Ethanol Intoxication of Young Children

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Abstract: Ethanol intoxication of infants and young children can be a challenging diagnosis in the pediatric emergency department, and features of the poisoning may differ in comparison with adolescents. The sources of ethanol exposures in this age are varied and include unintentional, malicious, and iatrogenic etiologies. Young children exposed to ethanol often present with mixed clinical signs and symptoms that may not fit the traditional ethanol or sedative-hypnotic toxidrome. Pediatric ethanol intoxications are often managed supportively, and recovery is usually rapid. The purpose of this review is to describe the sources of ethanol poisoning among children 6 years and younger, highlight presenting symptoms and pharmacokinetic considerations unique to this age group, and review management strategies. In addition, published cases of ethanol poisoning due to ingestion among young infants are compiled for presentation.

Key Words: ethanol, infant, ingestion, poisoning

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TARGET AUDIENCE

This CME activity is intended for health care providers who care for young children, including pediatric emergency medicine physicians, pediatric hospitalists, pediatric critical care physicians, general pediatricians, general emergency medicine physicians, family medicine physicians, nurses and nurse practitioners, and pharmacists.

LEARNING OBJECTIVES

After completion of this article, the reader should be better able to:

1. Identify common sources of ethanol and scenarios leading to ethanol exposure in children 6 years and younger.
2. Describe the clinical signs and symptoms of ethanol intoxication in young children.
3. Evaluate a pediatric patient with ethanol toxicity.

Inebriation with alcohol (ethanol) is a common problem among adolescents and adults presenting for emergency care; ethanol misuse and abuse are a major contributor to human morbidity and mortality worldwide. Because of the ubiquitous nature of ethanol in beer, wine, and spirits; in household products; and in pharmaceutical preparations, infants and young children may also be victims of alcohol poisoning. Ethanol poisoning as a cause of altered neurological functioning or metabolic derangement may not be diagnosed unless considered. The finding of alcohol poisoning during infancy or childhood may surprise both family members and clinicians. This review reminds clinicians that pediatric poisoning with ethanol occurs and is a clinical challenge. Sources and pathways of pediatric exposure to ethanol are presented, previously observed clinical effects are detailed, and management considerations are reviewed (*The research methodology for this review is presented in a footnote to this article).

SOURCES OF ETHANOL

Food and Beverages

Alcoholic beverages are the most recognizable source of ethanol and are widely available. Ethanol is also found in cooking wines, vinegars, flavor extracts, and fermented foods. Even foods considered to be nonalcoholic, such as breads, yogurts, and fruit juices, may contain small amounts of ethanol.[1] Among products that are not considered alcoholic beverages, the percentage of ethanol ranges from trace amounts to greater than 35% ethanol by volume in vanilla extracts.[1,2]

Household and Consumer Products

Given its utility as an antiseptic and an organic solvent, ethanol is present in many household products. Major ethanol-containing product categories include cosmetics, personal hygiene products, and cleaning products.[3,4] The percentage of ethanol can vary widely based on product type and brand, ranging from 0.1% to 100%.[3] Some of these products are rare sources of intoxication given their lack of palatability, difficulty of access, or small dispensable volume. However, several products have been recognized as potentially hazardous to young children—perfumes,[5–7] colognes,[7] mouthwashes,[8–10] hand sanitizers,[11–15] and reed diffusers.[16]

Medications and Health Care Settings

Ethanol is used as an excipient in over-the-counter and prescription medications. The American Academy of Pediatrics[17] and the United States Food and Drug Administration[18] have introduced guidelines in recent decades that have led manufacturers to shift toward producing reduced alcohol or alcohol-free over-the-counter medications. However, liquid formulations of some products still contain up to 10% ethanol by volume.[3,19] Prescription products, including forms of furosemide, morphine, and potassium chloride,

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*Research methodology for the review: In preparing this review, a search of academic medical literature was performed using 2 databases—PubMed and Embase—from inception until December 31, 2018. Articles were identified for retrieval using the search terms [ethanol *or* ethyl alcohol *or* alcoholic beverage] to identify the substance of interest, *and* [exposure *or* ingestion *or* overdose *or* poisoning *or* toxicity] to identify exposure, *and* that were indexed as involving children with age less than or equal to 6 years. Retrieved articles were limited to those that involved humans and were available as full text in English. Articles were discarded as being misidentified if they only described ethanol exposures among adults, adolescents, or children older than 6 years. Additional targeted searches were performed for ethanol pharmacokinetics and pharmacodynamics. Reference lists of applicable articles were reviewed, and further articles were identified and pursued as appropriate.

contain ethanol in varying amounts.[20–24] Liquid supplements and herbal remedies may also contain ethanol and are not subject to the same oversight or labeling as prescription medications.

