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Hypoglycemia in Pediatric Sulfonylurea Poisoning: An 8-Year Poison Center Retrospective Study

WHAT'S KNOWN ON THIS SUBJECT: Pediatric sulfonylurea ingestions are known to cause profound and prolonged hypoglycemia. The optimum observation period for asymptomatic children remains controversial.

WHAT THIS STUDY ADDS: Either food or intravenous glucose can delay the onset of hypoglycemia in pediatric sulfonylurea poisonings. The conventional 8-hour observation period with free access to food for pediatric sulfonylurea exposures is an unreliable screening method.

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abstract

OBJECTIVE: The goal of this study was to describe the clinical effects and time of onset of hypoglycemia in pediatric sulfonylurea poisoning.

METHODS: This was a retrospective, descriptive study of pediatric (<6 years old) sulfonylurea exposures with hypoglycemia (glucose concentration <60 mg/dL) that were consulted on by the California Poison Control System for the 8-year period between January 1, 2002, and December 31, 2009.

RESULTS: Of the 1943 consultations for pediatric sulfonylurea exposure in the study period, 300 children developed hypoglycemia. Ten percent had hypoglycemia occurring or persisting ≥12 hours after ingestion despite receiving treatment. All 5 children with seizures experienced these before hospital presentation. The mean (SD) time to onset of hypoglycemia in children not given any prophylactic treatment was 2.0 (1.2) hours. The mean (SD) times in children receiving prophylactic food only, intravenous glucose only, and both food and intravenous glucose were 5.9 (3.9), 5.7 (2.5), and 8.9 (3.6) hours, respectively. Ranges were 1 to 18, 1.5 to 9, and 2.5 to 15 hours. Seven of 40 patients (18%) receiving prophylactic food only had an onset of hypoglycemia >8 hours after sulfonylurea ingestion.

CONCLUSIONS: Pediatric sulfonylurea exposure can result in significant poisoning. Severe effects such as seizures occurred only in cases of unrecognized sulfonylurea ingestion. The onset of hypoglycemia after pediatric sulfonylurea ingestion can be delayed by as much as 18 hours by either free access to food or administration of intravenous glucose. *Pediatrics* 2011;127:e1558–e1564

Acute sulfonylurea exposure is a frequent cause of pediatric presentation to hospital emergency departments (EDs). The 2009 Annual Report of the American Association of National Poison Centers' National Poison Data System found 922 pediatric (<6 years old) exposures to sulfonylureas, the majority of which were referred to EDs for evaluation.[1]

Management of sulfonylurea exposure in toddlers is difficult. These exposures are potentially serious, as ingestion of a single pill by a child has been shown to cause hypoglycemia.[2–4] Sulfonylurea-induced hypoglycemia can persist for >24 hours.[3,5,6] Uncorrected hypoglycemia can lead to severe clinical effects such as coma and seizures.[6–13] However, ingestions in toddlers are often unwitnessed. In addition, hypoglycemia from sulfonylurea ingestion can often develop over several hours and be clinically insidious.[14] Unless the blood glucose is readily measurable, it is difficult to diagnose this dangerous ingestion.

A common, but contested, recommendation for screening of suspected sulfonylurea ingestions in asymptomatic children is frequent clinical observation and glucose monitoring for 8 hours with free access to food but without supplemental intravenous glucose.[2,15–18] Controversially, this approach assumes that food will not "mask" drug-induced hypoglycemia to the same degree as supplemental intravenous glucose. This assumption has been directly challenged.[19] Physiologically, the caloric content of food and snacks can easily exceed that of a continuous infusion of 5% dextrose. Moreover, the recommendation for an 8-hour observation time was based on 2 studies with a total of 76 pediatric patients experiencing hypoglycemia after sulfonylurea ingestion.[18,20] On the basis of these findings and other reports,

competing recommendations vary between >8 and 24 hours of observation.[4,14,20,],[27] The goals of the present study were to describe the clinical effects and time of onset of hypoglycemia in pediatric sulfonylurea poisoning.

