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Research Article

Emergency Management of Accidental Medication Ingestion: A Review of Clinical Risks, Assessment, and Multidisciplinary Interventions

Alhazeemi, Abdulrazaq Hudhayri A[1], Ahmed Abdullah Saeed Alshahrani[2], Waleed Hussain Mohsin Alharthi[3], Ali Hamed Dabbus Alanza[4], Jaber Hamdan A Alanazi[5], Mohammad Alamlah H Alenezi[6], Shuaa Juayf A Alanazi[7], Mohammed Omar Adi Alhamwan[8], Mamduhah Ratib Osaykir Alruwaili[9], Rana Hassan Almalki[10], Hana Aziz M Alruwaili[11]

[1]General Practice – Northern Border Health Cluster – Arar – Northern Border Province – Saudi Arabia
* **Corresponding Author Email:** Dr.Abdulrazaq93@gmail.com- **ORCID:** 0000-0002-5247-7850

[2]General Physician – PHC Moktt 1, Asir Health Cluster – Abha / Khamis Mushait – Asir Region – Saudi Arabia
Email: aalshahrani294@moh.gov.sa - **ORCID:** 0000-0002-5247-9050

[3]Emergency Medical Services Specialist – Children's Hospital – Taif – Makkah Region – Saudi Arabia
Email: Waleedalharthi22@gmail.com- **ORCID:** 0000-0002-5247-8050

[4]Emergency Medical Services – Ministry of Health – Hafr Albatin – Eastern Province – Saudi Arabia
Email: alhaalanazi@moh.gov.sa - **ORCID:** 0000-0002-5247-7050

[5]Emergency Medical Services – Ministry of Health – Hafr Albatin – Eastern Province – Saudi Arabia
Email: jahalanzi@moh.gov.sa- **ORCID:** 0000-0002-5247-6050

[6]Health Care Security Assistant – Erada Mental Health Complex, Northern Borders Health Cluster, Ministry of Health – Arar – Northern Border Province – Saudi Arabia
Email: MOALALENEZI@moh.gov.sa - **ORCID:** 0000-0002-5247-5050

[7]Clinical Pharmacy – Ministry of Health Branch, Northern Borders – Arar – Northern Border Province – Saudi Arabia
Email: nn8s@hotmail.com - **ORCID:** 0000-0002-5247-4050

[8]Pharmacy Technician – Al-Qurayyat General Hospital, Al-Jawf Health Cluster – Al-Qurayyat – Al-Jawf Region – Saudi Arabia
Email: malhamwan@moh.gov.sa - **ORCID:** 0000-0002-5247-3350

[9]Nursing Technician – Suwayr General Hospital, Al-Jawf Health Cluster – Suwayr – Al-Jawf Region – Saudi Arabia
Email: malrwwily@moh.gov.sa- **ORCID:** 0000-0002-5247-0250

[10]Pharmacist – Zahrat Hannen Pharmacies – Medina – Madinah Region – Saudi Arabia
Email: ran44.hn44@gmail.com- **ORCID:** 0000-0002-5247-0350

[11]Clinical Pharmacy – Turayf General Hospital, Northern Borders Health Cluster – Turayf – Northern Border Province – Saudi Arabia
Email: Haalruwili@moh.gov.sa- **ORCID:** 0000-0002-5247-1050

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Abstract:

Abstract should be about 100-250 words. It should be written times new roman and 10 punto. Accidental medication ingestion, particularly among children and vulnerable populations, presents significant clinical risks that necessitate prompt and effective emergency management. The types of medications involved can vary widely from over-the-counter drugs to potent pharmaceuticals, each carrying distinct toxicological profiles. A thorough assessment is crucial in the emergency setting, where healthcare providers must quickly ascertain the type and dosage of the ingested substance, as well as the time elapsed since ingestion. This rapid evaluation informs the decision-making process regarding decontamination procedures, the necessity for activated charcoal administration, and the potential requirement for advanced interventions such as antidote therapy or supportive care measures. The management of accidental medication ingestion requires a multidisciplinary approach, integrating the expertise of emergency physicians, toxicologists, nursing staff, and pharmacists to provide optimal care. Collaboration among these professionals facilitates the development of comprehensive treatment protocols tailored to individual cases, which include ongoing monitoring and re-evaluation of the patient's clinical status. Educational initiatives aimed at caregivers and the community are also vital to enhance awareness of medication safety and prevention strategies. By improving understanding of risks and proper storage practices, the incidence of accidental ingestions can be reduced, ultimately leading to better health outcomes and reduced strain on emergency resources.

