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Acute Cocaine Intoxication Leading to Multiorgan Dysfunction: A Case Report on Concealed Cocaine Use

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Short Running Title: cocaine intoxication with systemic complication

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Abstract: ***Introduction**: Cocaine abuse remains a significant global public health concern due to its widespread recreational use. It continues to pose substantial diagnostic challenges because of its unpredictable pharmacologic effects, potential for multisystem involvement, and the frequent concealment of usage by patients. **Case Report**: This case outlines the clinical course of a middle-aged female patient who, following travel to a high-altitude location, presented with fever, cough, dyspnea, and altered mental status. On evaluation she had multiorgan dysfunction—including transaminitis, bicytopenia, acute kidney injury, bilateral lung consolidation, gastrointestinal bleeding, altered sensorium, and mononeuropathy. The initial clinical suspicion was sepsis with multiorgan failure secondary to community acquired pneumonia, as both the patient and her family denied any substance abuse. After ruling out infectious and autoimmune causes, suspicion of cocaine toxicity was raised due to the presence of hyperthermia, accelerated hypertension, mydriasis, and clinical signs indicative of systemic vasoconstriction. A urine toxicology test was strongly positive for cocaine. The patient was managed in the intensive care unit, where she received a combination of antibiotics, steroids, nitroglycerin infusion, hemodialysis, multiple blood product transfusions, and intensive neuropsychiatric rehabilitation. Over the course of four weeks, she demonstrated significant clinical improvement and ultimately returned to her baseline functional status. **Conclusion**: This case report offers valuable prognostic insight into the importance of suspecting cocaine abuse in patients presenting with altered mental status, multiorgan failure, and signs of systemic vasoconstriction.*

Keywords: cocaine, intoxication, drug abuse, multiorgan dysfunction

1. Introduction

Cocaine, chemically known as benzoyl methyl ecgonine is a potent sympathomimetic agent derived from the leaves of the coca plant, Erythroxylum coca (1). Historically, cocaine was used as a topical anesthetic and as an active ingredient to treat depression and malaise, but it is now a commonly abused illegal narcotic (2).

The pathophysiology involves cocaine-induced vasoconstriction resulting in ischemia and tissue necrosis; augmented sympathetic activity through inhibition of monoamine reuptake, leading to elevated levels of dopamine, norepinephrine, and serotonin; prothrombotic effects via enhanced platelet aggregation; and direct cytotoxic effects on various tissues (3). It also antagonizes sodium channels, resulting in impaired nerve conduction. Clinical symptoms may arise hours to days after acute intoxication (4).

Currently there is no antidote for cocaine toxicity and hence aggressive supportive treatment is crucial in the management. This case highlights a complex instance of multi-organ failure, with the initial diagnosis obscured by a concealed history of substance abuse, which was ultimately identified as acute cocaine toxicity. It also outlines the clinical course and the successful management strategy that led to the patient's full recovery.

2. Case Report

A 43-year-old female was brought to the emergency department with complaints of cough with haemoptysis (less than 10 ml), fever, and progressive breathlessness for 5 days, which she developed during her travel to Char Dham, Uttarakhand (nearly 10,800 ft), with her family. Considering the possibility of high-altitude sickness, she was treated with diuretics, nebulization, supplemental oxygen, oral azithromycin (500 mg), and oral deflazacort (6 mg) for three days at Char Dham.

In view of progressive breathlessness and altered mental status they returned to Mumbai. She has no pertinent past medical history and denied any substance abuse. Clinical examination findings on arrival to the emergency department were as follows: Heart rate 104/minute, BP 120/80 mmHg, respiratory rate 28/minute, SpO2 88% on room air, GCS E2V3M4, and respiratory system examination revealed bilateral scattered crepitations. The cardiovascular and

remainder of the clinical examination were normal. After stabilization, she was transferred to the intensive care unit, and treatment was initiated, which included low-flow supplemental oxygen, fluid resuscitation with normal saline, nebulized bronchodilators, ceftriaxone and doxycycline.

