



Emergency Response to Cocaine Overdose: Roles of Clinicians, Emergency, and Health Information Professionals

¹Jafar Ahmed Mohammad Alnakhli, ²- Zaher Ibrahim Shahir Alziri, ³-Fadyah Faisal Alshareef, ⁴-Zainab Faisal Alshareef, ⁵-Ayoub Abed A Alnakhli, ⁶-Afnan Saleh Hassan Alharbi, ⁷-Rabab Ali N Alnakhli, ⁸- Zakri Jubran Ahmed Shanyah, ⁹- Abdullah Mussa Ahmad Sairafi, ¹⁰-Reem Hilal Break Al-Otaibi, ¹¹- Hamzah Shuwaier Ali Salwa, ¹²- Raed Slman Ahmad Sahari, ¹³-Hussein Hedar Abutalib, ¹⁴ Faisal Mohmmmed Eyada Alenazi,

1. Ksa, Ministry Of Health, Ksamc
2. Ksa, Ministry Of Health, Ksamc
3. Ksa, Ministry Of Health, Ksamc
4. Ksa, Ministry Of Health, Ksamc
5. Ksa, Ministry Of Health, Ksamc
6. Ksa, Ministry Of Health, Ksamc
7. Ksa, Ministry Of Health, Ksamc
8. Ksa, Ministry Of Health, Crisis And Disaster Management Center Of Jazan
9. Ksa, Ministry Of Health, Ahad Al-Masariha General Hospital
10. Ksa, Ministry Of Health, City Center Heath Center
11. Ksa, Ministry Of Health, Crisis And Disaster Management Center Of Jazan
12. Ksa, Ministry Of Health, Crisis And Disaster Management Center Of Jazan
13. Ksa, Ministry Of Health, Crisis And Disaster Management Center Of Jazan
14. Ministry of Health - King Salman Kidney Center

Abstract:

Background: Cocaine misuse is a significant global public health issue, leading to high rates of emergency department (ED) visits due to acute toxicity. Cocaine's effects on the cardiovascular and neurological systems can result in life-threatening complications, including myocardial infarction, stroke, hyperthermia, and excited delirium. The rise in polysubstance use further complicates clinical management.

Aim: This article examines the pathophysiology, clinical presentation, and emergency management of cocaine toxicity, emphasizing the roles of clinicians, Emergency, and health information professionals in improving patient outcomes.

Methods: A comprehensive review of cocaine's toxicokinetics, clinical manifestations, and evidence-based treatment strategies was conducted. The roles of emergency responders and healthcare informatics in diagnosis, treatment, and long-term care coordination were also explored.

Results: Cocaine toxicity requires prompt recognition and intervention. Benzodiazepines remain first-line for agitation and cardiovascular symptoms, while labetalol and calcium channel blockers are preferred for hypertension. Hyperthermia demands aggressive cooling. Health information professionals enhance care through electronic health records (EHRs), clinical decision support, and data tracking for public health surveillance.

Conclusion: Effective management of cocaine overdose relies on multidisciplinary collaboration, rapid pharmacologic intervention, and robust health informatics systems. Long-term strategies must integrate addiction treatment and mental health support to reduce relapse and mortality.

Keywords: Cocaine toxicity, emergency management, benzodiazepines, hyperthermia, health informatics, addiction treatment.

Received: 15 Aug 2024

Accepted: 04 oct 2024

Published: 15 oct 2024

Introduction:

Cocaine misuse remains a significant public health issue across the globe. Individuals experiencing acute cocaine toxicity frequently present to emergency departments (EDs), often requiring immediate medical intervention to address symptoms such as tachycardia, cardiac dysrhythmias, elevated blood pressure, and coronary vasospasm. These conditions can escalate into severe complications, including acute coronary syndrome, cerebrovascular accidents, and even sudden death, highlighting the urgent nature of emergency care in such cases [1][2][3]. In recent years, the clinical challenges associated with cocaine toxicity have expanded, particularly due to the emergence of "body packers" individuals who internally conceal drug packages for smuggling purposes. In instances where these packages rupture, patients present with life-threatening toxicity that demands rapid diagnosis and management in the emergency setting. Additionally, many of these patients have consumed other illicit substances such as alcohol, further complicating the clinical picture and impeding the standardization of treatment protocols. Polysubstance ingestion alters both the presentation and progression of toxicity, often requiring customized therapeutic approaches that address multiple physiological effects simultaneously. Cocaine's harmful impact extends throughout the body, but its most destructive and fatal outcomes predominantly affect the cardiovascular system. It can provoke a spectrum of serious cardiovascular conditions by enhancing sympathetic nervous system activity, increasing myocardial oxygen demand, and inducing vasoconstriction. These physiological changes may result in myocardial ischemia, arrhythmias, and infarction, even in young patients without underlying cardiac disease. The risk of sudden cardiac death is markedly elevated, underscoring the necessity of early recognition and aggressive intervention in ED settings. As cocaine continues to be widely abused, emergency professionals are confronted with increasing numbers of complex, high-risk cases that demand multidisciplinary coordination and advanced clinical judgment [1][3].

Etiology

The coca plant, *Erythroxylon coca*, has served as a natural stimulant for over four millennia in South American cultures. Traditionally, its leaves were chewed to combat fatigue and hunger, especially in high-altitude regions. In the mid-nineteenth century, scientists successfully isolated the active compound, cocaine, from these leaves. Following its isolation, cocaine gained widespread use in various over-the-counter remedies. It was marketed as an ingredient in products meant to relieve toothaches, reduce nausea, boost energy, and was famously included in the original formulation of the Coca-Cola beverage. Despite its widespread use during the late 1800s and early 1900s, awareness of its addictive potential and harmful effects gradually grew. By 1961, the International Single Convention on Narcotic Drugs formally classified recreational cocaine use as a criminal act on a global scale. However, the drug experienced a resurgence in popularity during the 1970s, particularly linked to the social and cultural dynamics of the disco era. This resurgence intensified in the 1980s with the emergence and mass distribution of crack cocaine, a more potent and inexpensive form of the drug. Currently, cocaine is categorized as a Schedule II controlled substance by the U.S. Drug Enforcement Administration (DEA). This classification recognizes its limited medical use—particularly as a topical anesthetic in specific surgical procedures—while acknowledging its high potential for abuse and dependence [4]. The legal restrictions reflect a balance between its therapeutic utility and the significant public health risks associated with non-medical consumption [4].

Epidemiology

Cocaine misuse continues to represent a substantial global public health concern. According to the latest data from the United Nations World Drug Report, the estimated number of cocaine users worldwide has reached approximately 20 million. This figure highlights the widespread nature of cocaine consumption and the persistent challenges it poses to healthcare systems and public health authorities. Although North America experienced a gradual decline in frequent cocaine use after 2006, recent trends indicate a

resurgence. Currently, about 1% of the population in that region reports using cocaine, signaling a reversal in the previous downward pattern and raising concern about future increases in usage rates [5]. In the United States, detailed surveillance from the Drug Enforcement Agency provides insight into the scope of the problem. In 2012, an estimated 639,000 individuals aged 12 or older reported using cocaine for the first time within the preceding year. This statistic reflects an average of nearly 1,800 new users per day, suggesting both a high level of exposure and the continuous influx of new individuals into cocaine use. These patterns underline the enduring appeal of the drug, despite well-documented health risks and decades of public health messaging and legal restrictions aimed at discouraging its use. Cocaine's impact on emergency medical services in the United States is particularly severe. Data from the Substance Abuse and Mental Health Services Administration for the year 2011 indicate that cocaine was the most frequent cause of drug-related emergency department (ED) visits. That year, cocaine accounted for 505,224 ED visits, representing 40.3% of all visits attributed to drug misuse. This figure translates to a rate of 162 emergency visits per 100,000 people in the general population. The high number of cocaine-related ED admissions reflects the acute physiological dangers the drug poses, including cardiovascular emergencies, neurological complications, and psychiatric crises. It also imposes a significant burden on emergency healthcare services, requiring specialized medical interventions and contributing to overcrowding and resource strain [5].

