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A review of common psychoactive Substances in Emergency Medicine: Clinical Patterns and Diagnostic Strategies in the ED

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Abstract

Introduction: The use of psychoactive substances in Europe has been steadily rising, resulting in a significant increase in emergency department (ED) visits related to acute intoxication. Substances such as cocaine, amphetamines, cannabinoids (including synthetic variants) and opioids, particularly potent analogues like fentanyl and nitazenes, pose complex diagnostic and therapeutic challenges in the emergency setting. Prompt recognition of clinical patterns and effective management are critical to reducing morbidity and mortality.

Materials and Methods: This narrative review is based on an extensive analysis of current scientific literature, institutional reports (including EMCDDA and Euro-DEN), and clinical toxicology guidelines published between 2018 and 2025. Sources include observational studies, retrospective analyses, and expert recommendations focused on substance-related emergencies.

The review discusses pharmacological mechanisms, clinical presentations, diagnostic strategies, and treatment protocols commonly employed in EDs.

Summary: Each substance class is examined in terms of its pharmacodynamics, typical toxidromic features, and associated complications. The review highlights the diagnostic limitations of rapid toxicology screening and emphasizes the role of toxidrome recognition and structured emergency assessment tools. It also addresses the growing problem of polysubstance use, which complicates clinical presentation and requires careful, symptom-driven treatment approaches.

Conclusions:The dynamic landscape of psychoactive substance use necessitates continuous updates in emergency care practices. Clinicians must rely on symptom-based management, targeted pharmacologic interventions such as naloxone or benzodiazepines, and vigilant patient monitoring. Broader availability of advanced toxicological testing and more prospective clinical data are essential for optimizing diagnosis and improving outcomes in substance-related emergencies.

Keywords: Psychoactive substances, Emergency medicine, Cocaine, Fentanyl, Synthetic cannabinoids, Toxidromes, Acute intoxication, Diagnostic strategies, Polysubstance use

1. Introduction

According to the European Union Drugs Agency (EUDA), the use of psychoactive substances in Europe has been steadily increasing in recent years, with a parallel rise in drug-related hospitalizations and emergency department (ED) visits. In 2023 alone, an estimated 7,459 fatal overdoses were reported across the European Union, compared to 7,145 in the previous year. Opioids remain the leading cause of these deaths, being involved in approximately 74% of all cases [1]. These numbers highlight the urgent need for effective strategies to recognize, diagnose, and manage substance-related emergencies in clinical practice.

Among the most frequently encountered substances in emergency departments (EDs) are cocaine, amphetamine and its derivatives, natural and synthetic cannabinoids, and opioids—including highly potent synthetic analogues such as fentanyl and nitazenes [2]. These substances differ in their pharmacological profiles, toxic effects, and treatment protocols.

Clinicians face considerable challenges in acute care settings due to the often non-specific presentation of intoxication, limited access to rapid toxicological testing, and the urgency of clinical decision-making [3,4]. Although not the primary focus of this review, it is important to note that many patients present with symptoms resulting from the combined use of multiple substances (polysubstance use). This practice, increasingly common across Europe, can intensify toxicity, complicate diagnosis, and increase the risk of severe outcomes [1].

The aim of this review is to provide a comprehensive overview of the most prevalent psychoactive substances encountered in emergency medicine, with emphasis on their mechanisms of action, clinical presentation, diagnostic strategies, and management within the ED setting.

2. Characteristics, Pharmacology and Clinical Presentation of Selected Psychoactive Substances

2.1 Cocaine

Cocaine, a tropane alkaloid derived from *Erythroxylon coca* leaves, is commonly abused in the form of cocaine hydrochloride (administered intranasally or intravenously) or as crack cocaine (inhaled as vapor) [5]. Its primary mechanism of action involves the inhibition of monoamine transporters: dopamine, norepinephrine and serotonin, resulting in elevated synaptic concentrations and enhanced sympathomimetic activity [6,7]. In addition to monoamine reuptake inhibition, cocaine blocks voltage-gated sodium channels, which accounts for its local anesthetic effects and contributes to arrhythmogenic potential through impaired myocardial conduction [5,8]. The substance also interacts with muscarinic, NMDA, sigma, and opioid receptors, potentially facilitating seizure activity and neuropsychiatric symptoms [8,9].

