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Cocaine-Induced Severe Rhabdomyolysis with Acute Kidney Injury: A Case Report of Massively Elevated Creatine Kinase Exceeding 1,000,000 U/L

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Abstract

Cocaine-associated rhabdomyolysis is a serious complication of stimulant intoxication characterized by skeletal muscle injury with the release of intracellular contents into the circulation, potentially resulting in acute kidney injury (AKI), severe electrolyte abnormalities, and other life-threatening complications. Fentanyl was also detected, but its contribution is difficult to determine.

A 33-year-old male with a history of polysubstance use disorder presented after being found unresponsive on the floor with a posterior scalp laceration. He was combative during transport and arrived confused and obtunded. Urine drug screening was positive for cocaine and fentanyl (presumptive, unconfirmed); serum ethanol was 19 mg/dL. Initial laboratory evaluation demonstrated severe rhabdomyolysis with creatine kinase (CK) of 67,705 U/L, rising to a peak of 1,233,505 U/L on hospital day 2; pigmenturia suggestive of myoglobinuria; hyperkalemia (8.3 mEq/L); hypocalcemia (5.6 mg/dL); hyperphosphatemia (9 mg/dL); high anion gap metabolic acidosis (anion gap 21 mEq/L, lactate 18.8 mmol/L); and stage 3 AKI, with creatinine of 1.3 mg/dL on arrival, peaking at 5.7 mg/dL on hospital day 11. Electrocardiography demonstrated peaked T-wave morphology consistent with hyperkalemia. Emergency management included intravenous calcium gluconate, insulin-dextrose therapy, sodium bicarbonate, fluid resuscitation, and emergent hemodialysis. Hemoadsorption therapy for myoglobin removal was considered but was unavailable. After improvement in mental status, left foot drop was recognized on hospital day 4. Evaluation revealed acute compartment syndrome (ACS) of the left lower extremity, requiring emergent anterior and lateral fasciotomy followed by multiple wound washouts, serial debridement, and wound vacuum-assisted closure exchanges. The patient was ultimately discharged on outpatient hemodialysis three times weekly, with wound care, surgical follow-up, and referral for substance use disorder counseling and harm reduction services. Renal recovery was documented about 12 weeks after presentation.

This case highlights an extreme presentation of cocaine-associated rhabdomyolysis complicated by profound rhabdomyolysis-associated AKI, life-threatening metabolic derangements, and delayed recognition of ACS. Early recognition, fluid resuscitation, timely renal replacement therapy, and continued neurological surveillance beyond the acute metabolic phase are critical for optimizing outcomes. Clinicians should maintain a high index of suspicion for compartment syndrome and should not rely on classic signs in patients with severe rhabdomyolysis.

Categories: Rheumatology, Internal Medicine
Keywords: acute cocaine intoxication, acute kidney injury, fentanyl co-exposure, hd (hemodialysis), myoglobinuria, rhabdomyolysis and compartment syndrome, severe hyperkalemia

Introduction

Rhabdomyolysis is a clinical syndrome arising from acute disruption of skeletal muscle, leading to disruption of muscle cell membranes and leakage of intracellular constituents, including myoglobin, creatine kinase (CK), potassium, phosphate, and purines, into the systemic circulation [1]. Although mild or atypical cases may go unrecognized, severe rhabdomyolysis carries significant morbidity, most notably acute kidney injury (AKI), electrolyte derangements, and coagulopathy [2,3].

Cocaine intoxication is among the most common toxicological causes of rhabdomyolysis [4,5]. A 2023 meta-analysis reported a pooled incidence of approximately 26.6% in cocaine-intoxicated patients [4]. These cases are driven by sympathomimetic vasoconstriction, hyperthermia, agitation, direct myotoxicity, and the frequent contribution of prolonged immobility or trauma [5,6]. Fentanyl is increasingly detected alongside stimulants [7] and may contribute through hypoxia and immobilization [8]; however, its contribution in an individual patient is often unclear.

Acute compartment syndrome (ACS) may coexist with or develop as a complication of severe

rhabdomyolysis. ACS results from elevated intracompartmental pressures that impair perfusion, classically following trauma, but nontraumatic cases, including those related to drug use, prolonged compression, or metabolic injury, have been described [9-11]. Cocaine-associated ACS in the absence of significant external trauma is particularly uncommon, posing diagnostic challenges and increasing the risk of delayed intervention, which can lead to irreversible neurovascular injury or limb loss [10-12].

