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Acaricide Poisoning and Newer Cocktails: A Review of Current Treatment Recommendations

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ABSTRACT

Acaricides are a group of compounds developed in the early 1960's to combat infestations of ticks and mites among livestock and plants. These parasites, left unchecked, have been known to cause multiple diseases among the host animal or plant leading to debilitating illness, livestock deaths and crop failures. Some of these insects are also natural reservoirs for multiple zoonotic diseases, easily spread to humans leading to major disease outbreaks. As a result, many farmers and horticultural experts pushed for chemical controls of these pests, but with time, some of these parasites have developed resistance to single agent use, necessitating the development of chemical cocktails and newer agents for effective pest control.

These chemicals, when applied correctly, were relatively harmless to livestock and plants and did not linger in the environment, but with the development of chemical resistance, some "super acaricides" have much higher levels of toxicity and are much easier to be absorbed after exposure. In East Africa, most acaricides poisoning is found during accidental ingestion, such as ingestion by unknowing children, exposure following poor handling, as well as intentional ingestion seen in suicide attempts. Some of these super acaricides present a huge challenge in patient care as there is little information on these newfound cocktails and their effective clinical management.

We discuss the presentation of the common acaricides seen on the East African Market, common approaches and potential pitfalls seen in the management of acaricide poisoning in most patients as a guide for most emergency and critical care providers as well as emergency room clinicians.

Pesticides and Acaricides

Acaricides are a form of pesticide, invented due to the high resilience of insects and arachnids to organic and natural methods of pest control, as well as to typical organophosphates [1,2]. These chemicals were specifically engineered to kill ticks and mites, to which they were developed as concentrated chemicals to be diluted and sprayed on both livestock and crops. They were heralded for their effectiveness and ability to increase horticultural yields by up to 30% and reduce costs of production by up to 13 billion dollars annually globally [1,3]. With this newfound tool, farmers flocked to its use, specifically in animal husbandry of cattle, goats and poultry, enabling them to maximize profits with little overhead [4]. They consist of a wide variety of chemicals spread over various classifications including some organophosphates.

Unfortunately, these chemicals killed mites, ticks and other insects/ arachnids indiscriminately leading to lack of natural and harmless predators as well as development of resistance. The resistance to these agents has led to development and use of various bespoke compounds which may contain two or more different acaricides or even other classes of toxins as a way around this.

The chemicals were also known to linger longer in the environment and in various food chains, poisoning multiple ecosystems all the way up to local human food supply [5,6]. Unfortunately, this also resulted in a new challenge to clinicians in the management of these poisonings as the typical presentation of these agents has become variable, especially since there are no widely available clinical guidelines on poisoning management more so with these bespoke compounds. This is more evident in low resource areas where chemicals banned in other countries at times wound up in the market [5]. This has led to confusion and challenges in management, especially in areas not receiving frequent similar cases or not previously exposed to these newer compounds.

Due to its extremely potent nature and its rapid absorption through skin or the gastrointestinal tract, patients exposed to the chemicals present with dramatic and severe symptoms. These agents are also extremely fat soluble, which then sequester in the body's fat cells, released during stress or lipolysis later on, presenting with variations in peaks and severity of symptomatology [6]. These patients also usually present out of the 1-hour advisory period recommended for gastric lavage [7]. With no known reliable antidote for these chemicals currently available or under testing, most clinicians are forced to treat patients symptomatically, often being admitted to ICU or HDU due to the severity of presentations.

Most countries including Kenya, Uganda and Tanzania have regulatory bodies mandated to give oversight over the different manufacturers, distributors and suppliers. While they provide these necessary services, they are typically overwhelmed by the sheer number of suppliers, rapid product turnover and illegal products. This is also demonstrated by the almost copy-paste data sheets, unmanned distress/call-center numbers seen in most of these products highlighting the probable lack of comprehensive product testing, especially in human safety.

INCIDENTAL POISONING

Patients may present with two different aspects of accidental poisoning. Most dramatic and common, more so in farmers, is when poor handling and storage of these chemicals leads to accidental ingestion of these chemicals by children. Typically, toddlers and other young children mistake these chemicals for soft drinks or medicines. These chemicals can be sold in bottles resembling syrup medications or similar packaging, confusing these children. It is also common for these chemicals to be sold in measured quantities or for farmers to share with others by dispensing some quantity in disposable water or soda bottles, or even farmers to mix various brands into one container. Sellers also take old soda or water bottles and dispense the compounds at quantities the buyer would like or can afford. These dispensed bottles don't retain the warning labels as well as any other safety information and neither does the modality come with child proof bottle tops/ safety locks. These chemicals have also been shown to denature when stored improperly in high heat or in direct sunlight leading to formation of various volatile and toxic compounds, compounding the problem [8].

