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Life-Threatening Single-Dose Toxic Agents: An Integrated Clinical Approach to Emergency Medicine, Laboratory Diagnostics, Nursing Management, and Medical Records Documentation.

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Abstract

Background: Potentially lethal single-dose toxic agents represent a significant healthcare concern because exposure to relatively small quantities of certain medications, chemicals, and household products can result in severe toxicity, life-threatening complications, or death. Early recognition and appropriate management are essential, particularly in pediatric patients and individuals with accidental or intentional exposures.

Aim: This review aims to provide an integrated overview of potentially lethal single-dose toxic agents, focusing on their mechanisms of toxicity, clinical manifestations, diagnostic investigations, emergency management, and interprofessional healthcare responsibilities.

Methods: A narrative review approach was adopted to examine selected high-risk pharmaceutical agents, toxic chemicals, and household substances. The review addresses their pharmacological mechanisms, characteristic toxicological manifestations, and evidence-based management principles, with particular attention to emergency medicine, clinical laboratory investigations, nursing interventions, and medical records management.

Results: The reviewed agents include alpha-adrenergic agonists, sulfonylureas, calcium channel blockers, beta-blockers, tricyclic antidepressants, opioids, antidiarrheals, salicylates, antipsychotics, antimalarials, antiarrhythmics, camphor, colchicine, acetylcholinesterase inhibitors, methylxanthines, toxic alcohols, and caustic substances. Their toxic effects involve cardiovascular instability, respiratory depression, neurological impairment, metabolic disturbances, and multiple-organ dysfunction. Effective management depends on early clinical assessment, appropriate laboratory testing, continuous monitoring, supportive treatment, and specific antidotes or interventions when indicated.

Conclusion: Multidisciplinary coordination between emergency physicians, laboratory professionals, nurses, pharmacists, medical toxicologists, and medical records personnel is essential for improving patient safety, treatment outcomes, and poisoning prevention.

Keywords: Single-dose toxic agents; acute poisoning; drug toxicity; emergency medicine; toxicology; laboratory diagnostics; nursing management; medical records; poisoning prevention.

Introduction

Acute poisoning represents a major public health concern and an important cause of preventable morbidity and mortality among children and adults. Certain pharmaceutical agents, chemicals, and household substances can produce severe toxic effects following exposure to relatively small quantities. These substances are commonly described as potentially lethal single-dose agents because even one tablet, a small amount of liquid medication, or limited accidental exposure may result in life-threatening complications. The clinical importance of recognizing these agents was highlighted by Dr. Gideon Koren in his landmark 1993 publication, *Medications Which Can Kill a Toddler with One Tablet or Teaspoonful*. Subsequent studies have supported this concept and emphasized the need to identify medications that pose substantial risks

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following accidental ingestion, particularly in young children. Although these investigations have contributed significantly to pediatric toxicology, many focus primarily on childhood exposures and provide limited coverage of poisoning in adults. Therefore, an integrated reference covering potentially dangerous substances across different age groups is essential for healthcare professionals involved in emergency assessment, laboratory investigation, nursing care, and medical documentation.[1–4] The toxicity of a substance depends on several factors, including its pharmacological properties, chemical structure, concentration, route of exposure, absorbed dose, and individual patient characteristics. Age, body weight, nutritional status, underlying diseases, hepatic and renal function, and concurrent medication use may influence the severity and clinical progression of poisoning. Children are particularly vulnerable because a quantity that represents a relatively small exposure in an adult may constitute a substantial dose per kilogram of body weight in an infant or toddler. Furthermore, their exploratory behavior, limited understanding of environmental hazards, and inability to recognize dangerous substances increase the likelihood of accidental ingestion. Adults may experience poisoning through medication errors, intentional overdose, occupational exposure, recreational substance use, or inappropriate administration of pharmaceutical preparations.

Several categories of medications deserve particular attention because of their potential to cause severe toxicity after exposure to small quantities. These include alpha-1 adrenergic agonists and imidazoline receptor agonists, such as clonidine, guanfacine, naphazoline, oxymetazoline, tetrahydrozoline, and xylazine. These substances may produce profound central nervous system depression, bradycardia, hypotension, and respiratory compromise. Sulfonylureas, including chlorpropamide, glyburide, glipizide, and glimepiride, can cause prolonged and potentially recurrent hypoglycemia. Calcium channel blockers, such as verapamil, diltiazem, nifedipine, amlodipine, nicardipine, and nimodipine, may cause severe hypotension, conduction abnormalities, bradycardia, and cardiovascular collapse. Similarly, beta-blockers, including propranolol, metoprolol, labetalol, and sotalol, can produce bradycardia, hypotension, hypoglycemia, and cardiac conduction disturbances. Other important groups include tricyclic antidepressants, opioids, antidiarrheal medications, salicylates, antipsychotics, antimalarials, and antiarrhythmic drugs. Tricyclic antidepressants, particularly amitriptyline and imipramine, may cause seizures, altered consciousness, hypotension, and life-threatening cardiac arrhythmias. Opioids such as morphine, codeine, methadone, hydrocodone, buprenorphine, and fentanyl can produce respiratory depression and coma. Loperamide and diphenoxylate-containing preparations may cause serious cardiac and neurological complications when taken in excessive quantities. Salicylates, including methyl salicylate found in certain topical preparations and traditional medicinal products, may produce metabolic disturbances, respiratory abnormalities, and neurological toxicity.

Additional substances of concern include chloroquine, hydroxychloroquine, quinine, primaquine, quinidine, disopyramide, procainamide, flecainide, and amiodarone. Their toxic effects may involve cardiac conduction abnormalities, ventricular arrhythmias, hypotension, and neurological manifestations. Camphor is another important toxic substance found in analgesic preparations, antipruritic products, cooling gels, ointments, mothballs, fragrances, and essential oils. Accidental oral exposure may cause seizures and central nervous system toxicity. Podophyllin, podofilox, colchicine, oral acetylcholinesterase inhibitors, carbamate pesticides, and methylxanthines such as theophylline and caffeine also require careful consideration. Toxic alcohols, including methanol, ethylene glycol, and diethylene glycol, represent another important category because they may produce severe metabolic acidosis, neurological injury, renal impairment, and visual complications. Finally, corrosive household products and industrial chemicals, including strong acids, alkalis, hydrofluoric acid, selenious acid, acetonitrile, and methylene chloride, can cause serious systemic or local tissue damage.[5,6] The identification of these agents does not mean that every exposure will result in fatal poisoning. Clinical outcomes depend on the quantity absorbed, formulation, time since exposure, patient characteristics, and availability of appropriate treatment. Conversely, the absence of a substance from a list of potentially lethal agents should not be interpreted as evidence of safety. Any suspected poisoning requires an individualized assessment based on the specific substance and circumstances of exposure.

Despite improvements in medication packaging, public awareness, poison prevention programs, and access to poison information services, accidental poisoning remains a frequent emergency presentation. Poison control centers in the United States receive more than 2.4 million calls annually, with children younger than six years accounting for more than half of reported exposures. Common pediatric poisoning incidents involve household cleaning products, analgesics, cosmetics, personal care products, foreign objects, and dietary supplements. In adults, poisoning may result from accidental medication duplication, incorrect dosing, drug interactions, occupational exposure, or intentional ingestion. Opioids, sedative-hypnotics, antidepressants, and cardiovascular medications represent important causes of serious adult toxicity. America's Poison Centers identifies analgesics,

household cleaning products, antidepressants, cosmetic and personal care products, and cardiovascular medications among the frequently reported substance categories.[7] The COVID-19 pandemic also highlighted the relationship between household behavior and poisoning risks. Increased use and inappropriate mixing of cleaning and disinfectant products contributed to a rise in reported exposures during the pandemic. Although these incidents subsequently declined, household chemicals continue to represent an important source of accidental poisoning.