1. Introduction

Accidental medication ingestion stands as a pervasive and persistent global public health issue, representing a significant source of preventable morbidity and mortality across all age groups. Its impact is most acutely felt in pediatric populations, where it consistently ranks as a leading cause of emergency department visits for children under six years of age [1]. The very nature of childhood—characterized by innate curiosity, oral exploration, and a lack of risk awareness—converges with the modern reality of households stocked with a wide array of pharmaceuticals. This creates a perfect storm for unintentional poisoning, where a moment of unsupervised access can lead to a life-threatening crisis. While statistical trends offer some reassurance that the majority of these exploratory incidents involve a single substance and result in minimal or no toxicity, this should not foster complacency. The potential for any single ingestion to rapidly escalate into a severe, life-altering, or fatal event demands that healthcare systems and providers maintain a constant state of readiness and a high index of suspicion [2].

The clinical landscape of accidental poisoning is not static; it has undergone a significant and challenging evolution over recent decades. The threats have shifted from traditional household chemicals to a new generation of pharmaceutical agents that are more potent, more complex, and more widely prescribed. Emerging threats continuously challenge clinical toxicology paradigms. Novel pharmaceuticals, such as targeted oncologic agents, new-generation antipsychotics, and sophisticated cardiovascular drugs, often have mechanisms of action and toxicity profiles unfamiliar to many frontline providers. Compounding this is the proliferation of high-potency formulations. A single tablet of a modern opioid like fentanyl, or a sustained-release calcium channel blocker, can contain a sufficient dose to be lethal to a small child [3]. Furthermore, the increasing prevalence of certain medication classes in home medicine cabinets has shifted the epidemiological profile of significant ingestions. The ongoing opioid epidemic has made potent agonists more accessible, while the rising management of chronic diseases like hypertension, diabetes, and hyperlipidemia means that cardiovascular drugs and oral hypoglycemic agents are now commonplace, introducing substances with narrow therapeutic indices into domestic environments [4].

The challenge of accidental ingestion extends far beyond the pediatric population, presenting with distinct etiologies and complexities in adults. In the geriatric demographic, accidental ingestions are frequently linked to unintentional dosing errors, often driven by visual impairment, cognitive decline, or functional limitations. The phenomenon of polypharmacy—the concurrent use of multiple medications—is a dominant risk factor in this group. The complex interplay of five, ten, or even fifteen different medications dramatically increases the risk of drug-drug interactions, dosing confusion, and cumulative toxicity [5]. An accidental double dose of an anticoagulant like warfarin or a direct oral anticoagulant (DOAC) can precipitate a catastrophic hemorrhage, while a mis-timed dose of an oral hypoglycemic may lead to profound and protracted hypoglycemia. In younger adults, accidental ingestion may occur in the context of substance misuse, medication diversion, or simple error, such as taking a roommate's medication in the dark. The common thread across all ages is that the medication was not taken with malicious intent, yet the consequences can be just as dire as those of an intentional overdose, requiring the same rigorous and attentive emergency management.

The fundamental clinical challenge in managing these presentations lies in the vast and ever-expanding arsenal of potential toxins, each possessing unique pharmacokinetic and pharmacodynamic properties. A medication's volume of distribution, protein binding, half-life, and active metabolites all dictate the timing, severity, and duration of its toxic effects. A one-size-fits-all approach is not only ineffective but can be profoundly harmful. The administration of activated charcoal, for instance, is a cornerstone of decontamination for many substances but is contraindicated and dangerous in others, such as caustic agents or hydrocarbons [6]. Similarly, the strategy of enhanced elimination through urinary alkalinization is highly effective for salicylates but is useless or even detrimental for many other toxins. This immense variability necessitates a tailored, poison-specific management strategy that must often be initiated empirically, based on limited initial information.