These epidemiological trends show that cocaine use is not only persistent but also evolving. Its widespread use across multiple demographics, including adolescents and young adults, points to the need for improved prevention and intervention strategies. The recurring pattern of new user initiation, combined with a resurgence in overall usage, suggests that current public health strategies may be insufficient or misaligned with emerging social behaviors and drug trafficking dynamics. Healthcare systems must remain adaptable, using real-time surveillance, community outreach, and evidence-based education to respond effectively. Emergency departments, in particular, play a central role in identifying high-risk individuals, delivering acute care, and connecting patients to substance use treatment services. The high number of ED visits tied to cocaine use highlights the need for integrated approaches that combine immediate medical management with long-term addiction support services. Without addressing both the acute and chronic aspects of cocaine abuse, the healthcare burden is likely to grow, and the cycle of harm may continue unchecked [5][6].

Pathophysiology

Cocaine use can result in multiple acute and chronic health complications. Its pharmacologic effects on various organ systems create a range of life-threatening conditions such as tachydysrhythmias, hypertensive crises, acute coronary syndromes, ischemic stroke, seizures, acute myocardial and renal failure, rhabdomyolysis, hyperthermia, and complications in pregnancy including fetal and maternal morbidity and mortality [7]. The diversity and severity of these consequences make cocaine a high-risk substance with complex pathophysiology. Cocaine's most significant physiological impact is on the cardiovascular system. It acts primarily by blocking the reuptake of catecholamines—especially norepinephrine, dopamine, and serotonin—at sympathetic nerve terminals. This inhibition leads to elevated levels of catecholamines in synaptic clefts, intensifying sympathetic stimulation. The result is a surge in heart rate, systemic vascular resistance, myocardial oxygen demand, and overall cardiac workload. These responses significantly increase the risk of arrhythmias, particularly tachydysrhythmias, and can trigger hypertensive emergencies. Furthermore, cocaine also possesses sodium channel-blocking properties similar to local anesthetics, which slow cardiac impulse conduction and can promote re-entry arrhythmias, increasing the likelihood of sudden cardiac arrest [7].

Long-term cocaine exposure introduces structural changes within the heart. Repeated sympathetic stimulation and vasoconstriction contribute to myocardial fibrosis, contraction band necrosis, and in some cases, myocarditis. These histological changes compromise cardiac contractility and increase susceptibility to heart failure and arrhythmic death. Even individuals with minimal or subclinical coronary artery disease are at risk of developing acute coronary syndrome due to cocaine's ability to cause intense coronary vasoconstriction. This vasospasm, combined with increased myocardial oxygen demand, creates an imbalance that precipitates ischemia and infarction. Cocaine can also increase platelet aggregation, further

raising the risk of thrombus formation in coronary arteries. The neurological consequences of cocaine are also severe. One of the major risks is ischemic stroke, which may result from vasospasm, accelerated atherosclerosis, or cardioembolic phenomena due to arrhythmias. Cocaine lowers the seizure threshold, increasing the likelihood of convulsions, even in individuals without prior neurologic conditions. In chronic users, alterations in the dopaminergic system occur, including reduced dopamine receptor density and function. These changes may result in movement disorders that resemble extrapyramidal symptoms, such as bradykinesia, dystonia, akathisia, and akinesia. These motor impairments reflect underlying neurochemical imbalances caused by persistent cocaine exposure [7].

Another dangerous neuropsychiatric outcome is cocaine-induced delirium. A subset of patients may develop a severe and fatal condition known as excited delirium. This condition is characterized by extreme agitation, incoherent screaming, aggressive behavior, physical strength beyond normal levels, paranoia, hyperactivity, and elevated body temperature. These patients are at high risk for sudden death, often related to cardiac arrest. Individuals experiencing excited delirium display heightened sensitivity to elevated catecholamine levels, which exacerbates cardiovascular stress and accelerates deterioration. Hyperthermia is another prominent and dangerous feature of cocaine toxicity. Body temperatures in severe cases can reach 45°C, a level that often signals a poor prognosis. Hyperthermia reflects a failure of thermoregulation and is typically accompanied by rhabdomyolysis, liver dysfunction, acute kidney injury, encephalopathy, and disseminated intravascular coagulation (DIC). Cocaine-induced hyperthermia often contributes to metabolic acidosis due to increased lactate levels and muscle breakdown, creating a vicious cycle of worsening organ function. Musculoskeletal complications include cocaine-induced rhabdomyolysis, in which skeletal muscle tissue breaks down rapidly, releasing myoglobin into the bloodstream. This condition places significant strain on renal function and can lead to acute kidney injury. It is frequently observed in patients with prolonged agitation, hyperthermia, or those suffering seizures. The kidneys, already vulnerable due to cocaine's vasoconstrictive effects on renal vasculature, may fail under the burden of excessive myoglobin and reduced perfusion [7].

Cocaine's impact on pregnancy further underscores its systemic dangers. Pregnant individuals who use cocaine are at higher risk of complications including placental abruption, fetal growth restriction, premature labor, and stillbirth. The vasoconstrictive action of cocaine reduces uteroplacental blood flow, which jeopardizes fetal oxygenation and development. Additionally, the drug crosses the placental barrier, directly exposing the fetus to toxic effects that may result in long-term neurodevelopmental disorders or neonatal abstinence syndrome. The multisystemic nature of cocaine toxicity requires emergency professionals to maintain a broad differential diagnosis and to act quickly in identifying and managing life-threatening manifestations. The diversity of cocaine's pathophysiological effects—ranging from direct cardiotoxicity and neurotoxicity to secondary complications such as renal failure and coagulopathies—necessitates coordinated, multidisciplinary care. Managing acute cocaine toxicity often involves airway protection, aggressive cooling measures, seizure control, cardiovascular stabilization, and renal function monitoring, alongside psychiatric support for long-term recovery. Without timely intervention, the progression of cocaine-induced complications often results in permanent disability or death (**Figure 1**) [7].

Toxicokinetics

Cocaine can be administered through various routes, including intranasal insufflation (snorting), oral ingestion, intravenous injection, and smoking. Each route produces different onset times and intensities of effect, but the pharmacokinetic behavior of the drug remains largely consistent in terms of its core properties. Cocaine has a relatively short plasma half-life, averaging approximately one hour, though its toxic effects may extend far beyond this window due to the activity of its metabolites and downstream physiological interactions. The central pharmacodynamic mechanism of cocaine involves competitive inhibition of monoamine reuptake transporters. It binds to the dopamine transporter (DAT), norepinephrine transporter (NET), and serotonin transporter (SERT) with comparable affinity. This blockade prevents the reabsorption of these neurotransmitters back into presynaptic neurons, resulting in their accumulation in the synaptic cleft. The subsequent overactivation of postsynaptic receptors leads to heightened sympathetic nervous system activity. This neurochemical environment produces the euphoria,

increased alertness, and energy commonly reported by users, but it also underpins the serious cardiovascular risks associated with cocaine toxicity [8].