Myoglobin released from injured muscle causes kidney injury through renal vasoconstriction, tubular obstruction, and heme-mediated tubular toxicity [13]. Rhabdomyolysis and compartment syndrome can each cause the other: muscle swelling raises compartment pressure, and ischemic compartments release more muscle contents [9,11].

We present a case of cocaine-associated rhabdomyolysis with a peak CK of 1,233,505 U/L, complicated by AKI requiring dialysis and ACS that was recognized once mental status had improved. Because the foot drop may have preceded recognition, we describe delayed recognition rather than delayed onset. We discuss management and teaching points for clinicians caring for patients with stimulant use disorder.

Case Presentation

A 33-year-old male with a known history of polysubstance use disorder presented to the emergency department after bystanders found him unresponsive on the floor. He had an approximately 4-cm posterior scalp laceration consistent with a fall, was combative during transport, and arrived in a confused and obtunded state. On arrival, he was afebrile, tachycardic (122/min), and tachypneic (46/min), with a blood pressure of 119/95 mmHg and oxygen saturation of 98% on room air. When briefly responsive, he reported recent cocaine use, and a strong odor of alcohol was noted.

The urine drug screen was positive for cocaine and fentanyl. This was a presumptive urine immunoassay collected in the ED at presentation, without confirmatory testing. No fentanyl was administered before collection. The source of the fentanyl is unknown, and we make no assumption that the cocaine was adulterated. Serum ethanol was 19 mg/dL, and no opioid toxidrome was documented. Urinalysis showed 3+ blood on dipstick with fewer than 5 red blood cells per high-power field, consistent with pigmenturia and suggestive of myoglobinuria in the setting of severe rhabdomyolysis. Myoglobin was not measured. The urine was dark and cloudy. Initial CK was 67,705 U/L, establishing the diagnosis of rhabdomyolysis.

Presenting laboratory abnormalities included hyperkalemia (8.3 mEq/L), hypocalcemia (total calcium 5.6 mg/dL; albumin 4.1 g/dL; ionized calcium 0.71 mmol/L on hospital day 2, 0.65 mmol/L on day 3, and 0.83 mmol/L on day 4; no symptoms attributable to hypocalcemia were documented), hyperphosphatemia (9 mg/dL), and high anion gap metabolic acidosis (anion gap 21 mEq/L, venous pH 7.10, bicarbonate 11.6 mmol/L). Lactate was 18.8 mmol/L, indicating severe lactic acidosis of undetermined mechanism. It fell to 7.6 mmol/L within about 3 hours, to 4.9 mmol/L by 20:07, and normalized to 1.8 mmol/L on the afternoon of hospital day 2. The patient developed AKI: creatinine was 1.3 mg/dL on arrival, increased to 2.2 mg/dL on hospital day 2, and peaked at 5.7 mg/dL on hospital day 11 (8/14). BUN was 13 mg/dL on arrival. These findings and the initial management are summarized in Table *1*.