Therefore, the emergency clinician must function as both a detective and a physiologist, adept at rapid risk assessment and the synthesis of disparate clues. The process begins with a meticulous history—though often incomplete or unreliable—and a focused physical examination aimed at identifying characteristic toxidromes, the clinical constellations of signs and symptoms that point to a specific class of intoxicants [7]. Is the patient agitated with dry skin and dilated pupils, suggesting an anticholinergic syndrome? Or are they somnolent, bradypneic, and miotic, pointing toward opioid toxicity? This initial clinical gestalt must then be supplemented by the intelligent use of diagnostic adjuncts, such as electrocardiography to detect sodium channel blockade or serum chemistry to reveal a metabolic acidosis. Crucially, the clinician is not operating in a vacuum. The utilization of specialized resources, most notably regional poison control centers, is an indispensable component of modern toxicological care. These centers provide immediate access to toxicology experts who can offer critical guidance on the expected clinical course, recommended workup, and most appropriate decontamination and antidotal strategies for thousands of commercial products and drugs [8].

2. Epidemiological Considerations and High-Risk Medications

Understanding the epidemiology of accidental poisonings is fundamental to directing resources and anticipating clinical challenges. Pediatric ingestions typically follow a bimodal pattern, involving exploratory behavior in toddlers and intentional overdose or risk-taking behavior in adolescents [5]. The home environment is the most common setting, with medications often accessed from purses, nightstands, or easy-to-open containers. In contrast, accidental ingestions in the elderly are frequently associated with visual or cognitive deficits, complex medication regimens, and the use of high-alert medications such as anticoagulants, oral hypoglycemics, and opioids [6].

Certain medication classes are disproportionately represented in severe and fatal outcomes. Cardiovascular medications, including calcium channel blockers, beta-blockers, and clonidine, are notorious for their narrow therapeutic index and capacity to induce rapid cardiovascular collapse, even with the ingestion of a single tablet in a small child [7]. Oral hypoglycemic agents, particularly sulfonylureas, can provoke profound and prolonged hypoglycemia requiring extended monitoring and repeated dextrose administration. Opioids pose a dual threat of respiratory depression and, with the rise of illicitly manufactured fentanyl, the potential for lethal potency in minute quantities [8]. Other high-risk agents include tricyclic antidepressants, which can cause anticholinergic toxicity, sodium channel blockade, and seizures; salicylates, which disrupt acid-base balance and metabolism; and toxic alcohols like methanol and ethylene glycol, whose metabolites cause severe metabolic acidosis and end-organ damage [9, 10].

3. Initial Clinical Assessment and Triage

The initial assessment of a patient with suspected accidental medication ingestion must be rapid, systematic, and focused on identifying immediate life threats. The primary survey follows the standard ABCDE (Airway, Breathing, Circulation, Disability, Exposure) approach, with particular attention to signs of compromise that may be directly attributable to toxin exposure [11].

A patent and protected airway is the first priority. Depressed level of consciousness, as seen in sedative-hypnotic or opioid overdose, can lead to loss of protective airway reflexes and risk of aspiration. Breathing assessment should evaluate rate, depth, and pattern, noting both hypoventilation (from opioids) and hyperventilation (from salicylates or metabolic acidosis). Circulation requires evaluation of heart rate, blood pressure, and perfusion; bradycardia and hypotension are hallmarks of calcium channel blocker and beta-blocker toxicity, while tachycardia and hypertension may be present with sympathomimetic or anticholinergic agents [12]. The disability assessment involves a rapid neurological evaluation using the Glasgow Coma Scale and checking pupil size and reactivity, which can provide crucial diagnostic clues. Finally, exposure involves a full-body examination to look for hidden pills, transdermal patches, or signs of trauma.