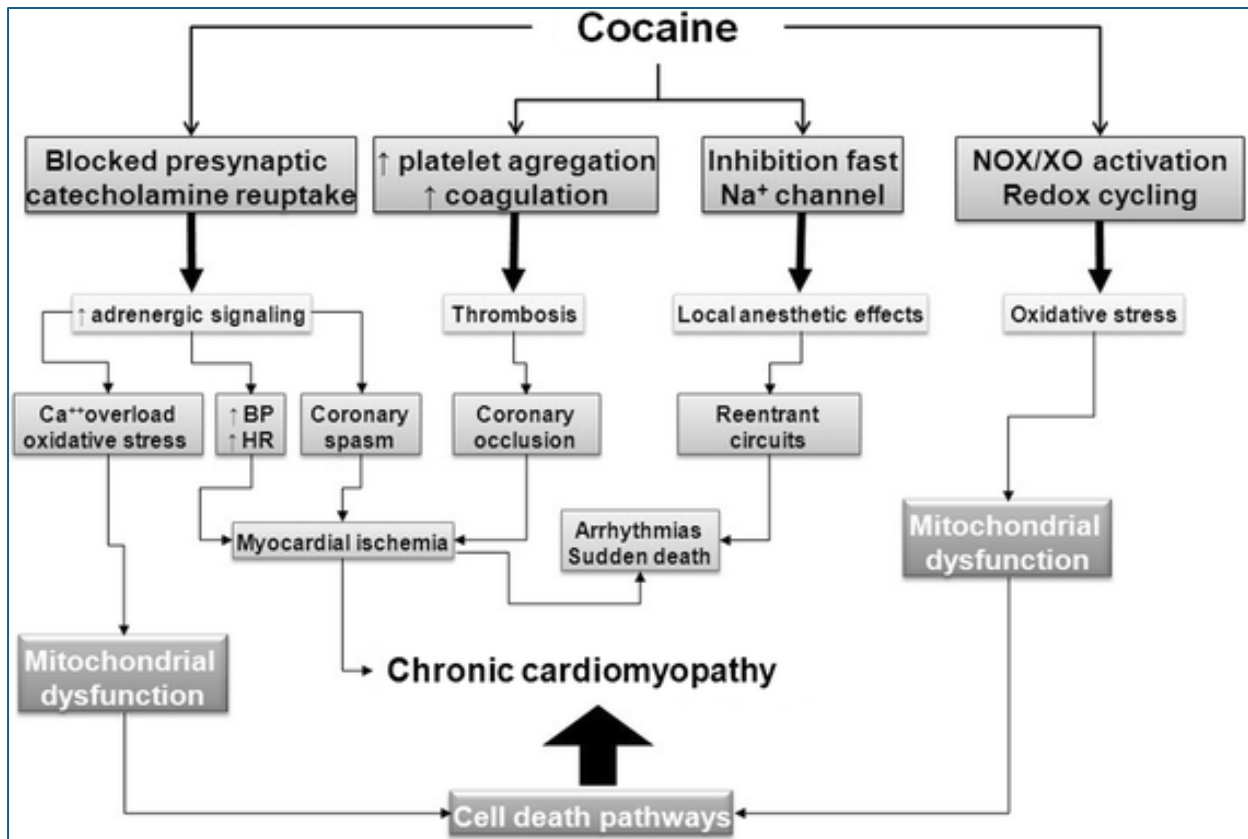


Figure 1: Pathophysiology of Cocaine Toxicity.

Cocaine exerts significant influence on the cardiovascular system through stimulation of alpha- and beta-1-adrenergic receptors. This stimulation elevates heart rate, increases systemic arterial blood pressure, and enhances myocardial contractility. These three factors—heart rate, arterial pressure, and contractility—are the principal determinants of myocardial oxygen consumption. The simultaneous increase in oxygen demand and reduction in oxygen delivery, due to cocaine-induced vasoconstriction, generates a critical imbalance that raises the risk of myocardial ischemia and infarction. Importantly, these cardiovascular effects are not limited to the period of acute intoxication. Vasoconstrictive properties of both cocaine and its metabolites can persist for several hours after the drug is used, especially affecting the epicardial coronary arteries, which are highly susceptible to reduced perfusion. Cocaine also contributes to thrombogenic processes. It promotes platelet aggregation through alpha-adrenergic receptor activation and stimulation of adenosine diphosphate (ADP)-mediated pathways. The increased aggregation predisposes users to thrombus formation, which can occlude coronary vessels and lead to acute coronary syndromes. Additionally, cocaine elevates levels of plasminogen activator inhibitor-1 (PAI-1), a protein that suppresses fibrinolysis. This inhibition of clot breakdown further supports a pro-thrombotic state, amplifying the cardiovascular dangers during and after cocaine exposure [8].

From a cellular electrophysiology perspective, cocaine shares properties with local anesthetics like lidocaine. It inhibits sodium channels in excitable tissues, particularly within cardiac myocytes. This action slows the rate of depolarization, delays conduction through the myocardium, and increases the likelihood of re-entrant arrhythmias. These effects are categorized as Vaughn-Williams class IC antiarrhythmic activity. In the context of cocaine toxicity, this sodium channel blockade becomes maladaptive, contributing to conduction disturbances and life-threatening tachyarrhythmias. In addition to its primary sympathomimetic and anesthetic actions, cocaine interacts with multiple other receptor systems. It binds to muscarinic acetylcholine receptors, N-methyl-D-aspartate (NMDA) receptors, and opioid receptors,

including sigma and kappa subtypes. These interactions add further complexity to its toxic profile. NMDA receptor binding may play a role in neuroexcitatory effects and seizure risk, while the impact on opioid and cholinergic systems may contribute to alterations in mood, perception, and autonomic function. Such receptor activity likely explains the variability in clinical presentation and complicates both diagnosis and management during acute intoxication [7][8]. Cocaine's toxicokinetics thus involves not only rapid onset and intense sympathomimetic effects but also prolonged cardiovascular, neurologic, and hematologic consequences. The continued action of its metabolites and engagement with multiple receptor pathways requires emergency clinicians to remain vigilant long after the initial period of intoxication. Comprehensive management must address both the immediate sympathomimetic surge and the delayed pro-arrhythmic and pro-thrombotic effects, all of which stem from its complex pharmacologic behavior [8].

History and Physical

Patients experiencing acute cocaine toxicity often arrive at the emergency department in a state of marked physiologic arousal. Many displays agitation, intense chest discomfort, or overwhelming anxiety. Others manifest overt psychosis. Some arrive after sustaining blunt or penetrating trauma, accidents that may stem from impaired judgment or violent encounters during intoxication. Clinicians typically note elevated blood pressure and rapid heart rate on initial assessment. Despite obvious clinical clues, some patients hesitate to admit recent cocaine use. This reluctance complicates early decision-making and demands that the care team keep a broad differential in mind. Could the agitation reflect alcohol or benzodiazepine withdrawal? Might the tachycardia signal thyrotoxicosis? Could psychosis represent a primary psychiatric disorder? Other possibilities include sepsis, pheochromocytoma, anticholinergic poisoning, serotonin or neuroleptic malignant syndromes, and intracranial hemorrhage. An inclusive diagnostic approach is therefore important during the first minutes of contact. The history should probe prior drug exposure, patterns of use, and earlier hospitalizations. Clinicians should document mode of administration—intranasal, oral, intravascular, or inhalational, because each route influences onset, peak effect, and complication pattern. Recent co-ingestion of alcohol or other stimulants deserves special attention, as these agents amplify cardiovascular strain and seizure risk. Past episodes of chest pain, arrhythmia, or cerebrovascular events suggest heightened vulnerability to recurrence. Social history should explore sleep deprivation, nutritional status, and psychosocial stressors that may precipitate binge episodes. A focused review of systems helps reveal subtle complications: visual disturbance may herald hypertensive retinopathy; abdominal pain can warn of mesenteric ischemia; diffuse pruritus sometimes accompanies severe diaphoresis and hyperthermia [9].