Children often don't have refined reflexes such as spitting out chemicals or foresight to distinguish foul smelling agents, and therefore whatever is in the mouth is usually swallowed [9]. For adults, this usually comes about by either lack of adequate personal protective equipment (PPE) or poor storage of these chemicals [5,9]. This is most evident during the mixing and preparation stages and among new farmers. Adequate PPE is often out of reach of most farmers, they sometimes use polythene bags as a barrier and these usually have holes or do not ensure adequate protection [5,10].

Incidental poisoning is on the rise with the above reasons but mostly putting into consideration the ease of absorption through various access routes/entry by these agents into the body such as by oral ingestion, inhalation, and dermal routes, making persons handling these agents prone to poisoning [7].

INTENTIONAL EXPOSURE

Mental health and wellness are a major problem worldwide as well as in East Africa. With the rising cost of living, stressful environments, lack of resources and unemployment, patients are evolving maladaptive behaviors including suicide and attempted suicide [7,11,12]. As these pesticides are readily available in most agrovet shops at a cheap price, patients with depression and other psychiatric illnesses tend to take them as a means of achieving a painless death [7,11-13]. Many cases come with co-ingestion with alcohol or other common prescription drugs including paracetamol, antidiabetic and antihypertensive drugs, more so among young adults [11].

METHODS AND SCOPE

The authors focused on looking into the most commonly available acaricides found on the East African market. This entailed discussions on product sale estimates by various high volume agrochemical suppliers, interviews with members of various regulatory bodies as well as interviews with local farmers to ascertain the product of choice in these communities. Interviews with major farmers

were able to demonstrate these high-volume suppliers confirmed with confidential information through regulatory bodies. We were able to sample multiple poultry farmers, many of which had over 10,000 commercially viable stocks as well as various major cattle, sheep, pig and goat farmers with substantial livestock classified as being over half a million yearly turnover by sales. Three major agricultural cities across Kenya, Uganda and Tanzania were sampled. Accordingly, three major suppliers in each city were reviewed to which the reviewers were able to either buy or access the highest sales volume acaricides by these distributors. The team was also able to access the data sheets of these compounds found on the sale items, correlate this with the online data sheets or other brand resources as well as compare this with worksheets of various regulatory and oversight authorities. The team also investigated farmers preference through in-person interviews, correlational data from poisoning cases at more than six major hospitals as well as through online farmers forums. No brands, suppliers or major distributors are mentioned for confidentiality and legal reasons.

The team was able to analyze the data collected through interviews and data sheet analysis as well as from information of various regulatory bodies. The team endeavored to include as many distinct chemical compounds found throughout the data collection period. Only chemicals actively being sold in the market were included. The team excluded any banned products, unlicensed products, chemicals not actively being sold or being phased out and any chemicals with regulatory advisory notices or recall. The whole population was sampled and over 48 brands were included with a large overlap within countries as most were sold across borders by the same manufacturer under similar if not the same trade names. We must insist that the compounds discussed are not exhaustive as the market remains volatile and new formulary are approved or withdrawn daily within these markets.

COMMON COMPOUNDS FOUND IN THE MARKET

The chemicals can be classified based on their mode of action and receptor preference. They may act on various receptors simultaneously and have multiple modes of action. Readers must remember combinations exist and some may be homemade mixtures of various brands. We also only took into consideration the effects of the active ingredients and did not include the possible effects of other solvents and preservatives not meant for human consumption that are used in these chemicals.

Table 1: Active agents

Compound	**Receptor activation**	**Literature/theoretical antidote**	**Key management**
Amitraz/ Actraz	Mainly Alpha 2 adrenergic agonist, multiple active metabolites- inhibition of MAO, H1 receptor, PGE2 synthesis, insulin release, LH release/ infertility.	Low dose atropine*	Decontamination, oxygen, arrhythmia prevention, BP control, atropine till secretions dry.
Chlorpyrifos	Requires CYP 450 activation to active metabolite- Acetylcholinesterase inhibition, other esterase inhibition through metabolites.	As per organophosphates, gastric lavage with sodium bicarbonate and activated charcoal.	Prevent liver and kidney failure. Atropine and pralidoxime as per need. Sodium bicarbonate is better given intravenously following BGA.
Chlorfenapyr/ tralopyril^	Inhibits mitochondrial oxidative phosphorylation by forming cyanide- reduction in ADP to ATP formation.	Hydroxocobalamin and sodium nitrate.	Decontamination, oxygen, seizure, bronchospasm mx, fluid mx.
Cymethrin/cypermethrin/ alpha cypermethrin / deltamethrin/ sumithrin/phenothrin/ cyhalothrin/ Deltamethrin	Organochlorine inhibits closure of sodium channels, causing continuous depolarization, inhibits GABA receptors and inhibits Ca2+and Mg 2+ channels.	No antidote, Sodium and magnesium supplementation. Some may be responsive to calcium supplementation.	Decontaminate, gastric lavage, oxygen, seizure management, anaphylaxis management, vitamin E ointment for skin, avoid zinc adhesive strapping.
Chlorfenvinphos	Organophosphate/ organochlorine inhibitor of acetylcholine.	As per organophosphate, Atropine, Glycopyrrolate, Pralidoxime.	As per organophosphate, the case for pralidoxime is high.
Fipronil	Blocks Chloride channels as well as production of Cyanide with mitochondrial toxicity.	High dose Vitamin C and E.	Decontamination, oxygen therapy, seizure management and other supportive therapies.
Ethylene dibromide/ dibromoethane/ glycol bromide	Unclear, extremely liver and kidney toxic metabolites.	None, aggressive decontamination, avoid bromine containing meds.	Extremely carcinogenic. Decontaminate, oxygen, seizure management, gastric lavage.

