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"Poison Severity Score of Accidental Acute Pediatric Intoxication among Cases Presenting to the Pediatric Department in Damietta General Hospital "

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ABSTRACT:

Pediatric intoxication, due to the high vulnerability of children and its frequent occurrence, is a serious worldwide health concern. One of the main causes of morbidity and mortality in children is acute poisoning; younger children are more likely to experience accidental poisoning, whereas adolescents are more likely to experience purposeful poisoning. There are regional, social, and age-related variations in poisoning patterns that occur all around the world. Effective management of pediatric poisoning requires understanding the pharmacokinetics and pharmacodynamics specific to children, which differ substantially from adults. Despite advancements in the understanding of pediatric toxicology, challenges remain in the accurate diagnosis and management of poisoning in children due to the variability in symptoms and the need for age-specific treatment protocols. Regular surveillance and updated research are crucial to improve prevention strategies and therapeutic outcomes in pediatric poisoning cases.

The aim of this study is to contribute to the global understanding and improvement of pediatric care by evaluating and enhancing the use of the Poison Severity Score (PSS) in acute pediatric intoxication cases at Damietta General Hospital, with the broader goal of reducing childhood morbidity and mortality worldwide.

Keywords: Pediatric, Acute intoxication, Poison severity score, Pediatric emergency.

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Introduction

Acute toxicity is the process by which cells are harmed or destroyed by the inhalation, ingestion, injection, or absorption of a hazardous chemical. It is a major health concern as it is one of the primary causes of morbidity and mortality globally [1].

Acute pediatric intoxication is still a major global health issue that poses a risk of morbidity and mortality and requires emergency room treatment. A 2008 World Health Organization (WHO) research estimated that acute poisoning accounts for 45,000 deaths annually among adolescents and young adults (under 20). 40% of toddlers under the age of three were among the over 1.3 million youngsters exposed to toxins in 2015, according to the American Association of Poison Control Center (AAPCC) [2].

Information about common poisoning compounds, common history elements, and the progression and effects of the poison in the body are helpful when evaluating patients. Decontamination, improved elimination, antidotes, and supportive care are frequently employed to treat poisoning instances [3].

Medical malpractice cases (MLC) involve doctors deciding to investigate further by law enforcement agencies, following local laws and health department directives. Causes and types of poisoning vary globally, influenced by factors like customs, demography, socioeconomic status, and education level [1].

ED doctors always have trouble treating children's poisoning because the demographics and etiology might change over time, even within the same locale. Regular surveillance is necessary to identify trends in certain agents and other factors linked to kid poisoning. This aids in the development of preventative measures and enables emergency department doctors to accurately diagnose and treat poisoning according to age and time [4].

Poisoning can be categorized as (0) none, (1) mild, (2) moderate, (3) severe, or (4) fatal based on the Poisoning Severity Score. It seeks to offer a thorough study of the circumstances, taking into consideration the most severe clinical manifestations [5].

Epidemiology of pediatric intoxication

Global estimates show that 13% of accidental poisoning deaths occur in children under 18, with mortality rates four times higher in developing countries. In the US, 37.4 poison exposures occurred in children under six years, with 43% of exposures unintentional [6].

Europe faces limited updated data on poisoning, but 3000 young children die annually from acute intoxication, with children aged 0-14 being particularly vulnerable. In India, accidental intoxication is the leading cause of hospital admission and emergency pediatric admissions. In 2016, only 17.2% of 58 African countries had poison information centers, with Eastern Africa reportedly accounting for 1,128,500 cases and 16,500 deaths due to unintentional intoxication [7].

Toxicokinetics and toxicodynamics in the pediatric population

Pediatric drug toxicokinetics and toxicodynamics are significantly impacted by physiological changes during childhood, necessitating age-specific classifications such as those outlined by the International Conference on Harmonization (ICH) E11. These classifications segment the pediatric population into groups ranging from preterm newborns to adolescents. Differences in absorption, distribution, metabolism, and excretion (ADME) processes between children and adults influence drug concentrations and their toxicokinetic profiles, making it crucial to understand these variations for effective pediatric therapy. For instance, neonates have distinct absorption and metabolism patterns due to factors like higher gastric pH, immature bile secretion, and different enzyme activity. Additionally, the volume of drug distribution (Vd) varies due to changes in body composition, while protein binding differences affect free drug concentrations. Metabolic enzyme activity also evolves with age, impacting drug metabolism, with enzymes like CYP450 playing a significant role. Finally, renal elimination is less efficient in newborns but can be more effective in older children, with drug

elimination influenced by factors like glomerular filtration rate and urinary pH. Understanding these complex, age-related physiological differences is essential for optimizing drug therapy in pediatric patients **[8].**

Toxicodynamics involves the physiological and biological responses to a drug or toxin. Pediatric dose selection aims to achieve similar internal drug exposure and pharmacodynamic effects as in adults. Still, adjustments must consider the specific drug properties and the developmental stage of the child. Most pediatric dosing methods rely on simple algorithms that extrapolate from adult doses based on body weight or surface area, but no single algorithm is suitable for all age ranges. In some cases, where pediatric dosage information is lacking, scaling from adult doses is necessary, requiring careful consideration of the drug's therapeutic index, toxicity profile, and the child's age. The FDA's pediatric guidelines emphasize understanding the similarities between disease progression and drug response in adults and children. Specific drug effects in children include heightened susceptibility to respiratory depression, hypoglycemia, cardiovascular collapse, QT prolongation, and paradoxical reactions to certain sedatives and antihistamines, particularly in infants and young children [9].