Apart from medications, ethanol is found in the health care setting in hand sanitizers, alcohol swabs, and ethanol intravenous catheter "locks." It is used as a sclerosing agent in embolization therapies[25] and historically in alimentation intravenous fluids.[26] Intoxication and adverse effects from ethanol-containing medications and health care–related ethanol sources are of particular concern in premature and term infants.[20–24,26–28]

EXPOSURE MECHANISMS

Unintentional Exposure

Unintentional or exploratory ingestion of ethanol or ethanol-containing substances is a common means of exposure for young children. A potentially vexing problem for pediatric emergency care providers is that clear alcoholic beverages, such as vodka or gin, may be mistaken for water and used to reconstitute infant formula or to dilute juice.[29–35] Risk for this injury mechanism increases when ethanol is stored in an accessible site, such as a refrigerator, or transferred into another container that is either unmarked or labeled as another product. An astute index of suspicion may be needed to consider this exposure pathway, which may be confirmed by thorough medical history, blood alcohol testing, and/or environmental investigation. Published cases of ethanol intoxication through ingestion, by infants younger than 9 months, are presented in Table 1. Older infants and toddlers, exhibiting developmentally appropriate oral and environmental exploratory behaviors, may ingest ethanol when items such as hand sanitizers, mouthwash, or opened alcoholic beverage containers are left in places accessible to a young child.[10,11,13,41–44] Medically complex children may have ethanol-containing substances accidentally administered via a nasogastric or gastric tube.

Breastfeeding infants whose mothers consume ethanol are at risk for exposure. Breastmilk alcohol levels generally parallel maternal blood alcohol levels.[45] Thus, the magnitude of exposure correlates with both maternal alcohol intake and infant breastfeeding frequency. Infants who nurse after their mothers have consumed as few as 1 or 2 alcoholic beverages have been observed to have decreased feeding efficiency, agitation, and poor sleep.[45] Maternal ingestion of larger quantities of ethanol-containing substances while breastfeeding has led to clinically significant intoxication of infants, including altered mental status, decreased reflexes, and gastrointestinal manifestations.[40,45] Similarly, pregnant mothers who consume ethanol peridelivery can expose infants transplacentally, leading to ethanol intoxication in the immediate postnatal period.[46–48]

Intentional Exposure, Abuse, and Neglect

Young children may be intentionally exposed to ethanol or ethanol-containing substances by their parent or guardian. Alcohol has a long history of medicinal use, and some communities may still use alcohol for this purpose.[40,49,50] Alcoholic spirits may be rubbed on infants' gums in a, perhaps, misguided attempt to relieve teething pain. Overuse of alcohol for medicinal purposes could lead to intoxication.

Malicious administration of alcoholic beverages or ethanol-laced substances to a young child is a documented cause of ethanol intoxication.[51–53] Recognizing pediatric poisonings as cases of abuse or neglect is considered more difficult compared with cases of physical abuse, as exposures could be erroneously labeled as unintentional.[51,54] Provider bias may exist with respect to the severity of presentation; clinicians are more likely to refer severe or fatal cases of poisoning to law enforcement and child services.[54] Maintaining a high index of suspicion is, again, critical

TABLE 1. Reported Cases of Nonmalicious Ethanol Intoxication by Direct Ingestion Among Infants 9 Months and Younger Published in Medical Journals

Author	Age	Highest Noted BAC, mg/dL	Ethanol Source	CNS Description	Respiratory Depression?	Blood Glucose <60 mg/dL
Boroughf et al (2015)[36]	4 mo	337	Rum	Normal	No	No
Chikwava et al (2004)[37]	7 mo	183	Vodka	Hyperactive	No	No
Edmunds et al (2014)[32]	9 mo	524	Vodka	Obtunded	No*	No
Fong and Muller (2014)[33]	29 d	301	Gin	Not focusing	No	No
Ford et al (2013)[38]	5 wk	270	Vodka	Obtunded	No	No†
Iyer et al (2009)[29]	3 mo	128‡	Vodka	Lethargy	No	No
McCormick (2013)[34]	8 wk	405	Gin	Floppy tone	Yes	No
McCormick (2013)[34]	2 mo	278	Unknown	Unresponsive	Yes	No
Minera and Robinson (2014)[31]	9 wk	330	Vodka	Dazed	No§	No
Palano et al (2007)[39]‖	30 d	—	Wine	Lethargy	No	No
Suneja et al (2016)[35]	7 mo	234	Vodka	Agitated	No	Yes¶
Zaitsu et al (2013)[30]	15 d	43	Sake	Somnolent	No	No

*Developed respiratory concern after medical administration of lorazepam.