Laboratory parameter	Value	Clinical significance and initial intervention
Creatine kinase (initial) (26-174 U/L)	67,705 U/L	Severe rhabdomyolysis confirmed; IV fluid resuscitation initiated; serial monitoring ordered.
Creatine kinase (peak) (26-174 U/L)	1,233,505 U/L (hospital day 2)	Extreme elevation; hemoadsorption considered but unavailable
Creatinine (arrival vs. peak) (0.5-1.1 mg/dL)	1.3 mg/dL on arrival vs. a peak of 5.7 mg/dL (hospital day 11)	Stage 3 AKI (KDIGO); emergent nephrology consultation; hemodialysis initiated
Blood urea nitrogen (6-20 mg/dL)	13 mg/dL on arrival vs. a peak of 59 mg/dL (hospital day 11)	Normal on arrival; rose with AKI
Potassium (3.4-5.1 mEq/L)	8.3 mEq/L	Severe hyperkalemia; IV calcium gluconate (cardiac membrane stabilization), insulin-dextrose, IV sodium bicarbonate; emergent HD
Total calcium (8.6-10.4 mg/dL)	5.6 mg/dL	Hypocalcemia likely related to rhabdomyolysis; IV calcium gluconate given for cardiac stabilization during hyperkalemia; albumin 4.1 g/dL; ionized calcium 0.71 (day 2), 0.65 (day 3), 0.83 mmol/L (day 4).
Phosphorus (2.7-4.5 mg/dL)	9 mg/dL	Hyperphosphatemia in the setting of rhabdomyolysis and AKI; managed with HD
Anion gap (3-14 mEq/L)	21 mEq/L	High anion gap metabolic acidosis; IV bicarbonate; HD
Lactate (0.5-2.2 mmol/L)	18.8 mmol/L	Severe lactic acidosis of undetermined mechanism; the patient was normotensive and normoxic, with no documented seizure; managed with resuscitation and HD.
Urine drug screen	Presumptively positive for cocaine and fentanyl	No hypoxia or opioid toxidrome; fentanyl contribution not established
EKG	Peaked T-waves	Consistent with hyperkalemia; urgent protocol initiated; repeat EKG after intervention

TABLE 1: Laboratory findings at presentation and corresponding initial management.

AKI, acute kidney injury; EKG, electrocardiogram; HD, hemodialysis; IV, intravenous; KDIGO, Kidney Disease: Improving Global Outcomes

The electrocardiogram revealed peaked, hyperacute T waves, supporting the finding of hyperkalemia (Figure *1*). CT of the abdomen showed no acute findings (Figure *2*). Plain radiography of the leg demonstrated nonspecific soft-tissue swelling at the level of the knee joint (Figure *3*).



FIGURE 1: EKG showing tall, peaked T waves with a narrow base in leads V2 and V3, a morphology characteristic of hyperacute T waves in severe hyperkalemia.

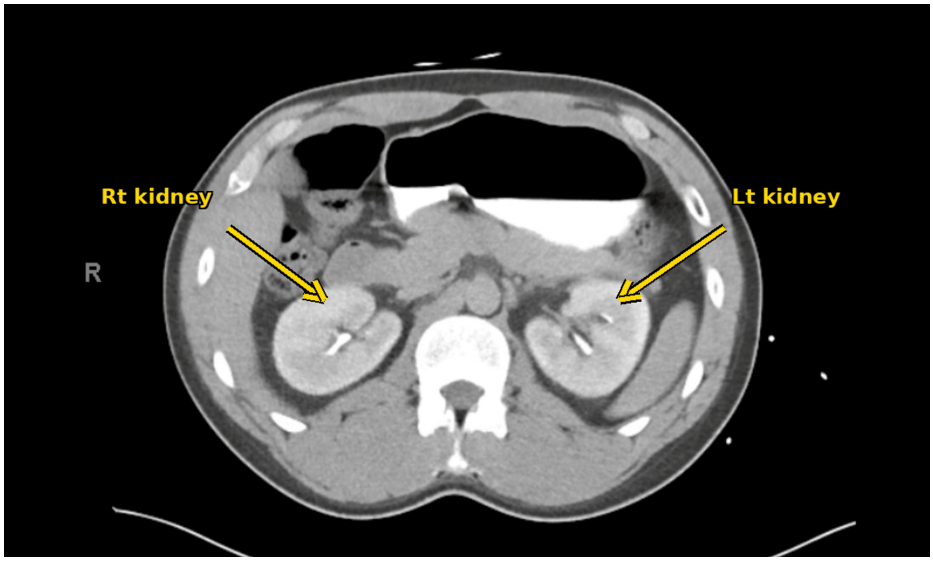


FIGURE 2: Axial CT of the abdomen at the level of the kidneys, showing no acute intra-abdominal or retroperitoneal findings.

Arrows mark the right and left kidneys for anatomical orientation; no focal abnormality is seen.