Approach to management of intoxicated pediatrics

History taking

Obtaining a thorough history from the patient or caregiver is crucial in cases of potential poisoning, though it can be challenging. Key questions include what was ingested, when, the amount, and any other available toxins, as well as what medications or products are present in the home. However, young children who have been poisoned often present to the emergency department with nonspecific symptoms rather than a clear history of toxin exposure. Indicators of possible hidden poisoning include the child's age (1-5 years), sudden onset of symptoms, multiple organ dysfunction, and altered mental status. Environmental and social history can also provide clues, such as recent exposure to new medications, visits to less childproofed homes, or high-risk situations like holiday gatherings. An inconsistent history or a concerning past medical or family history may suggest the possibility of intentional poisoning **[10].**

Physical examination

Physical examinations for poisoned pediatric patients should focus on vital signs, capillary perfusion, central and autonomic nervous system findings, pupillary size, and skin abnormalities. A detailed neurological assessment is essential for substance identification. Evaluation and treatment of airway, breathing, and circulation are mandatory. Detection of characteristic odors and classic clinical findings (toxidromes) are common in young children **[11].**

Laboratory investigations and ECG evaluation

Rapid overdose toxicology and drug-level issues apply to toddlers, adolescents, and adults. Toxicology screens have limited value in emergency management, especially for toddlers with witnessed ingestion. ECGs should be conducted for substances causing cardiac arrhythmias or where there is a significant possibility of their ingestion. Many drug classes result in sodium channel blockade or potassium efflux channel blockade [11].

Acid-base balance disturbances can be caused by various poisons, and blood gas analysis is recommended in cases of deliberate poisoning or suspected poisoning. X-rays may be used in radio-opaque drugs like chloral hydrate, calcium, opiates, iron, neuroleptic agents, and sustained-release preparations. In the UK, urine toxicology screens identify certain medications, but their role is limited and has not improved outcomes in recreational drug patients [10].

Assessment

Emergency physicians use clinical evaluation, laboratory data, and ECG interpretation to determine the possible severity of poisoning in children with known exposure. Commonly used standardized approaches include the Poison Severity Score, Toxic Exposure Surveillance System

(TESS), and Matthew-Lawson scale. Practitioners seeking aid for occult poisoning could consult with a local toxicology consultant or poison control center [12].

Poison Severity Score

The Poisoning Severity Score (PSS) was developed by the International Program on Chemical Safety (IPCS), the European Community (EC), and the European Association of Poisons Centers and Clinical Toxicologists (EAPCCT) as a qualitative tool to evaluate the morbidity caused by various forms of poisoning. It grades poisoning severity based on the most serious symptoms observed without considering factors like the amount of toxin ingested or serum concentrations [13].

The PSS is applicable to all types of poisonings, though it requires assessing multiple symptoms and signs. Treatment measures are not graded but may assist in evaluating severity, while preventive antidote use should not affect the grading. The PSS is primarily intended for acute poisoning, but chronic consequences like disabling sequelae or disfigurement may warrant a higher severity grade. Severe cases, including fatalities, are graded separately for accurate data representation, and the patient's medical history should be considered if it influences the severity of the poisoning [14].

Severity Grades classification [5].

NONE (0): No symptoms or signs related to poisoning
MINOR (1): Mild, transient, and spontaneously resolving symptoms
MODERATE (2): Pronounced or prolonged symptoms
SEVERE (3): Severe or life-threatening symptoms
FATAL (4): Death.

Table (1): Associations Between Clinical Conditions and Poisoning Severity Scores in Pediatric Patients. [5]:

Organ	Minor (1)	Moderate (2)	Severe (3)
Description	Mild, transient, and spontaneously resolving symptoms or signs	Pronounced or prolonged symptoms orsigns	Severe or life-threatening symptoms orsigns
GIT	- Vomiting, diarrhea, pain - Irritation, 1st degree burns, minimal	- prolonged vomiting, diarrhea, pain, ileus - 1st-degree burns of critical localization or 2nd and 3rd	- Massive hemorrhage, perforation - More widespread 2nd and 3rd degree burns
Organ	**Minor (1)**	**Moderate (2)**	**Severe (3)**
GIT	-ulcerations in the mouth -Endoscopy: erythema, edema	-degree burns in restricted areas -Dysphagia -Endoscopy: ulcerative transmucosallesions	-Severe dysphagia -Endoscopy: ulcerative transmural lesions, circumferential lesions, perforation
Respiratory system	-Irritation, coughing, breathlessness,mild dyspnea, mild bronchospasm -Chest X-ray: abnormal with minor orno symptoms	-Prolonged coughing, bronchospasm, dyspnea, stridor, hypoxemia requiring extra oxygen -Chest X-ray: abnormal with moderate symptoms	-Manifest respiratory insufficiency (due to, e.g., severe bronchospasm, airway obstruction, glottal edema, pulmonary edema, ARDS, pneumonitis, pneumonia, pneumothorax) - Chest X-ray: abnormal with severe symptoms

Nervous system	-Drowsiness, vertigo, tinnitus, ataxia -Restlessness -Mild extrapyramidal symptoms -Mild cholinergic/ anticholinergic symptoms -Paresthesia -Mild visual or auditory disturbances	-Unconsciousness with appropriate response to pain -Brief apnea, bradypnea -Confusion, agitation, hallucinations, delirium -Infrequent, generalized, or focal seizures -Pronounced extrapyramidal symptoms -Pronounced cholinergic/ anticholinergic symptoms -Localized paralysis not affecting vital functions -Visual and auditory disturbances	-Deep coma with inappropriate response topain or unresponsive to pain -Respiratory depression with insufficiency -Extreme agitation -Frequent, generalized seizures, status epilepticus, opisthotonos -Generalized paralysis or paralysis affecting vital functions -Blindness, deafness
Organ	**Minor (1)**	**Moderate (2)**	**Severe (3)**
Cardiovascular system	-Isolated extrasystoles - Mild and transient hypo/hypertension	-Sinus bradycardia (HR ~40-50 in adults, 60-80 in infants and children, 80-90 in neonates) -Sinus tachycardia (HR ~140-180 in adults, 160-190 in infants and children, 160-200 in neonates) -Frequent extrasystoles, atrial fibrillation/flutter, AV-block I-II, prolonged QRS and QTc-time, repolarization abnormalities -Myocardial ischemia - More pronounced hypo/hypertension	-Severe sinus bradycardia (HR ~<40 in adults, <60 in infants and children, <80 in neonates) -severe tachycardia (HR ~>180 in adults, >190 in infants and children, >200 in neonates) -Life-threatening ventricular dysrhythmias, AV block III, asystole. -Myocardial infarction - Shock, hypertensive crisis
Metabolic	-Mild acid-base disturbances (HCO_3 ~15-20 or 30-40 mmol/l;pH~7.25-7.32 or 7.50-7.59) -Mild electrolyte and fluid disturbances (K^+ 3.0-3.4 or 5.2-5.9mmol/l) -Mild hypoglycemia (~50-70 mg/dl or 2.8-3.9 mmol/l in adults) - Hyperthermia of short duration	-More pronounced acid-base disturbances (HCO_3 ~10-14 or >40 mmol/l; pH ~7.15-7.24 or 7.60-7.69) -More pronounced electrolyte and fluid disturbances (K^+ 2.5-2.9 or 6.0-6.9 mmol/l) -More pronounced hypoglycemia (~30-50 mg/dl or 1.7-2.8 mmol/l in adults) - Hyperthermia of longer duration	-Severe acid-base disturbances (HCO_3 ~<10 mmol/l; pH ~<7.15 or >7.7) -Severe electrolyte and fluid disturbances (K^+ <2.5 or >7.0 mmol/l) -Severe hypoglycemia (~<30 mg/dl or 1.7 mmol/l in adults) - Dangerous hypo- or hyperthermia
Liver	- Minimal rise in serum enzymes (ASAT, ALAT ~2-5 x normal)	- Rise in serum enzymes (ASAT, ALAT ~5-50 x normal) but no diagnostic biochemical (e.g., ammonia, clotting factors) or clinical evidence of liver	- Rise in serum enzymes (~>50 x normal) or biochemical (e.g., ammonia, clotting factors) or clinical

		dysfunction	evidence of liver failure
Kidney	- Minimal proteinuria/hematuria	- Massive proteinuria/hematuria - Renal dysfunction (e.g., oliguria, polyuria, serum creatinine of ~200-500 µmol/l)	- Renal failure (e.g. anuria, serum creatinine of >500 µmol/l)