Concurrently, every effort should be made to obtain a detailed history. Key information includes the specific medication(s) involved, the maximum possible quantity ingested, the time of ingestion, and any symptoms already exhibited. Collateral history from family members, paramedics, or the prescription bottle itself is invaluable. It is also critical to inquire about the co-ingestion of ethanol or other substances, which can alter the clinical presentation and course [13].

4. The Role of Toxidrome Recognition in Diagnosis

In many cases, the specific intoxicant is unknown at presentation. Here, the recognition of toxidromes—clusters of signs and symptoms characteristic of poisoning by a specific class of agents—becomes a vital diagnostic and therapeutic tool. The physical examination is the primary means of identifying these patterns [14].

The sympathomimetic toxidrome manifests as agitation, tachycardia, hypertension, hyperthermia, mydriasis, and diaphoresis. It is caused by agents such as amphetamines, cocaine, and decongestants like pseudoephedrine. In contrast, the sympatholytic toxidrome presents with sedation, bradycardia, hypotension, hypothermia, and miosis. Classic examples include opioids, clonidine, and barbiturates. The anticholinergic toxidrome is memorized by the phrase, "mad as a hatter, red as a beet, dry as a bone, hot as a hare, and blind as a bat," referring to delirium, flushing, dry skin and mucous membranes, hyperthermia, and mydriasis with blurred vision. This is seen with diphenhydramine, atropine, and tricyclic antidepressants [15]. The cholinergic toxidrome, caused by organophosphates or nerve agents, presents with the SLUDGE acronym (Salivation, Lacrimation, Urination, Defecation, Gastrointestinal upset, Emesis) along with muscle fasciculations and weakness.

Identifying a toxidrome allows for the initiation of empirical supportive care and specific antidotes even before confirmatory laboratory testing is available. For instance, the recognition of an opioid toxidrome should prompt the immediate administration of naloxone [16].

5. Diagnostic Adjuncts: Laboratory and Radiographic Evaluation

While clinical assessment is paramount, targeted laboratory and radiographic studies can confirm suspicions, quantify severity, and guide management. Basic studies should include a point-of-care glucose test to rule out hypoglycemia as a cause of altered mental status. An electrocardiogram (ECG) is essential for evaluating rhythm disturbances and signs of cardiotoxicity, such as QRS or QTc prolongation [17].

Serum acetaminophen and salicylate levels should be considered in any unknown or intentional overdose, given their ubiquity and the availability of effective antidotes. Basic metabolic panels can reveal anion gap metabolic acidosis, which can be caused by salicylates, toxic alcohols, and metformin. Elevated osmolar gaps suggest the presence of low molecular weight solutes like ethanol, methanol, or ethylene glycol [18]. Specific quantitative drug levels (e.g., for digoxin, lithium, valproic acid, iron) are indicated when there is a known or suspected ingestion of these agents.

Radiography has a limited but specific role. Abdominal radiographs can be useful for detecting radiopaque substances. While many pills are not visible, certain medications are radiopaque, including iron tablets, potassium chloride, phenothiazines, and some enteric-coated formulations [19]. A chest radiograph is indicated if there is concern for aspiration pneumonitis or non-cardiogenic pulmonary edema, a complication of opioid or salicylate overdose.

6. Gastrointestinal Decontamination:

The role of gastrointestinal decontamination has been refined over recent decades, with a shift away from routine use towards a more selective, risk-benefit approach. The chosen method depends on the timing of ingestion, the specific toxin, the patient's clinical status, and the potential risks of the procedure itself [20].

Activated charcoal (AC) is the most commonly employed decontamination method. It acts by adsorbing toxins within the gastrointestinal tract, preventing systemic absorption. AC is most effective when administered within one hour of ingestion and is contraindicated in patients with an unprotected airway, those at risk for gastrointestinal perforation or hemorrhage, and for ingestions of corrosives, hydrocarbons, or metals like iron and lithium, which it does not bind well [21]. The decision to use single-dose AC should be based on the potential for the ingested substance to cause significant toxicity and its likelihood of being adsorbed by charcoal.

