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RHABDOMYOLYSIS-ASSOCIATED ACUTE KIDNEY INJURY: A THREE-PATIENT CASE SERIES ILLUSTRATING DIFFERENT PRECIPITANTS, BIOCHEMICAL TRAJECTORIES AND RENAL OUTCOMES

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ABSTRACT

Background: Rhabdomyolysis results from skeletal muscle injury with release of intracellular contents into the circulation. It can lead to acute kidney injury (AKI), electrolyte and acid-base disturbances, and, in severe cases, multiorgan dysfunction. The clinical course is heterogeneous, and the extent of CK elevation does not necessarily reflect the severity of kidney injury.

Case presentation: We describe three adult cases of rhabdomyolysis-associated AKI arising from distinctly different clinical circumstances. The first patient, a 27-year-old man, developed exertional rhabdomyolysis following an unusually intense gym workout. His CK concentration peaked at 46,780 U/L, while serum creatinine increased from 0.9 to 3.2 mg/dL. He responded to early intravenous crystalloid therapy and did not require kidney replacement therapy. The second patient was a 41-year-old man admitted after a generalized tonic-clonic seizure followed by prolonged immobilization. He developed severe rhabdomyolysis, hyperkalemia, metabolic acidosis and oligo-anuric AKI, with CK peaking at 132,400 U/L and creatinine reaching 7.8 mg/dL. Intermittent hemodialysis was required for six sessions, followed by gradual renal recovery. The third patient, a 58-year-old woman receiving high-dose statin therapy, presented after several days of fever, poor oral intake and progressive proximal muscle weakness. Her initial CK was 18,940 U/L, but she developed marked AKI with creatinine rising to 6.4 mg/dL in the setting of volume depletion and delayed presentation. She required four hemodialysis sessions and subsequently recovered to a creatinine of 1.3 mg/dL at 3-month follow-up.

Conclusion: These cases show the clinical heterogeneity of rhabdomyolysis-associated AKI. Severe kidney injury may result from the combined effects of muscle injury, hypovolemia, acidemia, and other patient factors rather than from CK elevation alone. Management rests on early recognition, treatment of theprecipitating cause, appropriate crystalloid therapy, close biochemical monitoring, and kidney replacement therapy when standard indications arise. CK should therefore be interpreted alongside the overall clinical and biochemical picture.

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INTRODUCTION

Rhabdomyolysis results from disruption of skeletal muscle cell membranes and subsequent release of intracellular components, particularly CK, myoglobin, potassium, phosphate, urate and other intracellular metabolites into the systemic circulation. The syndrome encompasses a broad spectrum, ranging from an isolated biochemical abnormality to severe illness complicated by electrolyte disorders, disseminated intravascular coagulation, compartment syndrome, acute kidney injury and death [1-3].

The relationship between rhabdomyolysis and AKI is complex. Myoglobin-mediated tubular injury is an important component, but renal hypoperfusion caused by fluid sequestration within injured muscle and systemic volume depletion also contributes substantially. In addition, heme-mediated oxidative injury and tubular obstruction may be potentiated by academia [1-2]. Thus, the development of AKI is probably determined by the interaction between the extent and duration of muscle injury and the patient's

hemodynamic and metabolic state rather than by muscle injury alone.

The causes of rhabdomyolysis are diverse. Trauma and crush injury remain classical precipitants, but non-traumatic causes account for a substantial proportion of contemporary presentations. Exertional injury, generalized seizures, prolonged immobilization, drugs and toxins, hyperthermia, infection and metabolic disorders are increasingly recognized in emergency and nephrology practice [1-3].

Creatine kinase is widely used for diagnosis and monitoring. Although very high CK concentrations have been associated with an increased risk of AKI, CK is an imperfect predictor of renal outcomes. A systematic review and meta-analysis found an association between CK and AKI but reported an area under the receiver operating characteristic curve of 0.75, with predictive performance varying by etiology.[4]In a large cohort, McMahon and colleagues developed a prognostic model incorporating age, sex, etiology, creatinine, CK, phosphate, calcium and bicarbonate; this score performed better than CK alone for predicting severe outcomes, including renal replacement therapy or death [5].