^ Has very slow toxicokinetic's and may take 3 days to fully manifest due to metabolites. + Atropine of sufficient dose to clear respiratory secretions. + BGA- Blood gas analysis, ADP- Adenosine diphosphate, ATP- Adenosine triphosphate References include [14-17], Extensive credit to Pub Chem and the National Center for Biotechnology Information.

CLINICAL PRESENTATIONS

All presentations will be individualized with many variables increasing the severity of presentations. Some of the risk factors for severe presentations include:

- High amounts of chemicals have been ingested.
- Brands with multiple active chemicals and highly concentrated chemicals or mixtures of multiple brands.
- Method of exposure with ingestion being extremely potent followed by skin then inhalation.
- Age of presentation- worse in the extremes of age.
- Low body weight and low BMI.
- Prolonged time to present to the hospital.
- Co-ingestion with medications and alcohol.
- Continued exposure through skin or clothing.
- Comorbidities including diabetes, hypertension, psychiatric illness and others.

Death is usually due to respiratory related issues more so caused by sedation, seizures and secretions causing hypercapnia and hypoxia. Signs and Symptoms will generally be classified as cholinergic or anticholinergic depending on the agent. However, the symptoms vary depending on the drug, dosage and individual physiology or receptor activation.

Table 2: Receptor action

Receptor activation	Neurologic	Cardiovascular	Gastrointestinal	Neuromuscular	Others
Alpha 2 adrenergic agonist	Alpha 2-receptors: sedation, decreased sympathetic activity, analgesia (cholinergic).	Mild Alpha1-receptors: vasoconstriction leading to increased BP and eventually lowered CO, increased afterload.	Alpha2-receptors: decreased motility, contract esophageal and anal sphincter.	Alpha 2-receptors: Contract smooth muscles in the urinary bladder neck	
Acetylcholinesterase inhibition	Confusion, agitation, impaired cognition, memory impairment, convulsions, coma	Bradycardia, decreased BP, dizziness, fainting.	Increased secretions, nausea, vomiting, diarrhea, excessive bowel movement, abdominal cramps	Increased muscle contraction, muscle fasciculation, muscular paralysis, respiratory distress, excessive sweating, miosis.	Muscle weakness, twitching.
Mitochondrial inhibition	Headache, Learning disabilities, deafness, cerebellar dysfunction, coma	Arrhythmias, atherosclerosis, ischemic heart diseases, hypertrophic and dilated cardiomyopathy and heart failure.	Poor appetite, gastroesophageal sphincter dysfunction, constipation alternating with diarrhea, dysphagia, vomiting.	Muscle weakness hence fatigue, muscular atrophy, impairment in movement	
Acetylcholine inhibition	Confusion, delusion, difficult forming and recalling memories, seizures, blurry vision	Dizziness and syncope, bradycardia, atrial arrhythmias, angina, myocardial infarction, Sino-atrial and atrioventricular block.	Diarrhea and vomiting	Muscle twitching, fasciculations, paralysis.	Wheezing, respiratory failure, sweating, blurry vision, miosis
Chloride channel inhibition (disinhibition of the neurons)	Cerebellar dysfunction, altered sensory perception	Arrhythmias, especially VT/VF, impair myocardial contractility and hypotension.	Alter GIT secretions.	Muscle stiffness, seizure, hyperexcitability, tremors or muscle twitching/ataxia	
Sodium channel inhibition (anticholinergic effects)	Altered mental state, diaphoresis, paresthesia, seizure, lethargic, confused.	Dysrhythmia, dizziness, hypotension, bradycardia, widening QRS complex, prolonged QT interval, VT/VF or torsade des pointes.	Slowed gut motility, absent bowel sounds, nausea, vomiting, hypersalivation, abdominal pain.	Muscle rigidity, skin flushing, tremor	Mydriatic pupils, contact dermatitis

CO- Cardiac output, BP- Blood pressure, GIT- gastrointestinal tract, VT- Ventricular tachycardia, VF- Ventricular Fibrillation

References include- [16,18-26]

ADULT PRESENTATION

Depending on the severity, duration and method of poisoning, adult patients will present in a variety of ways. As stated above, depending on the poison exposure, multiple presentations exist, but more prominent are nausea and vomiting, respiratory depression, cardiac arrhythmia, blood pressure swings as well as excessive salivation and sweating [27,28]. Due to late presentations, patients may come in coma or impending respiratory arrest requiring urgent resuscitation. It is common for patients to have been in fights or accidents around the time of toxin ingestion leading to distracting injuries and complex presentation. As stated, prior co-ingestion is probable, more so alcohol, taken on an empty stomach. Due to complexities in psychiatric presentation, the history of similar presentations should be elicited with the use of relevant suicide screening tools and mental state exams.