Physical examination findings often correlate with sympathetic activation. Many patients exhibit severe hypertension with systolic readings exceeding 180 mm Hg. Tachycardia is common, though a rapid irregular rhythm may point toward atrial fibrillation triggered by catecholamine excess. Altered mental status ranges from mild confusion to frank delirium. Some patients seize during triage, while others complain only of throbbing headache or vague paranoia. Respiratory evaluation may uncover dyspnea caused by pulmonary edema or pneumothorax from barotrauma during intense Valsalva maneuvers. Epistaxis can result from repetitive intranasal insufflation and fragile mucosal vessels. A quick ocular inspection may reveal mydriasis, but more alarming signs include corneal ulcerations or sudden vision loss due to retinal artery vasospasm. Neurologic deficits require urgent imaging because cocaine-induced vasoconstriction can culminate in ischemic stroke even in young adults with no vascular risk factors. Hyperthermia may push core temperatures above 40 °C, signaling impending metabolic catastrophe. Extremity examination sometimes exposes pulseless limbs from peripheral arterial spasm. Skin is often drenched in sweat. Severe restlessness or violent thrashing suggests progressing excited delirium, a syndrome prone to sudden cardiovascular collapse. Some patients complain of formication and relentless itching. Others suffer gastrointestinal distress—vomiting, diarrhea, colicky abdominal pain—which can precede life-threatening bowel ischemia. The diversity of presentations calls for systematic, head-to-toe evaluation rather than a symptom-specific focus [9].

Stages of Acute Cocaine Toxicity

Clinicians have described three progressive stages of acute cocaine toxicity. Although real-world cases may blur these boundaries, the framework guides monitoring and anticipatory management. **Stage 1** marks the early sympathomimetic surge. Patients often report pounding headache, queasiness, or spinning sensations. Pupils dilate. Skeletal muscle twitches may appear, and some individuals exhibit brief pseudo-hallucinations. Fine tremors or jerky pre-convulsive movements demand prompt benzodiazepine therapy to forestall generalized seizures. Blood pressure climbs; auscultation may reveal occasional ectopic beats foreshadowing arrhythmia. Respiratory rate accelerates as catecholamine drive intensifies. Core temperature begins to rise. Psychiatrically, patients oscillate between exaggerated euphoria and sudden paranoia. Many remain argumentative, emotionally labile, and restless, challenging staff safety and making venous access difficult.

Stage 2 heralds neurologic and cardiovascular deterioration. Encephalopathy dominates, with disorientation giving way to frank seizure activity. Post-ictal periods may be brief or absent, and status epilepticus can emerge without warning. Deep tendon reflexes become brisk. Urinary or fecal incontinence suggests diffuse cortical involvement. Blood pressure may remain high, but pulse irregularities become more frequent and ominous. Ventricular tachycardia or supraventricular tachyarrhythmias threaten hemodynamic stability. Cyanosis of distal extremities or lips reflects impaired oxygen delivery from both vascular constriction and impaired cardiac output. Respiratory patterns grow erratic—gasping, periodic apnea, or shallow inhalations signal impending failure. Meanwhile, hyperthermia persists. Without aggressive cooling and sedation, multi-organ injury accelerates.

Stage 3 represents terminal decompensation. Brainstem reflexes vanish. Pupils fix and widen. Coma ensues, accompanied by absent gag and corneal responses. Cardiovascular collapse progresses from refractory hypotension to pulseless ventricular fibrillation or asystole. Respiratory arrest coincides, with cyanosis deepening and agonal gasps dwindling. Even with maximal supportive measures, survival at this point is rare, and neurologic recovery unlikely [9]. Understanding these stages underscores the importance of early recognition and intervention. Clinicians must act vigorously during Stage 1, using benzodiazepines to mitigate central nervous system excitation, nitrates or calcium channel blockers to relieve coronary vasospasm, and rapid cooling to prevent hyperthermia-mediated rhabdomyolysis. Continuous cardiac monitoring catches ectopy before it degenerates into malignant rhythm disturbances. Serial neurologic checks detect subtle progression toward seizure or encephalopathy. Laboratory tests—including creatine kinase, troponin, electrolytes, and arterial blood gases—guide therapy and reveal hidden complications such as lactic acidosis or renal impairment. Stage 2 demands escalation. Anticonvulsants beyond benzodiazepines, such as propofol or barbiturates, may be necessary. Intravenous fluids replace volume losses from diaphoresis and vomiting, but clinicians must balance resuscitation with the risk of precipitating pulmonary edema in compromised myocardium. If tachyarrhythmia proves refractory, cautious beta-blockade with agents possessing alpha-antagonist properties, like labetalol, can temper catecholamine excess without unopposed alpha stimulation. However, isolated beta-blockers remain contraindicated. Vasodilators such as nitroprusside or phentolamine may be required for severe hypertension. Mechanical cooling—ice packs, evaporative techniques, intravascular devices—combats relentless hyperthermia. When patients slip into Stage 3, the focus shifts to advanced life support. High-quality chest compressions, defibrillation for shockable rhythms, and epinephrine according to resuscitation guidelines are mandatory. Yet clinicians must recognize that ongoing sympathetic storm and metabolic derangements hinder success. Even if return of spontaneous circulation occurs, post-arrest care must address profound acidosis, refractory hyperthermia, and extensive organ damage, all of which portend poor neurologic outcome. These grim realities stress the need for preventive strategies and public health measures aimed at reducing cocaine use before catastrophe strikes. Why do some users deteriorate rapidly while others do not? Genetic differences in catecholamine metabolism, co-ingested substances, environmental stressors, and pre-existing cardiovascular pathology all contribute. Investigating these factors can help tailor risk stratification tools and improve prognosis. Are emergency departments equipped to deliver the full spectrum of care required? Staffing shortages, crowding, and limited access to cooling devices challenge many facilities. Integrating clear algorithms for cocaine toxicity management may streamline decision-making and improve survival [9].

Clinicians should remember that excited delirium may not progress through predictable stages. Some individuals bypass early warning signs and succumb suddenly to ventricular fibrillation. Early identification of extreme agitation, bizarre strength, and incoherent yelling should prompt aggressive sedation and temperature control. Collaboration with security personnel ensures safe restraint while minimizing positional asphyxia risk. Long-term sequelae deserve attention during discharge planning. Recurrent chest pain, latent cardiomyopathy, or neurocognitive decline may follow initial recovery. Referral to addiction services, cardiology, and neurology fosters continuity of care. What motivates sustained abstinence? Studies show that combining cognitive-behavioral therapy with contingency management improves outcomes. Linking patients to community resources before they leave the hospital can reduce readmissions and interrupt dangerous cycles of relapse. In summary, a meticulous history, comprehensive physical examination, and familiarity with the evolving stages of acute cocaine toxicity empower clinicians to deliver timely, life-saving therapy. Early intervention averts progression from sympathetic overdrive to multi-organ failure. Success hinges on interdisciplinary cooperation, rapid symptom control, and proactive planning for long-term rehabilitation [9].

Evaluation

Early laboratory assessment anchors safe management of acute cocaine toxicity. You aim to clarify organ function, reveal hidden complications, and rule out co-ingestants. Start with a complete blood count. Elevated white cells can reflect stress or infection. Hemoglobin shifts may indicate gastrointestinal bleeding or hemolysis from severe hypertension. A comprehensive chemistry panel highlights electrolyte derangements that can trigger arrhythmias or seizures. Pay close attention to potassium and magnesium levels before administering antiarrhythmic drugs. Creatinine and blood urea nitrogen provide a snapshot of renal perfusion because vasoconstriction and rhabdomyolysis threaten kidney function. Glucose guides seizure prophylaxis since hypoglycemia clouds neurologic interpretation [10]. Troponin testing carries high priority. Cocaine often causes silent ischemia. A normal initial value does not exclude injury. Repeat measurement at three-hour intervals if chest pain persists. Elevated B-type natriuretic peptide suggests ventricular strain and may influence fluid resuscitation when pulmonary edema complicates tachydysrhythmia. High creatine kinase implies skeletal muscle breakdown. You should suspect rhabdomyolysis when levels exceed five times the upper limit of normal and urine turns dark. Myoglobin tests in urinalysis confirm pigment nephropathy risk. Ask yourself whether aggressive intravenous hydration could prevent acute tubular necrosis in this patient. A urine drug screen broadens the diagnostic lens. Many users ingest alcohol, methamphetamine, or synthetic cannabinoids. Combined exposure alters hemodynamics and seizure threshold. Screening also detects benzoylecgonine, cocaine's main metabolite. Although cocaine itself vanishes within twenty-four hours, benzoylecgonine lingers for days to weeks and can still provoke neurotoxicity [9][10]. Positive results do not always confirm acuity, yet they raise suspicion for repeated binges. How will you integrate that timeline with reported symptoms? [10]