The pharmacokinetics of cocaine vary by route of administration. Intravenous injection typically produces effects within 3 minutes, with a half-life of approximately 0.5-1.5 hours [8]. Inhalation of crack cocaine yields a near-immediate onset (~1-1.5 minutes), while intranasal administration produces effects after about 15 minutes, with maximum psychological and physiological impact occurring around 20–40 minutes post-use [8].

Clinically, cocaine intoxication presents with a characteristic sympathomimetic toxidrome, including tachycardia, hypertension, mydriasis, diaphoresis, and hyperthermia [7,9].

Neuropsychiatric manifestations such as agitation, anxiety, paranoia, hallucinations, psychosis and seizures are frequently observed [7,9]. Seizures occur in approximately 3% of ED presentations involving cocaine intoxication [10]. Among cardiovascular complications, acute myocardial infarction (AMI) has been reported in 0.7-5.7% of patients presenting with cocaine-associated chest pain [11]. The risk of AMI may be up to seven times higher in cocaine users compared to non-users, particularly within the first hour after use [12]. Life-threatening ventricular arrhythmias, sudden cardiac death, ischemic and hemorrhagic strokes, aortic dissection, rhabdomyolysis and multiorgan failure have also been documented [9,10].

Cocaine is the second most commonly used illicit drug in Europe, with lifetime prevalence in adults (18–64 years) at approximately 7–8%, and past-year use around 2–3%, with an overall rising trend in many countries, including among socially marginalised groups [5].

Although in-hospital mortality for cocaine-induced AMI remains relatively low (<2%), long-term mortality is significantly elevated due to trauma, infections, and cardiovascular disease [12]. Global epidemiological estimates attribute several thousand deaths annually to cocaine-related causes [12].

2.2 Amphetamines and Derivatives

Amphetamines, including methamphetamine and MDMA, boost synaptic dopamine, norepinephrine, and serotonin by reversing and blocking their transporters (DAT, NET, SERT), leading to strong stimulant and sympathomimetic effects [13]. MDMA uniquely triggers large serotonin release and promotes oxytocin secretion, underpinning its empathogenic properties [14]. Methamphetamine administration causes oxidative stress, mitochondrial impairment, and neuroinflammatory responses, which can damage dopaminergic and serotonergic neurons [15].

The onset of action is rapid, within minutes if smoked or injected, while oral consumption delays peak effects to 1-2 hours; methamphetamine's long elimination half-life of ~10-12 hours sustains its psychoactivity [13,15].

In emergency care, patients often present with a sympathomimetic toxidrome: tachycardia, hypertension, hyperthermia, diaphoresis and dilated pupils with frequent neuropsychiatric manifestations such as agitation, paranoia, psychosis, hallucinations and possible seizures [13,16,17]. A large retrospective ED study reported that 78% of patients exhibited

behavioral disturbance, while 56% had tachycardia, 42% hypertension, 5% hyperthermia and notable complications, including rhabdomyolysis (30%), acute kidney injury (13%), seizures (2 cases), intracranial hemorrhages (3 cases) and one myocardial infarction [13,16].

Chronic complications include cardiovascular diseases (myocardial infarction, arrhythmias), stroke, pulmonary hypertension, cardiomyopathy, as well as rhabdomyolysis, renal and hepatic injury and infectious outcomes like endocarditis [15,17]. A systematic review further identified acute kidney injury as a common consequence of methamphetamine intoxication [17].

Methamphetamine related mortality has increased over the past decade, driven in part by cardiomyopathy, overdoses, and vascular events [15,16]. One cohort study reported two ED deaths, one attributed to cardiac arrest and another to subarachnoid hemorrhage [13].

Amphetamine use remains notable in Europe, with lifetime prevalence among adults (15-64 years) estimated at ~4.1% and past year use around 0.8%, trends that emphasize ongoing public health relevance [1].