Paralysis can commonly occur after ingestion of large doses, it can either be acute, intermediate or it can occur 2-3 weeks post exposure. Some patients may show signs of neuropsychiatric effects e.g. impaired memory, confusion, psychosis and irritability.

OBSTETRIC PRESENTATION

Obstetric treatment of most pesticide poisons follows the same principles afforded to adult patients with emphasis on various aspects of care with some caveats.

During exposure, poisons generally are absorbed faster in pregnant mothers due to a vastly more perfused skin and mucous membranes as well as delayed gastric emptying [29]. Once absorbed, the volume of distribution is usually higher leading to a slower but more prolonged symptomatology. Due to physiologic increases in lipid storage, as most acaricides tend to hide within fat cells, a longer toxidrome is thus expected [30,31]. It is unclear how well these compounds distribute into the amniotic fluid as well as to the fetus, and the effects thereof, but there have been reports of higher incidences of preterm delivery, malformations such as cleft lip/palate as well as fetal death [32]. In later life, they have been associated with developmental delay, respiratory diseases such as COPD/ asthma as well as neurologic items including psychomotor delay and attention deficit disorder [33]. Pregnant mothers are also more prone to psychiatric illness and earlier reviews are warranted [34].

Aggressive physical and chemical decontamination methods are at a premium due to the catastrophic effects of these compounds and their associated long-term effects. Due to delayed gastric emptying, supervised gastric leverage is key with use of activated charcoal even beyond the typical time-frames [30]. A lower threshold for hemodialysis and other heme-detoxification methods should also be used to ensure optimal outcomes. A longer duration of stay is expected at critical care units, slow and steady is the motto. Careful prescription is necessary to ensure fetal compatibility, with drugs such as pralidoxime being used for up to one week for those chemicals it is indicated for. Pregnant mothers should also have more frequent blood gas analysis due to their altered physiology [35].

PEDIATRIC PRESENTATION

Acaricide poisoning in children requires prompt and effective management due to its potential for serious toxicity and death. Pediatric patients justifiably present much earlier and more severe than adult patients. As such they usually skip the typical prodromal symptoms and rapidly develop respiratory failure or altered level of consciousness. The clinical manifestations of acaricide poisoning in children include central nervous system (CNS) depression, which is the most prominent feature, leading to symptoms such as unconsciousness and drowsiness [28,36,37]. Other common signs include miosis, bradycardia, hypotension, and respiratory insufficiency [28,36,37]. In some cases, children may also present with vomiting, disorientation, hypothermia, and generalized seizures [38].

Due to the relatively low volume of distribution, neurotoxicity is highly evident with early presentation of neuromuscular weakness as well as seizures in comparison to adults, necessitating a more aggressive approach. Also, due to the same, they are more prone to renal failure as contributed by excessive volume losses and inherent nephrotoxicity of the poison or its metabolites. Fortunately, due to the relatively low body fat percentage, little of the toxins are sequestered leading to severe but shorter hospital stay when considering toxin/kg body weight ingested. Amitraz is one of the most commonly used acaricide and insecticide, and poisoning can occur through accidental ingestion, especially in children. [28,36,38]

Laboratory findings can include hyperglycemia, glycosuria, and mild elevations in liver transaminases [36,39]. The onset of symptoms usually occurs within 30 to 150 minutes after exposure, and CNS depression usually resolves within 4 to 28 hours [36,37,39].

It is important to note that while the clinical features of amitraz poisoning are well-documented in children, the literature on other acaricides may vary, and specific presentations can depend on the chemical nature of the acaricide involved.

LABORATORY INVESTIGATIONS AND FINDINGS

Laboratory derangement will include features of acute stress response in the hemogram, liver and kidney function tests [40]. Changes in blood sugar with episodes of hyper and hypoglycemia, electrolyte changes especially in relation to the toxins, dehydration and diuresis (commonly hyponatremia) [39,41]. Variety of abnormalities in blood gases, typically respiratory acidosis following paralysis. It is also possible to find mild to moderately elevated D-dimers and C-reactive protein as well as free T3 [14].