Gastric lavage is rarely indicated due to a lack of proven clinical benefit and significant risks, including aspiration, electrolyte imbalance, and mechanical injury. It may be considered only in rare cases of a life-threatening ingestion of a non-caustic substance within the last 60 minutes, where the airway can be protected [22]. Whole bowel irrigation (WBI) involves the rapid administration of a polyethylene glycol electrolyte solution to flush the entire gastrointestinal tract. Its primary indications are for ingestions of sustained-release or enteric-coated medications, packets of illicit drugs ("body packers"), or toxins not bound by activated charcoal, such as heavy metals [23]. The patient must be alert and have a functional GI tract for WBI to be safe and effective. Syrup of ipecac is no longer recommended for use in either the home or clinical setting due to its poor efficacy and association with complications like persistent vomiting and drowsiness [24].

7. Antidotal Therapy and Enhanced Elimination

For a select group of toxins, specific antidotes are available that can directly counteract the poison's effects at the receptor or metabolic level. The timely administration of these antidotes is a cornerstone of modern toxicology and can be life-saving [25].

Naloxone is a competitive opioid receptor antagonist used to reverse respiratory and CNS depression in opioid overdose. It can be administered intravenously, intramuscularly, or via intranasal spray. N-acetylcysteine (NAC) is the specific antidote for acetaminophen poisoning, acting as a glutathione precursor and conjugate to prevent the formation of the toxic metabolite NAPQI. Flumazenil is a benzodiazepine receptor antagonist, but its use is controversial due to the risk of precipitating seizures in patients with co-ingestion of pro-convulsant drugs or benzodiazepine dependence [26]. For calcium channel blocker and beta-blocker toxicity, therapies beyond standard advanced cardiac life support include high-dose insulin euglycemia, calcium gluconate, and lipid emulsion therapy.

Enhanced elimination techniques are employed when a toxin has already been absorbed and the body's natural clearance mechanisms are insufficient to prevent toxicity. Urinary alkalinization, achieved with intravenous sodium bicarbonate, enhances the renal excretion of weak acids like salicylates and phenobarbital by ion trapping in the urine [27]. Extracorporeal methods, such as hemodialysis, are highly effective for removing toxins that are water-soluble, have a low volume of distribution, and low protein binding. Hemodialysis is a mainstay of treatment for severe poisonings with methanol, ethylene glycol, salicylates, lithium, and valproic acid [28].

8. Management of Specific Organ System Complications

The supportive care of a poisoned patient often revolves around managing complications affecting major organ systems. A proactive approach is required to anticipate and mitigate these effects.

Cardiovascular instability is common. Hypotension should first be managed with fluid challenges. If refractory, vasopressors such as norepinephrine or vasopressin may be required. For certain toxicants like beta-blockers, glucagon can be an effective inotrope. Dysrhythmias must be managed based on the underlying toxin; for instance, sodium bicarbonate is first-line therapy for wide-complex tachycardias from sodium channel blockers like tricyclic antidepressants [29]. Neurological complications range from agitation to coma and seizures. Agitation should be managed with benzodiazepines, not antipsychotics which can lower the seizure threshold. Seizures are also typically treated with benzodiazepines as first-line therapy. For persistent status epilepticus, second-line agents like propofol or phenobarbital may be needed [30].

Respiratory failure may necessitate endotracheal intubation and mechanical ventilation. The decision to intubate should be based on clinical criteria, not an arbitrary Glasgow Coma Scale score. Metabolic disturbances, such as the high anion gap metabolic acidosis seen in toxic alcohol or salicylate ingestion, require correction with sodium bicarbonate and, in severe cases, hemodialysis [31].

9. Special Populations: Pediatric and Geriatric Considerations

The management of accidental ingestion must be tailored to the patient's age and physiological status. Pediatric patients are not simply small adults; they have distinct pharmacokinetics, including higher metabolic rates and differences in body water and fat distribution, which can alter a drug's volume of distribution and half-life [32]. Furthermore, the differential diagnosis for altered mental status in a child is broad and includes infection and trauma, which must be ruled out. Dosing for antidotes and supportive medications must be meticulously weight-based.