†Blood glucose was 63 mg/dL; given IV dextrose.

‡Measured 19 hours post-ingestion (extrapolated peak level, >400 mg/dL).

§Initial respiratory rate 22; no interventions performed.

‖Palano et al was not available as a full-text English article but is included for completeness.

¶Blood glucose was 58 mg/dL; given IV dextrose.

Hon et al[40] described 2 infants who indirectly developed ethanol intoxication after breastfeeding; this article was excluded from the table.

BAC indicates blood alcohol concentration; CNS, central nervous system; IV, intravenous.

for identifying potential cases of child neglect or abuse related to ethanol poisoning.

Iatrogenic Exposure

Clinicians may be unaware of, or may underestimate, the potential for iatrogenic ethanol exposure. Iatrogenic ethanol exposure can be part of clinical care; even at routine doses, ethanol exposure in young children may be significant. Off-label use of adult formulations of medications may lead to higher than intended ethanol excipient exposure in children.[20] In one study of embolization therapy, post-ethanol administration blood alcohol levels were as high as 0.17 g/100 mL (170 mg/dL) in pediatric patients.[25] Some neonates were noted to have elevated levels of acetaldehyde, a byproduct of alcohol metabolism, after receiving medications with ethanol as an excipient,[21] and others were documented to have received weekly ethanol doses via medications that could be comparable with the recommended limit of adult alcohol consumption.[20] Thus, excessive use or dosing of ethanol or ethanol-containing medications could feasibly lead to ethanol toxicity.[26] Ethanol may be used therapeutically in the treatment of methanol or ethylene glycol poisoning, but in the United States, this practice has largely been replaced by the use of fomepizole.

Ethanol poisoning may also occur owing to medical error. Ethanol locks, if improperly administered, could be delivered into the bloodstream instead of the central catheter device. Medical errors leading to administration of ethanol to mothers in labor has led to transplacental intoxication of infants postnatally.[55] Incorrect use of alcohol gauze has been documented, in rare instances, to cause clinically significant percutaneous ethanol exposure.[28]

EPIDEMIOLOGY

Accurate estimates of ethanol exposures, or intoxications, in young children are difficult, given the variety of potential exposure sources and the challenges in diagnosis. The American Association of Poison Control Centers' National Poison Data System estimates that approximately 1% of reported poison exposures among children 5 years of age and younger involve alcohols, including ethanol.[56] This likely underestimates the percentage of exposures, given that alcohol-containing products and medications are categorized under different National Poison Data System product codes. Also, poison center data are derived from a voluntary reporting system; many pediatric ethanol exposures are likely to go unrecognized or unreported. Single institution experiences from the United States and international sites vary, but multiple studies focused on children and adolescents have identified the hazard of ethanol exposures and intoxication among infants and children 6 years and younger.[57–63]

PHARMACOKINETIC CONSIDERATIONS

An understanding of the pharmacokinetics of ethanol is valuable in framing the clinical presentation and course of an intoxicated child. Infants and young children have higher total body water content compared with adults.[64,65] Hydrophilic substances, including ethanol, have higher volumes of distribution in younger children owing to this difference in body composition. Thus, infants and young children may present with lower plasma concentrations of ethanol compared with adults for a given absorbed dose.[66] Ethanol absorption and subsequent distribution among various organs systems in infants may differ from adults owing to changes in the distribution of cardiac output in the immediate postnatal period,[66–68] potentially leading to atypical clinical presentations.