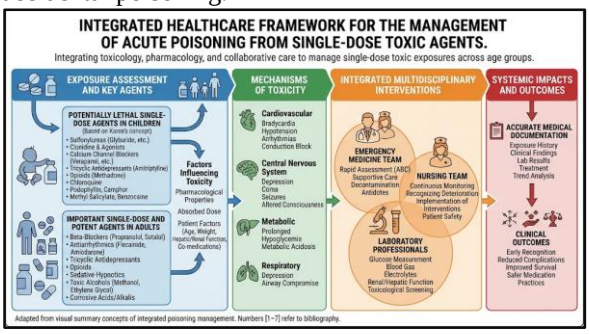


Figure-1: Overview of single dose toxic agents.

The effective management of potentially lethal single-dose exposures requires coordinated multidisciplinary intervention. Emergency physicians and healthcare teams must rapidly assess airway patency, breathing, circulation, neurological status, and possible cardiovascular instability. Laboratory professionals contribute through timely glucose measurement, blood gas analysis, electrolyte assessment, renal and hepatic function testing, toxicological screening when indicated, and measurement of specific drug concentrations. Nurses play an essential role in continuous monitoring, medication administration, recognition of clinical deterioration, implementation of supportive interventions, and communication with the medical team. Medical records professionals support patient safety through accurate documentation of exposure history, clinical findings, laboratory results, administered treatments, and patient outcomes. Reliable documentation also facilitates clinical decision-making, continuity of care, incident reporting, and future evaluation of poisoning trends. This review therefore examines potentially life-threatening single-dose toxic agents from an integrated healthcare perspective. It emphasizes their sources, toxicological mechanisms, clinical manifestations, diagnostic investigations, emergency management principles, and nursing responsibilities. It also addresses the importance of accurate medical documentation and effective communication among emergency, laboratory, nursing, and health information departments. Understanding these agents and establishing coordinated responses can improve early recognition, reduce preventable complications, and support safer medication handling and poisoning prevention across healthcare settings.

Clinical Overview

Potentially lethal single-dose toxic agents represent a diverse group of pharmaceutical compounds, chemicals, and naturally occurring substances that can produce severe physiological disturbances following exposure to relatively small quantities. Their toxicological effects depend on their pharmacological classification, molecular structure, receptor interactions, metabolic pathways, and target-organ distribution. Some agents primarily affect the cardiovascular system, whereas others produce neurological depression, respiratory failure, metabolic disturbances, gastrointestinal injury, or multiple-organ dysfunction. Understanding the mechanisms of toxicity and characteristic clinical manifestations is essential for early recognition, appropriate laboratory investigation, emergency treatment, and effective nursing management. The clinical presentation of poisoning may vary according to the patient's age, body weight, general health, exposure route, and time elapsed since ingestion. Children may develop severe manifestations following exposures that produce limited symptoms in adults. Furthermore, certain agents cause delayed toxicity, while others produce rapid deterioration within minutes. Healthcare professionals must therefore consider the possibility of serious poisoning even when the initial clinical examination appears relatively reassuring.

1. α-Adrenergic Agonists

Alpha-adrenergic agonists and imidazoline receptor agonists include clonidine, guanfacine, naphazoline, oxymetazoline, tetrahydrozoline, and xylazine. These substances interact with central α2-adrenergic and imidazoline receptors, reducing sympathetic nervous system activity and inhibiting norepinephrine release. Their pharmacological effects normally contribute to reductions in blood pressure and sympathetic stimulation. In poisoning, excessive receptor activation can produce profound central nervous system depression, bradycardia, hypotension, and respiratory depression. Clinical manifestations frequently resemble opioid intoxication and include reduced consciousness, pinpoint pupils, diminished respiratory effort, and decreased responsiveness. Some patients initially develop transient hypertension because of peripheral adrenergic receptor stimulation before central sympatholytic effects become predominant. Severe poisoning may progress to coma, respiratory failure, and cardiovascular instability.

2. Sulfonylureas

Sulfonylureas, including chlorpropamide, glyburide, glipizide, and glimepiride, are oral antidiabetic medications that stimulate insulin secretion from pancreatic beta cells. They bind to the sulfonylurea receptor 1 component of ATP-sensitive potassium channels, resulting in channel closure, membrane depolarization, calcium influx, and

increased insulin release. Excessive insulin secretion following poisoning can produce severe, prolonged, or recurrent hypoglycemia. The clinical manifestations range from sweating, tremors, palpitations, hunger, and weakness to irritability, confusion, behavioral changes, seizures, and coma. Young children are particularly vulnerable because relatively small quantities may produce clinically significant reductions in blood glucose. Recurrent hypoglycemia represents an important complication because insulin secretion may remain elevated after initial glucose correction. Continuous glucose monitoring and repeated laboratory assessment are therefore important components of clinical evaluation.

3. Calcium Channel Blockers

Calcium channel blockers include verapamil, diltiazem, nifedipine, amlodipine, nicardipine, and nimodipine. These medications inhibit L-type voltage-gated calcium channels in cardiac muscle, conduction tissue, vascular smooth muscle, and pancreatic beta cells. Their therapeutic effects include reduced myocardial contractility, decreased cardiac conduction, and relaxation of vascular smooth muscle. Overdose can produce severe cardiovascular toxicity characterized by hypotension, bradycardia, atrioventricular conduction abnormalities, and impaired myocardial contractility. Severe cases may progress to cardiogenic, vasodilatory, or mixed shock. Hyperglycemia may also develop because calcium-channel inhibition interferes with insulin secretion from pancreatic beta cells. Dihydropyridine agents, such as nifedipine and amlodipine, initially produce prominent peripheral vasodilation and may cause reflex tachycardia. However, this pharmacological selectivity may become less pronounced during severe poisoning, resulting in significant myocardial depression and cardiovascular instability.[8]

4. β-Blockers

Beta-blockers, including propranolol, metoprolol, labetalol, and sotalol, inhibit beta-adrenergic receptors and reduce the effects of circulating catecholamines. Their principal cardiovascular actions include decreased heart rate, reduced myocardial contractility, and delayed atrioventricular conduction. They may also interfere with physiological mechanisms responsible for maintaining blood glucose concentrations during hypoglycemia. Toxicity typically produces bradycardia, hypotension, atrioventricular block, and reduced cardiac output. Severe exposure can lead to cardiogenic shock and cardiac arrest. Hypoglycemia may occur, particularly in children, because beta-adrenergic blockade interferes with glucose regulation. Propranolol is highly lipophilic and can cross the blood-brain barrier, producing confusion, seizures, and CNS depression. It also blocks cardiac sodium channels at high concentrations, potentially causing QRS widening and ventricular dysrhythmias. Sotalol has additional potassium-channel-blocking properties that prolong cardiac repolarization and increase the risk of QT prolongation and torsades de pointes.

5. Tricyclic Antidepressants

Tricyclic antidepressants include amitriptyline, nortriptyline, imipramine, and desipramine. These medications primarily inhibit the reuptake of norepinephrine and serotonin. However, their toxic effects involve several additional pharmacological mechanisms, particularly cardiac sodium-channel blockade, muscarinic receptor antagonism, and alpha-1 adrenergic receptor antagonism. Sodium-channel blockade slows electrical conduction through the myocardium, resulting in QRS widening, conduction abnormalities, and potentially fatal ventricular arrhythmias. Antimuscarinic activity produces dry mouth, dilated pupils, urinary retention, reduced bowel activity, tachycardia, and altered mental status. Alpha-1 adrenergic antagonism contributes to hypotension through peripheral vasodilation. Neurological manifestations include agitation, confusion, delirium, seizures, and coma. Electrocardiographic abnormalities may include prolonged QRS duration, QT prolongation, and a prominent terminal R wave in lead aVR. Severe poisoning can result in ventricular tachycardia, ventricular fibrillation, cardiovascular collapse, and death. Early electrocardiographic assessment is particularly important in suspected tricyclic antidepressant poisoning.

6. Opioids

Opioids include morphine, codeine, methadone, hydrocodone, and heroin. These substances primarily activate mu-opioid receptors within the central nervous system. Their therapeutic effects include analgesia and sedation, but excessive receptor stimulation suppresses respiratory activity and reduces the brainstem response to increasing carbon dioxide concentrations. The characteristic clinical presentation is the opioid toxidrome, consisting of reduced consciousness, miosis, and respiratory depression. Patients may exhibit slow or irregular breathing, decreased oxygen saturation, reduced responsiveness, and diminished protective airway reflexes. Severe poisoning can produce hypercapnia, hypoxemia, respiratory arrest, and secondary cardiac arrest. Pulmonary edema and aspiration complications may also develop. Respiratory depression represents the most immediately life-threatening manifestation and requires prompt assessment and intervention.