Electrocardiography remains indispensable. You look for QRS widening from sodium channel blockade, QTc prolongation that heralds torsades, or ST elevation signaling coronary vasospasm. Monitor continuously because rhythms fluctuate as catecholamine surges wax and wane. Consider serial tracings if new chest discomfort arises. Imaging tailors itself to presentation. Obtain a chest x-ray when dyspnea, hypoxia, or abnormal lung auscultation appears. You may identify noncardiogenic pulmonary edema, a pneumothorax from barotrauma, or aspiration infiltrates in an obtunded patient. A widened mediastinum warrants further aortic evaluation. Abdominal x-ray serves a special role in suspected body packers or stuffers. You search for radiopaque capsules in the gastrointestinal tract. Plain films fail when packets contain low-density material or overlay bowel gas shadows. Do you switch to contrast-enhanced computed tomography when doubt persists? CT offers near-complete sensitivity, detects early leaks, and guides surgical consultation. Remember that rupture risk grows as gastric acids erode wrapping, so diagnostic delay invites catastrophe [10].

Neurologic red flags demand brain imaging. Perform non-contrast head CT after a witnessed seizure, focal deficit, or sudden alteration in mental status. Cocaine can precipitate hemorrhagic stroke through abrupt hypertension or rupture of berry aneurysms. Ischemic stroke also occurs via vasospasm or platelet

aggregation. Early imaging dictates thrombolytic eligibility and blood pressure targets. Some clinicians add CT angiography to visualize vascular narrowing and guide calcium channel blocker therapy. In patients with meningismus or fever, consider lumbar puncture after imaging excludes mass effect. Meningitis in this context may stem from endocarditis due to needle use or contamination from adulterants. Cerebrospinal fluid analysis differentiates infectious meningoencephalitis from drug-induced neuroinflammation. Point-of-care ultrasound augments bedside decision making. Cardiac views assess ejection fraction during suspected cardiomyopathy, visualize pericardial effusion, and aid volume status estimates. Doppler of peripheral arteries verifies pulses compromised by vasospasm. Pulmonary ultrasound reveals B-lines suggestive of edema sooner than radiography [10].

Interpret results against a ticking clock. Cocaine's rapid distribution means early labs may appear deceptively reassuring. Repeat testing six to eight hours later often unmasks worsening acidosis, rising troponin, or climbing creatine kinase. Are you prepared to escalate care when numbers trend unfavorably despite stable vital signs? When you order tests, integrate them into a structured pathway. Standardize panels for all stimulant exposures to reduce omissions during chaotic shifts. Incorporate reminders for serial troponin and electrolytes into electronic order sets. Engage toxicology specialists early to interpret atypical findings. Invite cardiology when arrhythmia persists despite benzodiazepine sedation and electrolyte correction. Finally, use evaluation as a teachable moment. Discuss findings with the patient once agitation resolves. Show how laboratory markers reflect organ injury. Ask whether witnessing physiologic data changes their view on continued use. Collaboration with addiction services during this window leverages objective evidence to motivate treatment engagement. Comprehensive evaluation therefore merges systematic laboratory analysis with focused imaging. Each data point illuminates a facet of cocaine's multifaceted toxicity. The process equips you to recognize hidden complications, personalize interventions, and anticipate deterioration. In a disorder notorious for sudden reversals, vigilant assessment becomes your best defense against preventable harm [10].

Treatment and Management

Management of cocaine toxicity begins with immediate stabilization, using the ABCDE approach—airway, breathing, circulation, disability (neurological status), and exposure (undressing to assess for signs of trauma, track marks, or hidden complications). Ensuring airway protection and adequate oxygenation is the first priority. Continuous pulse oximetry, cardiac monitoring, and blood pressure surveillance are essential from arrival. If the patient presents with altered mental status or seizures, rapid correction of reversible causes like hypoglycemia must be prioritized. A bedside glucose test should be performed without delay. In all women of reproductive age, a pregnancy test is essential to guide medication choices and anticipate potential fetal complications. Treatment must be symptom-based, guided by the severity and type of toxicity rather than laboratory values alone. Physical restraints should generally be avoided due to the risk of worsening agitation, hyperthermia, or positional asphyxia. Instead, early pharmacological control of symptoms, particularly agitation and cardiovascular instability, is more effective and safer. Benzodiazepines are the cornerstone of first-line treatment for both agitation and cardiovascular toxicity caused by cocaine. A large systematic review supports their use to reduce sympathetic nervous system outflow from the central nervous system [11]. Benzodiazepines such as lorazepam or diazepam help blunt the hyperadrenergic state, lower blood pressure, reduce heart rate indirectly, and prevent seizures. However, these agents must be dosed carefully, often in repeated and escalating doses, which carries a real risk of over-sedation and respiratory depression. Clinicians must be prepared to support ventilation, if necessary, especially in patients with compromised pulmonary function or co-ingestions such as opioids or alcohol [11].

For hypertension not controlled by benzodiazepines alone, several pharmacologic options exist. Non-dihydropyridine calcium channel blockers, particularly diltiazem and verapamil, are effective at lowering blood pressure but have minimal effect on heart rate. These agents can be used safely in patients without significant left ventricular dysfunction. Dihydropyridine calcium channel blockers like nifedipine are avoided due to their potential to trigger reflex tachycardia, which may worsen myocardial ischemia. Alpha-adrenergic blockers such as phentolamine have been used to reduce cocaine-induced hypertension through

alpha receptor antagonism. However, phentolamine is not commonly used due to limited clinician familiarity and poor titratability. It also does not treat tachycardia, making it less useful in combined cardiovascular presentations. More widely used vasodilators like nitroglycerin and nitroprusside are effective at reducing systemic vascular resistance and blood pressure, but the potential for reflex tachycardia should be monitored carefully, especially in patients with chest pain or suspected coronary ischemia (Figure 2) [11].

Table 6. Treatment Summary For Cocaine-Associated Acute Coronary Syndrome
Oxygen
Benzodiazepines (diazepam, midazolam)
Aspirin
Nitroglycerin
Morphine
Phentolamine
Verapamil
Fractionated or unfractionated heparin
Glycoprotein IIb/IIIa inhibitors
Percutaneous angioplasty
Fibrinolytic therapy

Figure 2: Treatment Lines of Acute Cocaine Toxicity.

Labetalol, a combined alpha- and beta-blocker, offers a balanced and evidence-based option. It addresses both hypertension and tachycardia without triggering “unopposed alpha stimulation”—a theoretical risk often cited in the past but not supported by clinical evidence. According to AHA/ACC guidelines, labetalol is safe for patients with cocaine- or methamphetamine-induced unstable angina or non-ST-segment elevation myocardial infarction [12]. It can be administered intravenously in repeated boluses or as a continuous infusion, depending on response and severity. In severely agitated or violent patients, benzodiazepines remain the first-line choice. When they fail to control symptoms, antipsychotic medications such as haloperidol or olanzapine can be added. Several studies show that a combination of benzodiazepines and antipsychotics provides better control of agitation than either agent alone [13]. Diphenhydramine is frequently co-administered to potentiate sedation and reduce the risk of extrapyramidal side effects, such as dystonia or akathisia. A commonly used emergency department cocktail, often referred to as the “B-52,” combines haloperidol 5 mg, diphenhydramine 50 mg, and lorazepam 2 mg. This combination can calm severely agitated patients quickly and reduce the need for physical restraints. For life-threatening ventricular arrhythmias, lidocaine has been used successfully as a Class IB antiarrhythmic. In severe cases, especially those involving cocaine-induced sodium channel blockade and wide QRS complexes, intravenous lipid emulsion therapy has shown success in case reports. This “lipid sink” mechanism may help remove lipophilic toxins like cocaine from myocardial tissue, though it remains a second-line treatment reserved for refractory cases [13].