Table 3- Laboratory Findings

Phase	Tests (Laboratory/Bedside)	Common Findings	Considerations
Acute Phase (0–24 hrs.)	1. ECG (arrhythmias, QT prolongation) POCUS (IVC collapsibility, lung B-lines, cardiac function) CBC, RFTs (U/E/Cr), LFTs, ABG, Blood glucose, Serum electrolytes, Coagulation profile, Serum lactate, Cardiac enzymes (if indicated), CK, Serum cholinesterase (organophosphates/carbamate suspicion)	1. ECG: Bradycardia, Tachyarrhythmias, QT prolongation, ST-T changes 2. POCUS: Hypovolemia, Pulmonary edema, Cardiac dysfunction 3. CBC: Leukocytosis (stress response), Hemoconcentration 4. RFTs: Prerenal azotemia, AKI 5. LFTs: Transaminitis 6. ABG: Metabolic acidosis, Respiratory acidosis 7. Electrolytes: Hyponatremia, Hyperkalemia, Hypokalemia 8. Glucose: Hyperglycemia, Hypoglycemia 9. Coagulation: Mild DIC in severe toxicity 10. Cholinesterase: Reduced in organophosphate/carbamate exposure	1. Dysrhythmias, respiratory failure and electrolyte imbalances often drive mortality 2. Monitor for aspiration pneumonitis 3. Early POCUS helps in assessing fluid responsiveness and cardiac function
In Intermediate Phase (24–72 hrs.)	1. ECG (continuous if QT prolongation) 2. POCUS (daily reassessment) 3. Repeat U/E/Cr, LFTs, ABG, Electrolytes, Serum lactate, Cardiac enzymes, CK, Inflammatory markers (CRP, D-dimers), Blood cultures if infection suspected	1. ECG : Persistent arrhythmias, QT prolongation, T-wave inversion 2. POCUS: Evolving pulmonary edema, cardiac dysfunction, volume overload 3. RFTs: Worsening AKI 4. LFTs: Progressing hepatocellular injury 5. Electrolytes: Ongoing hyponatremia, hypokalemia, or hyperkalemia 6. ABG: Respiratory acidosis in ventilatory failure, Metabolic acidosis with lactate elevation 7. CRP/D-dimers: Elevated (systemic inflammation, potential sepsis, SIRS) 8. CK: Elevated if rhabdomyolysis	- Watch for delayed toxicity from lipophilic agents in newer cocktails - Secondary organ damage (liver, kidneys) emerges - Volume overload common due to aggressive resuscitation; diuretics may be needed but cautiously - Early sepsis-like syndrome mimicking distributive shock may arise
Chronic Phase (>72 hrs.)	1. ECG (re-evaluate for persistent abnormalities) 2. POCUS (cardiac function, residual lung changes) 3. CBC, U/E/Cr, LFTs, Electrolytes, ABG, CK 4. Thyroid Function Tests	1. ECG: Persistent T-wave abnormalities, QT normalization 2. POCUS: Pleural effusions, residual pulmonary edema 3. RFTs: Recovery or progression to chronic kidney injury 4. LFTs: Improvement or transition to hepatic dysfunction 5. Electrolytes: Gradual normalization but may show hypokalemia with diuretics 6. ABG: Resolution of acid-base disturbance 7. TFTs: Low T3 syndrome in prolonged illness	1. Monitor for long-term organ dysfunction 2. Psychological effects (PTSD, depression) after severe poisoning 3. Follow up with fertility and hormonal testing in long term.

*ABG- arterial blood gasses, AKI- Acute Kidney Injury, CBC- Complete blood count, CK- Creatine Kinase, CRP- C reactive Protein, DIC- Disseminated Intravascular Coagulopathy, ECG- Electrocardiogram, IVC- Inferior Vena Cava, LFTs- Liver Function tests, POCUS- point of care Ultrasound, PTSD- Post traumatic stress disorder, QT- QT segment on ECG, RFT- Renal function tests, TFT's- Thyroid function tests. References include- [14,23,39,41-43]

EAST AFRICAN EXPERIENCE

After accidental ingestion, patients often try homemade remedies and homemade antidotes including drinking milk, tea, and baking soda. These typically have no neutralizing properties but may temporarily relieve the gastritis frequently associated with ingestion. Unfortunately, this also causes nausea and vomiting. As symptoms rapidly develop, inducing emesis in a patient with altered sensorium may lead to aspiration and chemical pneumonitis. This is worsened when associated with co-ingestion with other compounds, alcohol or drugs. For intentional exposure, patients are found several hours after ingestion with others disposing of the evidence prior to onset of symptoms.

Majority of patients present outside the one-hour window following ingestion of the chemicals. The symptoms may not be overtly present in the initial presentation, but if exposure is sufficient, deterioration is eminent. Patients worsen quickly and dramatically with neurological and gastrointestinal symptoms being evident. Cardiovascular symptoms usually indicate severe toxemia and need for rapid intervention.

Due to resource limitations and lack of specialized units, patients are usually admitted to general wards only to deteriorate in these wards with associated morbidity. Access to ICU and HDU services are limited or expensively out of reach for many, but necessary for even stable looking patients due to the possibility of protracted and irregular symptomatology.

Patients who have been poisoned with acaricides, especially those who have ingested a mixture of substances, often seek medical help many hours after exposure. This delay is common in patients with intentional

ingestion, where the information provided may be unclear due to evidence tapering or incomplete disclosure.