METHODS

Study Design and Setting

This was a retrospective cohort study of pediatric (<6 years old) sulfonylurea exposures with hypoglycemia that were consulted on by the California Poison Control System (CPCS) for the 8-year period between January 1, 2002, and December 31, 2009. The CPCS receives ~320 000 consultations annually from patients and health care providers. The CPCS consists of 4 linked call-answering sites that provide consultations for the state of California 24 hours per day. Each call is received by poison specialists who are trained pharmacists or nurses. Medical toxicologists are available for consultation on complex cases. Cases judged by the specialists in poison information to have potentially significant toxicity are followed up until the outcome is known. These are coded as no effect, minor effect, moderate effect, major effect, or death. The exception is cases in which minimal toxicity is expected or the case is lost to follow-up. These are coded as not followed, judged as nontoxic/not followed, minimal clinical effects possible, or unable to follow/judged as potentially toxic. For each consultation received, an electronic poison center case record is created in Visual Dotlab (VDL, WBM Software, Fresno, CA) according to the guidelines of the American Association of Poison Control Centers (AAPCC), which includes text-based notes of the case. Every note entered in VDL is permanent and automatically time stamped. The CPCS data system analyst is responsible for maintaining the

CPCS case record data set, which is uploaded to the national poison center database every 20 minutes.

Study Population

We searched 8 years of data in VDL from January 1, 2002, through December 31, 2009. Only patients younger than 6 years of age were included. We searched the VDL database for cases coded with AAPCC generic codes and POISINDEX (Micromedex, Thomson Reuters, New York, NY) product identification codes for sulfonylureas, including acetohexamide, chlorpropamide, gliclazide, glimepiride, glipizide, gliquidone, glisolamide, glisoxepide, glyburide, glyburide/metformin, tolazamide, tolbutamide, and unknown sulfonylurea. We searched for the AAPCC generic code, which for sulfonylurea oral hypoglycemic agents is 201119. Because both this AAPCC code and the POISINDEX product-specific code are saved in VDL for every case, no brand name product cases would be missed because both codes are in the electronic record. For the cases analyzed, the specific sulfonylurea was determined by review of the individual chart. We included all ingestions regardless of intent.

Of these cases, only those with the AAPCC symptom code for hypoglycemia were included. We extracted cases with all codes for hypoglycemia (related, unrelated, and unknown if related). We reviewed each case to determine that the coding had been correctly assigned and we included only those cases, based on the review of the record, in which hypoglycemia was related to the ingestion. We excluded cases that were duplicates, managed out of state, or lost to follow-up. We further reviewed each of the VDL text notes and excluded cases that had no recorded glucose measurement <60 mg/dL. This yielded the cases used for the descriptive analysis. For our hypoglycemia time-of-

onset analysis, we included cases only if they were immediate health care presentations, as defined here, and had text notes detailed enough to determine the timing of episodes of hypoglycemia.

Variables

We extracted the following information from the VDL charts: age, gender, drug ingested, estimated dose, estimated time of ingestion, times of episodes of hypoglycemia, number of episodes of hypoglycemia recorded, symptoms, treatments given prior to and after hypoglycemia, and disposition.

The health care presentation was defined as immediate if the patient was brought to a health care facility promptly after a witnessed or suspected ingestion. The presentation was defined as delayed if the patient was not brought to a health care facility promptly, which often occurred because the ingestion was not recognized or the caregiver attempted to manage the case at home. The delayed presentation group was excluded from the hypoglycemia time-of-onset analysis because these children arrived at the hospital already hypoglycemic, and we could not determine the time that the hypoglycemia had first occurred.

Hypoglycemia was defined as a measured serum glucose concentration <60 mg/dL. Altered mental status was defined as any change from the patient's baseline mental status, which included a broad range from unusual behavior to coma. If a child vomited while receiving activated charcoal, this action was attributed to the charcoal and not to hypoglycemia. Any treatment (food or intravenous glucose) that had been given before a measured serum glucose concentration <60 mg/dL was categorized as prophylactic. We defined "emergency department observation" as treatment in the ED for <12 hours. If a patient was treated in the ED for ≥12 hours, this was defined as an admission to a health care facility.