Ethanol metabolism begins with the oxidation of ethanol to acetaldehyde by several systems—alcohol dehydrogenase (ADH), microsomal ethanol oxidizing systems such as CYP2E1, and catalase (Fig. 1).[66] Acetaldehyde is subsequently oxidized by aldehyde dehydrogenase into acetate via the transformation of nicotinamide adenine dinucleotide (NAD) from its oxidized (NAD+) to reduced (NADH) form. Acetate is further converted to acetyl coenzyme A, which enters the Krebs cycle, where it is eventually broken down into carbon dioxide and water. Alcohol dehydrogenase is considered the main driver in the first step of alcohol metabolism in adults. In infants and younger children, ADH enzymatic activity is only a fraction of that of an adult.[66,69,70] Infants and young children rely on other pathways of alcohol metabolism, such as catalase, which has consequences on the rise of blood alcohol levels as well as the efficiency of alcohol metabolism and clearance.[70]

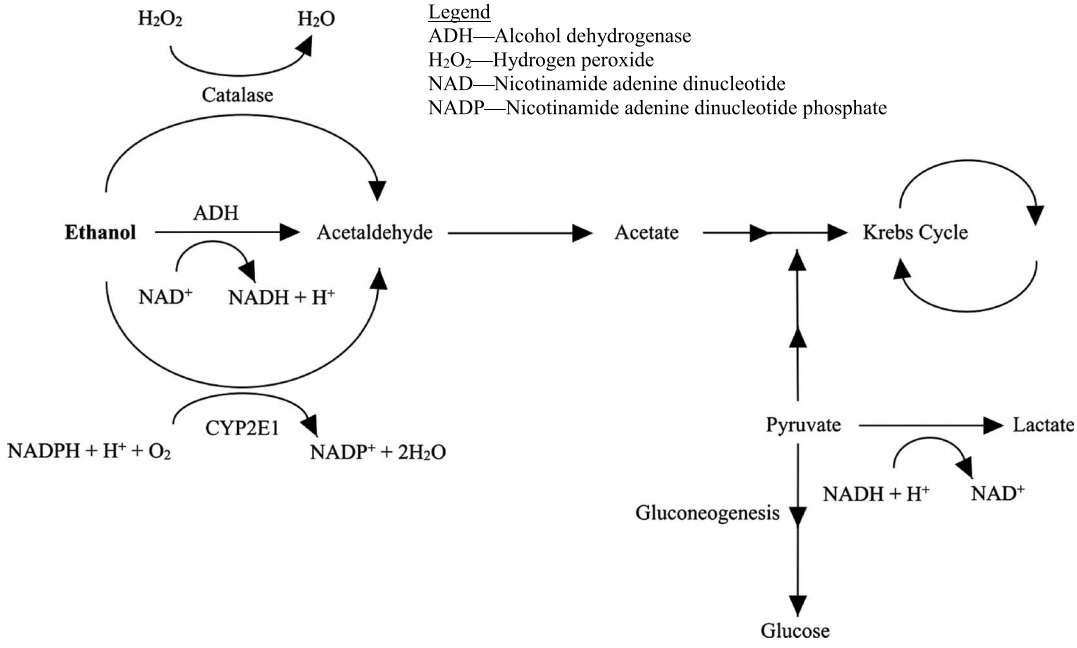

FIGURE 1. Biochemical pathways involved in the metabolism of ethanol and in ethanol-induced hypoglycemia.

Excess alcohol consumption or absorption leads to an imbalance in the biochemical reducing potential of the body secondary to NADH accumulation during metabolism. Consequences of this imbalance include impairment of gluconeogenesis, preferential conversion of pyruvate to lactate, and increase in ketogenesis due to disruption of endocrine homeostasis.[36,71] These biochemical changes contribute to the lactic acidosis, ketoacidosis, and hypoglycemia observed in some intoxicated patients.[71] The neurologic effects of ethanol intoxication may impair feeding, leading to unintended fasting, which can further contribute to hypoglycemia. Infants and young children exposed to ethanol are hypothetically at greater risk for the development of hypoglycemia owing to smaller hepatic glycogen stores.[36,43]

Elimination of ethanol from the body occurs primarily via metabolism, with less than 10% excreted via sweat, urine, or exhalation.[66] In adults, the elimination of ethanol is described by Michaelis-Menten zero-order kinetics in clinically significant exposures.[66,72] Elimination in adults typically occur at a rate of 10 to 25 mg/dL/h for nontolerant individuals.[73] Anecdotally, young children and infants were thought to clear ethanol at faster rates than adults.[36] Case reports present conflicting evidence—several studies describe elimination rates twice as fast as that of adults,[60,62] whereas others report elimination rates similar to the adult range.[33,48,74] Multiple factors affecting elimination rate that have yet to be elucidated likely influence the wide interindividual variation described in the literature.