During examinations, patients may display changes in mental status, features of severe gastritis with evidence of hematemesis or signs of respiratory distress like low oxygen saturations and use of accessory muscles of respiration. This could be coupled with skin changes e.g. dryness or diaphoresis, temperature changes and alterations in blood sugar levels.

Supportive care addresses specific symptoms such as antiemetics for vomiting or anticonvulsants for seizures. In cases of mixed ingestions, the toxic effects may be synergistic or staggered, presenting challenges in treatment and monitoring. Additionally, interactions between antidotes and co-ingested substances can complicate their efficacy.

Patients with significant instability require admission to an ICU or high-dependency unit for close observation and specialized care. Multidisciplinary management is essential, involving toxicologists or people with experience in managing poisoning, critical care teams, and mental health professionals, particularly in cases of intentional poisoning. Long-term follow-up is necessary to monitor complications such as organ damage or persistent post-toxic syndromes which mostly result from low dose or slowed exposure over time [44]. Some of the syndromes include cognitive impairment and dementia in the elderly. These cases highlight the challenges posed by resource limitations and the critical need for specialized care in regions like East Africa.

Current Treatment Recommendations

Severe Symptoms/ Exposure

Death usually results from airway compromise following vomiting or seizure. Hypoxia may also be as a result of central and peripheral respiratory muscle compromise. An ABCDE approach is required to ensure the most optimal results. The following guide must be supplemented with toxin specific data as highlighted above as well as local clinical guidelines.

Table 4- Management

SYSTEM	INTERVENTIONS	COMMENT
AIRWAY	Early assessment and consideration of early intubation is usually warranted more so in the patients with reduced levels of consciousness, elderly, pediatrics and obstetric patients with any signs of severe poisoning. Obese patients will be challenging.	Early intubation usually allows for early/late gastric leverage and may curtail severe poisoning. This also prevents challenges during seizure management. Do not induce emesis even in a fully conscious patient.
BREATHING	Adequate ventilation based on blood gas analysis. Due to Expected paralysis, volume modes on ventilators are key to ensure ventilation. (Link with Critical care teams for support)	Depending on co-ingestion and severity of poisoning, parameters could vary significantly. Co2 retention may be more of a challenge than oxygenation. Aspiration and hypoventilation are the most common causes of early cardiac arrest. Be wary of alcohol and drug co-ingestion responsible for hypoventilation.
CIRCULATION	Due to excessive losses, and cardiotoxicity of these compounds, adequate fluid resuscitation guided by urine output and Ultrasound are key. Patients may also require low dose vasopressors; renal supportive agents will be beneficial.	Multiple wide bore cannula or central lines are usually warranted. Cardiac arrhythmias are probable and should be approached in consideration of the mechanism of action of the toxin, electrolyte correction may curtail this.
DISABILITY	Seizures are common and should be managed aggressively to reduce incidences of permanent neurologic injury, rhabdomyolysis and renal failure. The use of electrolyte supplementation should be the first line of management. Rapid and wild changes in glycemic control can be seen, more so when associated with seizures. Frequent monitoring and replacement will be required. Delayed feeding should be employed for most moderate and severe cases.	Calcium and magnesium can be very useful in seizure management, as well as monitoring sodium levels. The use of benzodiazepines typically provides better seizure control than phenytoin. Avoid suxamethonium. Ensure adequate investigations to rule out other confounding injuries or illness. Stress ulcer protection required.
EXPOSURE	Excessive losses through urine, stool and sweat should be compensated for. These also help keep the patient cool when hyperthermic. Ensure mild diuresis to ensure adequate renal elimination. Ensure adequate and prompt decontamination including bodily fluids, as the toxins in them may be reabsorbed rapidly.	Physical cooling mechanisms are preferred due to potential interactions and side effects of chemical antipyretics. Chest physiotherapy, frequent nebulization with appropriate agents and positioning may reduce duration of intubation. Ensure frequent reviews for special subgroups such as obstetrics and pediatrics

For the above receptors, the following summary recommendations can be valuable:

Table 5- Receptor recommendation

RECEPTOR	RECOMMENDATION
Alpha receptors blockade	Fluid resuscitation then vasopressors if not responsive to fluids.
Acetylcholinesterase blockade	Supportive care, atropine, pralidoxime.
Mitochondrial Inhibition	Manage arrhythmia at presentation if present, vitamin supplements, avoid complication by treating the organs affected at presentation.
Acetylcholine inhibition	Avoid loop diuretic, theophylline and succinylcholine. Supportive measures plus atropine or glycopyrrolate and pralidoxime or obidoxime.
Chloride channel inhibition	Supportive plus replacement of the electrolytes.
Sodium channel inhibition	Supportive care, benzodiazepines preferred for seizure control, vitamin E in treatment of paresthesia.