Organ	**Minor (1)**	**Moderate (2)**	**Severe (3)**
Blood	-Mild hemolysis - Mild methemoglobinemia (metHb ~10-30%)	-Hemolysis -More pronounced methemoglobinemia (metHb ~30-50%) -Coagulation disturbances withoutbleeding - Anemia, leukopenia, thrombocytopenia	-Massive hemolysis -Severe methemoglobinemia (metHb >50%) -Coagulation disturbances with bleeding - Severe anemia, leukopenia, thrombocytopenia
Muscular	-Mild pain, tenderness - CPK ~250-1,500 iu/l	-Pain, rigidity, cramping and fasciculation - Rhabdomyolysis, CPK ~1,500-10,000 iu/l	-Intense pain, extreme rigidity, extensive cramping, and fasciculation -Rhabdomyolysis with complications, CPK ~>10,000 iu/l - Compartment syndrome
Skin	- Irritation, 1st degree burns (reddening) or 2nd degree burns in <10% of body surface area	- 2nd-degree burns in 10-50% of body surface (children: 10-30%) or 3rd-degree burns in <2% of body surface area	- 2nd degree burns in >50% of body surface (children: >30%) or 3rd degree burns in >2% of body surface area
Eye	- Irritation, redness, lacrimation, mild palpebral edema	-Intense irritation, corneal abrasion - Minor (punctate) corneal ulcers	-Corneal ulcers (other than punctate), perforation - Permanent damage
Local effects from bites and stings	-Local swelling, itching - Mild pain	-Swelling involving the whole extremity,local necrosis - Moderate pain	-Swelling involving the whole extremity and significant parts of the adjacent area, more extensive necrosis -Critical localization of swelling threatening the airways - Extreme pain

Common pediatric intoxications (clinical picture and management).

1- OTC preparations

- **Paracetamol**

Paracetamol poisoning is the most common pediatric poisoning seen in emergency departments, though most cases do not result in toxicity. Paracetamol is rapidly absorbed, especially in liquid form, and is metabolized primarily in the liver, with a small percentage forming the toxic metabolite NAPQI. In therapeutic doses, NAPQI is detoxified by glutathione, but in overdoses, it accumulates, leading to hepatotoxicity. Children are generally less susceptible to the hepatotoxic effects of acute overdose compared to adults, possibly due to differences in metabolism and a higher tendency to vomit after ingestion [15].

In cases of overdose, acetylcysteine is the preferred antidote, especially when administered within 8 hours. For chronic overdosing or repeated supratherapeutic ingestion, which is more concerning, the standard treatment guidelines are less applicable, and these cases often require consultation with a toxicologist. Activated charcoal may be used in specific situations, and late presentation still warrants treatment to improve survival chances. Additionally, education on proper dosing and storage of paracetamol is crucial for prevention, and mental health assessments are recommended for deliberate self-poisoning cases [16].

Salicylates

The incidence of acute salicylate poisoning has decreased due to improved medication packaging, the removal of aspirin from pediatric formulations, and the preference for paracetamol as an over-the-counter analgesic. Salicylates are available in various forms, including tablets, powders, and dermal preparations, which can cause systemic toxicity. Aspirin is absorbed from the gastrointestinal tract, with enteric-coated tablets taking longer to reach peak serum concentrations. In overdoses, salicylate absorption kinetics may be altered, leading to higher free salicylate levels as plasma protein binding becomes saturated. Salicylate poisoning disrupts oxidative phosphorylation, causing lactic acidosis, especially in young children, and presents with respiratory alkalosis, confusion, seizures, and mixed acid-base derangements [17].

Treatment focuses on stabilizing the patient, limiting absorption, enhancing elimination (e.g., with urinary alkalinization using IV sodium bicarbonate), and managing metabolic abnormalities. In severe cases, extracorporeal removal of salicylates and hemodialysis may be necessary, particularly if acute pulmonary edema or seizures develop. Symptomatic children or those with significant ingestions require observation and possibly hospital admission [18].

Non-steroidal anti-inflammatory drugs

Ibuprofen is the most common NSAID consumed by children, causing low-toxicity symptoms like headache, dizziness, tinnitus, and visual disturbances. Massive overdose can cause electrolyte disturbances, metabolic acidosis, central nervous system depression, and respiratory failure. Symptomatic children require hospital admission and monitoring of electrolytes, blood glucose, renal function, and acid-base status [19].

Antihistamines

Antihistamines are essential medications for treating hyperhistaminic conditions in children, but they can induce sedation and affect learning ability. Management is supportive, and anticholinergic effects are generally not life-threatening. Hypotension, convulsions, and delirium can be managed with IV fluids, benzodiazepines, continuous ECG monitoring, and sodium bicarbonate for ventricular arrhythmias [20].

Decongestant drugs for cold and flu.

High-dosage ingestion of pseudoephedrine can cause physical symptoms like decreased appetite, dry mouth, palpitations, and motor disorders. Dosage-dependent effects range from euphoria to psychotic symptoms [21].

2- Household chemicals

Organophosphates and carbamates Insecticides

Common and inexpensive pesticides known as organophosphate chemicals (OPCs) block acetylcholinesterase (AChE), which can cause systemic sickness when exposed to high concentrations of the substance. These substances influence cholinesterases other than synaptic acetylcholinesterase (S-AChE), such as butylcholinesterase (BChE) and erythrocytecholinesterase (E-AChE).