7. Antidiarrheal Medications

Loperamide and diphenoxylate are opioid receptor agonists used to reduce intestinal motility and treat diarrhea. Diphenoxylate is frequently combined with atropine, which contributes antimuscarinic effects intended to discourage excessive consumption. Excessive diphenoxylate-atropine exposure may produce a combination of

opioid and anticholinergic manifestations. Patients can develop CNS depression, respiratory suppression, tachycardia, dry mucous membranes, urinary retention, and altered mental status. Symptoms may appear after a delay and can recur because of prolonged absorption or drug elimination. Loperamide poisoning is particularly concerning because high systemic concentrations can affect cardiac electrical activity. In addition to opioid receptor stimulation, excessive exposure inhibits cardiac sodium and potassium channels. These effects can produce QRS widening, QT prolongation, bradycardia, syncope, ventricular tachycardia, and other potentially fatal dysrhythmias.[9]

8. Salicylates and Methyl Salicylate

Salicylates include aspirin, bismuth subsalicylate, and methyl salicylate-containing preparations. Methyl salicylate is present in certain topical analgesics, liniments, and traditional medicinal products, including oil of wintergreen. Salicylate toxicity involves stimulation of the medullary respiratory center and disruption of cellular energy production. Early respiratory stimulation produces hyperventilation and respiratory alkalosis. At the cellular level, salicylates uncouple oxidative phosphorylation, reducing ATP production and increasing heat generation and organic acid accumulation. These processes contribute to metabolic acidosis and disturbances in acid-base balance. Common clinical manifestations include nausea, vomiting, sweating, tinnitus, dizziness, fever, and rapid breathing. Severe poisoning may produce agitation, confusion, seizures, hypoglycemia within the central nervous system, pulmonary edema, cerebral edema, and coma. Mixed respiratory alkalosis and high-anion-gap metabolic acidosis represent characteristic laboratory findings. The clinical condition may deteriorate despite apparently stable initial vital signs.

9. Antipsychotic Medications

Antipsychotic medications include chlorpromazine, thioridazine, and loxapine. Most conventional antipsychotics exert their principal effects through dopamine D2 receptor antagonism. However, their interactions with histamine H1, muscarinic, alpha-1 adrenergic, and cardiac ion channels contribute to their adverse effects and toxicological profiles. Overdose commonly causes CNS depression, sedation, tachycardia, and hypotension. Muscarinic receptor antagonism may produce dry mouth, dilated pupils, urinary retention, and anticholinergic delirium. Some agents can cause extrapyramidal manifestations, including dystonia, rigidity, and abnormal involuntary movements. Cardiac toxicity may involve QT prolongation and ventricular arrhythmias. Chlorpromazine exposure can produce particularly prominent sedation and hypotension because of its antihistaminic and alpha-adrenergic blocking properties.[10]

10. Antimalarial Medications

Antimalarial medications include chloroquine, hydroxychloroquine, quinine, and primaquine. Their toxicological effects differ according to their molecular structures and pharmacological mechanisms. Chloroquine and hydroxychloroquine can interfere with cardiac sodium and potassium channels, producing conduction abnormalities and disturbances in myocardial repolarization. They may also promote intracellular potassium redistribution, resulting in marked hypokalemia. Severe poisoning can cause hypotension, QRS widening, QT prolongation, ventricular dysrhythmias, seizures, and coma. Quinine toxicity may produce hypoglycemia through increased insulin secretion. It can also cause cinchonism, characterized by tinnitus, headache, dizziness, nausea, hearing disturbances, and visual abnormalities. Cardiac conduction disturbances may accompany severe exposure. Primaquine and other oxidant antimalarials can cause methemoglobinemia and hemolytic anemia, particularly in individuals with glucose-6-phosphate dehydrogenase deficiency.

11. Antiarrhythmic Medications

Antiarrhythmic medications include quinidine, disopyramide, procainamide, flecainide, and amiodarone. Their therapeutic actions involve modification of cardiac ion channels and conduction pathways. However, excessive exposure can disrupt normal electrical activity and produce potentially fatal cardiovascular complications. Class I antiarrhythmics inhibit sodium channels, slowing myocardial depolarization and electrical conduction. Toxicity may result in QRS widening, hypotension, conduction block, and ventricular dysrhythmias. Class IA agents, including quinidine and procainamide, also inhibit potassium channels, prolonging repolarization and increasing the risk of QT prolongation and torsades de pointes. Other agents may produce bradycardia, atrioventricular block, myocardial depression, or additional conduction abnormalities. The specific manifestations depend on the individual drug and the extent of exposure.

12. Camphor

Camphor is a lipophilic terpenoid commonly incorporated into topical analgesics, antipruritic preparations, cooling gels, ointments, mothballs, fragrances, and essential oils. It is rapidly absorbed following oral exposure and readily crosses the blood-brain barrier. Although its precise mechanism of neurotoxicity remains incompletely understood, camphor is recognized as a potent neurological toxin. Initial manifestations may include nausea, vomiting, abdominal discomfort, agitation, restlessness, and confusion. Neurological deterioration can occur rapidly, with abrupt generalized seizures or status epilepticus. Subsequent CNS depression and respiratory compromise may develop in severe cases.

A characteristic camphoraceous odor may sometimes assist in identifying exposure, although its absence does not exclude poisoning.

13. Nonalkaloid Toxic Lignans

Podophyllin and podofilox are compounds associated with the treatment of certain dermatological conditions, including anogenital warts. Podophyllotoxin, their principal active toxicological component, interacts with tubulin and inhibits microtubule assembly. This prevents normal mitotic spindle formation and arrests cell division during metaphase. Systemic exposure can damage rapidly dividing tissues, particularly gastrointestinal epithelial cells and bone marrow. Initial manifestations may include nausea, vomiting, abdominal pain, and diarrhea. Progressive toxicity can produce mucosal injury, bone marrow suppression, leukopenia, thrombocytopenia, and neurological complications. Hepatic and renal injury may also occur, potentially progressing to multiorgan dysfunction.[11]

14. Colchicine

Colchicine is an anti-inflammatory medication commonly used in the management of gout and certain inflammatory disorders. It binds to tubulin and prevents microtubule polymerization, interfering with cellular processes that depend on an intact microtubule network. Colchicine poisoning typically develops in several clinical phases. Early manifestations predominantly involve the gastrointestinal system and include severe abdominal pain, nausea, vomiting, and diarrhea. Subsequent systemic toxicity may produce dehydration, hypotension, cardiovascular instability, and multiple-organ dysfunction. Bone marrow suppression can cause leukopenia, thrombocytopenia, and pancytopenia. Severe poisoning may also result in cardiac dysfunction, hepatic injury, acute kidney injury, neuromuscular complications, and respiratory failure. The development of multiorgan failure represents a major determinant of mortality.[12]

15. Oral Acetylcholinesterase Inhibitors

Oral acetylcholinesterase inhibitors include rivastigmine, donepezil, and galantamine. Carbamate pesticides also inhibit acetylcholinesterase through a related mechanism. These substances prevent the normal breakdown of acetylcholine, resulting in excessive stimulation of muscarinic, nicotinic, and central cholinergic receptors. The resulting cholinergic syndrome involves multiple organ systems. Muscarinic manifestations include excessive salivation, lacrimation, sweating, miosis, vomiting, diarrhea, abdominal cramping, bronchorrhea, bronchospasm, and bradycardia. Nicotinic effects include muscle weakness, fasciculations, and neuromuscular dysfunction. Central nervous system manifestations may include confusion, agitation, seizures, reduced consciousness, and coma. Respiratory compromise is particularly dangerous because it may result from several simultaneous mechanisms, including excessive bronchial secretions, bronchospasm, respiratory muscle weakness, and central respiratory depression. Early recognition of the cholinergic toxidrome is therefore essential for preventing respiratory failure and other serious complications.