Although most chemicals advocate for symptomatic management, some also recommend the supplementation of Magnesium and Calcium as means to stabilize the excessive receptor activation leading to less severe neurologic symptoms and indirectly less ICU stay [45]. Multiple and frequent lab assessments including poison assays are required with up to 3 hourly reviews to track patient trends or deterioration.

Overall, while poisoning can be serious, with appropriate and timely supportive care, the prognosis is generally good, and most adults and children recover without long-term sequelae [23,28,36].

MILD / MODERATE SYMPTOMS AND EXPOSURE

For such cases, admission and thorough assessment may not reveal any life-threatening derangements. Key is to ensure no further exposure, and high-level decontamination should be employed less the symptoms become pronounced. Admission for monitoring is key not only for psychological assessment but to ensure no second phase toxidrome. Late onset seizures are possible as well as mild derangements in lab work such as the hemogram, liver, kidney function test among others. Ensure proper counseling on pesticide handling as well as potential long term side effects. For obstetric patients, close follow up is warranted and a delivery plan in tertiary institutions should be established. For pediatric patients, transient muscle weakness, seizures or gastrointestinal challenges may manifest. These are usually transient and resolve within a few weeks. Permanent disabilities vary widely and are out of the scope of this article.

SUMMARY

Management of acaricide poisoning is primarily supportive, as there is no reliable antidote available. Key components of treatment include:

Table 6- Summary

INTERVENTION	COMMENT
Decontamination:	If ingestion is recent (within 60 minutes), gastric lavage and administration of activated charcoal may be considered to limit absorption. If intubated, a wider window of up to 6 hours is available. Activated charcoal is relatively safe especially in an intubated patient.
Atropine Administration	Atropine is used to relieve symptoms, especially if the poisoning is due to organophosphate or carbamate acaricides. Atropine is used to counteract the muscarinic effects of poisoning, such as bradycardia and hypotension. Multiple doses may be required depending on the severity of symptoms. Titrate to the respiratory symptoms and watch out for over atropinization.
Supportive Care	This includes symptomatic treatment: Address specific symptoms such as respiratory distress with oxygen therapy, seizures with anticonvulsants, dehydration/hypotension with intravenous fluids monitoring and supporting respiratory and cardiovascular function in some cases, vasopressors like dopamine may be necessary to maintain hemodynamic stability.
Close Monitoring	Patients should be closely monitored in a hospital setting, ideally in a critical care unit, to manage potential complications and ensure prompt intervention if the patient's condition deteriorates

References include - [7,16,23,27,28,36,42,43,46-49]

Potential Pitfalls and Recommendations

Most patients have had poor outcomes due to various issues including but not limited to:

- Underestimation of the toxemia due to relative stable presentation early on. Ensure that each suspected poisoning patient is admitted for 24-48 hr. observation. HDU and ICU utilization for moderate and severe cases, reduces the need for emergency interventions and looking for bed space in critical cases as toxemia evolves.
- Excessive use of atropine despite the lack of atropine sensitive poisons. Although its use should be encouraged for symptom management, overuse will confound the underlying toxidrome. Ensure identification of the active ingredient causing poisoning to minimize use of atropinization and associated complications. Over atropinization may typically present with other cardiovascular complications which may be prevented by frequent reassessment of patients for evolution of symptoms.

Use of triage and early warning scores may be sufficient to adequately classify patients.