Initial lab investigations (Table 1) revealed anemia (Hb 9.8 g/dl), thrombocytopenia (platelet 36,000/dl), leukocytosis (WBC 18,680/dl), acute liver injury (transaminases >7000, total bilirubin 1.43 mg/dl, INR 2.36), acute kidney injury (creatinine 2.84 mg/dl, BUN 39.9 mg/dl, urea 85.3 mg/dl), elevated CRP 98.7 mg/dl, uncompensated metabolic acidosis, elevated LDH (>2500 U/L), and elevated CPK (522 U/L). ECG showed sinus tachycardia, and echocardiography was normal. Chest X-ray was suggestive of an area of consolidation in the right mid-zone and lower zone with perihilar airspace opacification on the left side.
HRCT chest was suggestive of scattered ground glass opacities with confluent consolidation predominantly involving bilateral lower lobes (Figure 1). Our initial impression was sepsis with multiorgan failure secondary to community-acquired pneumonia. As the patient continued to have fever spikes (Tmax 103F), antibiotics were escalated to Meropenem and Teicoplanin. Patient was able to maintain her oxygen saturation with a low flow supplemental oxygen at 2-4 Litre per minutes via nasal cannula. Within 48 hours of hospitalization, she developed oliguria, necessitating furosemide infusion. Systemic workup for infectious etiologies, including blood culture, sputum aerobic culture, urine culture, and BioFire respiratory panel real-time PCR, was inconclusive (Table 2). Due to persistent drowsiness and irrelevant speech, a CT scan of the brain was performed, suggesting mild cerebral edema. In view of encephalopathy, dexamethasone (8mg IV q8h subsequently tapered) was given. The patient required three sessions of hemodialysis over one week as the renal functions further deteriorated. CT abdomen showed features of hepatomegaly with mild ascites. Her sensorium gradually improved; however, she continued to exhibit irrelevant speech and hallucinations. Blood ethanol level was normal. Possible autoimmune causes were ruled out by this time, as the ANA by IF, ANCA, and RA factor tests returned negative results. The patient required multiple transfusions of platelets, fresh frozen plasma (FFP), and packed red blood cells (PRBCs). She developed accelerated hypertension that was refractory to four antihypertensive agents, necessitating the initiation of a nitroglycerin infusion for blood pressure control. Renal artery doppler ruled out renal artery stenosis.

Given that the clinical presentation was inconsistent with septicemia, a multidisciplinary team concluded that, in the context of multi-organ involvement exhibiting vasoconstrictive features and negative autoimmune and infectious workups, drug-induced toxicity should be considered in the differential diagnosis. Urine test for Screening Drugs of Abuse was positive for cocaine and semi quantitative test for cocaine was 1888ng/ml. The patient and the family consistently denied any history of substance abuse, despite repeated counseling sessions and thorough psychological evaluation. Visible markers of drug abuse like IV track marks, inflamed nasal mucosa, perforated nasal septum, or body packing were not found.

In the course of hospitalization, she developed melena with a corresponding drop in hemoglobin to 5.2 g/dL, which warranted transfusion with packed red blood cells. Upper GI endoscopy revealed diffuse mucosal edema, multiple petechiae and Mallory- Weiss tear. She exhibited residual weakness in the right upper limb, with muscle strength rated at 3/5. Nerve conduction study of right upper limb demonstrated attenuated muscle action potential and sensory action potential.

She required total five sessions of hemodialysis, multiple blood product transfusion along with neuropsychiatric rehabilitation and aggressive conservative management. The patient was discharged after four weeks of care, with significant improvement and return to near-baseline function. Surprisingly, it wasn't until her follow-up visit a month later that she broke her silence—admitting to vaping and snorting cocaine, a revelation that reframed the entire case.

3. Discussion

According to the *National Survey on Extent and Pattern of Substance Use in India* (2018), the prevalence of cocaine use was estimated at 0.06% (approximately 0.2 million) among individuals aged 10–17 years and 0.11% (approximately 1 million) among those aged 18 years and above (5). Furthermore, data from the National Crime Records Bureau (NCRB) reported 704 drug overdose-related deaths in 2019 (6). Although these figures suggest a relatively low burden of cocaine use and associated mortality in India, the true prevalence is likely underestimated due to underreporting and limitations in national surveillance systems.

Clinical presentation of cocaine overdose is highly variable ranging from mild autonomic hyperactivity to multiorgan failure and even death. Acute cocaine-induced multiorgan dysfunction is a rare but potentially life-threatening condition primarily through intense sympathetic overdrive and widespread vasoconstriction, resulting in ischemia, oxidative stress, and direct cellular toxicity across multiple organ systems (7).