16. Methylxanthines

Methylxanthines include caffeine, theophylline, and related compounds. They exert their pharmacological effects primarily through adenosine receptor antagonism. At higher concentrations, they also inhibit phosphodiesterase enzymes, increasing intracellular cyclic adenosine monophosphate concentrations. These mechanisms enhance catecholaminergic activity and alter intracellular calcium regulation. Clinical manifestations include nausea, repetitive vomiting, abdominal discomfort, agitation, anxiety, tremors, tachycardia, and palpitations. Laboratory abnormalities may include hypokalemia, hyperglycemia, and metabolic disturbances. Severe poisoning can cause seizures, hypotension, supraventricular tachycardia, ventricular tachycardia, and cardiac arrest. Theophylline poisoning is particularly concerning because its narrow therapeutic index increases the risk of severe toxicity following excessive exposure.

17. Other High-Risk Opioids

Buprenorphine and fentanyl require particular attention because of their pharmacological potency and potential to produce severe respiratory depression. Buprenorphine is a high-affinity partial agonist at mu-opioid receptors. Despite its partial agonist activity, it can cause significant and prolonged respiratory and CNS depression, particularly in opioid-naive individuals and young children. Accidental pediatric exposure may result in reduced consciousness, miosis, hypoventilation, and respiratory failure. Fentanyl is a potent synthetic opioid used in medical anesthesia and pain management. Its high potency creates substantial risks when exposure occurs accidentally or outside controlled medical settings. Clinical manifestations include profound respiratory depression, unconsciousness, reduced protective airway reflexes, and potentially fatal hypoxia. Pediatric exposures are particularly concerning because relatively small quantities may produce severe clinical consequences.[13]

18. Toxic Alcohols

Toxic alcohols include methanol, ethylene glycol, and diethylene glycol. These compounds produce toxicity through both their parent molecules and their metabolic products. Initial manifestations may resemble ordinary alcohol intoxication, with dizziness, nausea, vomiting, and CNS depression. However, subsequent metabolism can generate highly toxic compounds that cause severe metabolic acidosis and target-organ injury. Methanol is metabolized into formaldehyde and formic acid. Formate interferes

with mitochondrial function and can cause optic nerve injury, visual disturbances, and permanent blindness. Ethylene glycol is metabolized into several organic acids, including glycolic and oxalic acids. These metabolites contribute to high-anion-gap metabolic acidosis, hypocalcemia, calcium oxalate crystal formation, and acute kidney injury. Diethylene glycol poisoning may cause gastrointestinal symptoms, metabolic acidosis, neurological complications, and renal impairment. Severe exposure to any of these substances can result in seizures, coma, cardiovascular instability, and death. Delayed recognition is particularly dangerous because substantial metabolic injury may develop after an initially nonspecific clinical presentation. Poisoning outbreaks have also occurred following contamination of medicinal products and other consumer substances.[14,15]

19. Caustics and Other Household Products

Caustic substances include strong acids, strong alkalis, and certain industrial or household chemicals. Their toxicity primarily results from direct chemical injury to exposed tissues. Alkalis generally produce liquefactive necrosis, allowing deeper tissue penetration, whereas acids typically cause coagulative necrosis. However, both categories can produce severe gastrointestinal injury, perforation, hemorrhage, and subsequent complications. Clinical manifestations depend on the substance, concentration, volume, physical form, and duration of contact. Common findings include oral pain, visible mucosal burns, excessive salivation, vomiting, difficulty swallowing, painful swallowing, abdominal pain, and respiratory distress. Airway involvement may produce hoarseness, stridor, bronchospasm, and respiratory failure. Inhalation of certain household chemicals can also cause pulmonary irritation and chemical pneumonitis. Some products produce additional systemic toxic effects. Artificial nail products containing specific oxidizing compounds may cause methemoglobinemia, reducing the oxygen-carrying capacity of blood. Acetonitrile can be metabolized into cyanide, resulting in cellular hypoxia and cardiovascular instability. Methylene chloride can undergo metabolism to carbon monoxide, producing impaired oxygen delivery and neurological manifestations. Hydrofluoric acid can cause profound fluoride-related toxicity, including hypocalcemia, hypomagnesemia, hyperkalemia, cardiac conduction abnormalities, and ventricular dysrhythmias.[16]

20. Integrated Clinical Assessment and Recognition

Although these toxic agents differ in their mechanisms and clinical manifestations, their evaluation requires a systematic approach that integrates clinical examination, laboratory investigations, electrocardiographic assessment, and exposure history. Emergency healthcare professionals should prioritize assessment of airway patency, respiratory effort, circulation, consciousness, and vital signs. Particular attention should be given to respiratory depression, abnormal pupils, seizures, hypotension, bradycardia, tachycardia, and altered mental status. The identification of recognizable toxidromes can help narrow the differential diagnosis and guide early clinical decisions. Laboratory investigations provide essential information about the physiological consequences of poisoning. Depending on the suspected agent, investigations may include:

- Blood glucose measurement to identify hypoglycemia or hyperglycemia.
- Arterial or venous blood gas analysis to evaluate acid-base disturbances.
- Serum electrolytes, particularly sodium, potassium, calcium, and magnesium.
- Renal function tests, including serum urea and creatinine.
- Liver function tests to identify hepatic injury.
- Serum lactate to assess tissue hypoperfusion and metabolic disturbances.
- Specific toxicological concentrations, including salicylate, acetaminophen, methanol, ethylene glycol, and theophylline when clinically indicated.
- Complete blood count to identify anemia, leukopenia, or thrombocytopenia.
- Electrocardiography to detect conduction abnormalities, QRS widening, QT prolongation, and cardiac dysrhythmias.

Nursing assessment and continuous monitoring are equally important. Nurses should document the patient's neurological condition, respiratory pattern, cardiovascular parameters, and response to treatment. Changes in consciousness, breathing, blood pressure, heart rate, or oxygen saturation require prompt communication with the treating medical team. Medical records personnel also contribute to the clinical management process by maintaining accurate information about the suspected toxic agent, exposure circumstances, symptom onset, laboratory findings, medical interventions, and patient outcomes. Accurate documentation supports continuity of care, clinical audit, adverse-event investigation, and the development of poisoning prevention strategies. Overall, understanding the mechanisms and clinical manifestations of potentially lethal single-dose agents is essential for effective multidisciplinary management. Early identification of characteristic toxicological patterns, timely laboratory assessment, continuous nursing observation, and accurate medical documentation can support appropriate clinical decisions and reduce the risk of preventable complications and mortality.

Key Clinical Considerations

The management of potentially lethal single-dose toxic agent exposures requires rapid

identification of the suspected substance, assessment of the patient's clinical condition, and implementation of appropriate supportive and specific therapeutic interventions. The initial approach should prioritize airway protection, adequate ventilation, cardiovascular stability, and neurological assessment. Because different toxic agents affect different physiological systems, treatment must be individualized according to the pharmacological mechanism, severity of exposure, time since ingestion, and presence of complications. Emergency healthcare professionals should obtain a detailed exposure history whenever possible, including the substance involved, estimated quantity, formulation, route of exposure, and time of ingestion. Information regarding the patient's age, body weight, medical history, and concurrent medications is also important. However, treatment of life-threatening manifestations should not be delayed while waiting for complete exposure information. Laboratory investigations and continuous clinical monitoring are essential components of poisoning management. Depending on the suspected agent, investigations may include blood glucose, serum electrolytes, renal and hepatic function, blood gas analysis, lactate, complete blood count, and specific toxicological concentrations. Electrocardiography is particularly important following exposure to cardiovascular medications, antidepressants, antipsychotics, and other substances associated with conduction abnormalities or ventricular dysrhythmias. Activated charcoal may be considered in selected cases when ingestion has occurred recently and the airway remains protected. However, gastrointestinal decontamination is not appropriate for every toxic substance. Some agents are absorbed rapidly, while others carry substantial aspiration or tissue-injury risks. Therefore, decontamination decisions should follow substance-specific recommendations and consultation with a medical toxicologist when necessary. The following sections describe the principal management considerations for selected high-risk toxic agents.