Hyperthermia is another critical issue in cocaine toxicity. Patients can reach body temperatures above 40 °C, which may lead to rhabdomyolysis, renal failure, and disseminated intravascular coagulation. Prompt and effective cooling is necessary. The safest and simplest method in most clinical settings involves tepid water misting and forced-air convection using fans. This can be done rapidly at the bedside and does not require advanced equipment. In more severe cases, cooling blankets, ice packs, or even invasive cooling catheters may be considered. Antipyretics like acetaminophen are ineffective because the elevated temperature is not due to hypothalamic set-point change but rather to excessive muscle activity and metabolic rate.

Throughout treatment, clinicians must monitor for delayed complications, including myocardial infarction, stroke, acute kidney injury, and seizures. Patients should remain under observation for several hours even if initial symptoms subside. Continuous cardiac monitoring is especially important in those with chest pain, abnormal ECG findings, or elevated troponins. Seizure prophylaxis may be considered in patients with known high risk or past seizure history, particularly when hyperthermia or hyponatremia is present. Ultimately, treatment of cocaine toxicity requires a flexible, symptom-driven approach. Early and aggressive pharmacologic sedation with benzodiazepines, appropriate cardiovascular support with agents like labetalol or calcium channel blockers, and rapid cooling in hyperthermic patients are the foundation of care. Proper monitoring, supportive care, and interdisciplinary coordination, especially with toxicology and psychiatry—can significantly improve outcomes and reduce long-term complications [13].

Differential Diagnosis

When evaluating a patient with suspected cocaine toxicity, it is essential to maintain a broad differential diagnosis due to the overlapping signs and symptoms with other acute medical and psychiatric conditions. Agitation, delirium, hallucinations, hyperthermia, and autonomic instability are not exclusive to stimulant intoxication. Each alternative condition requires distinct management strategies, making accurate diagnosis critical. Hypoglycemia can be presented with confusion, agitation, diaphoresis, tachycardia, and even seizures. Unlike cocaine toxicity, these symptoms rapidly resolve with glucose administration. Point-of-care blood glucose testing should be performed early to exclude this reversible cause, especially in patients with a history of diabetes, alcohol use, or malnutrition. Anticholinergic toxicity shares features such as agitation, mydriasis, tachycardia, hyperthermia, and dry skin. However, patients with anticholinergic syndrome often have dry mucous membranes, urinary retention, absent bowel sounds, and may display picking or grasping motions (carphologia). Unlike cocaine toxicity, sweating is typically absent [13].

Central nervous system (CNS) hemorrhage, particularly subarachnoid or intracerebral bleeding, can cause sudden headache, vomiting, altered consciousness, and focal neurologic signs. Cocaine can precipitate such events, but a spontaneous hemorrhage in the absence of stimulant use must be considered. Non-contrast head CT is essential when there is altered mental status, seizures, or focal deficits. Delirium tremens occurs in patients undergoing alcohol withdrawal and can mimic cocaine toxicity with agitation, tremor, tachycardia, and hallucinations. A detailed substance use history is key. In contrast to cocaine use, delirium tremens often develops 48–96 hours after cessation of alcohol in chronic users and is accompanied by autonomic instability and confusion. Acute schizophrenia may present paranoia, hallucinations, disorganized speech, and agitation. However, physical findings such as tachycardia, hypertension, hyperthermia, or diaphoresis are typically absent unless co-ingestion is involved. A psychiatric history and lack of autonomic signs help differentiate the two. Phencyclidine (PCP) toxicity closely resembles cocaine intoxication, with agitation, combativeness, hallucinations, and nystagmus. PCP can also cause vertical nystagmus and muscle rigidity, which are not typical with cocaine. Urine toxicology may help confirm the diagnosis. Neuroleptic malignant syndrome is a rare, life-threatening reaction to antipsychotic medications and presents hyperthermia, muscle rigidity, altered mental status, and autonomic instability. Unlike cocaine toxicity, it is typically associated with recent antipsychotic use and marked elevations in creatine kinase [13].

Prognosis

Cocaine toxicity carries a high risk of mortality, especially when diagnosis or treatment is delayed. Fatal outcomes are often linked to acute cardiovascular or neurologic complications such as myocardial infarction, arrhythmia, stroke, or status epilepticus. Severe hyperthermia and rhabdomyolysis can also lead to multi-organ failure and death if not recognized and managed rapidly. Injection of cocaine introduces additional risks. Intravenous use may result in pneumothorax from deep tissue trauma or vascular puncture. Thrombosis of major veins and arteries is common due to repeated endothelial injury and hypercoagulability. Infective endocarditis may develop from contaminated needles or injection sites, particularly in those with underlying valvular heart disease. Central vascular complications include

pseudoaneurysms and arteriovenous fistulas, especially in patients who inject near large vessels without sterile technique. These structural abnormalities carry the potential for rupture or high-output heart failure. Soft tissue complications are also frequent. Repeated injections can lead to cellulitis, abscess formation, and necrotizing infections. Infections such as tetanus and HIV remain significant concerns among users who share or reuse needles. Thrombophlebitis, both superficial and deep, can progress to systemic illness. Necrosis of skin and underlying tissues may require surgical intervention or amputation in severe cases. Neuropsychiatric sequelae may persist even after acute symptoms are resolved. Chronic use and repeated episodes of toxicity can result in long-term cognitive impairment, persistent psychosis, or affective instability. Recovery of neurologic function is often slow and may be incomplete. Many individuals require prolonged rehabilitation, psychiatric care, and substance use treatment. The prognosis improves with early intervention, abstinence, and long-term multidisciplinary follow-up [14].

Complications

Cocaine use, particularly through intravenous injection, is associated with multiple systemic complications that increase both morbidity and mortality. Thrombophlebitis is common due to repeated trauma to veins and the irritant nature of injected substances. It presents pain, swelling, and erythema along the veins and may progress to deep vein thrombosis or septic thrombophlebitis if untreated. Human immunodeficiency virus (HIV) transmission is a significant risk among individuals who share needles or engage in unprotected sex under the influence of cocaine. The high-risk behaviors associated with stimulant use further elevate the chance of acquiring or spreading HIV in this population. Cellulitis frequently occurs at injection sites, especially when non-sterile techniques are used. Skin infections can rapidly evolve into abscesses or necrotizing fasciitis, requiring surgical drainage or debridement. Hepatitis B and C infections are also prevalent among cocaine users due to needle sharing and blood exposure. These infections can lead to chronic liver disease and cirrhosis over time. Infective endocarditis is a life-threatening complication in intravenous cocaine users. The combination of bacteremia and endothelial damage from foreign particles introduced during injection increases the risk of valvular infection, particularly involving the tricuspid valve. Signs include fever, murmur, and septic emboli. Pulmonary emboli may occur from thrombus formation in peripheral veins or from particulate matter injected into the venous system. These emboli can obstruct pulmonary arteries and lead to hypoxia, chest pain, or sudden death. Vascular aneurysms, including pseudoaneurysms, may form due to repeated arterial trauma or infection at injection sites. These abnormal vessel dilations carry a risk of rupture and hemorrhage, requiring urgent surgical intervention [14].

Other Issues

The concept of “unopposed alpha-stimulation” in cocaine toxicity has generated debate among clinicians for decades. This theoretical phenomenon suggests that when beta-adrenergic receptors—particularly beta-2—are blocked in a patient who has used cocaine, the resulting unopposed alpha-adrenergic activity may lead to worsened hypertension or coronary vasospasm. This idea emerged from early reports and animal studies suggesting adverse cardiovascular responses when beta-blockers were administered in the setting of cocaine use. As a result, many clinicians have historically viewed the use of beta-blockers, especially non-selective agents like propranolol, as contraindicated in patients presenting with cocaine-associated chest pain or acute coronary syndrome. This caution extended to cardioselective beta-blockers and even mixed alpha/beta agents like labetalol. However, over time, a growing number of studies and clinical experiences have challenged this view.