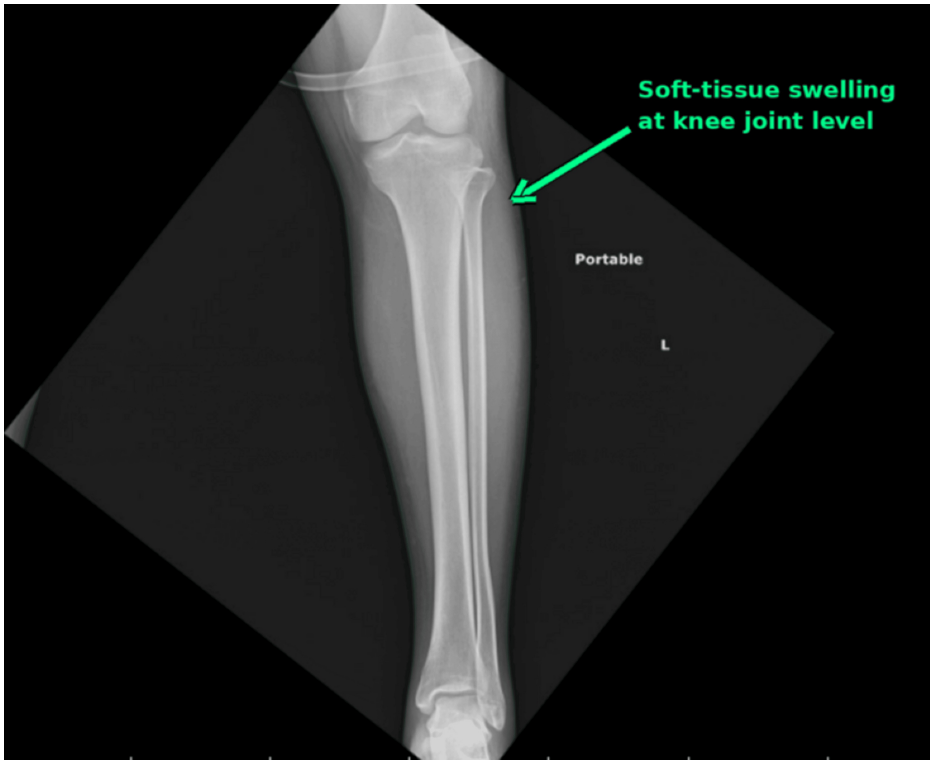


FIGURE 3: Portable anteroposterior (AP) radiograph of the left leg.

The arrow indicates nonspecific soft-tissue swelling at the level of the knee joint/proximal tibiofibular region, without acute fracture or dislocation.

Emergency resuscitation was initiated simultaneously with the diagnostic workup. Intravenous calcium gluconate was administered for cardiac membrane stabilization in the setting of electrocardiographic hyperkalemic changes. A combined insulin-dextrose infusion was initiated for acute transcellular potassium shifting. The presenting venous pH was 7.10, with a bicarbonate level of 11.6 mmol/L. Intravenous sodium bicarbonate was administered for severe acidemia and hyperkalemia, not for urine alkalinization, and was discontinued on hospital day 4 once the serum bicarbonate level had normalized. Despite these temporizing measures, the severity of renal failure, oliguria, and refractory hyperkalemia necessitated the emergent initiation of hemodialysis within hours of presentation.

Potassium fell from 8.3 to 4.7 mEq/L within about 14 hours of the first measurement, rebounded to 6.0 mEq/L on hospital day 2, and then normalized. The patient received about 4 L of crystalloid boluses, followed by 0.9% saline at 200 mL/hour and a sodium bicarbonate infusion (75 mEq in 1 L of 0.45% saline) at 200 mL/hour. Urine output fell to 75 mL per 24 hours on hospital day 3. On hospital day 4, nephrology reduced the saline infusion rate to 75 mL/hour and discontinued the bicarbonate infusion. Weight increased from 88.5 to 92 kg. A bumetanide trial on hospital day 7 produced no urine, and pulmonary edema subsequently developed, requiring up to 4 L/min of oxygen and ultrafiltration during dialysis (14 sessions in total). CK peaked at 1,233,505 U/L on hospital day 2, then fell to 604,010 U/L on day 3 and 136,037 U/L on day 4 before rising again to 238,595 U/L on day 5. Hemoadsorption was considered but was unavailable. Serial values are shown in Table *2*.