Organophosphate poisoning results in a cholinergic toxidrome that includes coma, convulsions, psychosis, and neuromuscular dysfunction, in addition to profuse secretions [22].

Early signs in youngsters include hypotonia, unconsciousness, and lethargy; severe instances worsen quickly. In order to combat muscarinic symptoms, initial care entails decontamination, atropine injection, and cardio-respiratory stability. Though its efficacy is debatable, oxime therapy, such as pralidoxime, may aid in the restoration of respiratory function. Children exposed to organophosphates should be observed for at least 12 hours; those exhibiting severe symptoms should receive immediate medical attention [23].

Warfarin and Rodenticides

Domestic rodenticides, primarily containing superwarfarins, pose a management challenge due to their combination of short-acting and long-acting superwarfarins. Short-acting warfarin ingestion in children is concerning at doses greater than 0.5 mg/kg. Treatment depends on dose, prothrombin time, and bleeding signs. Coagulopathy occurs in massive or chronic ingestions, requiring therapy with vitamin K and serial coagulation tests. Coagulopathy may not manifest until 48 hours post-exposure [24].

Corrosive ingestions

Caustic ingestions, often involving strong acids and alkalis, can cause severe gastrointestinal and respiratory tract injuries, affecting the esophagus, pharynx, larynx, and mouth. Symptoms can range from pain and drooling to airway compromise and potential esophageal perforation. CT scans or endoscopy are crucial for assessing injury severity [25].

Complications include esophageal strictures, with 80% developing within two months of ingestion, and a significantly increased risk of esophageal carcinoma decades later. Immediate treatment involves securing the airway, IV fluids, and avoiding vomiting. Surgical intervention and antibiotics may be necessary for suspected perforations, while steroids are not recommended. Patients with severe burns should be closely monitored and may require intensive care [26].

Toxic alcohols

Ethanol, commonly found in household products and alcoholic beverages, is frequently ingested by children but rarely causes severe toxicity. Toxic alcohols like ethylene glycol (in antifreeze) and methanol (in various household products) pose greater risks. These substances are rapidly absorbed and metabolized, with methanol being particularly dangerous due to its conversion to toxic formic acid, which can cause severe metabolic acidosis, visual disturbances, and potentially lethal outcomes [27].

Ethylene glycol's toxicity stems from its metabolites, leading to CNS depression, metabolic acidosis, and renal damage. Children are especially vulnerable to ethanol-induced hypoglycemia due to inhibited gluconeogenesis. Management involves the use of antidotes like fomepizole or ethanol, which inhibit the conversion of methanol and ethylene glycol to their toxic metabolites. Hemodialysis is indicated in severe cases. Monitoring of serum ethanol levels, glucose, electrolytes, and acid-base status is crucial, and symptomatic children should be admitted to the PICU for observation and treatment [28].

kerosine

Kerosine, a substance found in foods, can cause chemical pneumonitis in children who ingest it. This can lead to irritation in the lung airways, causing loss of lung surfactant and potentially necrotizing hemorrhagic pneumonitis. Symptoms include fever, vomiting, cough, decreased breath sounds, crepitations, drowsiness, stupor, and convulsions. Treatment involves oxygen support, respiratory monitoring, and fluid balance monitoring [29].

Phenol (carbolic acid)

Phenol, a disinfectant and chemical precursor, can cause acute phenol toxicity through unintentional exposure. Its protoplasmic properties allow it to break through cellular membranes, leading to cell death and necrosis. Management should focus on supportive measures like patent airway, ventilation, oxygenation, and hemodynamic support. Activated charcoal is not recommended for gastrointestinal decontamination due to risks of aspiration and confusion with endoscopic findings [30].

Essential oils

Essential oils, derived from plants like cloves, eucalyptus, lavender, and tea tree, can be highly toxic and cause vomiting, chemical pneumonitis, and respiratory complications. Treatment involves supportive care, airway management, and benzodiazepines for seizures. Asymptomatic patients may be discharged [31].

Naphthalene

Naphthalene, a key ingredient in mothballs, poses significant toxicity risks, especially to children. Even a single mothball can cause poisoning. Ingestion can cause symptoms like headache, vomiting, diarrhea, and severe complications like acute intravascular hemolysis, especially in children with glucose-6-phosphate dehydrogenase (G6PD) deficiency. Treatment focuses on managing anemia, fluid balance, and preventing kidney damage. No specific antidote is available [32].

Nitrates

Nitrate, found in environmental media and some medicines, can cause methemoglobinemia, which can cause severe symptoms like gastroenteritis, abdominal pain, and blood in urine and feces. Patients with asymptomatic methemoglobinemia require supportive care, while those with significant oxygen delivery issues may need antidotal treatment. Methylene blue is an effective antidote for most patients [33].