1. α-Adrenergic Agonists

Management of alpha-adrenergic agonist poisoning is primarily supportive because no universally reliable specific antidote exists. The main therapeutic objectives are maintaining adequate ventilation, correcting cardiovascular instability, and preventing complications associated with profound central nervous system depression. Continuous cardiorespiratory monitoring is necessary because patients may develop significant bradycardia, hypotension, and respiratory depression. Airway protection and ventilatory support should be provided when the patient's respiratory effort becomes inadequate or consciousness deteriorates. Intravenous crystalloid administration may help correct hypotension, although fluid therapy should be guided by the patient's cardiovascular condition. Atropine may be administered when bradycardia produces hemodynamic instability. Patients who remain hypotensive despite appropriate fluid administration may require vasopressor therapy. Naloxone has been reported to improve CNS depression and respiratory manifestations in some cases of clonidine poisoning. However, the clinical response is inconsistent, and naloxone should be regarded as an adjunct rather than a dependable antidote.[17] Nursing responsibilities include frequent assessment of respiratory rate, oxygen saturation, heart rate, blood pressure, and neurological status. Any deterioration in consciousness or respiratory function requires immediate communication with the emergency physician. Accurate recording of the timing of interventions and subsequent physiological responses is important for evaluating treatment effectiveness.

2. Sulfonylureas

Sulfonylurea poisoning requires prompt recognition and treatment because excessive insulin secretion may produce prolonged or recurrent hypoglycemia. Initial assessment should include immediate blood glucose measurement, followed by repeated monitoring throughout the observation period. Patients who remain conscious and can swallow safely may receive oral carbohydrates. Intravenous dextrose is indicated when hypoglycemia produces neurological symptoms or when oral intake is unsafe or impossible. However, dextrose administration alone may stimulate additional insulin secretion and contribute to recurrent hypoglycemia. Octreotide should therefore be administered intravenously or subcutaneously to suppress pancreatic insulin release. Its use reduces the likelihood of repeated hypoglycemic episodes and the need for repeated dextrose administration. Activated charcoal may be considered following recent ingestion when the patient's airway is adequately protected. Suspected pediatric exposure generally warrants at least 24 hours of observation because hypoglycemia may develop or recur after the initial presentation. Discharge should be deferred until the patient maintains normal blood glucose concentrations without intravenous dextrose or octreotide treatment.[18] Nursing staff should monitor blood glucose according to the prescribed schedule, observe for sweating, tremors, confusion, behavioral changes, and seizures, and document every hypoglycemic episode. Laboratory personnel should ensure timely processing and communication of abnormal glucose results. Medical records should include glucose measurements, treatment times, administered dextrose, octreotide doses, and the patient's response.

3. Calcium Channel Blockers

Calcium channel blocker poisoning represents a serious cardiovascular emergency that may progress rapidly to profound hypotension, myocardial depression, conduction abnormalities, and cardiovascular collapse. Management requires continuous electrocardiographic and hemodynamic

monitoring. Blood glucose and serum electrolyte concentrations should also be assessed regularly because hyperglycemia and potassium disturbances may accompany severe poisoning. Early treatment of hypotension may involve carefully administered intravenous fluids, intravenous calcium, and vasopressor therapy. High-dose insulin-euglycemia therapy (HIET) is an established treatment strategy for severe calcium channel blocker toxicity. This intervention improves myocardial carbohydrate utilization and supports cardiac contractility. Because HIET can produce hypoglycemia and hypokalemia, frequent monitoring of blood glucose and serum potassium is essential. Dextrose supplementation may be necessary to maintain appropriate glucose concentrations throughout treatment. Atropine and cardiac pacing may be attempted in selected patients with bradycardia or conduction disturbances, although their effectiveness is often limited in severe poisoning. Methylene blue and intravenous lipid emulsion have uncertain benefits and should generally be reserved for refractory cardiovascular instability after consideration of other interventions. Venoarterial extracorporeal membrane oxygenation (VA-ECMO) may provide circulatory support in patients with refractory cardiogenic shock or cardiovascular collapse despite conventional treatment.[19–23] Other extracorporeal approaches have been investigated. Single-pass albumin dialysis has been proposed for amlodipine poisoning, although its clinical role remains controversial.[24] Therapeutic plasma exchange has also been suggested because some calcium channel blockers exhibit substantial plasma protein binding. Molecular adsorbent recirculating systems have been considered in selected cases of refractory shock.[25,26] Nurses play an important role in monitoring blood pressure, cardiac rhythm, glucose, potassium, and fluid balance. Laboratory professionals should prioritize urgent electrolyte and glucose measurements during HIET. Accurate documentation of vasopressor requirements, insulin administration, glucose supplementation, and hemodynamic responses supports treatment evaluation.

4. β-Blockers

Beta-blocker poisoning may cause severe bradycardia, hypotension, impaired myocardial contractility, atrioventricular block, and cardiogenic shock. Immediate management focuses on restoring adequate cardiac output and maintaining tissue perfusion. Patients with clinically significant bradycardia, hypotension, or cardiogenic shock may require vasopressors and high-dose insulin-euglycemia therapy. Intravenous calcium and glucagon may also be considered as additional therapeutic interventions. Continuous electrocardiographic monitoring is necessary to identify conduction disturbances and ventricular dysrhythmias. Blood glucose and electrolyte concentrations should be assessed regularly, particularly during insulin-based treatment. Propranolol-associated QRS widening requires particular attention because of its additional sodium-channel-blocking activity. Intravenous sodium bicarbonate is recommended for this complication. Seizures should be treated with benzodiazepines. VA-ECMO may be considered as a rescue intervention when severe cardiovascular instability persists despite conventional treatment. Hemodialysis may also be useful for selected beta-blockers with sufficient dialyzability, including atenolol, nadolol, sotalol, and acebutolol.[27] Nursing responsibilities include continuous assessment of heart rate, blood pressure, consciousness, and peripheral perfusion. Patients receiving HIET require close glucose and potassium monitoring. Any development of seizures, worsening hypotension, or significant electrocardiographic changes must be reported immediately.

5. Tricyclic Antidepressants

Tricyclic antidepressant poisoning is a medical emergency because these medications can produce seizures, severe hypotension, cardiac conduction abnormalities, and potentially fatal ventricular dysrhythmias. Initial management includes airway assessment, cardiorespiratory stabilization, and immediate electrocardiography. Activated charcoal may be considered early after ingestion when the airway is protected and aspiration risk is controlled. Intravenous sodium bicarbonate represents the first-line intervention for clinically significant QRS widening, ventricular dysrhythmias, or hypotension associated with tricyclic antidepressant toxicity. Its therapeutic effects involve serum alkalinization and sodium loading, which help reduce the consequences of cardiac sodium-channel blockade. Seizures should be treated with benzodiazepines. Persistent hypotension requires appropriate cardiovascular support, including norepinephrine infusion when indicated. Lidocaine may be considered for ventricular dysrhythmias that remain refractory to standard treatment. Intravenous lipid emulsion therapy and VA-ECMO may be considered rescue options in selected patients who fail to respond to conventional interventions. However, extracorporeal removal techniques, including hemodialysis, hemoperfusion, and conventional continuous renal replacement therapy, are ineffective for removing these medications because of their pharmacokinetic properties.[28] Other proposed interventions, including esmolol infusion and CytoSorb-assisted hemoadsorption, have been described in isolated clinical reports but should not be considered established treatment strategies.[29–31] Laboratory assessment should include electrolytes, blood gas analysis, glucose, and renal function. Nurses should monitor neurological status, seizures, blood pressure, and

electrocardiographic changes. Medical records should document QRS duration, administered sodium bicarbonate, seizure treatment, and cardiovascular responses.