Date (hospital day)	CK (U/L)	Creatinine (mg/dL)	BUN (mg/dL)	Potassium (mEq/L)	Lactate (mmol/L)
8/4 (1)	67,705	1.3, 1.9	13, 22	8.3, 7.6, 7.8, 7.2	18.8, 7.6, 4.9
8/5 (2)	1,233,505; 716,155	1.8, 2.2	27-39	6.0, 6.8, 6.2, 4.7, 6.0, 4.1	4.8, 1.8, 2.1
8/6 (3)	604,010	2.3-2.9	21-41	3.5-4.6	2.8, 1.6, 1.2
8/7 (4)	136,037	2.8-2.9	25-26	3.4-3.6	1.4, 1.6
8/8 (5)	238,595	5.3	47	3.8	-
8/9 (6)	112,710	4.1	34	4.3	-
8/10 (7)	46,572	3.7	27	5.5	-
8/11 (8)	25,234	5.4	51	4.1	-
8/12 (9)	12,760	2.8	22	3.4	-
8/13 (10)	7,321	4.2	34	3.9	-
8/14 (11)	-	5.7	59	4.0	-
8/15 (12)	-	4.5	35	3.5	-
8/16 (13)	2,035	3.8	24	3.9	-
8/17 (14)	2,166	3.3	20	4.7	-
8/18 (15)	1,689	5.4	33	4.3	-
8/19 (16)	1,251	4.4	21	4.2	-
8/20 (17)	981	3.7	20	-	-
8/21 (18)	902	5.3	30	-	-
8/22 (19)	755	4.1	20	-	-
8/23 (20)	684	5.5	38	-	-
8/24 (21)	-	3.6	23	-	-
8/25 (22)	-	4.1	25	-	-
8/26 (23)	-	3.9	35	-	-
10/27 (85)	-	0.8	9	4.1	-

TABLE 2: Serial creatine kinase, creatinine, BUN, potassium, and lactate levels.

Creatinine and BUN varied with the timing of hemodialysis sessions.

Mental status had improved by hospital day 2. Physical therapy then observed left foot drop on hospital day 4. The patient reported left leg pain, paresthesias, and lateral leg tenderness, and the left dorsalis pedis pulse was 1+ compared with 2+ on the right. The examination on the morning of hospital day 4 showed no leg asymmetry, and swelling was noted later that day. Radiography showed soft-tissue swelling without fracture.

On surgical evaluation, the left anterior and lateral compartments were tense, the dorsalis pedis pulse was present, and active dorsiflexion was absent. He denied pain at rest, and passive stretch did not elicit pain. Lactate was normal. CK rose from 136,037 to 238,595 U/L over 21 hours. Based on the tense compartments, persistent motor deficit, and rising CK, he underwent left lower leg fasciotomy through anterior and lateral incisions on hospital day 5 (Figure *4*). Compartment pressures were not measured; the diagnosis was made clinically. Washouts, debridement, and wound VAC exchanges followed on hospital days 7 and 10. The full course is summarized in Table *3*.