The timing of episodes of hypoglycemia was determined from the VDL text record by ≥1 of the following: (1) a discrete mention; (2) recorded serial glucose measurements; or (3) a proximate episode of hypoglycemia leading to a telephone call to the CPCS. The first 2 situations were assumed to be reliable because of specifically noted times. We believed the third situation required some adjustment. We recognized that there might be any number of events that could occur between a glucose measurement at the patient's bedside and the time the CPCS specialist logged his or her VDL text note. Thus, directly correlating an episode of hypoglycemia with the time stamp of a VDL note could lead to an overestimation of the time of its actual occurrence. To correct for this possibility, the following assumptions were made. The first recorded glucose measurement was assumed to have been made at the time of the child's arrival to the ED. For callbacks to the CPCS regarding a low blood glucose level but without an explicitly denoted time of measurement, we assumed that it had been obtained 1 hour before the time-stamped VDL record. We chose a 1-hour time adjustment because it was methodologically easier to subtract a simple, arbitrary number rather than making case-specific modifications. Our goal was to err toward underestimation of the timing and duration of hypoglycemia to give greater credibility to any notable findings. If none of these details were discernible from the VDL record, we did not use the case for the time-of-onset analysis.

Data Analysis

Primary outcome measures were the incidence of clinical effects and the time to onset of hypoglycemia. All VDL records were reviewed by 1 of the authors (Dr Lung). All cases that lacked clearly documented times of hypoglycemia onset ($n = 45$) were also reviewed by the second author (Dr Olson) to verify exclusion from the time analysis. All cases with an onset of hypoglycemia >8 hours ($n = 17$) were also reviewed by the second author (Dr Olson) to verify the times of hypoglycemia onset. The intraclass correlation coefficient was calculated as 0.9436. Descriptive statistics were used for categorical data. Student's t test was used to compare mean times of onset of hypoglycemia. Microsoft Excel 2003 (Microsoft Corporation, Redmond, WA) was used for data collection and analysis.

This study was approved by the University of California, San Francisco, Committee on Human Research and by the California Poison Control System Research Committee.

RESULTS

There were 1943 CPCS consultations for pediatric sulfonylurea exposure in the 8-year study period. Based on our inclusion criteria, there were 300 cases of pediatric sulfonylurea exposure that resulted in hypoglycemia, which we examined in our descriptive study. Of these 300 cases, 221 met the criteria for our time-of-onset analysis (Fig 1).

Specific drugs involved were glimepiride, $n = 19$ (6.3%); glipizide, $n = 179$ (60%); glyburide, $n = 75$ (25%); tolazamide, $n = 7$ (2.3%); multiple or combination products, $n = 18$ (6%); and unknown, $n = 2$ (0.67%).

Table 1 describes common clinical characteristics of the 300 cases of pediatric sulfonylurea exposure causing hypoglycemia. It is noteworthy that children with documented hypoglycemia often did not exhibit other signs and symptoms of poisoning; 251 (84%) of the hypoglycemic children were oth-
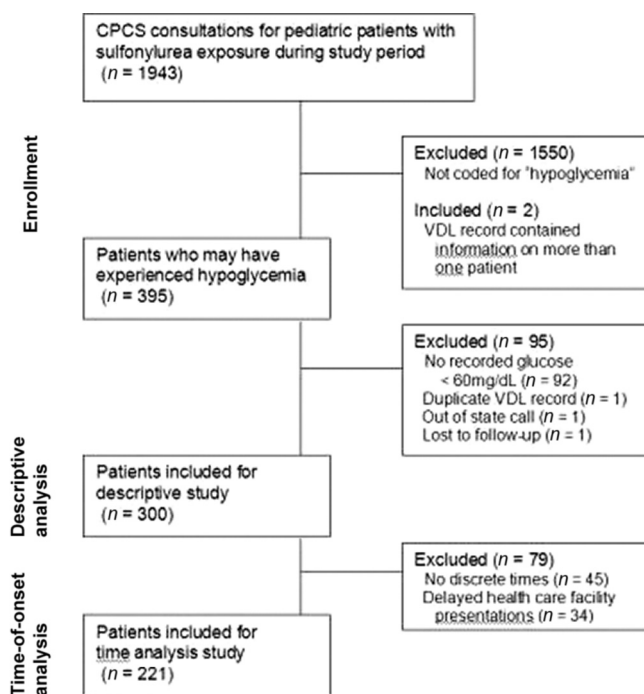
FIGURE 1
Flow diagram of patients enrolled in the study.

