

Severe Amphetamine-Associated Rhabdomyolysis With Dialysis-Requiring Acute Kidney Injury in Two Patients: A Case Report

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Abstract

Amphetamine-associated rhabdomyolysis is an uncommon but potentially life-threatening condition. Massive release of myoglobin and intracellular electrolytes may result in severe metabolic, cardiovascular, and renal complications, particularly acute kidney injury (AKI).

We report two cases of severe amphetamine-associated rhabdomyolysis complicated by dialysis-requiring AKI. The first patient, a 46-year-old man, presented with a serum creatine kinase (CK) level of 947,699 U/L, serum creatinine of 734 μmol/L, hyperkalemia of 6.8 mmol/L, and transient left ventricular systolic dysfunction. Management consisted of supportive therapy and eleven sessions of intermittent hemodialysis. The clinical course was complicated by severe delayed hypercalcemia (4.10 mmol/L), followed by cardiac arrest and death. The second patient, a 42-year-old man, presented with a serum CK level of 525,796 U/L, serum creatinine of 544 μmol/L, and hyperkalemia of 7.5 mmol/L. He received supportive treatment and ten sessions of intermittent hemodialysis, resulting in near-complete recovery of renal function. Urine toxicology screening was positive for amphetamines in both patients. Alternative traumatic and non-traumatic causes of rhabdomyolysis were systematically excluded, supporting amphetamine exposure as the most likely explanation for the rhabdomyolysis, based on the clinical presentation, toxicological findings, and exclusion of alternative causes. Amphetamine-associated rhabdomyolysis is a rare but severe cause of AKI that may require renal replacement therapy.

These cases highlight the heterogeneous clinical course of amphetamine-associated rhabdomyolysis, ranging from renal recovery to fatal metabolic and cardiovascular complications, and emphasize the importance of early toxicological screening, systematic evaluation of alternative causes of rhabdomyolysis, and prompt multidisciplinary management in patients presenting with myalgia, dark urine, and AKI.

Categories: Internal Medicine, Nephrology

Keywords: acute kidney injury, amphetamines, hemodialysis, hypercalcemia, rhabdomyolysis

Introduction

Rhabdomyolysis is a clinical and biochemical syndrome resulting from acute skeletal muscle injury and necrosis, leading to the release of intracellular constituents into the circulation, including myoglobin, creatine kinase (CK), potassium, and other intracellular electrolytes [1,2]. This process may result in myoglobinuria and acute kidney injury (AKI), mainly through direct tubular toxicity, renal vasoconstriction, and intratubular cast formation [1,3].

The clinical presentation of rhabdomyolysis is highly variable, ranging from asymptomatic elevations of muscle enzymes to severe forms associated with hypovolemia, electrolyte disturbances, metabolic abnormalities, and AKI [1,2]. The classic triad of myalgia, muscle weakness, and dark-colored urine is not consistently present. Diagnosis is generally established by a marked elevation of serum CK levels, usually exceeding five times the upper limit of normal [1,2].

Currently, non-traumatic causes account for the majority of rhabdomyolysis cases and include strenuous exercise, alcohol abuse, medications, toxins, illicit drug use, and ischemic muscle injury [1,2]. Early recognition and prompt fluid resuscitation remain the cornerstone of management to prevent or limit renal injury [1,2].

Amphetamines are potent central nervous system stimulants whose sympathomimetic effects may induce hyperthermia, psychomotor agitation, and increased muscular activity, thereby predisposing patients to rhabdomyolysis [4,5]. Massive myoglobin release may subsequently lead to AKI through direct tubular toxicity, intrarenal vasoconstriction, oxidative injury, and tubular obstruction by myoglobin casts [3,6]. In

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addition, amphetamine intoxication may be associated with cardiovascular, neurological, and metabolic complications that may contribute to poor outcomes [4,7]. Because rhabdomyolysis has numerous traumatic and non-traumatic etiologies, establishing amphetamine intoxication as the underlying cause requires careful clinical assessment, toxicological confirmation, and exclusion of alternative causes.

Although amphetamine-associated rhabdomyolysis has been reported, cases complicated by dialysis-requiring AKI, transient myocardial dysfunction, and fatal delayed hypercalcemia remain uncommon.