The present three-patient case series isillustrating three different presentations of rhabdomyolysis-associated AKI: exertional rhabdomyolysis managed without dialysis, seizure-associated rhabdomyolysis requiring dialysis, and statin-associated rhabdomyolysis complicated by marked volume depletion and delayed presentation. The cases underscore the importance of clinical context and serial assessment when estimating renal risk.

CASE 1: EXERTIONAL RHABDOMYOLYSIS WITH NON-DIALYSIS-REQUIRING AKI

A 27-year-old previously healthy man presented with severe bilateral thigh pain, generalized myalgia and cola-colored urine approximately 18 hours after performing an unusually strenuous lower-limb workout. He reported that he had recently increased the intensity of his exercise regimen and had completed multiple sets of heavy squats and deadlifts for the first time.

He had consumed relatively little fluid during the workout and had continued to work for several hours afterward. There was no history of trauma, seizure, alcohol binge, recreational drug use, or use of medications known to cause muscle injury.

On arrival, he was alert and hemodynamically stable. Blood pressure was 108/68 mmHg, pulse rate 104/min and temperature 37.1°C. There was marked bilateral quadriceps tenderness without tense compartments or pain on passive stretch. The remainder of the systemic examination was unremarkable.

Initial investigations showed hemoglobin 15.1 g/dL, white-cell count 12,600/µL, serum creatinine 1.4 mg/dL, urea 38 mg/dL, potassium 5.2 mmol/L, sodium 137 mmol/L, bicarbonate 21 mmol/L, phosphate 4.7 mg/dL, calcium 8.5 mg/dL, uric acid 8.2 mg/dL and CK 31,600 U/L. Urinalysis was strongly positive for blood with only 1–2 red blood cells per high-power field. Serum myoglobin was elevated at 8,940 ng/mL.

The patient was started on isotonic crystalloid at approximately 300 mL/hour with adjustment according to blood pressure, urine output and clinical volume status. His urine output during the first 12 hours was 1.8 mL/kg/hour. Electrolytes and renal function were monitored every 6–8 hours initially.

CK continued to rise during the next 24 hours, reaching a peak of 46,780 U/L on hospital day 2. Despite the rise in CK, renal function began to improve after restoration of effective circulating volume. Serum creatinine peaked at 3.2 mg/dL on day 2 and subsequently declined. Potassium remained below 5.5 mmol/L, and there was no clinically significant pulmonary edema, refractory acidosis or other indication for dialysis.

Intravenous fluid was progressively reduced as the CK concentration declined and oral intake improved. CK fell below 5,000 U/L by day 6. The patient was discharged on day 7 with a creatinine of 1.1 mg/dL and was advised to avoid strenuous exercise until complete biochemical recovery. At 6-week follow-up, he was asymptomatic, with serum creatinine 0.9 mg/dL and CK 118 U/L.

Clinical interpretation

Despite the marked CK elevation, this patient had a favorable clinical course. Reduced oral intake and early volume depletion likely contributed to the AKI in the setting of intense exertional muscle injury. CK continued to rise after the patient had clinically stabilized, demonstrating that CK kinetics may lag behind changes in renal function.

CASE 2: SEIZURE-ASSOCIATED RHABDOMYOLYSIS WITH DIALYSIS-REQUIRING AKI

A 41-year-old man with a history of poorly controlled epilepsy was brought to the emergency department after a generalized tonic-clonic seizure witnessed at home. According to family members, the convulsion lasted approximately 6–7 minutes. He remained confused afterward and was found several hours later to have severe generalized muscle pain and markedly reduced urine output.

At admission, blood pressure was 94/58 mmHg, heart rate 118/min and temperature 37.8°C. He was drowsy but arousable. There was diffuse muscle tenderness, particularly involving the shoulders and thighs. No obvious traumatic injury or compartment syndrome was found.