TABLE 1 Frequency of Clinical Characteristics in 300 Cases of Pediatric Sulfonylurea Exposure Causing Hypoglycemia

Characteristic	No. (%) of Cases
Age, y	
<1	6 (2.0)
1–2	142 (47)
2–3	110 (37)
3–4 y	38 (13)
≥4	4 (1.3)
Symptoms	
Asymptomatic (other than hypoglycemia)	251 (84)
Altered mental status	41 (14)
Diaphoresis	10 (3.3)
Vomiting	5 (1.7)
Seizure	5 (1.7)
Time to health care facility presentation	
Immediate	265 (88)
Delayed	35 (12)
Episodes of hypoglycemia	
1	194 (65)
2	61 (20)
3	30 (10)
≥4	13 (4.3)
Last reported episode of hypoglycemia	
1–12 h	269 (90)
12–24 h	28 (9.3)
>24 h	3 (1.0)

erwise asymptomatic. Among the subset of children who were brought promptly to the hospital after exposure, only 20 of 265 (7.5%) exhibited other clinical signs and symptoms. Hypoglycemia persisted ≥12 hours in 31 children (10%), despite receiving treatment.

Very few patients experienced serious clinical effects such as seizures. All 5 children who experienced seizures presented to a health care facility only after the seizure occurred and are further described in Table 2. The only child that received endotracheal intubation was 1 of these patients. No patients experienced seizures or coma after initiation of treatment in a health care facility.

Table 3 describes the common treatments provided and disposition of the 300 cases.

Our time-of-onset analysis shows the effect of prophylactic food and intravenous glucose on the onset of hypoglycemia in 221 cases of pediatric sulfonylurea poisoning (Fig 2). The mean

(SD) time to onset of hypoglycemia in patients not given any prophylaxis was 2.0 (1.2) hours, with a range of 0.5 to 7 hours. The mean (SD) times to onset of hypoglycemia in patients receiving prophylactic food, intravenous glucose, and both food and intravenous glucose were 5.9 (3.9), 5.7 (2.5), and 8.9 (3.6) hours, respectively. Ranges were 1 to 18, 1.5 to 9, and 2.5 to 15 hours. The difference between each group receiving prophylaxis compared with the group not receiving prophylaxis was significant (Student's t test, $P < .01$ for all groups). Notably, there was no significant difference (Student's t test, $P = .83$) between the time of onset of hypoglycemia of those patients receiving prophylactic food and those receiving prophylactic intravenous glucose. Several children receiving either prophylactic food or intravenous glucose had a recorded onset of hypoglycemia >8 hours after ingestion. This occurred in 18% (7 of 40) of patients who received prophylactic food only and 27% (3 of 11) of patients who received intravenous glucose only.

We also noted a striking incidence of recurrent hypoglycemia during the period of the "overnight fast" when children were sleeping. Among the 221 patients in the time-of-onset analysis, we found that 74% (28 of 38) of patients who were experiencing sulfonylurea-induced hypoglycemia for >8 hours had glucose nadirs between the hours of 10:00 PM and 7:00 AM. Notably, this pattern was also seen among 7 children receiving prophylactic food who had a recorded onset of hypoglycemia >8 hours after ingestion (Table 4). Six of these 7 children ingested a sulfonylurea in the early evening and experienced hypoglycemia overnight or in the early morning. A unique case, the child with hypoglycemia onset at 18 hours post-ingestion experienced this at home after being discharged by an emergency department after a few

TABLE 2 Seizures in Children with Sulfonylurea-Induced Hypoglycemia

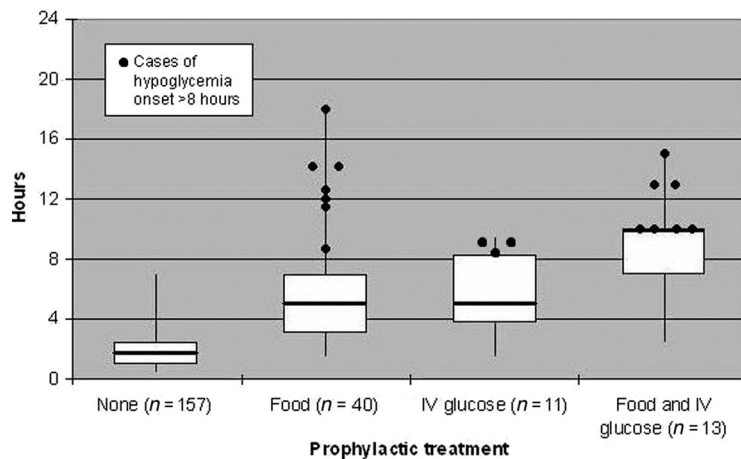| Age, mo	Gender	Sulfonylurea	Dose, Estimated, mg	Delay to Health Care Presentation	Reason for Delay	Presenting Signs and Symptoms	Initial Glucose Level, mg/dL	Treatments in Addition to IV Glucose
11	M	Glimepiride	2	Unknown	Unknown	Seizure	15	None specified
13	M	Glipizide	50	2 d	Glipizide mistakenly given every 4 h as acetaminophen	Seizure	20	Intubation, benzodiazepine
18	F	Glipizide	35	15 hours	Unknown	Seizure, vomiting	35	None specified
20	F	Glipizide	Unknown	Unknown	Unknown	Seizure	34	Activated charcoal
>24	F	Glipizide	Unknown	Unknown	Unwitnessed ingestion	Seizure	14	Octreotide