We report two cases of severe amphetamine-associated rhabdomyolysis complicated by dialysis-requiring AKI. In both patients, urine toxicology screening confirmed amphetamine exposure, while comprehensive clinical evaluation excluded other common causes of rhabdomyolysis, supporting amphetamine exposure as the most likely etiology based on the clinical presentation, toxicological findings, and exclusion of alternative causes. These cases illustrate the broad clinical spectrum and potentially life-threatening complications of amphetamine-associated rhabdomyolysis and emphasize the importance of considering this diagnosis in patients presenting with severe AKI associated with marked CK elevation and no identifiable conventional cause.

Case Presentation

Case 1

A 46-year-old man with no known medical history and no reported history of illicit drug use presented to the Emergency Department with general malaise, marked asthenia, high-grade fever (40°C), dyspnea, and severe diffuse myalgia that had progressively developed over several days. On admission, the patient was conscious with a Glasgow Coma Scale score of 15/15 but experienced episodes of delirium. Vital signs revealed a heart rate of 96 beats/min, blood pressure of 130/90 mmHg, and oxygen saturation of 94% on room air.

Physical examination demonstrated diffuse muscle weakness and dark-colored urine. Abdominal examination revealed diffuse abdominal tenderness without guarding. The patient presented with oliguria without peripheral edema.

Initial laboratory investigations demonstrated severe AKI with a serum creatinine level of 734 $\mu\text{mol/L}$, severe hyperkalemia of 6.8 mmol/L associated with electrocardiographic abnormalities, severe hypocalcemia of 1.30 mmol/L, markedly elevated C-reactive protein (351 mg/L), and markedly elevated serum CK levels reaching 947,699 U/L (normal range: 30–200 U/L). Urine toxicology screening, performed using a multidrug panel, was positive for amphetamines. The patient denied recent strenuous exercise, trauma, prolonged immobilization, seizures, alcohol binge drinking, and the use of statins or other myotoxic medications. Comprehensive clinical evaluation revealed no evidence of other identifiable traumatic or non-traumatic causes of rhabdomyolysis, supporting amphetamine exposure as the most likely etiology.

Renal ultrasonography demonstrated kidneys of normal size and echogenicity without hydronephrosis. Cardiac evaluation at admission revealed elevated cardiac biomarkers, including high-sensitivity cardiac troponin T levels of 62 ng/L and 104 ng/L (reference range: <14 ng/L) and an NT-proBNP level of 4,120 pg/mL (reference range: <125 pg/mL).

Transthoracic echocardiography demonstrated a nondilated left ventricle with anterior and anteroseptal hypokinesia associated with moderately impaired left ventricular systolic function, with a left ventricular ejection fraction (LVEF) of 47%. Left ventricular filling pressures were not elevated. Right ventricular systolic function was preserved, and no pericardial effusion was detected.

Because of oligoanuric AKI complicated by life-threatening hyperkalemia and electrocardiographic abnormalities, emergency renal replacement therapy was immediately initiated. The patient received aggressive intravenous hydration with isotonic saline and underwent a total of eleven intermittent hemodialysis sessions.

Serum CK levels progressively decreased from 947,699 U/L on day 1 to 630,672 U/L on day 2 and to 4,310 U/L on day 10. Despite this biochemical improvement, renal function remained severely impaired, with serum creatinine levels of 813 $\mu\text{mol/L}$ on day 10 and 1,017 $\mu\text{mol/L}$ on day 12, in the setting of persistent anuria requiring ongoing hemodialysis.

The biological evolution is summarized in Table 1.

Parameter	Unit	Hospital day 1	Hospital day 2	Hospital day 10	Hospital day 12	Hospital day 28	Reference range
Creatinine	μmol/L	734	751	813	1017	-	64-110
Urea	g/L	1.54	1.60	2.67	2.47	-	0.15-0.45
Potassium	mmol/L	6.8	6.0	4.8	4.5	-	3.5-5.2
Corrected calcium	mmol/L	1.30	1.25	1.80	1.93	4.10	2.10-2.55
CK	U/L	947,699	630,672	4,310	2,742	-	30-200
CRP	mg/L	351	-	12.4	25	-	<5
Hemoglobin	g/dL	-	-	-	10.9	-	13-18
Hematocrit	%	54	-	-	33	-	40-54
Platelets	×10 ⁹ /L	152	-	-	145	-	150-450
AST	U/L	-	-	90	53	-	5-34
ALT	U/L	-	-	98	60	-	0-55

TABLE 1: Laboratory findings during hospitalization in Case 1
CK, creatine kinase; CRP, C-reactive protein; AST, aspartate aminotransferase; ALT, alanine aminotransferase

A follow-up transthoracic echocardiogram performed before the subsequent complication demonstrated complete recovery of left ventricular systolic function, with an LVEF of 57% and no residual regional wall motion abnormalities.