In geriatric patients, age-related physiological changes such as decreased renal and hepatic function, reduced lean body mass, and altered protein binding can significantly increase the susceptibility to adverse drug events and prolong the effects of an accidental ingestion [33]. Polypharmacy is a major risk factor, increasing the potential for drug-drug interactions. The clinical presentation of toxicity in the elderly may be atypical, manifesting as delirium or a functional decline, which can lead to misdiagnosis. A high degree of suspicion and a thorough medication reconciliation are critical in this population.

10. Risk Stratification and Disposition Planning

Not all patients with accidental medication ingestion require hospital admission. Safe disposition relies on accurate risk stratification, which integrates the toxicity of the substance, the amount ingested, the time since ingestion, and the patient's clinical trajectory [34].

Patients who remain completely asymptomatic after a known low-risk ingestion over a sufficient observation period (typically 4-6 hours) may be considered for discharge. This observation period must be extended for ingestions of sustained-release formulations, agents with delayed toxicity (e.g., acetaminophen, sulfonylureas), or drugs that can cause delayed cardiovascular collapse (e.g., calcium channel blockers) [35]. Any patient who received naloxone for an opioid overdose should be observed for a minimum of 4 hours after the last dose due to the risk of re-sedation as the antidote wears off.

Indications for admission to a general medical floor include the development of mild, non-life-threatening symptoms that require ongoing monitoring. Admission to an intensive care unit is warranted for patients with altered mental status requiring airway protection, hemodynamic instability, significant dysrhythmias, recurrent seizures, or the need for continuous antidotal infusions or hemodialysis [36]. For all discharges, particularly in pediatric cases, providing robust poison prevention education and ensuring a safe home environment are essential components of care.

11. Preventative Strategies and Public Health Implications

Prevention is the most effective strategy for reducing the incidence and impact of accidental medication ingestions. A multi-layered approach involving public education, regulatory policy, and technological innovation is required [37].

On a household level, the consistent and correct use of child-resistant packaging is paramount. Medications should be stored up, away, and out of sight, ideally in a locked cabinet. Parents and caregivers must be educated to never refer to medicine as "candy." For the elderly and those on complex regimens, medication adherence aids like pill organizers should be used with caution and ideally under the supervision of a caregiver or pharmacist to prevent dosing errors [38]. A key preventative measure is the safe disposal of unused, expired, or unneeded medications, utilizing take-back programs to prevent them from remaining in the home.On a broader scale, public health campaigns can raise awareness about the dangers of medication misuse and unsafe storage. Regulatory actions, such as the mandated use of unit-dose packaging for high-risk medications and the reformulation of drugs to reduce their abuse potential or toxicity, have shown promise [39]. The integration of clinical toxicologists and poison control centers into the healthcare system provides a vital resource for both the public and providers, offering immediate, expert guidance during poisoning emergencies and contributing to surveillance and data collection on emerging trends [40].

12. Conclusion:

The emergency management of accidental medication ingestion is a dynamic and challenging field that requires a synthesis of rapid assessment, clinical knowledge, and tailored interventions. From the initial recognition of a potential poisoning through to decontamination, antidote administration, and supportive care, the approach must be systematic and guided by the best available evidence. The decline in routine use of certain decontamination methods and the expanded role of specific antidotes and enhanced elimination techniques reflect an evolving evidence base that prioritizes patient safety and outcomes.

Ultimately, optimal care extends beyond the emergency department. It hinges on seamless collaboration between emergency physicians, clinical toxicologists, intensivists, pharmacists, and social workers. Furthermore, a sustained commitment to preventative strategies at the individual, community, and regulatory levels is crucial to mitigate the risk of these often preventable events. Future efforts should focus on continued education, research into novel antidotes and treatment modalities, and the strengthening of poison control infrastructure to effectively confront the changing landscape of toxicological threats.

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