Laboratory investigations revealed creatinine 2.6 mg/dL, urea 68 mg/dL, potassium 6.4 mmol/L, sodium 138 mmol/L, bicarbonate 16 mmol/L, phosphate 7.1 mg/dL, corrected calcium 7.7 mg/dL, uric acid 10.6 mg/dL, AST 540 U/L, ALT 128 U/L and CK 72,300 U/L. Urine microscopy demonstrated granular casts and fewer than 5 red blood cells per high-power field despite 3+ heme positivity on dipstick. Serum myoglobin was 19,700 ng/mL.

The patient received rapid isotonic crystalloid resuscitation together with standard emergency treatment for hyperkalemia. Despite fluid administration, urine output remained below 0.3 mL/kg/hour over the subsequent 12 hours. Serum potassium repeatedly increased to 6.6 mmol/L, and bicarbonate decreased to 13 mmol/L.

CK peaked at 132,400 U/L on hospital day 3. Serum creatinine increased to 7.8 mg/dL, accompanied by persistent oliguria. Because of refractory hyperkalemia, metabolic acidosis and progressive azotemia, intermittent hemodialysis was initiated. A right internal jugular temporary dialysis catheter was placed under ultrasound guidance. Hemodialysis was performed with a high-flux dialyzer, initially for approximately 2.5 hours. Six dialysis sessions were required over the following nine days. No dialysis was initiated solely because of the CK level; treatment decisions were based on the patient's biochemical and clinical complications.

Urine output began to recover on hospital day 10. Serum creatinine gradually declined, reaching 2.4 mg/dL by discharge on day 17. Dialysis was discontinued after sustained improvement in urine output and stabilization of potassium and acid-base status. At 3-month follow-up, the patient's serum creatinine was 1.2 mg/dL and eGFR had returned close to his pre-illness baseline.

Clinical interpretation

The second patient had a substantially more severe course. The prolonged generalized seizure caused extensive muscle injury, while transient hypotension and prolonged immobilization likely compounded renal hypoperfusion. Persistent oliguria, hyperkalemia, and metabolic acidosis subsequently developed. Dialysis was therefore undertaken for the complications of AKI, not because the CK had crossed a particular threshold.

CASE 3: STATIN-ASSOCIATED RHABDOMYOLYSIS WITH SEVERE AKI AFTER DELAYED PRESENTATION

A 58-year-old woman with hypertension, type 2 diabetes mellitus and dyslipidemia had been receiving atorvastatin 80 mg daily for approximately eight months. She developed fever and anorexia associated with an acute febrile illness during the preceding week. During this period, her oral intake decreased substantially. She gradually developed proximal muscle pain and difficulty climbing stairs but did not seek medical attention initially.

On the fourth day of illness, she developed marked generalized weakness and dark urine and was brought to hospital because of progressive reduction in urine output.

At presentation, she appeared dehydrated. Blood pressure was 92/60 mmHg, pulse 108/min and temperature 37.6°C. Mucous membranes were dry and peripheral pulses were mildly diminished. There was symmetrical proximal muscle tenderness but no focal neurological deficit.

Initial laboratory studies showed creatinine 4.1 mg/dL, urea 96 mg/dL, potassium 5.7 mmol/L, bicarbonate 18 mmol/L, phosphate 5.8 mg/dL, calcium 8.1 mg/dL, uric acid 9.3 mg/dL, AST 372 U/L, ALT 96 U/L, and CK 18,940 U/L. Urinalysis showed 3+ blood with minimal erythrocytes. Serum myoglobin was 6,780 ng/mL.

Atorvastatin was discontinued immediately. The patient received carefully monitored isotonic crystalloid replacement. The initial impression was that both muscle injury and pronounced volume depletion were contributing to the renal dysfunction.

During the next 48 hours, urine output remained low despite correction of hypotension. Serum creatinine increased to 6.4 mg/dL and potassium to 6.1 mmol/L, with bicarbonate decreasing to 15 mmol/L. CK reached a peak of 37,850 U/L on day 2.

Because of persistent oliguria accompanied by hyperkalemia, metabolic acidosis and progressive azotemia, intermittent hemodialysis was initiated. Four sessions were required. The patient's urine output improved substantially during the second week, and serum creatinine declined progressively without further dialysis.