On hospital day 28, the patient developed severe corrected hypercalcemia (4.10 mmol/L), occurring after the profound hypocalcemia observed during the initial phase of rhabdomyolysis. Computed tomography and an extensive etiological work-up for hypercalcemia were performed. Imaging revealed diffuse muscular calcifications consistent with metastatic muscular calcinosis secondary to severe rhabdomyolysis (Figure 1).

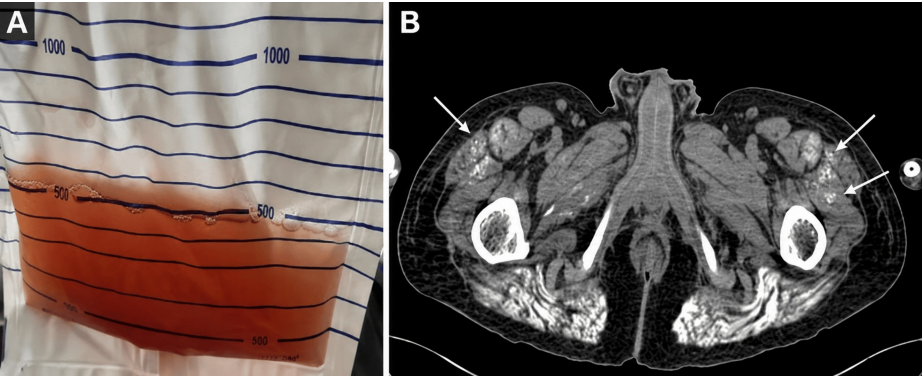


FIGURE 1: Clinical and radiological findings in Case 1
(A) Dark-brown urine at presentation, suggestive of myoglobinuria secondary to severe rhabdomyolysis.
(B) Axial pelvic computed tomography showing diffuse bilateral muscular calcifications (arrows), consistent with muscular calcinosis secondary to severe rhabdomyolysis.

Despite aggressive medical management, including intravenous hydration, corticosteroid therapy, zoledronic acid administration, and subsequent recovery of diuresis, the patient developed cardiac arrhythmia three days later, followed by refractory cardiac arrest despite advanced resuscitation measures, resulting in death.

Case 2

A 42-year-old man with a history of asthma treated with inhaled β_2 -agonists and no reported history of illicit drug use was admitted to our Nephrology Department after referral from a peripheral hospital for the management of severe rhabdomyolysis complicated by AKI.

The illness had begun 20 days before admission with a flu-like syndrome characterized by fever (38.6°C), severe diffuse myalgia, dark-colored urine, and progressive deterioration of the general condition. The clinical course subsequently worsened with oliguria, confusion, dyspnea, and generalized edema, prompting hospitalization at a peripheral hospital, where laboratory investigations revealed AKI, severe hyperkalemia (7.5 mmol/L), rhabdomyolysis, and positive urine toxicology for amphetamines. The patient underwent six sessions of intermittent hemodialysis before being transferred to our institution because of persistent anasarca, anuria, and ongoing renal failure despite initial management.

Upon admission, the patient was fully conscious with a Glasgow Coma Scale score of 15/15. Blood pressure was 137/71 mmHg, heart rate was 79 beats/min, and oxygen saturation was 85% on room air, improving with supplemental oxygen at 3 L/min.

Physical examination revealed generalized edema and bilateral pulmonary crackles. Urine output was estimated at 800 mL/24 h.

Laboratory investigations demonstrated severe rhabdomyolysis with markedly elevated serum CK levels of 525,796 U/L, 521,848 U/L, and 192,509 U/L (normal range: 30–200 U/L), AKI with an initial serum creatinine level of 544 μ mol/L that subsequently increased to 875 μ mol/L, and severe hyperkalemia (7.5 mmol/L). The phosphocalcic profile demonstrated a corrected serum calcium level of 2.17 mmol/L and vitamin D deficiency, with a 25-hydroxyvitamin D concentration of 9.5 ng/mL (reference range: 30–70 ng/mL).