M indicates male; F, female.

TABLE 3 Treatments and Disposition in 300 Cases of Pediatric Sulfonylurea Exposure Causing Hypoglycemia

Treatment or Disposition	No. (%) of Cases
Treatment	
Activated charcoal	71 (24)
Syrup of ipecac or induced emesis	7 (2.3)
Gastric lavage	2 (0.67)
Prophylactic food only	53 (18)
Prophylactic intravenous glucose only	16 (5.3)
Prophylactic food and intravenous glucose	16 (5.3)
Food only	36 (12)
Intravenous glucose	260 (87)
Octreotide, first-line treatment	31 (10)
Octreotide, second-line treatment	23 (8)
Disposition	
Admission to standard unit	196 (65)
Admission to ICU	87 (29)
ED observation	14 (5)
Unknown	3 (1.0)

FIGURE 2
Median and range of time to onset of hypoglycemia among 221 cases of pediatric sulfonylurea exposure. IV indicates intravenous.

hours of observation. When returning CPCS telephone messages the following morning, the mother measured her daughter's glucose to be 48 mg/dL. Since the family was not performing hourly glucose checks, the onset of hypoglycemia in this child may have occurred earlier than 18 hours post-ingestion.

DISCUSSION

Our results demonstrate that prophylactic administration of either food or intravenous glucose can delay the onset of hypoglycemia in pe-diatric sulfonylurea poisoning. The common 8-hour observation period allowing free access to food is an un-reliable method to screen pediatric sulfonylurea exposures.

Our results validate some previous ob-servations. Although hypoglycemia is fairly common after pediatric sulfonyl-urea ingestion, the clinical severity is generally benign if promptly recog-nized and treated. Poisonings causing severe clinical effects such as seizures or coma seem to be limited to cases of delayed recognition and treatment.[6–13] Consistent with previous studies and case reports,[4,18,20,22] we found that the administration of intravenous glucose delayed the onset of hypoglycemia.

Importantly, our study demonstrated that giving free access to food to chil-dren with sulfonylurea poisoning can delay the onset of hypoglycemia be-yond the common 8-hour observation period. As seen by examining cases managed using prophylactic food only, it is notable that 7 of the 40 cases (18%) would have had their hypoglyce-mia "missed" if serial glucose mea-surements were stopped at 8 hours' postingestion. This finding is consis-tent with a previous poison center re-port of a child with onset of hypoglyce-mia >8 hours after ingestion who had not received intravenous glucose.[22]

Our results suggest a more conserva-tive approach to pediatric sulfonylurea exposures; for example, a 24-hour ob-servation period or an overnight ad-mission.[14,20,21,26,27] We could not deter-mine a reasonable period of time to withhold food from children for screening purposes. We found that children being fasted for screening purposes had an onset of hypoglyce-

TABLE 4 Children Receiving Prophylactic Food With Onset of Sulfonylurea-Induced Hypoglycemia >8 Hours After Ingestion

Age (mo)	Gender	Sulfonylurea	Time of Ingestion	First Recorded Episode of Hypoglycemia	Onset of Hypoglycemia (h)	Glucose Nadir (mg/dL)	Decontamination	Other Treatments (besides food) Before Onset of Hypoglycemia
20	F	Glyburide	5:30 PM	3:00 AM	9.5	59	Activated charcoal	Octreotide 12 μg SC given at 1:50 AM for glucose 64 mg/dL
>24	F	Glipizide	5:00 PM	4:45 AM	11.75	40	None specified	None specified
21	F	Glipizide	7:00 PM	8:00 AM	13	46	None specified	None specified
18	F	Glipizide	6:00 PM	8:00 AM	14	53	Induced vomiting at home	None specified
>24	F	Glimepiride	6:00 PM	8:00 AM	14	40	Activated charcoal	None specified
22	F	Glipizide	2:30 PM	8:30 AM	18	48	None specified	"Monitored and released" by first health care facility
12	M	Glipizide	7:30 AM	7:30 PM	12	38	None specified	None specified