She was discharged after 14 days with a creatinine of 2.0 mg/dL. At one-month follow-up it had decreased to 1.5 mg/dL, and at three months it was 1.3 mg/dL. The patient remained off statin therapy for several weeks. After discussion of cardiovascular risk and potential recurrence, lipid-lowering therapy was subsequently reconsidered using an alternative regimen with close CK and symptom monitoring.

CLINICAL INTERPRETATION

The third case is notable because the CK concentration was considerably lower than in patient 2, yet kidney dysfunction was severe. Delayed presentation, substantial intravascular volume depletion, diabetes, and ongoing medication exposure likely contributed to the renal injury. The case reinforces the limitation of using CK alone to estimate either the likelihood or severity of AKI.

Table 01: Comparative clinical characteristics

Variable	Patient 1	Patient 2	Patient 3
Age, years	27	41	58
Sex	Male	Male	Female
Major precipitant	Intense exercise	Generalized seizure	Statin + dehydration
Relevant comorbidity	None	Epilepsy	DM, HTN, dyslipidemia
Initial CK, U/L	31,600	72,300	18,940
Peak CK, U/L	46,780	132,400	37,850
Initial creatinine, mg/dL	1.4	2.6	4.1
Peak creatinine, mg/dL	3.2	7.8	6.4
Initial potassium, mmol/L	5.2	6.4	5.7
Lowest bicarbonate, mmol/L	19	13	15
Initial phosphate, mg/dL	4.7	7.1	5.8

Variable	Patient 1	Patient 2	Patient 3
Myoglobin, ng/mL	8,940	19,700	6,780
Oliguria	No	Yes	Yes
KDIGO AKI stage	2	3	3
Dialysis	No	Yes	Yes
Number of HD sessions	0	6	4
ICU admission	No	Yes	No
Hospital stay, days	7	17	14
Creatinine at discharge, mg/dL	1.1	2.4	2.0
Creatinine at follow-up	0.9	1.2	1.3
Follow-up	6 weeks	3 months	3 months
Renal recovery	Complete	Near-complete	Near-complete

Serial biochemical course

Table 02: Patient 1 clinical characteristics

Hospital day	CK (U/L)	Creatinine (mg/dL)	K (mmol/L)	Urine output
1	31,600	1.4	5.2	1.8 mL/kg/h
2	46,780	3.2	5.3	1.5 mL/kg/h
3	41,200	2.7	4.8	2.0 mL/kg/h
4	22,600	1.9	4.4	2.2 mL/kg/h
6	4,780	1.2	4.2	2.0 mL/kg/h
7	2,210	1.1	4.1	Clinically adequate

Table 03: Patient 2 clinical characteristics

Hospital day	CK (U/L)	Creatinine (mg/dL)	K (mmol/L)	Urine output
1	72,300	2.6	6.4	0.25 mL/kg/h
2	109,600	5.4	6.6	0.18 mL/kg/h
3	132,400	7.8	5.1 after HD	<0.2 mL/kg/h
5	108,700	6.9	4.8	0.3 mL/kg/h
7	63,400	5.8	4.5	0.5 mL/kg/h
10	24,100	4.1	4.3	1.1 mL/kg/h
14	6,120	2.9	4.2	1.8 mL/kg/h
17	2,040	2.4	4.1	Adequate

Table 04: Patient 3 clinical characteristics

Hospital day	CK (U/L)	Creatinine (mg/dL)	K (mmol/L)	Urine output
1	18,940	4.1	5.7	0.35 mL/kg/h
2	31,500	6.1	6.0	0.25 mL/kg/h
3	37,850	6.4	4.9 after HD	0.3 mL/kg/h
5	25,600	5.2	4.5	0.5 mL/kg/h
7	11,800	3.9	4.3	1.0 mL/kg/h
10	4,320	2.7	4.1	1.6 mL/kg/h
14	1,480	2.0	4.0	Adequate

MCMAHON RISK ASSESSMENT

For illustrative purposes, the McMahon score was calculated using admission parameters. The original model incorporates age, sex, etiology, creatinine, CK, phosphate, calcium and bicarbonate and was developed to predict the composite outcome of kidney failure requiring renal replacement therapy or in-hospital mortality [5].