Urine toxicology screening, performed using a multidrug panel at the referring hospital, was positive for amphetamines. The patient denied recent strenuous exercise, trauma, prolonged immobilization, seizures, alcohol binge drinking, and the use of statins or other myotoxic medications. Comprehensive clinical evaluation excluded the common traumatic and non-traumatic causes of rhabdomyolysis. Although concomitant pulmonary and urinary tract infections were documented and treated appropriately, the marked elevation in serum CK levels together with the positive toxicological findings supported amphetamine exposure as the most likely etiology of rhabdomyolysis.

Renal ultrasonography demonstrated kidneys of normal size with preserved corticomedullary differentiation and no evidence of urinary tract obstruction.

Chest radiography showed findings consistent with interstitial pulmonary edema. Computed tomography pulmonary angiography excluded pulmonary embolism but revealed left basal pneumonia associated with bilateral bronchiectasis and moderate bilateral pleural effusions. Transthoracic echocardiography demonstrated preserved left ventricular systolic function with an LVEF of 65%.

Management consisted of individualized intravenous fluid therapy, oxygen supplementation, ceftriaxone, levofloxacin, prophylactic anticoagulation, and intermittent hemodialysis. A total of 10 intermittent hemodialysis sessions were required during hospitalization. The clinical course was marked by transient worsening of renal function, with a peak serum creatinine level of 1,096 μ mol/L associated with volume overload.

Progressive clinical and biochemical recovery was subsequently observed, with improvement in urine output beginning on day 10, regression of edema, and gradual normalization of serum CK levels to 601 U/L and subsequently 420 U/L.

At discharge, serum creatinine had decreased to 521 μ mol/L, and hemodialysis had been discontinued. The biological evolution is summarized in Table 2.

Parameter	Unit	Admission to our department	Peak value during hospitalization	At discharge	1-month follow-up	Reference range
Creatinine	μmol/L	544	1096	521	133	64-110
Urea	g/L	1.28	2.13	1.80	-	0.15-0.45
Potassium	mmol/L	7.5	5.8	4.4	-	3.5-5.2
Corrected calcium	mmol/L	2.17	-	-	-	2.10-2.55
CK	U/L	525,796	521,848	420	-	30–200
CRP	mg/L	82	93	34	-	<5
Hemoglobin	g/dL	8.9	-	10.2	-	13-18
Platelets	×10 ⁹ /L	178	-	-	-	150-450
AST	U/L	90	114	-	-	5-34
ALT	U/L	99	55	-	-	0-55

TABLE 2: Laboratory findings during hospitalization and follow-up in Case 2

CK, creatine kinase; CRP, C-reactive protein; AST, aspartate aminotransferase; ALT, alanine aminotransferase

The patient underwent a total of 10 intermittent hemodialysis sessions, including six sessions before transfer and four sessions in our department. Progressive renal recovery was observed during follow-up.

At the one-month follow-up visit, serum creatinine had decreased to 133 μmol/L, indicating near-complete recovery of renal function without the need for further renal replacement therapy.

Diagnostic assessment

In both cases, the absence of an initially disclosed history of illicit drug use represented the principal diagnostic challenge. The diagnosis was established only after multidrug urine toxicology screening demonstrated amphetamine exposure. Because rhabdomyolysis has numerous traumatic and non-traumatic etiologies, a systematic diagnostic evaluation was undertaken to exclude more common causes, including trauma, strenuous exercise, prolonged immobilization, seizures, alcohol binge drinking, and medication-induced myotoxicity.

In the second patient, the presence of concomitant pulmonary and urinary tract infections represented an additional diagnostic challenge. However, the extreme elevation of serum CK levels, positive toxicological findings, and exclusion of other common causes supported amphetamine exposure as the most likely etiology.

These cases emphasize the importance of considering toxicological screening in patients presenting with severe rhabdomyolysis and AKI when no obvious cause is identified.

Discussion

Our two cases illustrate the broad clinical spectrum of severe amphetamine-associated rhabdomyolysis complicated by dialysis-requiring AKI. Despite similar initial biochemical severity, including exceptionally high serum CK concentrations and life-threatening electrolyte abnormalities, the clinical outcomes differed markedly, ranging from near-complete renal recovery in one patient to fatal evolution in the other. In addition to dialysis-requiring AKI, both patients presented uncommon features, including CK concentrations approaching one million U/L, while the first patient developed transient left ventricular systolic dysfunction, extensive muscular calcifications, and severe delayed hypercalcemia. These observations highlight the potential severity and multisystem involvement of amphetamine-associated rhabdomyolysis.