While pulmonary complications such as acute lung injury and hemorrhage are recognized in cocaine users, the bilateral consolidation in this case raised differential considerations including infection, high-altitude pulmonary edema, and aspiration. Diagnostic challenges were heightened by the patient's denial of substance use, as well as the nonspecific nature of presenting symptoms. Interstitial pneumonitis, bronchiolitis obliterans, pulmonary oedema, ARDS, thermal injury, bullous lung disease, hilar lymphadenopathy, organizing pneumonia, and pulmonary hypertension are some of the other reported pulmonary complications that are related to direct lung injury from crack cocaine or its associated substances (such as talc, lactulose, mannitol, sucrose, heroin, silica, flour, etc) that are inhaled with it (8).

Encephalopathy was initially considered secondary to septic and metabolic causes. Classic neurological presentations of acute cocaine toxicity include intracerebral and subarachnoid hemorrhage, cerebral vasculitis, optic neuropathy, stroke, neuropsychiatric sequelae such as agitation, akathisia, formication and other hallucinations. It may induce reversible

cerebral vasoconstriction syndrome, posterior reversible encephalopathy syndrome (PRES), thunderclap headaches, and seizures through vascular mechanisms(4,9). Effects of cocaine toxicity, including coma, leukoencephalopathy, neurogenic stunned myocardium, transaminitis, acute kidney injury, rhabdomyolysis, and mononeuropathy, are well-documented in the literature, as demonstrated by Hong and Kromm in a case involving a 23-year-old polysubstance abuser (9).

The causes of acute kidney injury associated with cocaine toxicity include rhabdomyolysis, thrombotic microangiopathy, vasculitis, acute interstitial nephritis, and renal infarction(10). Five sessions of hemodialysis, in addition to other supportive measures, successfully restored our patient's renal function to normal.

The persistence of hyperthermia despite declining inflammatory markers, along with the presence of accelerated hypertension, prompted us to reconsider sepsis as the primary diagnosis and explore alternative etiologies. The negative autoimmune markers, ongoing delirium and hallucinations, along with systemic signs of vasoconstriction and sympathomimetic overdrive, led us to suspect that while the patient might conceal information, the laboratory results would provide clarity, compelling us to conduct a drug abuse screening test.

The common cardiovascular effects associated with acute cocaine intoxication are tachycardia, hypertension, arrhythmia and chest pain whereas long term intoxication can rarely cause coronary artery disease, heart failure, aortic dissection, myocardial infarction and endocarditis(7). Neurological complications of acute cocaine toxicity include intracerebral and subarachnoid hemorrhage, cerebral vasculitis, optic neuropathy, stroke, neuropsychiatric sequelae such as agitation, akathisia, formication and other hallucinations. It may induce reversible cerebral vasoconstriction syndrome, posterior reversible encephalopathy syndrome (PRES), thunderclap headaches, and seizures through vascular mechanisms (4, 9).

Acute cocaine intoxication causes liver toxicity, which is characterized by a disproportionate increase in lactate dehydrogenase (LDH) and aminotransferases relative to alkaline phosphatase as seen in our patient. Hyperbilirubinemia appears two to three days following cocaine ingestion, which usually resolves in one to two weeks (11).

Cocaine typically exists in two forms: cocaine hydrochloride, which is a white powder, and 'crack' cocaine, which is the freebase form (1). It is primarily metabolized in the liver by esterases and cytochrome P450 enzymes with a half-life of around 1 hour, but its metabolites can remain detectable in urine for several days (12). In this case cocaine was detected in blood after 7 days from symptom onset. Although urine drug quantification cannot precisely determine the amount of cocaine consumed due to pharmacokinetic variability, the markedly elevated levels strongly indicate significant or heavy substance use. The family confirmed that the patient was in their presence throughout the journey, effectively ruling out the possibility of surreptitious drug administration or substance adulteration.

Patient reluctance to reveal substance use is a major diagnostic challenge that often delays necessary evaluation and treatment. This case highlights the importance of considering cocaine toxicity in patients presenting with unexplained multiorgan dysfunction, especially when conventional causes such as infections or autoimmune disorders are ruled out. The patient's clinical improvement with timely supportive management, including hemodialysis and neuropsychiatric care, emphasizes the potential for recovery even in severe cases of acute cocaine intoxication. Clinicians should maintain a high index of suspicion for drug-induced toxicity, particularly in cases with altered mental status and signs of systemic vasoconstriction, as early diagnosis and intervention are key to improving outcomes.