6. Opioids

The immediate priority in opioid poisoning is restoring adequate ventilation and preventing hypoxic injury. Respiratory depression may progress rapidly, particularly following exposure to potent synthetic opioids or long-acting opioid formulations. Initial management involves assessment of respiratory effort, oxygen saturation, airway patency, and consciousness. Bag-mask ventilation should be provided when spontaneous breathing is inadequate. Advanced airway management may be necessary in patients who cannot maintain adequate ventilation or protect their airway. Naloxone should be administered and titrated to restore sufficient respiratory function rather than necessarily achieving complete consciousness. This approach aims to reverse life-threatening respiratory depression while avoiding unnecessary opioid withdrawal symptoms. Repeated naloxone doses or continuous intravenous infusion may be required when respiratory depression recurs or when the opioid has a longer duration of action than naloxone. Patients must remain under observation until their consciousness, ventilation, and vital signs remain stable without further naloxone administration for an interval appropriate to the specific opioid, dose, and formulation. Nursing staff should monitor respiratory rate, oxygen saturation, consciousness, and response to naloxone. Documentation should include the administered dose, route, time, clinical response, and any recurrence of respiratory depression.

7. Antidiarrheal Medications

Poisoning involving diphenoxylate-atropine or loperamide requires particular attention because these agents may produce opioid-related respiratory depression and, in severe cases, cardiovascular complications. Initial management should prioritize airway protection and adequate ventilation. Naloxone should be administered when opioid-related respiratory depression is present. Repeated administration may be necessary if respiratory suppression recurs. Diphenoxylate-atropine poisoning may produce delayed and recurrent respiratory depression. Therefore, patients, particularly children, should remain under observation until they are asymptomatic without naloxone treatment. Large loperamide exposures require continuous electrocardiographic monitoring because of the risk of QRS widening, QT prolongation, and ventricular dysrhythmias. Serum potassium and magnesium should be measured and corrected when abnormalities are identified. Sodium bicarbonate may be administered for clinically significant QRS widening. Torsades de pointes and other ventricular dysrhythmias require appropriate cardiac management, including intravenous magnesium, defibrillation when indicated, and chronotropic treatment or overdrive pacing in consultation with a medical toxicologist or cardiologist.[32]

8. Salicylates and Methyl Salicylate

Salicylate poisoning requires serial assessment because serum concentrations and clinical manifestations may change over time. Initial evaluation should include serum salicylate concentration, acid-base status, blood glucose, potassium, renal function, and neurological condition. Repeated salicylate measurements should continue until concentrations demonstrate a clear and sustained decline. Sodium bicarbonate is a central treatment intervention because alkalinization increases urinary salicylate elimination and reduces the movement of salicylate into the central nervous system. Intravenous bicarbonate therapy requires careful monitoring of blood pH, potassium, glucose, and renal function. Hemodialysis is indicated in severe poisoning, particularly when patients develop significant CNS toxicity, pulmonary edema, renal failure, severe acidemia, or clinical deterioration despite alkalinization. Endotracheal intubation may become necessary in patients with severe neurological deterioration or respiratory compromise. However, intubation carries considerable risks because these patients often maintain high minute ventilation to compensate for metabolic acidosis. If intubation cannot be avoided, clinicians must maintain alkalemia and immediately reproduce the patient's preintubation ventilation and low carbon dioxide tension.[33] Nurses should monitor respiratory patterns, mental status, fluid balance, and neurological changes. Laboratory personnel must provide timely salicylate concentrations and acid-base results to support treatment decisions.

9. Antipsychotic Medications

No specific antidote is available for most antipsychotic poisonings. Management therefore focuses on supportive treatment and prevention of neurological and cardiovascular complications. Airway and ventilatory support should be provided when consciousness deteriorates or respiratory function becomes inadequate. Significant exposures require continuous electrocardiographic monitoring because of the possibility of QT prolongation and ventricular dysrhythmias. Intravenous fluids may be administered for hypotension, while norepinephrine infusion may be necessary when hypotension persists despite appropriate fluid resuscitation. Benzodiazepines are used to manage agitation and seizures. Intravenous magnesium is generally administered when the corrected QT interval exceeds 500 milliseconds, particularly when additional risk factors for ventricular dysrhythmias are present. Activated charcoal may be considered early following ingestion when airway protection is adequate.[34] Nursing monitoring should include consciousness, agitation, muscle movements, blood

pressure, heart rate, and cardiac rhythm. Documentation of electrocardiographic findings and administered interventions is important for continuity of care.

10. Antimalarial Medications

Management of antimalarial poisoning depends on the specific drug involved. Chloroquine and hydroxychloroquine poisoning require particularly urgent intervention because severe cardiovascular and neurological complications may develop rapidly. Immediate airway and ventilatory support should be provided when necessary. Continuous electrocardiographic monitoring is essential to identify QRS widening, QT prolongation, and ventricular dysrhythmias. Epinephrine is used to manage shock, while benzodiazepines are administered for seizures. High-dose diazepam has been used in severe chloroquine poisoning. Blood glucose and potassium concentrations require frequent assessment. Hypokalemia may be pronounced because potassium shifts into cells. Replacement should therefore be performed cautiously because rebound hyperkalemia may occur as the toxic effects resolve. Sodium bicarbonate or hypertonic sodium administration may be considered in patients with marked QRS widening. VA-ECMO may be considered when cardiovascular collapse persists despite conventional treatment. Extracorporeal drug removal is generally ineffective. Quinine-associated hypoglycemia requires intravenous dextrose with continuous glucose monitoring. Clinically significant primaquine-associated methemoglobinemia may require methylene blue, although this treatment is contraindicated or requires particular caution in patients with glucose-6-phosphate dehydrogenase deficiency.

11. Antiarrhythmic Medications

Antiarrhythmic poisoning requires continuous electrocardiographic and hemodynamic monitoring because these agents can directly interfere with cardiac electrical conduction and myocardial repolarization. Class I sodium-channel blocker toxicity is managed with intravenous sodium bicarbonate when QRS widening, ventricular dysrhythmias, or hypotension occurs. This intervention is particularly important in flecainide poisoning. Lidocaine infusion may be considered for refractory toxicity associated with class IA or IC agents. VA-ECMO may provide circulatory support in patients with refractory cardiogenic shock. Class IA-associated torsades de pointes requires intravenous magnesium administration and correction of potassium abnormalities. Chronotropic treatment or overdrive pacing may be necessary in selected cases. Intravenous lipid emulsion has been proposed as a salvage intervention for severe toxicity, although its clinical benefit remains unproven. Amiodarone is generally contraindicated in the management of sodium-channel blocker poisoning because it may aggravate conduction abnormalities and prolong cardiac repolarization. Nurses should monitor cardiac rhythm, blood pressure, and neurological status continuously. Laboratory investigations should include serum potassium, magnesium, calcium, and acid-base measurements.

12. Camphor

No specific antidote exists for camphor poisoning. Management is supportive and focuses on preventing respiratory complications and controlling seizures. Patients require prompt assessment of airway patency, respiratory function, and neurological status. Benzodiazepines are the first-line treatment for seizures. Persistent seizures may require additional antiseizure medications or anesthetic management according to standard emergency protocols. Contaminated clothing should be removed, and exposed skin should be washed to reduce further absorption. Activated charcoal and gastric lavage should generally be avoided because camphor is rapidly absorbed and carries a substantial aspiration risk. Nurses should monitor for sudden neurological deterioration because seizures may occur abruptly. Continuous observation and documentation of seizure activity, consciousness, and respiratory status are essential.[17]

13. Nonalkaloid Toxic Lignans

Podophyllin and podofilox poisoning require early recognition because systemic toxicity may affect gastrointestinal tissues, bone marrow, and multiple organs. Gastrointestinal decontamination may be considered following significant exposure when ingestion occurred recently and the airway is protected. Decisions should be made in consultation with a medical toxicologist. Treatment primarily consists of intensive supportive care. Serial complete blood counts are necessary to detect leukopenia, thrombocytopenia, and other manifestations of bone marrow suppression. Electrolytes, renal function, and hepatic function should also be monitored. Management should address gastrointestinal fluid losses, seizures, infections, bleeding, and organ dysfunction. Patients with severe poisoning may require intensive care monitoring because complications can develop progressively. Nursing responsibilities include monitoring temperature, hydration, bleeding manifestations, neurological condition, and signs of infection. Laboratory results should be reviewed regularly to identify developing hematological abnormalities.