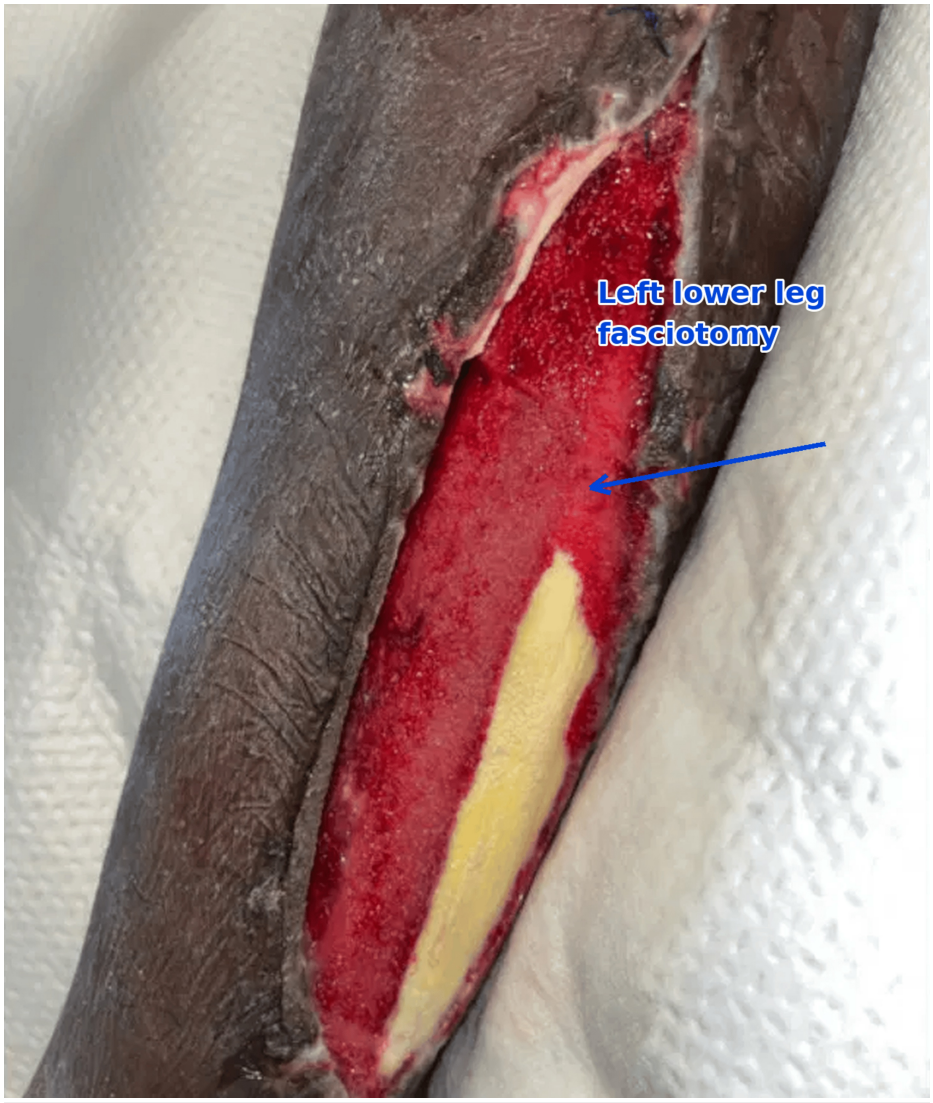


FIGURE 4: Intraoperative photograph following fasciotomy of the left lower leg (anterior and lateral incisions).

The arrow indicates the open fasciotomy wound bed, with exposed, viable-appearing musculature (yellow) and surrounding granulation tissue (red), consistent with decompression of elevated intracompartmental pressures.

Date	Hospital day	Events
8/4	1	Found unresponsive with a scalp laceration; CK 67,705 U/L, K 8.3 mEq/L, lactate 18.8 mmol/L, and pH 7.10; emergent hemodialysis (first session)
8/5	2	Peak CK 1,233,505 U/L (02:31); alert and oriented ×4; persistent oliguria; repeat hemodialysis; potassium rebounded to 6.0 mEq/L; lactate normalized (1.8 mmol/L); ionized calcium 0.71 mmol/L; calcium acetate started
8/6	3	CK 604,010 U/L; AST 7,235 U/L; ALT 1,549 U/L; ionized calcium 0.65 mmol/L; urine output 75 mL/24 hours
8/7	4	Left leg pain and paresthesias, foot drop, and left DP pulse 1+; saline reduced to 75 mL/hour; bicarbonate stopped; dialysis held; CK 136,037 U/L; swelling noted later in the day
8/8	5	CK 238,595 U/L; tense anterior and lateral compartments; absent active dorsiflexion; left leg fasciotomy
8/10	7	Washout, debridement, and wound VAC placement; bumetanide trial without urine output
8/13	10	Repeat washout, debridement, and wound VAC exchange
8/14	11	Peak creatinine 5.7 mg/dL
8/20	17	Tunneled dialysis catheter placed
8/26	23	Creatinine 3.9 mg/dL
8/31	28	Discharged on outpatient hemodialysis (Tuesday/Thursday/Saturday), with a wound VAC and PRAFO boot
10/27	85	Follow-up approximately 12 weeks after presentation; creatinine 0.8 mg/dL, BUN 9 mg/dL, and eGFR >60 mL/min/1.73 m²

TABLE 3: Hospital course timeline.

ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CK, creatine kinase; DP, dorsalis pedis; eGFR, estimated glomerular filtration rate; K, potassium; PRAFO, pressure-relieving ankle-foot orthosis; VAC, vacuum-assisted closure

A tunneled dialysis catheter was placed on hospital day 17. Creatinine was 3.9 mg/dL on hospital day 23. The patient was then stabilized and discharged on hospital day 28. The discharge plan included outpatient hemodialysis three times weekly (Tuesday, Thursday, and Saturday), wound VAC care with surgical follow-up, and referral for substance use disorder counseling and harm reduction services in accordance with current guideline recommendations [14]. An ankle-foot orthosis boot was ordered for the foot drop. At follow-up about 12 weeks after presentation, creatinine was 0.8 mg/dL, with an estimated glomerular filtration rate (eGFR) above 60 mL/min/1.73 m², consistent with renal recovery.

Discussion

This case illustrates the extreme end of the spectrum of stimulant-associated rhabdomyolysis, combining cocaine and alcohol use, massive muscle breakdown, severe metabolic derangements, stage 3 AKI, and delayed recognition of ACS. Several features of this case merit focused discussion.

Pathophysiology of cocaine-associated rhabdomyolysis and contributing factors

Cocaine-associated rhabdomyolysis is driven by multiple overlapping mechanisms: sympathomimetic vasoconstriction induces skeletal muscle ischemia; agitation, hyperthermia, and excessive sympathetic drive increase metabolic demand and promote direct myotoxicity; and trauma or prolonged immobility exacerbates mechanical muscle injury [5,6]. The pooled incidence of rhabdomyolysis among cocaine-intoxicated patients is approximately 26.6% according to large meta-analytic data [4], suggesting that this complication is common.

Fentanyl was also detected on urine screening. Opioid co-exposure can contribute to rhabdomyolysis through respiratory depression, hypoxia, and prolonged immobilization [7,8]. In this patient, however, there was no hypoxia or clinical opioid toxicity on presentation, the screen was presumptive, and cocaine, alcohol, and an unwitnessed fall with unknown down time were each plausible contributors to the muscle injury. We therefore cannot attribute the rhabdomyolysis to fentanyl, although a contribution cannot be excluded. Cocaine is a probable but not the sole cause; alcohol use may also have contributed.

Extreme CK elevation and prognostic limitations

The peak CK of 1,233,505 U/L is markedly elevated. Although CK has historically been used as a surrogate marker for muscle injury severity, emerging data suggest that CK elevation alone may not reliably predict AKI severity or mortality. A 2025 retrospective cohort analysis proposed a multivariable predictive model for AKI in rhabdomyolysis that incorporates additional clinical variables beyond CK, including pH, serum bicarbonate, urine output, and volume status, potentially enabling more nuanced risk stratification in future clinical practice [15]. CK also rose again, from 136,037 to 238,595 U/L, before fasciotomy, suggesting ongoing muscle injury associated with compartment syndrome. In extreme presentations, clinical monitoring of urine output, myoglobin levels when available, and early signs of organ dysfunction remains essential and should not be replaced by CK trending alone..

Management of severe rhabdomyolysis-associated AKI

Management of stimulant-associated rhabdomyolysis rests on goal-directed volume resuscitation, which should be reduced or stopped if oliguria or anuria persists, with reassessment based on weight, examination, and oxygenation. Management also includes control of hyperthermia and agitation and avoidance of nephrotoxins, as reaffirmed by the 2024 ASAM/AAAP guideline [14]. Routine bicarbonate administration to prevent rhabdomyolysis-associated AKI is not supported by strong evidence. Emergent hemodialysis is indicated in oliguric AKI with refractory hyperkalemia or severe acidosis unresponsive to medical therapy [16].

Hemoadsorption for myoglobin removal is an emerging adjunct with limited evidence. The 2024 Hemoadsorption in Rhabdomyolysis Task Force (HRTF) consensus suggests considering it when myoglobin exceeds 10,000 ng/mL or when CK is markedly elevated with ongoing muscle injury, ideally within 24 hours of admission [17]. Myoglobin was not measured in this case, so eligibility is unknown. Hemoadsorption was considered but was unavailable at our facility. Randomized evidence is lacking, and hemoadsorption should supplement, not replace, standard care.