CLINICAL PRESENTATION

Clinical signs and symptoms of ethanol intoxication in toddler-aged children follow a dose-dependent response, similar to that seen in adolescents and adults. The thresholds for clinically significant and life-threatening intoxications for young children, however, appear to be lower than adults. A serum ethanol level of 50 mg/dL is considered a toxic dose in an infant or young child.[75] Fatal hypoglycemia has been noted in infants with blood ethanol concentrations that have metabolized below 100 mg/dL.[60,76] In contrast, stupor and respiratory depression are not expected in older individuals until blood ethanol concentrations reach 200 to 300 mg/dL.[77] Paradoxically, some clinicians have noted that infants younger than 1 year may demonstrate an incongruence between the neurological examination and the blood ethanol concentration—some young infants with relatively mild central nervous system symptomatology have been found to have surprisingly high blood ethanol concentrations.[36]

Young children exposed to ethanol may be asymptomatic or have minimal clinical effects.[4,7,12,14–16,40] In these cases, the amount of ethanol absorbed is often small. Mild ethanol intoxication may present with nonspecific symptoms, requiring a high index of suspicion for diagnosis. Infants and young children exposed to relatively small amounts of ethanol have been documented to present with gastritis, abdominal distension, or fussiness.[4,40] Infants may present with subtle changes in sleep and feeding patterns without other overt clinical symptoms.[78–80]

More severe presentations of ethanol intoxication in young children are generally consistent with a sedative-hypnotic toxidrome (Table 2). Intoxication can be broadly conceptualized as central nervous system depression leading to altered mental status, along with a concurrent risk of ethanol-induced hypoglycemia given dysregulation of normal biochemical pathways.[36,71,75] Vital sign abnormalities, including hypothermia, hypotension, bradycardia, and respiratory depression, may be observed.[29,30,32,81,82] Initial laboratory testing may be notable for acid-base disturbances, hypoglycemia, and electrolyte derangements (Tables 2, 3).[29,71]

As was noted previously, infants appear less likely to present with the traditional signs or symptoms of ethanol intoxication.[36] Several case studies detail only subtle depression of neurologic

TABLE 2. Signs and Symptoms of Ethanol Intoxication in Infants and Young Children

Organ System Involved	Pathophysiology
Neurologic*	
Somnolence, lethargy, coma, ataxia, decreased tone and reflexes, nystagmus, slurred speech	CNS depression
Seizures	Usually related to hypoglycemia
Cardiovascular	
Bradycardia	Related to CNS depression
Tachycardia	Hypovolemia, possible sympathetic response
Respiratory*	
Respiratory depression	Related to CNS depression
GI	
Emesis/hematemesis, gastritis, abdominal distension	Irritation of GI tract if ingested
Endocrine	
Hypoglycemia†	Dysregulation of endocrine homeostasis, impaired feeding
Fluids/electrolytes	
Hypotension, volume depletion	Inhibition of ADH with polyuria, impaired feeding, peripheral vasodilation
Hypokalemia	In cases of significant emesis
Anion gap metabolic acidosis	Directly related to ethanol toxicity
Environmental/toxin	
Hypothermia	Inhibition of shivering, environmental exposure
Sweet/alcoholic odor on breath	Ethanol elimination via respiration

*Central nervous system depression and respiratory depression may not be observed in the intoxicated infant.

†Hypoglycemia is not always observed in cases of ethanol intoxication.

CNS indicates central nervous system; GI, gastrointestinal.

TABLE 3. Laboratory Testing and Diagnostic Imaging in Young Children Presenting With Suspected Ethanol Intoxication

Laboratory Testing		Diagnostic Imaging	
Test	**Rationale**	**Test**	**Rationale**
Recommended		Adjunct testing	
Blood glucose	Risk of ethanol-induced hypoglycemia	Chest x-ray	If concern for aspiration
Blood ethanol	Determination of exposure severity, estimation of duration of clinical effect	Abdominal x-ray	Evaluation of emesis, hematemesis, or abdominal signs, if present
Serum electrolytes	Risk of metabolic acidosis and hypokalemia, calculation of anion gap	Head computed tomography	If concern for child abuse, head trauma, or acute intracranial process
Arterial/venous blood gas	Evaluation of ventilatory effort, evaluation of acid-base disturbances	Skeletal survey	If concern for child abuse
Adjunct testing		Dilated eye examination	If concern for child abuse
Serum osmolality	Determination of osmol gap		
Liver function tests	Risk of hepatic involvement		
Complete blood count	If concern for infection or sepsis		
Rapid/comprehensive urine drug screen	If concern for coingestion		
Methanol and ethylene glycol levels	If concern for alternative "alcohol" exposure		