4. Conclusion

This case underscores the diagnostic complexity and potentially fatal nature of acute cocaine intoxication, particularly when the history of substance use is concealed. Cocaine's multisystem effects—mediated through intense vasoconstriction, sympathetic overactivity, and direct cellular toxicity—can mimic infectious or autoimmune etiologies, leading to diagnostic delay. A high index of suspicion, thorough multidisciplinary evaluation, and prompt supportive management are crucial for favorable outcomes. Clinicians should consider cocaine toxicity in patients presenting with unexplained multiorgan dysfunction and neuropsychiatric manifestations, even in the absence of an evident history of drug use. Early recognition and aggressive supportive therapy can be lifesaving and facilitate complete recovery, as demonstrated in this case.

Conflict of Interest
Nil

Author's Contributions

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GROUP 1: Conception of the work **AND/OR** Design of the work **AND/OR** Acquisition of data **AND/OR** Analysis of data **AND/OR** Interpretation of data
GROUP 2: Drafting the work **AND/OR** Revising the work critically for important intellectual content
GROUP 3: Final approval of the version to be published
GROUP 4: Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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GROUP 4: Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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References

[1] Roque Bravo R, Faria AC, Brito-da-Costa AM, Carmo H, Mladěnka P, Dias da Silva D, et al. Cocaine: An Updated Overview on Chemistry, Detection, Biokinetics, and Pharmacotoxicological Aspects including Abuse Pattern. Toxins. 2022 Apr 13;14(4):278.
[2] Brain PF, Coward GA. A Review of the History, Actions, and Legitimate Uses of Cocaine. J Subst Abuse. 1989 Jan 1;1(4):431–51.
[3] Goel N, Pullman JM, Coco M. Cocaine and kidney injury: a kaleidoscope of pathology. Clin Kidney J. 2014 Dec 1;7(6):513–7.
[4] Douglas SL, D'Arsigny CL. Cocaine Toxicity in the Intensive Care Unit. 2020;
[5] SURVEY ON ADDICTION OF DRUGS [Internet]. [cited 2025 Apr 14]. Available from: https://pib.gov.in/pib.gov.in/Pressreleaseshare.aspx?PRID=1985911
[6] Deaths Due to Drug Abuse [Internet]. [cited 2025 Apr 14]. Available from: https://pib.gov.in/pib.gov.in/Pressreleaseshare.aspx?PRID=1744800
[7] Jr JVP, Magnusson P, LeQuang JAK, Breve F, Varrassi G, Jr JP, et al. Cocaine and Cardiotoxicity: A Literature Review. Cureus [Internet]. 2021 Apr 20 [cited 2024 Dec 17];13(4). Available from: https://www.cureus.com/articles/55486-cocaine-and-cardiotoxicity-a-literature-review
[8] Restrepo CS, Carrillo JA, Martínez S, Ojeda P, Rivera AL, Hatta A. Pulmonary Complications from Cocaine and Cocaine-based Substances: Imaging Manifestations1. RadioGraphics [Internet]. 2007 Jul 1 [cited 2024 Dec 18]; Available from: https://pubs.rsna.org/doi/10.1148/rg.274065144
[9] Hong ZM, Kromm JA. Neurological and systemic effects of cocaine toxicity: A case report and review of the literature. Med Int. 2024 Oct 8;4(6):72.
[10] Filho JCCL, Ogawa MY, de Souza Andrade TH, de Andrade Cordeiro Gadelha S, Fernandes PFCBC, Queiroz AL, et al. Spectrum of acute kidney injury associated with cocaine use: report of three cases. BMC Nephrol. 2019 Mar 20;20:99.
[11] LiverTox: Clinical and Research Information on Drug-Induced Liver Injury [Internet]. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases; 2012 [cited 2024 Dec 18]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK547852/
[12] Jastrzębska J, Daniel WA. Cocaine-Induced Time-Dependent Alterations in Cytochrome P450 and Liver Function. Int J Mol Sci. 2023 Jan 13; 24(2):1632.