In practice, many physicians now safely administer beta-blockers in the context of cocaine-related cardiovascular complications, particularly when tachycardia and demand ischemia are present. A systematic review analyzing 1,744 patients found only seven adverse events that could potentially be attributed to unopposed alpha-stimulation. These events involved the use of propranolol (n=3), esmolol (n=3), and metoprolol (n=1). Importantly, none of these events were conclusively proven to result from beta-blockade itself, and the severity of outcomes varied. The authors of the original case reports that introduced the concept of unopposed alpha-stimulation revisited the issue in a 2017 review. They

concluded that the adverse hemodynamic effects observed in early reports may have been due to the direct effects of cocaine alone, regardless of beta-blocker administration. This re-evaluation has further weakened the case for blanket avoidance of beta-blockers in all cocaine-using patients. Current evidence supports the careful use of beta-blockers—especially mixed alpha/beta agents like labetalol—in select cases of cocaine-induced cardiovascular complications, particularly when heart rate control is necessary. Clinical judgment, patient-specific factors, and response to initial therapies should guide treatment rather than reliance on outdated theoretical concerns [14].

Enhancing Healthcare Team Outcomes

Cocaine overdose rates have shown a troubling trend over the past thirty years. In the United States, cocaine-related mortality ranged from 0.9 to 1.6 deaths per 100,000 people, reflecting widespread and persistent use. Only in the last five years have these numbers declined, with recent estimates falling to approximately 0.78 deaths per 100,000 population. Despite this reduction, the burden remains significant, particularly among those who use cocaine through high-risk routes. For instance, individuals injecting into the neck veins face serious complications such as pneumothorax, hemothorax, thrombophlebitis, and localized infections like myositis. Vascular damage may also occur, including the development of arterial aneurysms, which increase the risk of rupture, obstruction, and arteriovenous fistulas [14].

The co-use of cocaine with other substances compounds the danger. Combining cocaine with prescription drugs or illicit agents such as heroin or alcohol results in a markedly increased risk of overdose and death. Notably, when cocaine is used with alcohol, the risk of suicide increases dramatically—by a factor of 16 compared to using either substance alone. Users under this influence tend to select more violent methods of self-harm. Cocaine also has serious implications during pregnancy. Its use can lead to preterm birth, placental abruption, and low birth weight, posing risks to both maternal and fetal health. Improving outcomes for patients experiencing cocaine toxicity requires coordination among interprofessional healthcare teams. Emergency department nurses are often the first point of contact and play a critical role in early identification. A high index of suspicion is essential, especially when patients are present with chest pain, altered mental status, or agitation. Rapid triage, vital signs assessment, and recognition of patterns consistent with stimulant use must occur in the first minutes of care. Physicians and nurse practitioners in emergency or urgent care settings initiate treatment based on presenting symptoms. They stabilize the patient, manage cardiovascular and neurologic complications, and initiate cooling if hyperthermia is present. Critical care physicians and hospitalists take over in cases requiring intensive monitoring or inpatient admission. Cardiologists may be consulted for suspected myocardial infarction, arrhythmias, or ongoing chest pain. Toxicologists support decision-making on pharmacologic interventions, especially in complex cases involving polydrug use [14][15].

Pharmacists are integral to the safe management of these patients. They review medication regimens, identify potential interactions, and advise on correct dosing—particularly when benzodiazepines, antipsychotics, or antihypertensives are required. This is crucial in preventing over-sedation, hypotension, or adverse effects from drug combinations. Emergency and intensive care nurses administer medications, monitor the response to treatment, and promptly report changes in clinical status. They ensure ongoing assessments of cardiac rhythm, temperature, neurologic signs, and fluid status. These nurses also engage with patients and families, offering education on the risks of cocaine use and reinforcing discharge instructions. Long-term success depends not only on acute stabilization but also on early intervention and referral for substance use treatment. No pharmacological agent currently exists to prevent or cure cocaine addiction. This places significant responsibility on frontline healthcare providers to initiate conversations about substance use during the emergency encounter. When the patient is stabilized, healthcare workers should provide information about local addiction treatment programs and support groups [15]. Patients who present following suicide attempts require mental health evaluation before discharge. A referral to a mental health counselor should be made for anyone with suicidal ideation or a history of self-harm. This step is vital, particularly in patients using cocaine and alcohol concurrently, due to the high risk of repeated attempts. Follow-up care with mental health services improves the likelihood of recovery and reduces the risk of future crises. In summary, managing cocaine toxicity and its complications requires coordinated

effort across multiple healthcare disciplines. From triage through stabilization, monitoring, education, and referral, each team member plays a defined role. Effective interprofessional collaboration enhances patient safety, improves outcomes, and creates an opportunity to connect individuals with resources that address the root of their addiction [15].

Roles of Emergency and Healthcare Information Professionals:

Emergency professionals play a pivotal role in the acute management of patients presenting with cocaine toxicity. These professionals, including emergency physicians, nurses, and advanced practice providers, are responsible for the rapid recognition and stabilization of patients suffering from life-threatening effects of cocaine use. Their duties begin at triage, where clinical suspicion must be high, especially in cases of unexplained agitation, chest pain, hyperthermia, or seizures. Emergency professionals are trained to implement the ABCDE approach immediately—ensuring airway patency, adequate ventilation, circulatory support, neurologic assessment, and exposure evaluation. Their prompt actions are critical in preventing fatal complications such as arrhythmias, myocardial infarction, or respiratory failure. Beyond stabilization, emergency staff initiate appropriate pharmacologic therapy, monitor treatment responses, and escalate care when required, involving critical care or cardiology teams when necessary. They are also instrumental in recognizing psychiatric emergencies, including suicidal ideation, and ensuring safe referrals to mental health services before discharge. In parallel, healthcare information professionals contribute to improving outcomes by managing and safeguarding clinical data essential for decision-making. These professionals maintain electronic health records (EHRs), develop clinical decision support systems, and ensure data accuracy for timely access by all care team members. Their work enables rapid retrieval of patient histories, medication profiles, and diagnostic test results, which is particularly crucial in emergency settings. Healthcare information professionals also contribute to the design and maintenance of drug alert systems that help clinicians avoid adverse drug interactions, especially when patients have used cocaine in combination with other substances. Their role extends to ensuring compliance with data privacy regulations and supporting the integration of evidence-based guidelines into clinical workflows through digital tools. This includes embedding alerts or protocols related to substance use screening, suicide risk assessments, and referral pathways within the EHR interface. Furthermore, healthcare information professionals support quality improvement and public health surveillance by collecting and analyzing data on cocaine-related emergencies. Their reports can inform institutional policies, contribute to national databases, and aid in tracking trends in drug-related morbidity and mortality. By collaborating with emergency teams, these professionals facilitate coordinated care, optimize clinical efficiency, and contribute to long-term strategies aimed at addressing the broader implications of cocaine use through accurate data analysis and reporting systems. This integration of clinical and informatics roles is essential for delivering timely, safe, and effective care in the context of substance-related emergencies [15].