3- Low therapeutic index drugs (Dangerous drugs)

Digoxin

Digoxin, a cardiac glycoside used to treat heart failure and arrhythmias, can cause toxicity in children, especially accidental intake. Chronic toxicity may result from overdosing or renal impairment, leading to arrhythmias and hyperkalemia. Management involves monitoring ABCs, blood pressure, ECG, and serum digoxin and potassium levels. Activated charcoal may be used, though repeated vomiting may limit its efficacy. Hyperkalemia is treated with sodium bicarbonate and insulin/glucose, avoiding calcium due to the risk of myocardial tetany. The specific antidote, digoxin Fab antibodies, is used in cases of severe toxicity, with a clinical response typically seen within 20-30 minutes. Patients should be observed for at least 12 hours, and those showing symptoms or instability should be monitored in intensive care [34].

Lithium

Lithium is a common treatment for bipolar disorder, but toxicity can occur due to minor changes in medications or health status. Symptoms include nausea, vomiting, cramping, and diarrhea, with neuromuscular signs and cardiac dysrhythmias rarely occurring. Supportive therapy is crucial, with airway protection and seizures controlled with benzodiazepines, phenobarbital, or propofol. Serial lithium levels should be obtained every 6 hours, and patients should not be discharged until asymptomatic **[35].**

Tricyclic antidepressants (TCAs)

Tricyclic antidepressants pose a significant pediatric toxicity risk due to their low therapeutic index and potentially lethal outcomes despite declining prescriptions. Overdoses can lead to severe toxicity, including anticholinergic effects, cardiac dysrhythmias, seizures, and coma. Management involves securing the airway, continuous cardiac monitoring, and GI decontamination. Sodium bicarbonate is the antidote for cardiotoxic effects, while benzodiazepines are preferred for seizure control. Patients with significant toxicity require intensive care monitoring, and those ingesting over 5 mg/kg should be observed for at least 6 hours [36].

Aminophylline (Theophylline)

Theophylline, a methylxanthine once widely used to treat asthma, still causes significant morbidity and mortality due to its narrow therapeutic window despite decreased usage. Severe theophylline toxicity can lead to life-threatening seizures or arrhythmias that are resistant to standard treatments and cause hemodynamic instability. In contrast, milder toxicity may involve vomiting, tremors, or cardiac disturbances without instability. Treatment is guided by clinical symptoms and blood theophylline levels, with activated charcoal used for decontamination based on the level of theophylline in the blood [37].

Carbamazepine (Tegretol) ®

Carbamazepine is a common antiepileptic medication used in adults and children. It can cause arrhythmias, anticholinergic toxidrome, and central nervous system depression after an acute overdose. Its half-life is 30 hours, and peak blood concentrations occur 6-24 hours after therapeutic dose. Decontamination and supportive care are crucial for mild cases [38].

Selective serotonin reuptake inhibitors (**SSRIs**)

SSRIs, first introduced in the US in 1988, have become a crucial treatment for various psychiatric conditions, including depression and anxiety disorders. Acute toxicity can cause symptoms like nausea, headaches, and sleep disturbances, with serious complications like serotonin toxidrome. Management involves stopping medications, providing supportive care, sedating patients, and administering serotonin antagonists [39].

Opioid analgesics and CNS depressants

Opioids

Opioid overdoses in children, often due to family medications or iatrogenic errors, can cause respiratory depression, pinpoint pupils, and hypotension due to toxicity. Children are particularly sensitive to these effects. Methadone, diphenoxylate, and sustained-release opioids require extended monitoring due to delayed or prolonged toxicity. Naloxone, an opioid antagonist, is the primary treatment for reversing respiratory and neurological depression, but its short half-life may necessitate continuous infusion. Observation for at least 6 hours is recommended, with longer monitoring for certain opioids like methadone [40].

Clonidine

Clonidine, an antihypertensive medication often prescribed for pediatric conditions like ADHD, autism, anxiety, and sleep disorders, acts as a central α2-adrenergic agonist, inhibiting sympathetic outflow and potentially causing transient hypertension followed by hypotension. Clonidine, an opiate-like substance, can cause CNS depression, miosis, and hypothermia. Its rapid absorption can cause cardiovascular and respiratory compromise. Treatment involves atropine and naloxone for bradycardia and respiratory depression [41]**.**

Benzodiazepines

Benzodiazepine overdose is common in toddlers and adolescents, affecting the γ-aminobutyric acid (GABA) type-A receptor complex in the central nervous system. Sedation duration varies, with drowsiness, slurred speech, and ataxia being common symptoms. Acute benzodiazepine toxicity requires supportive care, including endotracheal intubation. Laboratory investigations are not routine, and flumazenil, a competitive antagonist, may avoid intubation and mechanical support. Most children can be discharged after 4-6 hours if vital signs are satisfactory and the child can walk unaided [42].