2.3 Cannabinoids (THC and Synthetic)

Δ9 Tetrahydrocannabinol (THC), the primary psychoactive compound in cannabis, exerts its effects by acting as a partial agonist at CB1 and CB2 receptors, modulating neurotransmission in both central and peripheral systems [18]. Synthetic cannabinoids (SCs), by contrast, often function as full agonists at these receptors and may also interact with non-cannabinoid targets such as GPR55, PPARs and TRPV1, resulting in more potent and unpredictable pharmacological effects [19,20].

After inhalation or ingestion, THC's psychoactive effects typically begin within minutes to hours, lasting 2–6 hours and sometimes longer, depending on dose and formulation [18]. SCs exhibit rapid onset similar to THC but may have variable duration due to diverse chemical structures and metabolic pathways [19].

Clinically, THC intoxication commonly presents with tachycardia, anxiety, dry mouth, red eyes, altered perception, and in rare cases acute psychosis or cannabinoid hyperemesis syndrome (CHS) [18,21]. CHS is characterized by cyclical vomiting, abdominal pain and rare cardiovascular events and is the result of long-term use of cannabinoids.[21]

SCs exposure, on the other hand, frequently leads to severe neuropsychiatric symptoms, including agitation, psychosis, seizures and coma, often accompanied by cardiovascular

manifestations such as tachycardia, hypertension or hypotension, chest pain, and chest tightness [19,20,22]. SCs related toxicity is linked to multiorgan effects including rhabdomyolysis, acute kidney injury, liver damage, severe cardiovascular compromise (e.g., myocardial ischemia, arrhythmias), intracranial events, and respiratory failure [19,20,22,23]

Mortality from cannabis intoxication alone is extremely rare; however, deaths associated with SCs have occurred, often due to cardiovascular collapse, respiratory depression, seizures, or multi-organ failure [19,22,24]. ICU series report mortality rates up to 3% among SC intoxications, frequently following lethal systemic complications [22].

Cannabis remains the most commonly used illicit drug in Europe, with approximately 8.4% of adults (15–64 years) reporting cannabis use in the past year [1].

2.4 Opioids (Natural, Semi synthetic, and Synthetic: Fentanyl, Nitazenes)

Opioids exert their primary effects by agonizing μ opioid receptors, providing analgesia and euphoria but posing significant risk of respiratory depression, especially with high-potency synthetic variants [25]. Fentanyl, a potent phenylpiperidine opioid, demonstrates approximately 50–100 times greater potency than morphine, which significantly increases the risk of overdose and respiratory suppression [26]. Recently emerging nitazenes, benzimidazole opioids, exhibit μ receptor affinity comparable to or exceeding fentanyl, with limited human data indicating extreme potency and unpredictable toxicity [27,28].

Natural and semi-synthetic opioid overdoses typically present with reduced consciousness, respiratory depression, miosis, hypotension, bradycardia, and possible hypoxic neuronal injury [25].

Complications from opioid overdose include pulmonary edema, chest wall rigidity (notably with fentanyl), aspiration pneumonia, prolonged hypoxic coma, rhabdomyolysis, and multi-organ failure; nitazene toxicity further exacerbates cardiovascular collapse and neurologic complications [26,27,28]. The unpredictable pharmacokinetics and high potency of fentanyl analogues and nitazenes complicate emergency airway management and naloxone dosing [26,27]. Compared to fentanyl, nitazene overdoses have exhibited increased incidences of cardiac arrest in emergency settings- metonitazene alone was linked to cardiac arrest in all reported ED cases [25,28].

Mortality associated with fentanyl and its analogues has surged, contributing to the majority of opioid-related deaths in North America and Europe [26]. Nitazenes are increasingly implicated in fatal and non-fatal overdoses, with reports from ED and postmortem data revealing cases numbering in the dozens per region and escalating rapidly [27,28].

3. Diagnostics in Acute Intoxication

In emergency settings, precise and timely diagnostics in suspected acute intoxication are critical due to the frequently non-specific clinical picture and the unreliability of patient history sources [4].

The structured ABCDE approach remains the cornerstone of initial assessment. Securing the airway, assessing respiration and circulation, evaluating neurological function, and performing a full body examination including checking for signs of hyperthermia, trauma or skin changes are all essential components of early care [4,29].

Simultaneously, a targeted history should be obtained, focusing on the substance used, route of administration, estimated quantity and time of exposure. In unconscious or uncooperative patients, information from emergency responders or witnesses may be crucial to diagnosis and management [4].