tone or mental status in infants,[29,33,38] except in situations with very high blood alcohol levels.[32,34] Respiratory depression among infants is also rarely observed and was noted in only 2 of the cases presented in Table 1.[34] The etiology for these differences is unknown and may be related to limitations of the neurologic examination or relative neurologic immaturity in this age group.[36] Hypoglycemia, a serious and potentially fatal complication of pediatric ethanol intoxication, was described in early case reports.[43,76,83] In the contemporary literature, the relative incidence of hypoglycemia with ethanol intoxication in young children appears to be low.[11,30,32,33,59] Prompt presentation to care after exposure, ingestion of ethanol with glucose or glucose-containing fluids (eg, wine, ethanol mixed with formula), and lack of prior or prolonged fasting are possible explanations for this observation.

Fatalities secondary to ethanol intoxication during childhood are rare but documented (Table 4). Poor prognosis has been noted in cases where there was a delay in seeking care for intoxicated young children. In these cases, the infant or young child presented with symptomatic hypoglycemia, often with progression to hypoglycemic seizure or coma.[10,76]

DIFFERENTIAL DIAGNOSIS

Ethanol intoxication should be considered by emergency care providers in the differential diagnosis of altered mental status, hypothermia, or hypoglycemia. If ethanol intoxication is suspected, providers should first obtain a targeted history of the presenting events to assist in the differential diagnosis and risk stratification of the intoxicated young child. *Alcohol* exposure should be clarified, as the exposure may have been to other toxic alcohols, such as isopropanol, methanol, or ethylene glycol. The likelihood of coexposures to other substances should be ascertained. Obtaining

TABLE 4. Summary of Case Reports and Case Series Characterizing Pediatric Ethanol Intoxication Fatalities in Children 6 Years and Younger

Author	Ethanol Type	Reported Coexposures	Sex	Age
Case et al (1983)[84]	Cologne	Salicylate	M	5 y
Cummins (1961)[76]	Gin	—	M	6 y
Parker (1967)[85]	Gin	—	M	2 y
Peden et al (1973)[26]	Medical grade ethanol in total parenteral alimentation fluid	—	F	Premature neonate
Selbst et al (1984)[10]	Mouthwash	—	M	4 y

CPR indicates cardiopulmonary resuscitation.

history regarding the child's medical problems, home environment, and recent illnesses may help inform the differential diagnosis. For formula-fed infants, a careful detail of how formula is prepared may be enlightening. If ethanol exposure is confirmed, confirming the type of ethanol-containing substance, timing of exposure, and amount ingested can help estimate blood alcohol levels and duration of clinical effect.[86] Time of last enteral intake is useful in assessing risk of hypoglycemia.

Confirmed ethanol exposure combined with characteristic signs or symptoms of intoxication on presentation often is sufficient to diagnose ethanol intoxication. Exposure or coexposure to other toxicants or drugs should be considered (a wide variety of drugs can cause central nervous system depression; other drugs and chemicals associated with hypoglycemia are presented in Table 5). Providers should rule out other etiologies of altered mental status, including metabolic derangements, meningitis/encephalitis, sepsis, seizure, accidental or non-accidental trauma, or structural brain abnormalities, as part of their evaluation of the intoxicated patient.

EVALUATION AND MANAGEMENT

Young children who are asymptomatic after exposure can be observed for onset of symptoms and be discharged home with return criteria if stable and safe disposition exists.[75] Symptomatic patients demonstrating signs or symptoms of ethanol intoxication will likely warrant active medical observation, blood glucose monitoring, and care until sober and risk of metabolic injury has passed. Diagnostic tests should be obtained during initial stabilization and include rapid blood glucose measurement, blood ethanol concentration, measurement of serum electrolytes, and venous blood gas assessment. Additional laboratory testing or imaging may be guided based on clinical suspicion for clinical complications or other diagnoses (Table 3).