Establishing amphetamine exposure as the underlying etiology of rhabdomyolysis represented the principal diagnostic challenge in both cases because neither patient initially disclosed illicit drug use. The diagnosis was supported by positive multidrug urine toxicology screening, compatible clinical manifestations, and systematic exclusion of the most common traumatic and non-traumatic causes of rhabdomyolysis, including trauma, strenuous exercise, prolonged immobilization, seizures, alcohol binge drinking, and medication-

induced myotoxicity. In the second patient, concomitant pulmonary and urinary tract infections represented potential confounding factors. However, although bacterial infections have been recognized as occasional triggers of rhabdomyolysis, the exceptionally high CK concentrations observed in our patient, together with positive amphetamine toxicology and the absence of other established etiologies, strongly supported amphetamine intoxication as the most plausible cause of rhabdomyolysis, while the documented infections may have contributed to the severity of the clinical presentation rather than representing the primary cause of rhabdomyolysis [1,4,7].

Amphetamines induce rhabdomyolysis through several complementary mechanisms. Excessive sympathetic stimulation results in hyperthermia, psychomotor agitation, increased muscular activity, peripheral vasoconstriction, dehydration, oxidative stress, mitochondrial dysfunction, and direct myocyte toxicity, all of which contribute to skeletal muscle necrosis and the massive release of intracellular constituents, including myoglobin, CK, potassium, and phosphate [4,7]. Myoglobin-mediated renal injury subsequently develops through intrarenal vasoconstriction, oxidative tubular injury, direct tubular toxicity, and intratubular cast formation, explaining the frequent occurrence of severe AKI in patients with massive rhabdomyolysis [1,3,6].

Severe rhabdomyolysis is one of the leading causes of pigment-induced AKI and is associated with substantial morbidity and mortality. Previous studies have consistently shown that the risk of AKI increases with the severity of muscle injury, particularly in patients with markedly elevated CK levels, persistent oliguria, electrolyte disturbances, and metabolic complications [1,3]. Both of our patients fulfilled these severity criteria and required intermittent hemodialysis because of oligoanuric AKI complicated by life-threatening hyperkalemia. Nevertheless, their renal outcomes differed considerably. While the second patient experienced progressive renal recovery with complete discontinuation of dialysis, the first patient remained dialysis-dependent until death. These contrasting outcomes emphasize that the prognosis of rhabdomyolysis depends not only on the degree of muscle injury but also on the occurrence of systemic complications, associated organ dysfunction, and the patient's overall clinical course.

One of the most remarkable findings in the first patient was the development of severe delayed hypercalcemia following profound hypocalcemia during the acute phase of rhabdomyolysis. Hypocalcemia is frequently observed early in the disease because calcium is deposited within damaged skeletal muscle. During recovery, progressive mobilization of these calcium deposits back into the circulation may result in delayed hypercalcemia, particularly in patients with extensive muscle necrosis and prolonged AKI [2,3]. In our patient, computed tomography demonstrated diffuse muscular calcifications, providing radiological support for this mechanism. Because of the severity of hypercalcemia, an extensive etiological investigation was undertaken, including endocrine and hematological evaluation. Although the diagnostic work-up could not be completed because of the patient's fatal outcome, the temporal sequence of profound hypocalcemia followed by delayed severe hypercalcemia together with diffuse muscular calcifications strongly suggested that rhabdomyolysis was the principal mechanism underlying the delayed hypercalcemia. This observation highlights the importance of continued monitoring of calcium metabolism during the recovery phase of severe rhabdomyolysis, particularly in patients requiring prolonged renal replacement therapy.

Cardiac involvement represented another noteworthy feature of the first case. The patient presented with elevated cardiac biomarkers and transient left ventricular systolic dysfunction, which completely resolved during hospitalization. Amphetamine-associated cardiovascular toxicity has been well documented and includes reversible cardiomyopathy, stress-induced cardiomyopathy, Takotsubo syndrome, malignant arrhythmias, and acute coronary vasospasm [8,9]. Several mechanisms have been proposed, including excessive catecholamine release, coronary vasoconstriction, oxidative stress, mitochondrial dysfunction, and direct myocardial toxicity [8,9]. The precise cause of the fatal cardiac arrest could not be established from the available data. Although the event occurred in the setting of severe delayed hypercalcemia and previous transient left ventricular systolic dysfunction, a direct causal relationship between these abnormalities and the cardiac arrest cannot be confirmed. A multifactorial contribution of metabolic disturbances and possible amphetamine-associated cardiotoxicity cannot be excluded.