When specific substance identification is not possible, recognizing toxidromes such as opioid, sympathomimetic or anticholinergic presentations enables empirical, life-saving interventions in the absence of laboratory confirmation [29].

Baseline laboratory workup, including glucose, electrolytes, liver and renal function tests, arterial blood gases, creatine kinase, coagulation profile, and ECG, provides valuable insight into both the toxic effects and any coexisting pathologies [29].

Immunoassay-based screens typically performed on urine or serum, are widely used in ED due to their speed and ease of use. However, they have limited sensitivity and detect only selected drug classes, leading to frequent false negatives, especially for benzodiazepines and cocaine. This limits their reliability in identifying the full scope of substance use and may hinder accurate clinical assessment in cases of suspected intoxication. [31]

Confirmatory tests using gas chromatography-mass spectrometry (GC-MS) or liquid chromatography–mass spectrometry (LC-MS/MS) offer higher accuracy and broader detection, making them valuable in emergency care. They can identify a wide range of substances,

including serotonergic drugs linked to serotonin syndrome, often missed by immunoassays. Although less available in real-time, their use can improve diagnostic accuracy in complex or unclear presentations. [31]

Notably, the clinical value of toxicology testing is limited if the results do not influence management decisions. The lack of robust prospective data on how test availability alters clinical outcomes reinforces the need for primarily symptom-driven care [30].

The differential diagnosis in patients with altered mental status or unexplained symptoms must remain broad. Conditions such as trauma, hypoglycemia, infection, or metabolic disorders may mimic intoxication or occur concurrently, and must not be overlooked [30].

Decision-support strategies, such as the PIRATE mnemonic (Primary survey, Investigations, Risk, ADME, Therapy, Evaluation), are increasingly recognized for structuring the toxicological workup and guiding emergency physicians through acute presentations of unknown origin [4].

4. Emergency Management and Treatment Options

Effective management of acute intoxication relies on a structured approach that balances empirical symptomatic care with targeted interventions when specific antidotes are available [32,33].

Immediate airway and ventilatory support are critical in the management of opioid overdose. According to the guidelines of the Royal College of Emergency Medicine, naloxone should be administered in incremental doses preferably intravenously at 100-200 µg per minute targeting the reversal of respiratory depression without precipitating abrupt opioid withdrawal. Alternative routes, such as intramuscular or intranasal administration, may also be employed. Titration should continue until the respiratory rate exceeds 10 breaths per minute and oxygen saturation reaches ≥92 %. Fentanyl and nitazenes frequently necessitate higher cumulative naloxone doses for reversal, with nitazene cases often requiring multiple naloxone boluses and prolonged observation [25,27]. Naloxone administration has been associated with adverse cardiovascular and pulmonary effects, including atrial and ventricular fibrillation, pulmonary edema, hypotension or hypertension, and cardiac arrest. Following emergency department intervention, patients should be observed for a minimum of four hours after the last naloxone

dose and provided with take-home naloxone kits and referral to evidence-based addiction treatment services. [32]

For stimulant intoxication (cocaine, amphetamines, some synthetic cannabinoid) no specific antidote exists. Instead benzodiazepines are recommended as first-line agents to reduce agitation, hypertension and tachycardia. Sedation may be required for patients with severe agitation from sympathomimetic overdose in order to treat hyperthermia and acidosis to avoid rhabdomyolysis and injury, and enable screening for additional potentially fatal issues. If high blood pressure or chest pain remains calcium channel blocker, α_1-adrenergic receptor antagonists and nitrates may be used. Beta blockers should be avoided [8,33]. External cooling techniques, such as an ice pack, cooled intravenous fluids, and gastric lavage, should be started in the event of hyperthermia [8]. Sodium bicarbonate, particularly in hypertonic solutions, has shown success in treating severe cocaine-induced cardiac arrhythmias, including wide-complex tachycardia and in cardiac arrest. Additionally, lidocaine is advised for the treatment of wide-complex tachycardia. [33]. If acute coronary syndrome arises, aspirin (if no contraindication) alongside benzodiazepines and nitrates is recommended [33]. Airway protection and oxygen supplementation are essential whenever respiratory compromise is identified. In stimulant-induced agitation or when high-dose benzodiazepines are required, patients may still need airway monitoring and potentially mechanical ventilation.[33]