The Airway, Breathing, Circulation, Disability, Exposure paradigm, or *ABCs*, used to assess critically ill or injured patients can be applied to cases of ethanol intoxication.[87] Evaluation of the airway involves determining airway patency and presence of airway edema. A patient's breathing, including presence and rate of respirations should be assessed, given the possibility of respiratory depression and acid-base disturbances. Interventions may range from supplemental oxygen and suctioning to endotracheal intubation, depending on the degree of airway and ventilatory compromise.[32,34,41]

TABLE 5. Other Drugs and Chemicals Commonly Implicated in Hypoglycemia in Children

B-adrenergic antagonists (eg, propranolol)
Insulin
Monoamine oxidase inhibitors
Quinine
Salicylate
SGLT2 inhibitors
Sulfonylureas
Thiazolidinediones
Valproic acid

SGLT2 indicates sodium-glucose cotransporter 2.

During assessment of circulatory function, vascular access may be warranted. Given the possibility of hypotension secondary to dehydration and peripheral vasodilation, fluid resuscitation with isotonic fluids may be necessary. Electrocardiographic changes in heart rhythms have been noted in intoxicated adolescents and adults,[88,89] and monitoring for potential arrhythmia may be warranted.

The disability assessment should evaluate the child's level of responsiveness and neurologic function. Ethanol-induced hypoglycemia should be treated aggressively given the risk of life-threatening complications; bolus intravenous dextrose administration and subsequent continuous infusion of dextrose containing fluids are usually warranted. Seizures, if present, can often be addressed by correcting hypoglycemia, unless another underlying etiology is the cause. Antiepileptics may be indicated in cases of status epilepticus. Glucagon administration for ethanol-induced hypoglycemia may be ineffective owing to ethanol-related dysregulation of gluconeogenesis and is not recommended.[90,91] Naloxone, although ineffective for resolving ethanol-related respiratory depression, may be helpful in improving respiratory drive in cases of opioid coingestion.

Evaluation of exposure involves assessment of environmental effects as well as thorough evaluation of the suspected toxidrome. Hypothermia is of concern in intoxicated young children,[10,11,13,29] and patients should receive external warming as needed to maintain normothermia. Ambiguous or mixed presentations should raise

Exposure Mechanism	Presenting Signs/Symptoms	Hypoglycemia Present	Advanced Therapies	Outcome
Unintentional ingestion—child with access to container	Cardiorespiratory arrest	—	CPR	Death
Unintentional ingestion—child with access to container	Active clonic convulsions, Cheyne-Stokes respirations, pinpoint pupils	Yes	Anticonvulsants, mechanical ventilation	Death
Unintentional ingestion—child with access to container	Altered mental status, coma, convulsions, hyperthermia, tachycardia, respiratory distress,	—	Anticonvulsants, mechanical ventilation, inotropic support	Death
Iatrogenic administration	Altered mental status, edema	—	Discontinuation of alimentation infusate	Death (unknown if related to intoxication)
Unintentional ingestion—child with access to container	Altered mental status, respiratory depression, hypothermia, tonic clonic seizures, decorticate/ decerebrate posturing	Yes	Anticonvulsants, mechanical ventilation	Death

suspicion of coingestion or an alternative diagnosis. Additional diagnostic testing or imaging should be pursued as clinically indicated.

After initial stabilization, care of the intoxicated young child is primarily supportive until ethanol is cleared from the body. Most young children recover rapidly within 24 hours[30,32,33,81] and without neurologic sequelae.[30] Gastric decontamination, via lavage, induced emesis, or activated charcoal, is not recommended, given rapid alcohol absorption after exposure and risk of aspiration.[75,92] Hemodialysis may be considered in cases of severe ethanol intoxication, with extremely high blood alcohol concentration and acidemia, but its benefits over supportive care in ethanol intoxication have not been definitively demonstrated.[93,94] Referral to child protective services or law enforcement may be warranted to determine safe disposition if there is suspicion of neglect or intentional administration.

SUMMARY

- Infants and young children are at risk of ethanol intoxication; caregivers need to maintain an index of suspicion when evaluating children with altered mental status.
- Young infants have been observed to maintain surprising levels of alertness despite high blood alcohol concentration.
- Improper storage of clear alcoholic spirits in alternative beverage containers has been a repeatedly documented route of exposure for ethanol poisoning among infants.
- With decreased hepatic glycogen stores, young children are at risk of hypoglycemia owing to inhibited gluconeogenesis during the metabolism of ethanol.
- Case reports of ethanol intoxication among children 6 years and younger are summarized in tabular form in the appendix available in the online version of this article (Table 6, Supplemental Digital Content, http://links.lww.com/PEC/A477).

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