14. Colchicine

Colchicine poisoning requires early intervention because gastrointestinal symptoms may progress to severe systemic toxicity, shock, bone marrow suppression, and multiorgan failure. Activated charcoal may be administered early following significant ingestion when the airway and gastrointestinal tract permit. Multiple-dose activated

charcoal may be considered because colchicine undergoes enterohepatic recirculation. Patients with significant exposure require intensive supportive management. Treatment should address fluid losses, hypotension, cardiac dysrhythmias, and organ dysfunction. Continuous monitoring of complete blood counts, renal function, hepatic function, and serum electrolytes is essential. Severe neutropenia may warrant consideration of granulocyte colony-stimulating factor. Limited evidence suggests that hemodialysis or plasma exchange may provide benefit in selected cases of severe poisoning, although these interventions are not established methods of reliably removing colchicine.[35] Nurses should monitor gastrointestinal symptoms, urine output, hemodynamic parameters, and signs of infection or bleeding. Accurate documentation of laboratory trends is important for identifying progressive bone marrow suppression and organ injury.

15. Oral Acetylcholinesterase Inhibitors

Poisoning involving oral acetylcholinesterase inhibitors and carbamate pesticides produces excessive cholinergic activity and may cause life-threatening respiratory complications. Airway management is a primary priority. Early airway protection should be considered when bronchorrhea, bronchospasm, central respiratory depression, or neuromuscular weakness threatens adequate ventilation. Excessive respiratory secretions should be removed through appropriate suctioning. Atropine should be administered promptly and titrated according to improvement in bronchial secretions, oxygenation, and ventilation. Treatment should not be guided solely by pupil size or heart rate. Pralidoxime should be administered early when organophosphate poisoning is suspected or when the responsible agent remains uncertain. Routine pralidoxime administration is not required for uncomplicated carbamate poisoning. Benzodiazepines are indicated for seizures and severe agitation. Healthcare professionals must use appropriate personal protective equipment when external contamination is possible. Contaminated clothing should be removed, and exposed skin should undergo appropriate decontamination. Succinylcholine and mivacurium should be avoided because their neuromuscular blocking effects may become prolonged in patients with cholinesterase inhibition. Nursing staff should monitor respiratory secretions, oxygen saturation, respiratory muscle function, heart rate, and blood pressure. Laboratory assessment should support evaluation of the patient's metabolic condition and, when appropriate, cholinesterase activity.

16. Methylxanthines

Methylxanthine poisoning, particularly theophylline toxicity, may produce severe neurological and cardiovascular complications. Management includes seizure control, cardiovascular monitoring, and reduction of systemic drug exposure when indicated. Benzodiazepines are used as first-line treatment for seizures. Activated charcoal may be administered following oral exposure when the airway is protected. Multiple-dose activated charcoal can enhance the elimination of theophylline and may be useful in significant poisoning. Tachydysrhythmias may require specific cardiovascular management. Beta-blocker administration remains controversial, although a short-acting agent such as esmolol may be considered in selected patients. Intermittent hemodialysis is recommended for severe theophylline poisoning associated with seizures, life-threatening dysrhythmias, shock, increasing serum concentrations, or clinical deterioration. Hemodialysis may also be considered in massive caffeine poisoning that does not respond adequately to conventional treatment.[36,37] Laboratory monitoring should include serum theophylline concentrations, glucose, potassium, renal function, and acid-base status. Repeated drug concentration measurements are important for assessing the response to treatment.

17. Partial Opioid Agonists and Synthetic Opioids

Buprenorphine and fentanyl poisoning require immediate attention because both substances can cause significant respiratory depression. The primary management priorities are adequate ventilation and naloxone administration when opioid-related respiratory depression is present. Patients with inadequate respiratory effort should receive bag-mask ventilation or advanced airway support as clinically indicated. Buprenorphine-associated respiratory depression may respond incompletely or slowly to conventional naloxone doses because of its high receptor affinity. Higher cumulative naloxone doses or continuous infusion may therefore be necessary in selected patients. Prolonged observation is recommended following symptomatic buprenorphine exposure because respiratory depression may persist or recur. Fentanyl poisoning is managed according to the same general principles, with particular attention to rapid respiratory deterioration and the possible need for repeated naloxone administration. Nurses should document the patient's respiratory response, naloxone administration, oxygen requirements, and recurrence of symptoms.

18. Toxic Alcohols

Methanol and ethylene glycol poisoning require urgent treatment because their metabolites can cause severe metabolic acidosis and irreversible organ damage. Fomepizole is used to inhibit alcohol dehydrogenase, thereby reducing the formation of toxic metabolites from methanol and ethylene glycol. Sodium bicarbonate may be administered to correct significant metabolic acidosis. Additional supportive treatments include folinic acid or folic acid for methanol poisoning and thiamine and pyridoxine as adjunctive treatments for ethylene glycol poisoning.

Intermittent hemodialysis is indicated in selected patients with severe poisoning. Clinical indications include severe acidemia, a substantially increased anion gap, coma, seizures, methanol-associated visual abnormalities, significant ethylene glycol-associated kidney injury, or sufficiently high toxic alcohol concentrations according to applicable clinical guidelines.[38,39] Laboratory investigations should include blood gas analysis, electrolytes, anion gap, renal function, glucose, and specific toxic alcohol concentrations when available. Serial monitoring is essential because metabolic abnormalities may progress despite initial supportive treatment. Isopropanol poisoning generally requires supportive management. Fomepizole and other alcohol dehydrogenase inhibitors are not indicated because they may prolong the presence of the parent alcohol.

19. Caustics and Other Household Products

Caustic poisoning requires immediate assessment of airway involvement and potential gastrointestinal injury. Management should prioritize airway protection because progressive edema and tissue damage may compromise ventilation. Vomiting should never be induced following caustic ingestion because repeated exposure can increase tissue injury. Initial management should include assessment of oral and pharyngeal injuries, respiratory symptoms, swallowing difficulties, and evidence of gastrointestinal complications. Consultation with medical toxicology or gastroenterology is recommended according to the substance and clinical presentation. Certain household and industrial chemicals require specific treatment. Hydroxocobalamin may be indicated for cyanide toxicity associated with acetonitrile exposure. Methylene blue may be required for clinically significant methemoglobinemia caused by oxidizing products. However, its use requires consideration of contraindications, including glucose-6-phosphate dehydrogenase deficiency. Methylene chloride-associated carbon monoxide toxicity requires administration of 100% oxygen and appropriate monitoring. Hydrofluoric acid exposure requires immediate irrigation and calcium therapy to counteract fluoride-related toxicity. Electrocardiographic monitoring and repeated assessment of calcium, magnesium, and potassium are necessary because severe electrolyte disturbances may produce life-threatening cardiac complications.[16] Nursing staff should document the exposure route, affected body areas, respiratory condition, and treatment response. Laboratory professionals should prioritize electrolyte measurements and other investigations indicated by the suspected chemical.

20. Multidisciplinary Responsibilities in the Management of Toxic Exposures

Effective management of potentially lethal single-dose agents depends on coordination between emergency physicians, laboratory professionals, nurses, pharmacists, and medical records personnel. Each discipline contributes to early recognition, treatment selection, monitoring, and evaluation of patient outcomes.

Emergency Department Responsibilities

Emergency department personnel are responsible for initial stabilization and identification of life-threatening complications. Their activities include:

- Obtaining an exposure history and identifying the suspected toxic agent.
- Assessing airway patency, breathing, circulation, and neurological status.
- Identifying characteristic toxidromes.
- Initiating appropriate supportive treatment.
- Ordering relevant laboratory investigations and electrocardiography.
- Consulting medical toxicology and other specialties when necessary.
- Arranging intensive care admission when the patient's condition requires advanced monitoring or organ support.

Clinical Laboratory Responsibilities

The clinical laboratory provides information essential for assessing the severity of poisoning and monitoring treatment responses. Important responsibilities include:

- Performing urgent blood glucose measurements.
- Assessing electrolytes and renal and hepatic function.
- Conducting blood gas analysis and evaluating acid-base disturbances.
- Measuring specific toxicological concentrations when indicated.
- Performing complete blood counts to identify hematological complications.
- Communicating critical laboratory findings promptly to the treating team.
- Maintaining quality control and accurate reporting of analytical results.