Table 05: McMahon score calculated by using admission parameters.

Patient	Estimated McMahon score	Interpretation
1	4	Lower predicted risk
2	10	High predicted risk
3	9	High predicted risk

In the original derivation and validation cohorts, the composite outcome occurred in 19% of patients. In the validation cohort, the risk was 2.3% among those with scores below 5 and 61.2% among those with scores above 10 [5]. A later 10-year cohort also found that a McMahon score of at least 6 was more useful than peak CK alone for identifying patients at risk of renal replacement therapy [6].

The serial laboratory trajectories are illustrated in Figure 01. The relationship between peak creatine kinase and peak serum creatinine is shown in Figure 02.

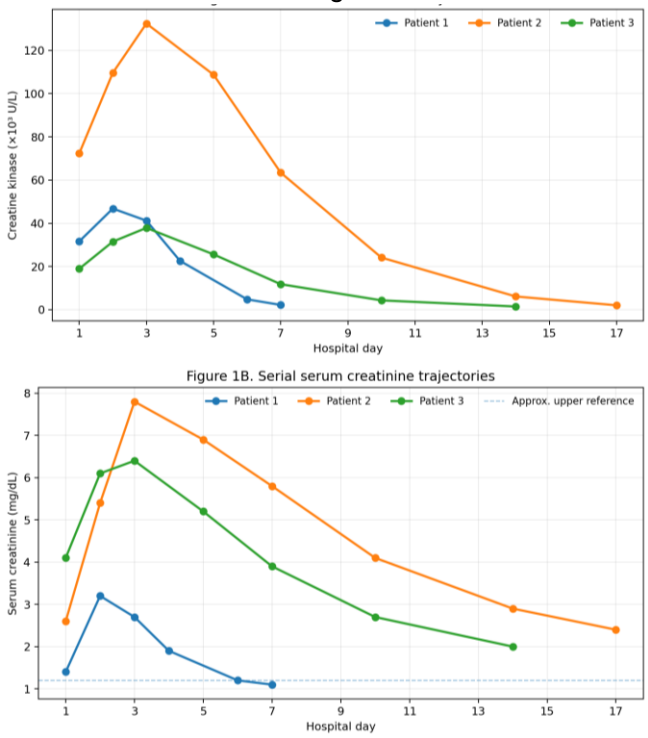


Figure 01. Serial biochemical trajectories. (A) Creatine kinase concentrations and (B) serum creatinine during hospitalization. The three patients show distinct temporal patterns of muscle injury and renal dysfunction.

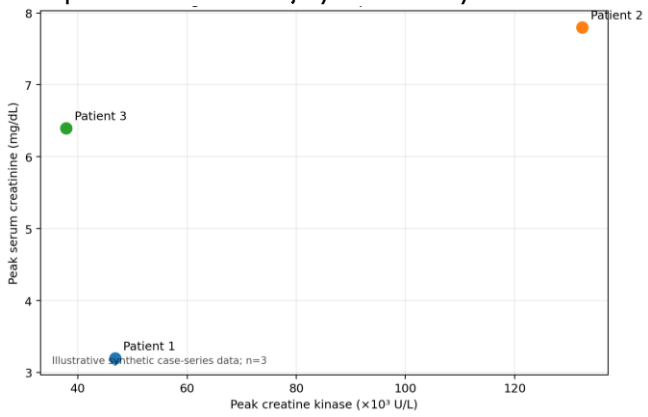


Figure 02: Peak creatine kinase versus peak serum creatinine. The cases demonstrate that the severity of AKI cannot be inferred reliably from CK concentration alone.

DISCUSSION

This three-patient case series illustrates three important aspects of rhabdomyolysis-associated AKI. First, the causes of rhabdomyolysis are heterogeneous. Second, CK concentration alone does not adequately describe renal risk. Third, the decision to initiate kidney replacement therapy depends on the systemic consequences of AKI rather than the degree of CK elevation itself.