Acute intoxication from cannabis and synthetic cannabinoids is typically managed with supportive care, as most cases are self-limiting. While natural cannabis may cause mild symptoms such as dizziness or anxiety, synthetic cannabinoids are often associated with more severe effects, including agitation, psychosis, seizures, and cardiovascular instability. Management begins with monitoring vital signs, assessing mental status, and ensuring airway and circulatory stability. Benzodiazepines are considered the treatment of choice for agitation, anxiety, or seizures related to these substances. Antipsychotics may be used cautiously if severe psychosis persists, though benzodiazepines remain first-line. Patients should be observed until symptoms resolve, which usually occurs within several hours. [34]

If the substance is unidentified, empirical symptomatic treatment based on toxidromic patterns remains the priority: benzodiazepines for sympathomimetic or anticholinergic features; naloxone for opioid signs; and appropriate circulatory or temperature management as clinical

circumstances dictate. Diagnostic testing and expert (poison center) consultation should run in parallel [29,33].

5. Polysubstance Use and Its Clinical Implications

Polysubstance use, defined as the concurrent or sequential use of two or more psychoactive substances, is increasingly common and presents significant challenges in emergency medicine [35,36]. According to data from the EMCDDA, a substantial proportion of emergency department intoxication cases involve more than one substance, often unknowingly, due to contamination of illicit drugs [35,37]. The most frequent combinations include stimulants (e.g., cocaine, amphetamines) with central nervous system depressants (e.g., opioids, benzodiazepines, alcohol), which may amplify toxicity and increase the risk of severe complications [36,38].

Clinically, such combinations can mask typical toxidromes or create mixed and atypical presentations. For instance, "speedballing", a mix of cocaine with heroin or fentanyl, can initially mask opioid-induced respiratory depression due to the stimulating effect of cocaine, delaying appropriate diagnosis and intervention [36,39]. Similarly, combining benzodiazepines with opioids may result in profound CNS depression, respiratory compromise and even cardiac arrest [38,39].

Additional diagnostic challenges arise with long half-life agents or delayed onset substances, such as certain new psychoactive substances (NPS), which may not be detected by standard toxicology panels [35,40].

Management should rely on a cautious, symptom-based approach. Administration of specific antidotes, such as naloxone in suspected opioid intoxication, remains critical but must be accompanied by close clinical monitoring, supportive care and early consultation with a poison control center [40,41]. Moreover, tracking local substance use trends and patient education are essential to reduce harm associated with polysubstance use [37,38].

6. Limitations

This review is narrative in nature and is primarily based on data derived from literature reviews, reports from European institutions, and findings from observational and retrospective studies. A key limitation is the absence of comprehensive data from prospective studies, which may

affect the generalizability of the presented conclusions across various clinical settings. Given the dynamic evolution of the psychoactive substance market, including the emergence of novel psychoactive substances (NPS), some information may rapidly become outdated. Furthermore, differences in the availability of toxicological diagnostic tools and local treatment protocols limit the direct applicability of the described therapeutic strategies to all emergency care settings. Sociodemographic factors and psychological or environmental determinants influencing the course of intoxication and therapeutic decisions were not fully addressed in this work.

7. Conclusion

The increasing incidence of intoxications involving psychoactive substances in Europe represents a significant challenge for prehospital and emergency care. Cocaine, amphetamines, cannabinoids (including synthetic variants), and opioids, particularly high-potency analogues such as fentanyl and nitazenes, are associated with diverse clinical presentations and require tailored therapeutic approaches. In settings with limited access to rapid laboratory diagnostics, the recognition of toxidromes and the implementation of supportive care based on emergency medicine principles remain crucial. Pharmacological interventions, such as benzodiazepines or naloxone, should be administered in accordance with current guidelines, while patient monitoring must account for the risk of multiorgan complications. Given the continually evolving landscape of psychoactive substances and the growing prevalence of polysubstance use, further prospective studies and the development of rapid diagnostic tools are essential to enhance the safety and effectiveness of acute intoxication management.

Disclosure

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