Laboratory turnaround time is particularly important in poisoning involving hypoglycemic agents, salicylates, toxic alcohols, and medications associated with severe electrolyte abnormalities.

Nursing Responsibilities

Nurses provide continuous bedside assessment and implement prescribed interventions. Their responsibilities include:

- Monitoring vital signs and oxygen saturation.
- Assessing consciousness and neurological changes.
- Observing respiratory effort and airway secretions.
- Monitoring cardiac rhythm and hemodynamic stability.

- Administering medications and intravenous fluids as prescribed.
- Monitoring glucose and electrolyte results.
- Recording treatment responses and adverse events.
- Communicating clinical deterioration immediately.
- Supporting safe patient transfer and continuity of care.

Medical Records and Health Information Management Responsibilities

Accurate medical documentation is essential for clinical communication, patient safety, and institutional quality improvement. Medical records personnel support the management process through:

- Recording the suspected toxic agent and exposure circumstances.
- Documenting the time of presentation and initial clinical findings.
- Maintaining accurate records of laboratory and electrocardiographic results.
- Recording medications, doses, routes, and administration times.
- Documenting consultations, clinical decisions, and treatment outcomes.
- Maintaining confidentiality and appropriate access to patient information.
- Supporting clinical audits and analysis of poisoning-related incidents.

Standardized documentation can help healthcare institutions identify recurrent exposure patterns, evaluate treatment delays, and develop preventive policies.

21. Clinical Monitoring and Patient Safety

Monitoring should continue throughout the period of potential toxicity rather than ending after initial symptomatic improvement. Some substances produce delayed complications, prolonged absorption, recurrent hypoglycemia, or persistent respiratory depression. Therefore, observation periods should reflect the pharmacological characteristics of the suspected agent and the patient's clinical condition.

Particular attention should be directed toward the following parameters:

- Neurological status, including consciousness, behavior, and seizure activity.
- Respiratory rate, oxygen saturation, and adequacy of ventilation.
- Heart rate, blood pressure, and electrocardiographic findings.
- Blood glucose and electrolyte concentrations.
- Renal and hepatic function.
- Acid-base status and metabolic abnormalities.
- Urine output and fluid balance.
- Evidence of bleeding, infection, or bone marrow suppression when relevant.

Before discharge, healthcare professionals should confirm that the patient's clinical condition has stabilized and that agent-specific observation requirements have been satisfied. Patients and caregivers should receive appropriate information about medication storage, prevention of accidental ingestion, and the importance of seeking medical assessment after suspected exposure. Overall, the management of deadly single-dose agents requires early recognition, appropriate supportive treatment, targeted interventions, and continuous reassessment. Coordination between emergency medicine, clinical laboratory services, nursing teams, and medical records departments supports timely clinical decisions and reduces the risk of preventable complications. Establishing standardized assessment and documentation procedures can also improve institutional preparedness and strengthen medication and chemical safety practices.

Summary and Interprofessional Team Considerations

The management of potentially lethal single-dose toxic agents requires a coordinated, multidisciplinary approach that extends from initial exposure assessment and prehospital stabilization to emergency treatment, hospital admission, discharge, and subsequent follow-up. Because these substances can affect multiple organ systems and produce rapid clinical deterioration, effective communication and collaboration among healthcare professionals are essential for reducing morbidity and mortality. The management process involves emergency medical services, emergency physicians, critical care specialists, nurses, clinical laboratory professionals, pharmacists, poison control centers, medical toxicologists, mental health professionals, and addiction specialists. Emergency medical services play an important role in the early recognition of poisoning, initial assessment of vital signs, stabilization of the patient, and safe transportation to an appropriate healthcare facility. Emergency department clinicians are responsible for identifying the suspected toxic agent, evaluating the severity of exposure, recognizing characteristic toxidromes, and initiating supportive or specific treatment. Patients with respiratory depression, cardiovascular instability, seizures, or altered consciousness may require immediate advanced interventions and admission to an intensive care unit.

Nurses contribute throughout the treatment process through continuous monitoring, medication administration, assessment of neurological and respiratory status, and early identification of clinical deterioration. Their role is particularly important in patients receiving antidotes, vasopressors, intravenous fluids, glucose supplementation, or other interventions requiring repeated clinical and

laboratory assessment. Clinical laboratory professionals support decision-making by providing timely and accurate results for blood glucose, electrolytes, renal and hepatic function, blood gases, toxicological concentrations, and other relevant investigations. Pharmacists contribute through medication reconciliation, identification of potentially toxic drug interactions, assessment of medication formulations, and support for appropriate antidote selection and dosing. Poison control centers and medical toxicologists provide specialized guidance regarding exposure identification, expected clinical progression, appropriate observation periods, laboratory investigations, and treatment strategies. Early consultation is particularly valuable when the suspected substance is unknown, the exposure involves multiple agents, or the patient develops unusual or severe manifestations. Such consultation can help healthcare teams avoid unnecessary interventions while ensuring that potentially serious exposures receive appropriate monitoring and treatment. The interprofessional approach should continue after the patient's clinical condition stabilizes. Intentional medication ingestion requires additional assessment because it may indicate suicidal behavior, psychological distress, substance use disorders, or other underlying problems. Patients with suspected intentional poisoning should undergo an appropriate suicide risk assessment and receive psychiatric evaluation when indicated. Mental health professionals and addiction specialists can contribute to treatment planning, counseling, rehabilitation, and strategies to prevent recurrent poisoning. Discharge planning should also address medication safety, appropriate storage of pharmaceutical products, caregiver education, and access to follow-up services.

Evidence-based triage pathways can improve the efficiency of emergency services by identifying patients who require hospital assessment, prolonged observation, or intensive treatment. Selected low-risk exposures may be managed through poison control guidance and structured follow-up without unnecessary emergency department attendance, provided that appropriate clinical criteria are satisfied and patient safety is maintained.[40] Emerging technologies, particularly artificial intelligence and machine learning, may provide additional opportunities to improve toxicological surveillance, identify exposure patterns, predict clinical deterioration, and support risk stratification. These technologies may assist healthcare institutions in analyzing large datasets, identifying high-risk medication exposures, and improving the coordination of emergency responses. However, their clinical application requires appropriate validation, assessment of accuracy, and consideration of patient safety. Artificial intelligence should complement professional expertise rather than replace clinical judgment, laboratory confirmation, or consultation with medical toxicologists.[41] Medical records and health information management professionals also contribute to the quality of poisoning care by ensuring accurate documentation of exposure history, clinical findings, laboratory results, treatment interventions, consultations, and patient outcomes. Standardized documentation facilitates communication between departments, supports continuity of care, and provides information for clinical audits, institutional surveillance, and preventive healthcare strategies. Overall, effective management of deadly single-dose toxic agents depends on coordinated interprofessional collaboration, timely clinical assessment, reliable laboratory services, appropriate nursing interventions, and accurate medical documentation. Integrating these responsibilities across the healthcare continuum provides the best opportunity to improve acute clinical outcomes, prevent recurrent poisoning, reduce avoidable complications, and support long-term patient recovery.

Conclusion:

Potentially lethal single-dose toxic agents represent an important cause of preventable morbidity and mortality among children and adults. Exposure to relatively small quantities of certain medications, chemicals, and household products can produce severe cardiovascular, respiratory, neurological, and metabolic complications. Early recognition of characteristic toxicological manifestations, accurate exposure assessment, and timely laboratory investigations are essential for appropriate clinical management. Treatment may involve supportive care, specific antidotes, cardiovascular stabilization, respiratory assistance, and continuous monitoring. Nurses contribute through patient observation and timely intervention, while laboratory professionals provide essential diagnostic information. Emergency clinicians coordinate treatment, and medical records personnel ensure accurate documentation and continuity of care. Collaboration among these professionals, supported by poison control services and medical toxicologists, improves clinical decision-making and patient safety. Strengthening medication storage practices, public awareness, standardized emergency protocols, and interprofessional communication can further reduce accidental exposures and prevent recurrent poisoning. Ultimately, an integrated healthcare approach is essential to improving outcomes and reducing the complications associated with potentially lethal single-dose toxic agents.

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