Tables

Table 1: Laboratory Investigations

Category	Investigations	Day 1	Day 5	Day 10	Day 14	Day 20	Day 28	Reference Range
Hematological	Hemoglobin (g/dL)	9.8	9.3	9.0	5.2	9.0	8.7	12-15
	Total leukocyte count (×10 3 /µL)	18.68	14.50	18.21	12.04	10.04	6.46	4-11
	Platelet (×10 3 /µL)	36	55	93	125	261	251	150-410
Inflammatory	CRP[mg/dL]	98	35	8.19	12	-	-	0-5
	Procalcitonin	0.52	-	-	-	-	-	0-0.5
Hepatic	SGOT (U/L)	>7000.0	1564	354	80.2	71.7	34	10-35.0
	SGPT (U/L)	>7000.0	2371	373	115	322	35	10-35.0
	GGT (U/L)	99.9	96.5	-	144	-	-	5-36.0
	Total bilirubin (mg/dL)	1.43	1.26	-	0.33	-	-	0.30-1.20
	ALP (U/L)	114.0	84.3	-	-	110	-	35.0-104.0
Renal	BUN (mg/dL)	39.9	87.9	109.4	79.9	100	-	6-20.0
	Serum creatinine (mg/dL)	2.84	5.25	6.58	5.73	3.4	0.89	0.51-0.95
Cardiac	hstrop I (ng/L)	14.9	-	-	-	-	-	0.0 - 16.0
	NT proBNP (pg/ml)	2736	-	-	-	-	-	5 -125

Abbreviations: (ALP: Alkaline Phosphatase; GGT: Gamma-Glutamyl Transferase; INR: International Normalized Ratio; PT: Prothrombin Time; SGOT: Serum Glutamic-Oxaloacetic Transaminase; SGPT: serum glutamic-pyruvic transaminase; BUN (Blood Urea Nitrogen); APTT: Activated Partial Thromboplastin Time; CRP: C-Reactive Protein; CPK: Creatine Phosphokinase; LDH: Lactate Dehydrogenase)

Table 2: Diagnostic Workup for Infectious and Autoimmune Causes

Tropical Fever Real-time PCR Panel	No organism detected
Nasopharyngeal swab in VTM: Biofire respiratory realtime PCR panel	Not detected
Rapid Malarial Antigen Test	Negative
Dengue NS-1, Dengue IgM & IgG:	Negative
Hepatitis A,B,C,E serology	Negative
Mycoplasma IgM	Negative
Blood culture	Negative
Urine culture	Negative
HIV antibody	Non reactive

Figure Legends

Figure 1: HRCT chest axial cut lung window shows centrilobular and tree-in-bud nodules in the right upper and middle lobes, subpleural ground-glass opacities with septal thickening in both upper lobes and the lingula, and perihilar ground-glass opacities with confluent consolidations and diffuse septal thickening in both lower lobes. Mild right pleural effusion is also noted.

Figures

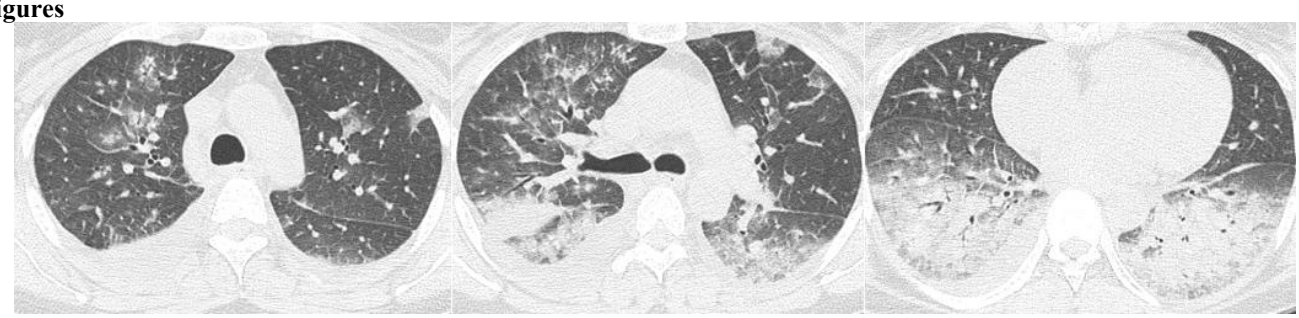

Figure 1: HRCT chest Plain