The pathophysiology of pigment-associated AKI is multifactorial. Destruction of skeletal muscle releases myoglobin and other intracellular solutes into the circulation. Myoglobin is filtered through the glomerulus and may contribute to tubular toxicity through heme-mediated oxidative stress. It can also interact with uromodulin and contribute to tubular obstruction.[1,2] In parallel, injured muscle can sequester considerable quantities of fluid, reducing the effective circulating volume and renal perfusion. Acidemia further enhances the adverse effects of myoglobin within the tubular environment.

The first patient developed AKI despite being young and previously healthy. His relatively rapid presentation, restoration of intravascular volume and preserved urine output probably contributed to his recovery without dialysis. Exercise-associated rhabdomyolysis is often self-limiting when promptly recognized, although severe exertional cases can still cause substantial AKI.

The second patient illustrates a more severe phenotype. Generalized seizures can produce extensive muscle injury over a short period. In addition to the muscle breakdown itself, transient hypotension and prolonged immobilization probably intensified renal hypoperfusion. He subsequently developed persistent oliguria, refractory hyperkalemia and metabolic acidosis. These are conventional indications for kidney replacement therapy, and dialysis was therefore initiated because of the consequences of AKI rather than because the CK exceeded a particular value.

The third patient is perhaps the most instructive from a prognostic standpoint. Her peak CK was much lower than that observed in patient 2, yet kidney dysfunction was severe. A meta-analysis of studies examining CK and AKI demonstrated that although CK is associated with AKI risk, its predictive characteristics are insufficient to use CK as a stand-alone prognostic marker [4]. This is consistent with the clinical contrast illustrated by these cases.

This apparent dissociation is biologically plausible. CK reflects muscle-cell injury but does not directly measure intravascular volume depletion, renal vasoconstriction, tubular injury, or the efficiency of myoglobin clearance. Consequently, patients with comparable CK values may have very different renal outcomes when their hemodynamic state or comorbidity burden differs.

The McMahon score addresses this problem by combining admission creatinine, phosphate, calcium and bicarbonate with demographic and etiologic factors and CK. In its derivation and validation cohorts, the score showed good discrimination for severe outcomes [5]. The pattern illustrated by patients 2 and 3 supports the practical value of considering the broader biochemical context.

FLUID MANAGEMENT

Early volume resuscitation is generally considered the cornerstone of treatment in patients with significant rhabdomyolysis who do not have established anuria or severe volume overload. The objective is restoration of effective circulating volume and maintenance of renal perfusion while facilitating urinary clearance of filtered

muscle breakdown products. Current consensus guidance supports starting crystalloid therapy and titrating the rate according to clinical status and urine output rather than using an indiscriminate fixed-volume strategy.

The rate of fluid administration needs to be individualized. The American Association for the Surgery of Trauma consensus document describes an initial rate around 400 mL/hour, with a broad range of 200–1,000 mL/hour depending on the clinical circumstances. A commonly cited urine-output target is approximately 1–3 mL/kg/hour, with an upper practical target of around 300 mL/hour.

The cases also demonstrate why excessive fluid administration can be harmful once renal clearance becomes severely impaired. In anuric patients, continued aggressive fluid administration can lead to pulmonary edema and other complications [1]. Fluid therapy should therefore always be accompanied by frequent reassessment of urine output, cumulative fluid balance, respiratory status, electrolyte concentrations and renal function.

ROLE OF BICARBONATE AND MANNITOL

Urinary alkalinization and mannitol have historically been included in protocols for rhabdomyolysis. However, clinical evidence supporting routine use is limited. Current consensus literature does not support routine bicarbonate or mannitol administration specifically for prevention of rhabdomyolysis-induced AKI, and evidence for loop diuretics as a renal-protective intervention is similarly weak [1].

This does not preclude the use of bicarbonate or diuretics for specific indications. Sodium bicarbonate may be appropriate for clinically significant metabolic acidosis, and loop diuretics may help manage volume overload when residual renal function is present. Neither should be viewed as a substitute for restoration of effective circulating volume or for kidney replacement therapy when standard indications are met.