Conclusion:

Cocaine overdose remains a critical medical emergency requiring rapid, coordinated intervention to prevent fatal outcomes. The drug's complex pathophysiology—primarily driven by excessive catecholamine release—leads to cardiovascular collapse, hyperthermia, seizures, and multi-organ failure. Emergency clinicians must prioritize early stabilization, utilizing benzodiazepines to mitigate sympathetic overdrive, vasodilators for hypertensive crises, and aggressive cooling for hyperthermia. The historical reluctance to use beta-blockers has been challenged by recent evidence supporting labetalol in select cases, though caution remains necessary in severe intoxication. The management of cocaine toxicity extends beyond acute care. Polysubstance use, particularly with alcohol or opioids, complicates treatment and increases mortality risk. Emergency departments must adopt standardized protocols, including toxicology screening, serial troponin measurements, and continuous cardiac monitoring, to identify complications promptly. Additionally, psychiatric evaluation is crucial, as cocaine users exhibit high rates of suicidal ideation, especially when combined with alcohol. Health information professionals play a pivotal role in optimizing care. EHR systems enhance clinical decision-making by providing real-time access to patient histories, drug interactions, and treatment guidelines. Data analytics enable public health tracking of overdose trends, informing preventive strategies and resource allocation. Integrating substance use disorder (SUD) referrals

into EHR workflows can bridge the gap between emergency care and long-term addiction treatment. Despite advances in acute management, the lack of pharmacotherapy for cocaine addiction underscores the importance of behavioral interventions. Emergency visits serve as critical touchpoints for initiating motivational interviewing and connecting patients with rehabilitation services. Community-based programs, naloxone distribution for opioid co-ingestion, and mental health support are essential in reducing relapse. Future efforts should focus on interdisciplinary training for emergency teams, ensuring proficiency in managing stimulant-related complications. Public health initiatives must address socioeconomic determinants of drug use while expanding access to evidence-based treatment. By combining acute medical care, health informatics, and sustained addiction support, healthcare systems can mitigate the devastating impact of cocaine overdose on individuals and communities.

References:

1. Hantson P. Mechanisms of toxic cardiomyopathy. *Clin Toxicol (Phila)*. 2019 Jan;57(1):1-9.
2. Mikhail A, Tanoli O, Légaré G, Dubé PA, Habel Y, Lesage A, Low NCP, Lamarre S, Singh S, Rahme E. Over-the-Counter Drugs and Other Substances Used in Attempted Suicide Presented to Emergency Departments in Montreal, Canada. *Crisis*. 2019 May;40(3):166-175.
3. Narula N, Siddiqui F, Katyal N, Krishnan N, Chalhoub M. Cracking the Crack Dance: A Case Report on Cocaine-induced Choreoathetosis. *Cureus*. 2017 Dec 22;9(12):e1981.
4. Mladěnka P, Applová L, Patočka J, Costa VM, Remiao F, Pourová J, Mladěnka A, Karlíčková J, Jahodář L, Vopršalová M, Varner KJ, Štěrbá M., TOX-OER and CARDIOTOX Hradec Králové Researchers and Collaborators. Comprehensive review of cardiovascular toxicity of drugs and related agents. *Med Res Rev*. 2018 Jul;38(4):1332-1403.
5. Paulozzi LJ. Opioid analgesic involvement in drug abuse deaths in American metropolitan areas. *Am J Public Health*. 2006 Oct;96(10):1755-7.
6. Turner C, Chandrakumar D, Rowe C, Santos GM, Riley ED, Coffin PO. Cross-sectional cause of death comparisons for stimulant and opioid mortality in San Francisco, 2005-2015. *Drug Alcohol Depend*. 2018 Apr 01;185:305-312.
7. Zimmerman JL. Cocaine intoxication. *Crit Care Clin*. 2012 Oct;28(4):517-26.
8. Richards JR, Hollander JE, Ramoska EA, Fareed FN, Sand IC, Izquierdo Gómez MM, Lange RA. β -Blockers, Cocaine, and the Unopposed α -Stimulation Phenomenon. *J Cardiovasc Pharmacol Ther*. 2017 May;22(3):239-249.
9. Agrawal PR, Scarabelli TM, Saravolatz L, Kini A, Jalota A, Chen-Scarabelli C, Fuster V, Halperin JL. Current strategies in the evaluation and management of cocaine-induced chest pain. *Cardiol Rev*. 2015 Nov-Dec;23(6):303-11.
10. Maraj S, Figueredo VM, Lynn Morris D. Cocaine and the heart. *Clin Cardiol*. 2010 May;33(5):264-9.
11. Richards JR, Garber D, Laurin EG, Albertson TE, Derlet RW, Amsterdam EA, Olson KR, Ramoska EA, Lange RA. Treatment of cocaine cardiovascular toxicity: a systematic review. *Clin Toxicol (Phila)*. 2016 Jun;54(5):345-64.
12. Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE, Chavey WE, Fesmire FM, Hochman JS, Levin TN, Lincoff AM, Peterson ED, Theroux P, Wenger NK, Wright RS, Jneid H, Ettinger SM, Ganiats TG, Philippides GJ, Jacobs AK, Halperin JL, Albert NM, Creager MA, DeMets D, Guyton RA, Kushner FG, Ohman EM, Stevenson W, Yancy CW. 2012 ACCF/AHA focused update incorporated into the ACCF/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2013 Jun 11;61(23):e179-347.
13. Zun LS. Evidence-Based Review of Pharmacotherapy for Acute Agitation. Part 1: Onset of Efficacy. *J Emerg Med*. 2018 Mar;54(3):364-374.
14. Modisett KL, Walsh SJ, Heffner AC, Pearson DA, Kerns W. Outcomes in Cardiac Arrest Patients due to Toxic Exposure Treated with Therapeutic Hypothermia. *J Med Toxicol*. 2016 Sep;12(3):263-9.
15. Alfa-Wali M, Atinga A, Tanham M, Iqbal Q, Meng AY, Mohsen Y. Assessment of the management outcomes of body packers. *ANZ J Surg*. 2016 Oct;86(10):821-825.

الملخص:

الخلفية: يُعد إساءة استخدام الكوكايين مشكلة صحية عامة عالمية خطيرة، تؤدي إلى معدلات مرتفعة من زيارات قسم الطوارئ بسبب التسمم الحاد. يمكن أن تسبب تأثيرات الكوكايين على الجهاز القلبي الوعائي والعصبي مضاعفات تهدد الحياة، بما في ذلك النوبة القلبية، والسكتة الدماغية، وفرط الحرارة، والتهديان الاهتياجي. كما أن زيادة استخدام متعدد للمخدرات يجعل الإدارة السريرية أكثر تعقيداً.

الهدف: تبحث هذه المقالة في الفيزيولوجيا المرضية، والمظاهر السريرية، والإدارة الطارئة لتسمم الكوكايين، مع التركيز على أدوار الأطباء والمتخصصين في المعلومات الصحية لتحسين نتائج المرضى.

المنهجية: تم إجراء مراجعة شاملة لحركية السموم الخاصة بالكوكايين، والمظاهر السريرية، واستراتيجيات العلاج المبنية على الأدلة. كما تم استكشاف أدوار فرق الطوارئ والمعلوماتية الصحية في التشخيص، والعلاج، وتنسيق الرعاية طويلة المدى.

النتائج: يتطلب تسمم الكوكايين التعرف السريع والتدخل الفوري. تظل البنزوديازيبينات الخط الأول لعلاج الاهتياج والأعراض القلبية الوعائية، بينما يُفضل استخدام لايبتالول وحاصرات قنوات الكالسيوم لعلاج ارتفاع ضغط الدم. كما يتطلب فرط الحرارة تبريداً فورياً. يعمل متخصصو المعلومات الصحية على تحسين الرعاية من خلال السجلات الصحية الإلكترونية، ودعم القرارات السريرية، وتتبع البيانات لمراقبة الصحة العامة.

الاستنتاج: تعتمد الإدارة الفعالة لجرعة الكوكايين الزائدة على التعاون متعدد التخصصات، والتدخل الدوائي السريع، وأنظمة معلومات صحية قوية. يجب أن تستند الاستراتيجيات طويلة المدى إلى دمج علاج الإدمان والدعم النفسي لتقليل الانتكاس والوفيات.

الكلمات المفتاحية: تسمم الكوكايين، الإدارة الطارئة، البنزوديازيبينات، فرط الحرارة، المعلوماتية الصحية، علاج الإدمان.