Cocaine-associated kidney injury beyond pigment nephropathy

Cocaine-associated renal injury may manifest through mechanisms independent of myoglobin toxicity. Recent case series have described acute interstitial nephritis (AIN) and acute tubular injury (ATI) in patients with cocaine use, even in the setting of only modest CK elevations, driven by direct nephrotoxicity, interstitial inflammation, and vasoconstriction-mediated ischemia [18]. Myoglobin-mediated AKI in rhabdomyolysis results from several interacting mechanisms, including renal vasoconstriction, tubular obstruction, and direct heme-mediated tubular injury [13]. If creatinine fails to improve despite falling CK and adequate resuscitation, or if urine studies demonstrate persistent abnormalities, further evaluation for non-myoglobin-mediated renal injury is warranted. In this patient, creatinine recovered by about 12 weeks, which is consistent with acute tubular injury related to rhabdomyolysis, although myoglobin was not measured and no biopsy was obtained.

Delayed recognition of ACS

The most instructive feature of this case is the delayed recognition of ACS of the left leg, identified as foot drop once mental status had improved. Non-traumatic ACS in rhabdomyolysis, including cocaine-associated cases, is uncommon and likely underrecognized [9,11,12]. Massive muscle breakdown and edema raise compartment pressure, vasoconstriction compromises perfusion, and altered sensorium from intoxication or encephalopathy can mask pain. In this patient, however, pain with passive stretch was absent despite a normal sensorium, showing that classic signs can be unreliable. A motor deficit, tense compartments, and rising CK prompted surgery.

Neurological surveillance must continue beyond metabolic stabilization. Any new motor deficit, sensory change, or disproportionate pain in severe rhabdomyolysis warrants urgent surgical evaluation. Because encephalopathy may have masked earlier symptoms, the foot drop may have been present before it was noticed, which is why we describe delayed recognition rather than delayed onset. Fasciotomy was performed for presumed ACS.

Public health considerations and discharge planning

The 2024 ASAM/AAAP guideline emphasizes the importance of secondary and tertiary prevention in stimulant use disorder, including patient education on harm reduction strategies, naloxone provision where appropriate, substance use disorder counseling, and linkage to medication-assisted treatment where indicated [14]. These interventions are important components of discharge planning for patients with stimulant-associated toxicity and may help reduce the risk of recurrence.

Limitations

This report has several limitations. The fentanyl result was a presumptive urine immunoassay without confirmatory testing. Myoglobin was not measured, so myoglobinuria is inferred rather than confirmed.

Cocaine cannot be established as the sole cause, given concurrent alcohol use, an unwitnessed fall, and unknown down time. Early fluid administration was followed by volume overload during oliguria, and complete daily fluid balances were not available. Compartment pressures were not measured, and the diagnosis of compartment syndrome was clinical. Long-term wound healing and neurological recovery, including the outcome of the foot drop, are unknown. The date dialysis was discontinued is also unknown.

Conclusions

This case describes cocaine-associated rhabdomyolysis with a peak CK of 1,233,505 U/L in the setting of cocaine and alcohol use, with concurrent fentanyl detection. It was complicated by rhabdomyolysis-associated AKI requiring dialysis, life-threatening electrolyte derangements, and ACS that was recognized late and treated with fasciotomy. Cocaine is a probable, but not the sole, contributor, given concurrent alcohol use and an unwitnessed fall. Kidney function recovered, with a creatinine level of 0.8 mg/dL about 12 weeks after presentation. Several teaching points emerge.

First, heme-positive urine with few red blood cells suggests pigmenturia, often myoglobinuria, and should prompt a rhabdomyolysis workup. Second, CK alone is an imperfect predictor of AKI severity, and pH, urine output, and volume status should inform risk stratification. Third, opioid co-detection should be interpreted cautiously; in this case, there was no evidence that it drove the clinical course. Fourth, hemoadsorption is an investigational adjunct with limited evidence. Fifth, ACS may be recognized late in severe rhabdomyolysis and can be missed if neurological surveillance stops after the metabolic crisis; classic signs, such as pain with passive stretch, may be absent. Finally, discharge planning that includes substance use counseling and harm reduction is an important part of care for these patients.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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