Barbiturates

Barbiturates are used in pediatric critical care settings for sedation and in the PICU for critically unwell children with hypertension or refractory status epilepticus. They cause neuroinhibitory mechanisms in the brain, including GABA. Barbiturate poisoning can cause depression in the central nervous and cardiovascular systems, leading to muscle weakness and hypoventilation. Treatment is supportive, with no specific antidote. Early treatment with activated charcoal may be helpful, and other options include forced alkaline diuresis and hemodialysis for severe cases [43].

CNS Stimulating Drugs

Psychostimulants

Amphetamines, cocaine, and ecstasy (MDMA) are psychostimulants that enhance central and peripheral sympathetic outflow, causing symptoms such as hyperactivity, agitation, tachycardia, and serotonergic effects like hyperreflexia in children. Ecstasy also produces psychoactive effects, and its ingestion can lead to severe complications such as coma, convulsions, arrhythmias, malignant hyperthermia, rhabdomyolysis, hypertension, and multiorgan failure, with hyponatremia causing intractable seizures [44].

Symptomatic patients, particularly those with cardiac or CNS toxicity, require pediatric intensive care with careful monitoring, while asymptomatic children can be discharged after 12 hours. Convulsions and agitation are treated with benzodiazepines, and ventricular tachyarrhythmias are managed with sodium bicarbonate [45].

Anticholinergics

Anticholinergic poisoning can result from therapeutic substances, plants, and natural remedies, including over-the-counter medications, plants like Jimsonweed and angel's trumpet, and alkaloids. The anticholinergic toxidrome results from competitive inhibition of muscarinic receptors and presents with hyperthermia, dilated pupils, dry skin, erythema, and central anticholinergic delirium, characterized by agitation, confusion, hallucinations, and seizures. Management focuses on Supportive care, which includes monitoring vital signs, administering activated charcoal, using short-acting medications, and using physostigmine for delirium and coma, with careful monitoring for potential cardiac side effects [46].

Sympathomimetics

Sympathomimetic medicines are substances that mimic or alter the activities of catecholamines in the sympathetic nervous system. They can cause hypertension, vasoconstriction, ischemic injury, and neuropsychiatric effects like agitation, psychosis, and seizures. Treatment strategies should be tailored to the drug's clinical effects, considering gastrointestinal contamination early and ensuring adequate hydration. Benzodiazepines are often given intravenously for agitation, psychosis, and seizures, while non-selective beta/beta-adrenergic blockers should be administered for end-organ ischemia or hypertensive crisis [47].

Toxic gases

Carbon monoxide

Carbon monoxide poisoning occurs when nitrogen-containing polymers are burned, causing tissue hypoxia and cellular damage. Symptoms include headaches, nausea, confusion, ataxia, and collapse. CO poisoning can manifest in acute exposure and delayed neurologic sequelae, with death often due to cardiac arrhythmias, cerebral edema, and severe metabolic acidosis. Treatment involves immediate removal, 100% oxygen administration, and possibly hyperbaric oxygen therapy [48].

Cyanide

Cyanide is a naturally occurring compound found in various plants and fruits but can be released by modern synthetic materials when exposed to high temperatures. It binds to ferric iron, inhibiting oxidative phosphorylation and causing cyanosis with high anion gap metabolic acidosis and elevated lactate. Diagnosis is difficult due to limited clinical signs and co-existing CO poisoning. Treatment involves high-flow oxygen, supportive care, and cyanide antidotes. Hydroxocobalamin is the most effective cyanide antidote with few side effects [49].

Hydrogen Sulphide

Hydrogen sulfide (H2S) is a toxic gas found in waste plants and sewers. It causes endothelium disruption, cellular instability, respiratory dysfunction, and cardiovascular compromise. Exposure severity determines clinical signs, with respiratory symptoms being the main cause. Confirmation requires detecting thiosulphate or sulphate metabolites within seven days. Treatment options include hyperbaric oxygen and Sodium Nitrite or Cobamamide, which convert H2S to less toxic sulfmethaemoglobin [50].

Food Poisoning

Bacterial food poisoning

Food poisoning is a condition resulting from eating food contaminated with bacteria or toxins. Over 80% of cases are caused by bacteria. Common types include Clostridium perfringins and Staph. Aureus, Vibio Species, Bacillus Cereus, Salmonella, Clostridium Batulinum, Shigella, Toxigenic E.coli, Campylobacter, Yersinier, Listeria, and Aeromonas. Symptoms are delayed due to bacteria multiplying, often appearing 12-72 hours after consumption. Exotoxins, secreted during bacteria growth, can cause disease even when the microbes producing them die [51].

Viral food poisoning

Foodborne illnesses are primarily caused by enteric viruses, with hepatitis A and Norovirus being the most significant. Noroviruses are the leading cause globally, causing five million cases and 150 deaths annually in the United States. Symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Hepatitis A, an RNA-enveloped virus, has a 14-50-day incubation period and is more prevalent in underdeveloped countries with inadequate sanitary infrastructure [52].