KIDNEY REPLACEMENT THERAPY

No CK concentration, by itself, mandates dialysis. Kidney replacement therapy should instead be based on established clinical indications, including refractory hyperkalemia, severe metabolic acidosis, clinically significant fluid overload, uremic complications, or persistent severe oliguria/anuria in the appropriate clinical context.

Patients 2 and 3 developed severe oliguric AKI with persistent hyperkalemia and metabolic acidosis and consequently underwent hemodialysis. Patient 1, despite a CK concentration approaching 47,000 U/L, did not develop an indication for dialysis. The contrast illustrates why the decision to initiate kidney replacement therapy should follow the patient's renal and systemic physiology rather than the CK value alone.

Extracorporeal techniques capable of removing circulating myoglobin have generated interest, particularly high cut-off membranes and other enhanced extracorporeal strategies. However, a reduction in serum myoglobin does not automatically translate into improved survival or renal recovery, and evidence remains insufficient to recommend extracorporeal myoglobin removal as routine therapy for rhabdomyolysis-associated AKI.

ELECTROLYTE ABNORMALITIES

Electrolytes require close surveillance because injured skeletal muscle releases substantial intracellular stores of potassium and phosphate. Hyperkalemia can develop rapidly and may be life-threatening, particularly in oliguric patients. Hyperphosphatemia, hyperuricemia, and early hypocalcemia may occur with severe muscle injury, with serum calcium sometimes rising during recovery.

The second patient had the most prominent electrolyte abnormalities and required urgent treatment of hyperkalemia. In practical terms, potassium monitoring is often more immediately consequential than CK measurement because potassium can change over hours, whereas CK may remain markedly elevated for several days.

PROGNOSIS AND RECOVERY

All three patients recovered kidney function, although at different rates. Patient 1 recovered within days, whereas patients 2 and 3 required dialysis before renal recovery became evident. This course is consistent with the potential reversibility of pigment-associated tubular injury when the precipitating factors are addressed and complications are appropriately managed.

Follow-up remains important because severe AKI may carry a later risk of chronic kidney disease even when serum creatinine returns close to baseline. Recovery by hospital discharge should therefore not be considered the end of renal follow-up.

LEARNING POINTS

- Consider rhabdomyolysis in patients with acute muscle pain or weakness, dark urine, unexplained aminotransferase elevation, or AKI after a compatible precipitating event.
- A markedly elevated CK confirms substantial muscle injury but does not, by itself, determine the severity of AKI or the need for dialysis.
- AKI reflects the combined effects of myoglobin-mediated tubular injury, renal hypoperfusion, fluid sequestration, acid-base disturbances, and patient-specific factors.
- Early crystalloid therapy is important in volume-depleted patients, but the rate should be guided by urine output and repeated assessment of volume status to limit fluid overload.
- Dialysis should be initiated for standard clinical indications-such as refractory hyperkalemia, severe metabolic acidosis, fluid overload, or uremic complications-rather than an isolated CK threshold.

LIMITATIONS

This report has the inherent limitations of a three-patient case series.The small number of cases precludes meaningful statistical inference.

CONCLUSION

Rhabdomyolysis-associated AKI is clinically heterogeneous. These cases show that marked CK elevation may be followed by relatively rapid renal recovery, while severe

AKI may occur with a substantially lower CK when volume depletion, delayed presentation, and other risk factors are present. Assessment should therefore incorporate hemodynamics, urine output, creatinine, potassium, phosphate, calcium, bicarbonate, and the precipitating cause rather than CK concentration alone. Early identification of the precipitating cause, appropriate crystalloid therapy, close monitoring, and timely kidney replacement therapy for standard indications remain central to management. Risk-prediction tools such as the McMahon score may add useful context when CK alone does not adequately capture renal risk.

PATIENT PERSPECTIVE

Patient consent taken.

CONFLICTS OF INTEREST

None.

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AUTHOR CONTRIBUTIONS

Dr. G. K. Mukhiya conceived and supervised the manuscript and provided overall academic guidance. Dr. Nilanjan Mukherjee contributed to the data collection, clinical interpretation, literature review, and critical revision of the manuscript. Dr. Patel Parth Rajendrakumar contributed to data collection, literature review. All authors reviewed and approved the final manuscript.

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