaling 12 mg of ergotamine and 1200 mg of caffeine).⁴ This child had a rapid onset of symptoms including respiratory distress, CNS depression, and peripheral vasospasms (cold

blue extremities). The estimated dose range in our series was 0.2–11 mg with a median dose of 1 mg of ergotamine.

Our study suggests that unintentional ergot alkaloid ingestions by children most commonly result in transient symptoms involving the gastrointestinal system as well as minor CNS symptoms (lethargy). The concern of persistent vasoconstriction was not supported in any of these cases regardless of agent or route of exposure. Therefore, the general recommendation to admit methylergonovine exposed pediatric patients for 24–48 h of hospitalization does not seem warranted.⁵ Home management of unintentional pediatric ergot alkaloid ingestions by poison centers may be a viable option in cases that involve a single agent and the patient is asymptomatic. Exposures involving ingestion of unknown amounts of any ergot alkaloid by pediatric patients or those that are symptomatic should be evaluated and managed in an ED. Important for emergency providers is noting that in our study, if no symptoms were present on initial ED evaluation, they did not develop later. It is thus anticipated that most of these patients will require supportive care and short observation periods. The small number of parenteral exposures in our study makes any new conclusions on that route of exposure difficult. Therefore, accidental parenteral ergot alkaloid exposures in neonates still warrant observation periods of at least 24 h.^{5,11–15}

There are several major limitations to this study. First, the retrospective study design and data source used (poison control case reports) were an inherent limitation to completeness of the data. SPIs and related personnel responsible for documenting PCC cases were not under protocol to collect information specific to this study which were not necessary for patient management (e.g., specific product name, ergot alkaloid quantity in mg, co-ingredients, and co-ingestants). As a result, much information is missing in our data set. Another factor for missing information in PCC reports is incomplete patient follow-up. Patients are frequently lost to follow-up due to various reasons beyond the control of PCC personnel (e.g., patient leaving against medical advice or having been discharged upon follow-up call). It is not known if any patients who were lost to follow-up died. Another limitation is that the CPCS data do not include measurements of the serum concentration of the drug involved in the ingestions. The milligram quantity of an ingested drug is recorded in the CPCS database in only a small majority of cases. Even for those cases, the ingested amount was usually based on patient's self-report and not independently verified through witnessed ingestion or pill counts.

Conclusion

Pediatric ergot exposures were associated with few transient adverse effects but multiple hospitalizations. Current poison control send-in protocols and ED guidelines should base home management on the agent, dose, and patient symptoms. We recommend evaluating all symptomatic patients and ingestions of an unknown amount in the ED. For those patients evaluated in an ED, most will require a short stay with general supportive care as opposed to lengthy critical

care bed admissions. We do not recommend routine critical care bed admissions for asymptomatic patients.

Acknowledgments

The authors would like to thank Erika Gray, Pharm.D. for her assistance in data collection.

Declaration of interest

The authors report no declarations of interest. The authors alone are responsible for the content and writing of the paper.

References

1. Lee MR. The history of ergot of rye (*Claviceps purpurea*) I: from antiquity to 1900. *J R Coll Physicians Edinb* 2009; 39:179–184.
2. Van Dongen PW, De Groot AN. History of ergot alkaloids from ergotism to ergometrine. *Eur J Obstetr Gynecol Reprod Biol* 1995; 60:109–116.
3. Sanders-Bush E, Hazelwood L. Chapter 13. 5-Hydroxytryptamine (Serotonin) and dopamine. In: Brunton LL, Chabner BA, Knollmann BC, eds. *Goodman & Gilman's The Pharmacological Basis of Therapeutics*. 12th ed. New York: McGraw-Hill; 2011. <http://www.accessmedicine.com/content.aspx?aID=16662305>. Accessed September 17, 2012.
4. Jones EM, Williams B. Two cases of ergotamine poisoning in infants. *Br Med J* 1966; 1:466.
5. Aeby A, Johansson A, De Schuiteneer B, Blum D. Methylergometrine poisoning in children: review of 34 cases. *J Toxicol Clin Toxicol* 2003; 41:249–253.
6. Ergot Alkaloids. In: *Poisindex® System* [Internet database]. Greenwood Village, Colo: Thomson Healthcare. Updated June 5, 2013.
7. Goldstein M, Lew JY, Hata F, Lieberman A. Binding interactions of ergot alkaloids with monoaminergic receptors in the brain. *Gerontology* 1978; 24:76–85.
8. Orton DA, Richardson RJ. Ergotamine absorption and toxicity. *Postgrad Med J* 1982; 58:6–11.
9. Zavaleta EG, Fernandez BB, Grove MK, Kaye MD. St. Anthony's fire (Ergotamine induced leg ischemia): a case report and review of the literature. *Angiology* 2001; 52:349–356.
10. Musikataporn K, Suteparuk S. Ergotism unresponsive to multiple therapeutic modalities, including sodium nitroprusside, resulting in limb loss. *Clin Toxicol* 2008; 46:157–158.
11. Donatini B, Le Blaye I, Krupp P. Inadvertent administration of uterotonics to neonates. *Lancet* 1993; 341:839–840.
12. Pandey S, Haines C. Accidental administration of ergometrine to a newborn infant. *Br Med J* 1982; 285:693.
13. Whitfield MF, Salfield SAW. Accidental administration of syntometrine in adult dosage to the newborn. *Arch Dis Childhood* 1980; 55:68–72.
14. Baum CR, Hilpert PL, Bhutani VK. Accidental administration of ergot alkaloid to a neonate. *Pediatrics* 1996; 98:457–458.
15. Bangh SA, Hughes KA, Roberts DJ, Kovarik SM. Neonatal ergot poisoning: a persistent iatrogenic illness. *Am J Perinat* 2005; 22: 239–243.
16. Product Information: Ergotrate® injection, ergonovine maleate injection. Pharmacist Pharmaceutical, LLC, Roanoke, VA, 2006.
17. Product Information: Methylergonovine maleate IM,IV injection. PharmaForce, Inc., Columbus, OH, 2008.