Botulism

Clostridium botulinum is a Gram-positive bacteria found in soil, aquatic sediments, and dust. It produces seven botulinum neurotoxins, each with distinct immunological characteristics. Symptoms include double vision, vomiting, constipation, and difficulty swallowing. In severe cases, botulism can lead to heart and respiratory failures. Treatment involves decontamination, administration of the antidote, and support for respiratory function. The CDC recommends skin tests for hypersensitivity and epinephrine for allergic reactions. Adults and pediatric patients have varying infusion rates, with adults starting at 0.5 mL per minute and pediatrics at 0.01 mL/kg per minute [53].

Animal toxins

Snake bites

Snakebite envenomation is a significant medical issue in many tropical and subtropical regions, causing over 100,000 deaths annually. Snake venoms are complex mixtures of toxic and pharmacologically active proteins, including phospholipase A2 enzymes that cause edema, metalloproteinases that lead to bleeding, clotting, and necrosis, and hyaluronidases that facilitate tissue digestion and leakage from blood vessels [54].

Bites from most viperid species and many African cobras result in severe local tissue damage, such as edema, blistering, hemorrhage, and necrosis. If not promptly treated with antivenom, this can lead to permanent disability or require surgical intervention. While antivenom is the primary treatment for systemic effects, it is often ineffective against local tissue damage, making it crucial to understand the specific venom's lethal potency and pathophysiological effects for effective treatment [55].

Scorpion stings

Scorpion envenomation is a common and life-threatening condition in tropical and subtropical regions, particularly in Egypt. It can cause neuro-muscular and cardiovascular toxic effects, leading to death. The severity of envenomation is influenced by factors like age and body weight, with children showing more severe symptoms and higher mortality rates. Mild envenomation management involves pain management with oral medications, cleansing of the sting site, and tetanus prophylaxis. Severe systemic symptoms, such as restlessness and muscle fasciculations, require immediate intervention. Antivenom is reserved for patients with skeletal muscle or cranial nerve dysfunction [56].

Non-toxic exposures

Minimally toxic substances are those which produce little toxicity, minor self-limited toxicity, or clinically insignificant effects at most doses. Examples of these substances include silica gel, A&D ointment, chalk, lipstick, non-camphor lip balms, watercolors, hand dishwashing detergents, non-salicylate antacids (excluding magnesium or sodium bicarbonate-containing products), calamine lotion, clay, crayons, diaper rash creams and ointments, fabric softeners/sheets, glow products, glue (white, arts, and crafts type), household plant food, oral contraceptives, pen ink, pencils, starch/sizing, throat lozenges without local anesthetics, topical antibiotics, topical antifungals, topical steroids, topical steroids with antibiotics, and water-based paints [57].

Conclusion

In conclusion, pediatric poisoning remains a significant global health concern, particularly due to the unique vulnerabilities of children and the complexity of managing acute intoxication cases. This study underscores the importance of using the Poison Severity Score (PSS) as a valuable tool in assessing the severity of poisoning and guiding treatment decisions. The findings highlight the need for emergency physicians to be well-versed in age-specific toxicology and the differing pharmacokinetics and pharmacodynamics between children and adults.

Moreover, the study emphasizes the necessity of a tailored approach in managing pediatric poisoning, which considers not only the medical aspects but also the psychological and social implications for the child and their family. The integration of ethical considerations, including the process of obtaining assent, ensures that pediatric patients are treated with the utmost respect for their developing autonomy.

Looking forward, ongoing research is needed to refine the PSS further and develop more precise treatment protocols that can be applied in diverse clinical settings. Additionally, preventive measures, including public education on the risks of household substances and the safe storage of medications, are crucial in reducing the incidence of accidental poisoning among children.

Ultimately, this study contributes to the broader goal of enhancing pediatric care and reducing childhood morbidity and mortality due to poisoning. Continued vigilance, research, and education are essential to achieving this objective and improving outcomes for children worldwide.

Ethical consideration

In conducting this study, ethical considerations were thoroughly addressed to ensure the safety, well-being, and rights of the pediatric participants. Given the vulnerability of children in research settings, special attention was paid to obtaining both informed consent from the parents or legal guardians and assent from the children themselves, where appropriate.

Assent, which involves explaining the study to the child in a manner they can understand and seeking their agreement to participate, was particularly important in this study. This process respects the child's developing autonomy and ensures that their participation is voluntary. The assent process was designed to be age-appropriate, with explanations tailored to the cognitive and emotional maturity of each child.

Furthermore, the study protocol was reviewed and approved by an institutional ethics committee, ensuring that all procedures were in compliance with ethical standards for research involving minors. The rights of the participants to withdraw from the study at any time without consequence were emphasized, and confidentiality of all patient data was strictly maintained.

By integrating these ethical considerations, the study not only adheres to high ethical standards but also contributes to the growing body of pediatric research in a manner that respects and protects its young participants.

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