- Lack of cutaneous decontamination. This should be resolved with early skin and mucous membrane decontamination using simple running water with adequate soap. For those with critical parameters, avoid cold water, if possible, to prevent hypothermia while a simple sponge bath can be used in the initial stages.
- Attempting gastric leverage and activated charcoal outside the one-hour window without adequate airway protection. With early intubation and HDU/ICU utilization, activated charcoal may be attempted as late as 6 hours post ingestion [48]. In suspected large volume exposure, rapid decontamination including supervised gastric leverage and activated charcoal with advanced airway techniques is lifesaving.
- Inadequate quantities used for activated charcoal. Judicious and extensive use of activated charcoal is warranted with up to 40 times the ingested volume or up to 1 gm/kg oral dosing [48].
- Use of Suxamethonium in the emergency intubation of patients as this leads to prolonged paralysis of patients due to similar mechanisms of action. Use of alternative paralytic agents, or preparation for prolonged paralysis including access to a ventilator is required.
- Lack of accounting for comorbidities, more so the expected derangements in glycemic control which may thus present with DKA and other complications. Switch diabetic patients to injectable insulin analogues early, with frequent blood sugar measurements. Transient hyperglycemia is expected and should be managed conservatively.
- Early initiation of feeding. The bowel usually requires rest as well as most of these compounds may cause transient paralytic ileus. Therefore, it is usually advisable to maintain Nil Per Oral at least for 24hrs to minimize the risk of aspiration or other gastrointestinal complications [50].
- Lack of gastric ulcer protection and use of PPIs as mainstay. This should start early on as intravenous medications.
- Early extubation with the need for re-intubation due to variability of symptoms and inevitable muscle weakness. Ensure slow ventilator weaning processes with minimum of 6 hrs. observation on spontaneous ventilation with good laboratory parameters prior to extubation.
- Distracting injuries and co-ingestion. Many patients may receive injuries from convulsions, falls and even other methods of attempted suicide. Extensive physical examinations, head CT scans and toxicology assessment may be necessary to reduce incidences of missed diagnosis.
- Early mental state examinations, when combined with psychiatrist review may be key in improving health outcomes as patients may re-initiate suicide attempts while within the hospital. Use of mechanical and chemical restraints may be prudent with agents such as haloperidol being considered.
- Seizure control is key to reducing ongoing damage. The benzodiazepines have been shown to be the most effective at controlling these seizures, Phenobarbital, Magnesium sulphate and propofol have also been used especially with refractory seizures. Phenytoin has been shown to be less effective and should be avoided [51,52].
- Multiple organ support. Early and aggressive renal/liver salvage therapies should be employed to ensure these chemicals are rapidly metabolized and excreted. Glutathione, vitamin B complex, penicillamine as well as N-acetyl cysteine have been theoretically shown to improve liver functioning post poisoning while aggressive renal replacement therapy may ensure continued elimination of these poisons [53-57]. While we don't advocate for the judicious use of these compounds, in cases of severe life-threatening poisoning or those with underlying derangements, these options as well as renal dialysis, liver dialysis and exchange transfusions may be attempted in the correct setups.
- Carcinogenesis. Most of these compounds have been shown to cause multiple cancers, chief being leukemias and colorectal cancers. Active counseling and follow up is key to ensure symptoms are caught early [58].
- ECG monitoring. Due to multiple ion channel disruption by various poisons, their toxicity may be evident on ECG due to electrolyte changes. These changes may aid in monitoring, prevention of arrhythmias as well as guide electrolyte supplementation in real time in conjunction with lab analysis. ECG changes typically warrant ICU/HDU care as this may indicate early signs of severe toxicity.
- Lack of laboratory diagnosis. While lab assays of various types of these poisons are not available in low resource environments, serial quantitative measurements of these compounds can be useful in guiding management especially in the use of more aggressive modalities such as dialysis and other detoxification methods.

GENERAL RECOMMENDATIONS ALSO INCLUDE

- Early patient arrival- Social education and health information sharing through various forums will greatly reduce this time, more so in rural and semi-urban environments.
- Local policies and guidelines. Development of local and national guidelines and policies will ensure a more comprehensive approach and help create tools, foster research as well as lobby for improving health outcomes in this condition.
- Strengthening local poison centers and emergency response teams. Most countries have regulatory bodies for agricultural products as well as medicines and poisons. Strengthening these institutions to even offer emergency response calls and online consultation to ensure healthcare providers have access to a reliable and updated information source will ensure better outcomes and continuity of care.

AREAS OF FURTHER RESEARCH AND DISCUSSION

- DVT prophylaxis. The body's response to various acaricides in regard to coagulation is complex. Various agents have been known to cause kidney

and liver damage which may indirectly lead to hypo-coagulative states. It has also been stipulated that the chemical agents themselves may cause oxidative damage and general tissue damage leading to tissue inflammation and pro-coagulative changes [59]. It is therefore prudent to perform coagulation profiles frequently and intervene according to local hematological guidelines or with review from a hematologist.

- Pralidoxime. Oximes have been shown to offer mixed results even among the organophosphate group. The most recent meta-analysis shows minimal benefit with even some cases of harm among some populations, even in compounds to which it was developed as an antidote for. We cannot therefore recommend its empirical use outside the organophosphate group, especially in pediatrics until such time more compelling evidence is presented [46,60,61].
- N-acetyl cysteine- Although this compound has shown some theoretical promise among some pesticides it remains unclear the extent to which this can be translated to acaricides. We therefore don't recommend its routine use until stronger evidence is available [62-64].
- Endocrinological effects- Many pesticides including acaricides have been shown to cause infertility, menstrual irregularities as well as changes to thyroid hormone levels. This is due to interruption in hormonal metabolism as endocrine disruptive chemicals. While this effect may not be overtly evident in the acute phase, it is postulated that these changes may become evident as early as 2 weeks [65]. As severe cases may still be admitted in the hospital at this stage, the development of these new symptoms should not be confused with evolving toxicology. This may compound the situation with negative cardiovascular changes. More research is needed to show direct correlation, nonetheless, endocrinology follow up is usually warranted.
- Protective paralysis- There have been various trials showing some benefit by offering protective paralysis using rocuronium, sequestering the neurosynaptic junction from the harmful effects of continuous exposure to these pesticides. Further research will enrich management.

Conclusion

Acaricide poisoning remains a huge challenge in management as we still do not have very effective antidotes. Symptomatic management remains key, with rapid early decontamination as the most important intervention. Identification of the poison and multidisciplinary reviews will allow for a targeted approach and may improve patient outcomes.

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Conflict of Interest

The authors declare no conflict of interest

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