Compared with previously published reports, our cases highlight several uncommon and clinically relevant features. First, both patients presented with exceptionally high CK concentrations, exceeding 500,000 U/L, with one reaching nearly one million U/L, a finding only rarely reported in amphetamine-associated rhabdomyolysis [4,5]. Second, both developed severe AKI requiring intermittent hemodialysis. Third, the first patient experienced transient left ventricular systolic dysfunction with complete recovery, diffuse muscular calcifications, and severe delayed hypercalcemia (4.10 mmol/L), followed by cardiac arrest and death. In contrast, the second patient achieved near-complete renal recovery despite profound rhabdomyolysis and severe hyperkalemia. The educational value of these cases lies in illustrating the markedly different clinical trajectories that may occur in severe amphetamine-associated rhabdomyolysis, as well as the potential for delayed, life-threatening metabolic and cardiovascular complications. These observations emphasize the importance of continued monitoring of calcium homeostasis and cardiac function throughout the clinical course, including after initial stabilization. Although these individual complications have been described previously, their presentation alongside exceptionally severe muscle injury and contrasting outcomes provides a useful clinical perspective.

From a clinical perspective, our observations underscore several practical implications. First, amphetamine intoxication should be considered in the differential diagnosis of severe rhabdomyolysis, particularly in younger patients presenting with marked CK elevation and AKI without an obvious traumatic or medical cause. Because patients may not voluntarily disclose illicit drug use, early multidrug urine toxicology screening can substantially facilitate etiological diagnosis. Second, patients with severe rhabdomyolysis should undergo close monitoring for metabolic complications, including delayed disturbances in calcium homeostasis, even after improvement in muscle enzyme levels. Finally, management should extend beyond aggressive fluid resuscitation and include careful assessment of electrolyte abnormalities, cardiac involvement, and the timely initiation of renal replacement therapy when indicated.

Overall, these cases expand the limited literature on severe amphetamine-associated rhabdomyolysis and emphasize the importance of early toxicological screening, multidisciplinary management, and close monitoring for delayed metabolic and cardiovascular complications.

Strengths and limitations

The present report has several strengths. It provides a detailed description of two patients with severe amphetamine-associated rhabdomyolysis complicated by dialysis-requiring AKI, documents exceptionally high CK concentrations, and illustrates uncommon complications including transient myocardial dysfunction, extensive muscular calcifications, and fatal delayed hypercalcemia. The availability of serial biochemical measurements, cardiac imaging, and radiological findings further strengthens the clinical characterization of both cases.

This report also has important limitations. First, it describes only two patients and therefore cannot establish causality or estimate the frequency of these complications. Second, the diagnosis of amphetamine exposure relied on qualitative multidrug urine toxicology screening without confirmatory chromatographic testing. Third, neither patient acknowledged illicit drug use, precluding precise determination of the timing, dose, or formulation of amphetamine exposure. Consequently, the temporal relationship between amphetamine exposure and the onset of the observed complications, as well as any potential dose-response relationship, could not be established. In addition, serum myoglobin concentrations were not available, precluding correlation between myoglobin burden and the severity of AKI. Finally, although an extensive investigation for alternative causes of hypercalcemia was initiated in the first patient, the diagnostic work-up could not be completed because of the patient's fatal outcome. Nevertheless, the overall clinical course, toxicological findings, radiological evidence of diffuse muscular calcifications, and exclusion of more common causes strongly support amphetamine-associated rhabdomyolysis as the most plausible diagnosis.

Conclusions

Amphetamine-associated rhabdomyolysis is an uncommon but potentially life-threatening condition that may result in severe AKI requiring renal replacement therapy and multisystem complications. Our two cases illustrate the heterogeneous clinical course of this condition, ranging from near-complete renal recovery to fatal evolution complicated by delayed hypercalcemia and transient myocardial dysfunction. In patients presenting with severe rhabdomyolysis, AKI, myalgia, and dark-colored urine without an obvious traumatic or medical cause, amphetamine intoxication should be considered in the differential diagnosis. Early multidrug urine toxicology screening, systematic exclusion of alternative etiologies, and prompt multidisciplinary management are essential to facilitate accurate diagnosis, optimize patient outcomes, and prevent severe renal, metabolic, and cardiovascular complications.

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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