F indicates female; SC, subcutaneously; M, male.

mia up to 7 hours after ingestion. Although ideally this observation could be verified prospectively, it would pragmatically and ethically difficult to withhold food from very young children for >3 or 4 hours. Therefore, in children who have potentially ingested a sulfonylurea, we do not believe there is a "fasting trial" that would be both practical and able to reliably provide a safe disposition. Second, children seem particularly vulnerable to significant drug-induced hypoglycemia during the "overnight fast." Six of the 7 children receiving prophylactic food only who had an onset of hypoglycemia >8 hours after ingestion did so overnight or in the early morning. In addition, patients with multiple episodes of hypoglycemia often had repeated episodes overnight, even while receiving continuous dextrose infusions. Even if serious complications such as seizures or coma are rare, it does not seem reasonable to subject a child to the risk of having these at home during the night when their caregivers may be asleep. Finally, we believe the downsides of a conservative approach are minimal. Most concerns center around excessive resource utilization.[28] Although a common exposure, the actual incidence of pediatric ex-

ploratory sulfonylurea exposures per health care facility is small. A large, tertiary care children's hospital that routinely admits pediatric sulfonylurea exposures reported an average of <2 such admissions per year over a 7-year period.[23]

This study raises a number of unanswered questions in the management of pediatric sulfonylurea ingestions. Does activated charcoal or octreotide affect hypoglycemia frequency, duration, or nadir? Are episodes of hypoglycemia during the "overnight fast" caused by peaking drug levels or insufficient gluconeogenesis? Does a patient's weight or age affect the effectiveness of specific treatment modalities and strategies? What, if any, treatment errors occur in patients experiencing multiple episodes of hypoglycemia? Unfortunately, our data were not collected in a way that lent itself to these analyses.

Finally, it is worth emphasizing that the most effective strategy in pediatric exploratory pharmaceutical exposures is prevention. The medical community should continue to engage parents, caretakers, and industry in devising strategies to further decrease unintentional pharmaceutical access to children.

Limitations

Because our study was a retrospective, observational study, it has some limitations. Our patient population consisted of a convenience sample of patients who were referred to the CPCS by family members or health care providers. Because the CPCS does not capture all consecutive exposure cases at individual health care facilities, this patient population may misrepresent the incidence of exposure and clinical effects. Hospital and clinic medical records were not available for review, limiting our observations to verbal reports to our telephone hotline staff by family members and health care providers. Thus, the VDL record might underreport signs, symptoms, clinical events, and treatments. In addition, as an observational study, treatments were not controlled. Patients received varying combinations of decontamination, food, intravenous glucose, and octreotide. Although we can make generalized comparisons, we cannot make more precise comparisons of treatment measures, such as total caloric intake.

We took significant steps to mitigate error in the estimation of time to onset of hypoglycemia. Patients in this suba-

nalysis were included only if the child was brought promptly to the hospital after a witnessed or suspected ingestion so that accurate timing of glucose measurements would be recorded. We adhered to a detailed protocol for time estimation, including an arbitrary correction factor that we believed would overcorrect for delays in recorded time points. The subset of patient records that contained the most contro- versial time estimations (ie, >8-hour delay in onset of hypoglycemia) were reviewed by a second investigator (Dr Olson), who reported an excellent intraclass correlation coefficient.

CONCLUSIONS

Pediatric sulfonylurea exposures can result in significant poisoning with sustained hypoglycemia. Severe effects such as seizures seem isolated to cases of unrecognized sulfonylurea ingestion and delayed presentation. The onset of hypoglycemia after pediatric sulfonylurea ingestion can be delayed by as much as 18 hours by either free access to food or administration of intravenous glucose. We believe that the common 8-hour observation rule allowing free access to food is unreliable and recommend 24-hour or overnight hospital admission for pediatric sulfonylurea exposures.

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