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Review Article

Acute Kidney Injury Secondary to Exertional Rhabdomyolysis: A Review of Published Cases

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Abstract

Background: Exertional rhabdomyolysis is a recognized cause of acute kidney injury (AKI) in otherwise healthy individuals undertaking unaccustomed or extreme physical exertion, yet reported renal risk varies enormously across settings.

Objective: To synthesize published case reports, case series, and cohort data on AKI complicating exertional rhabdomyolysis.

Methods: Narrative review of individual published cases, athletic and military case series, population-based cohorts, and management guidelines.

Findings: Population-based incidence is roughly 1-36 per 100,000 person-years depending on the cohort, while AKI complicating hospitalized or severe cases ranges from 1% (spin-class cohorts) to over 75% (pooled published case reports), reflecting strong publication bias. Peak creatine kinase (CK) correlates imperfectly with AKI; sickle cell trait and inadequate hydration are recurrent predisposing factors. Fluid resuscitation remains the mainstay of prevention; bicarbonate and mannitol lack proven benefit.

Conclusions: Renal outcomes after exertional rhabdomyolysis are generally favorable with early recognition and fluid therapy, but severe cases can require dialysis; individualized risk assessment beyond CK alone is warranted.

Keywords: Rhabdomyolysis; Acute kidney injury; Exercise; Creatine kinase; Dialysis

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Introduction

Exertional rhabdomyolysis (ER) is skeletal muscle breakdown precipitated by strenuous or unaccustomed physical activity, releasing myoglobin, creatine kinase (CK), and intracellular contents into the circulation [1,2]. Although most cases in healthy athletes and military recruits resolve without sequelae, a subset progresses to acute kidney injury (AKI) through myoglobin-mediated tubular injury, vasoconstriction, and intratubular obstruction [1,3]. The condition spans a spectrum from asymptomatic CK elevation in trainees to fulminant AKI

requiring renal replacement therapy (RRT) [4]. Because the published literature is dominated by case reports of unusually severe presentations, apparent AKI rates in that literature substantially exceed rates observed in unselected clinical cohorts [5,6]. This review synthesizes published individual cases, case series, and population cohorts of ER-associated AKI, and examines epidemiology, pathophysiology, clinical presentation, diagnostic approach, management, and outcomes, with particular attention to where the published case literature and consecutive clinical cohorts diverge.

Epidemiology

Population-based incidence estimates vary widely by population studied. In an Olmsted County, Minnesota cohort of 431 rhabdomyolysis cases (2003-2015), only 20 (4.9%) were exertional, yielding an age- and sex-adjusted incidence of 1.06 per 100,000 person-years (95% CI 0.59-1.52); kidney injury occurred in 5 of 20, with no deaths [7]. By contrast, U.S. military surveillance recorded 464 incident ER cases among active-component service members in 2024 alone, a crude rate of 35.9 per 100,000 person-years, with recruit trainees exceeding 13 times the rate of officers and other enlisted personnel, and service-specific rates as high as 84.7 per 100,000 in the Marine Corps [8]. This roughly 30-fold disparity underscores that unaccustomed intense exertion in structured training environments, rather than exercise per se, drives most ER.

Sickle cell trait is an established risk factor: among 47,944 Black active-duty soldiers, sickle cell trait carried an adjusted hazard ratio of 1.54 (95% CI 1.12-2.12) for exertional rhabdomyolysis, comparable to hazard ratios associated with tobacco use or elevated BMI, though it did not independently increase mortality [9]. Among cohorts of hospitalized or severe rhabdomyolysis, renal outcomes diverge sharply by population: AKI complicated only 1 of 93 (1.1%) hospitalized spin-class-induced ER cases [10], 8.5% of 200 hospitalized ER patients in a large health

system [6], 16.4% of 813 pooled endurance-sport participants [11], 58% of 126 patients with severe rhabdomyolysis of mixed etiology (CK >5,000 IU/L) [12], and 75.4% of 65 pooled published athlete case reports [5]. This gradient illustrates that the published case literature substantially overrepresents severe renal outcomes relative to consecutive clinical cohorts, an important caveat when a narrative review such as this one draws heavily on individually reported cases.

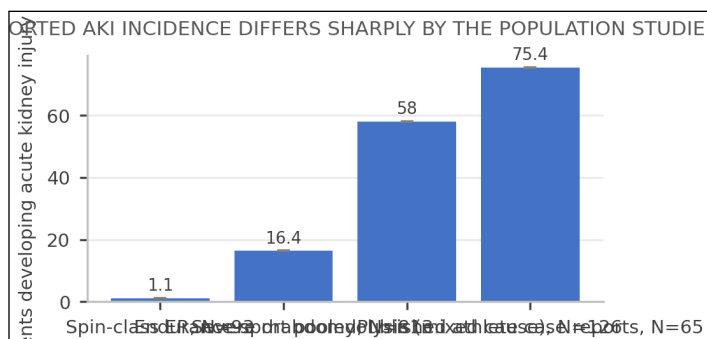


Figure 1: Reported AKI incidence across cohorts of exertional rhabdomyolysis, from 1.1% in a hospitalized spin-class cohort to 75.4% among pooled published athlete case reports, illustrating publication bias in the case-report literature [10,11,12,5].

Activity-specific case series further refine this picture. Among 93 hospitalized patients with spin/indoor-cycling-induced ER, mean age was 28.6 years, 71.0% were female, and 87.1% were first-time participants; despite peak CK exceeding 20,000 U/L in 89.2%, only one patient developed AKI, which resolved within 24 hours, and none required dialysis or developed compartment syndrome [10]. In a single academic center, 11 of 523 (2.1%) CrossFit-related presentations over six years met criteria for rhabdomyolysis, with a mean age of 34.9 years, 81.9% male, and over half beginners to the activity [13]. A broader systematic review of high-intensity functional training identified 63 published cases across 26 studies, with CK ranging from 7,816 to 232,579 U/L and dark urine reported in 40% [14].

Pathophysiology

Muscle injury releases myoglobin, potassium, phosphate, and CK into the circulation. Myoglobin is filtered and can

precipitate as intratubular casts, particularly in acidic, concentrated urine, while its heme moiety generates reactive oxygen species that injure tubular epithelium directly [1,3]. Concurrent volume depletion, common after prolonged exertion in hot environments, promotes renal vasoconstriction and reduces tubular flow, compounding obstruction [2]. Underlying metabolic myopathies — including carnitine palmitoyltransferase II (CPT II) deficiency — can unmask ER at unexpectedly modest exertional loads and should be considered when episodes recur or occur with trivial triggers [15,4]. Genetic risk modifiers such as sickle cell trait potentiate exertional muscle injury through microvascular sickling under conditions of dehydration, acidosis, and heat [9,16]. The three-part mechanism most often invoked — direct tubular toxicity, renal vasoconstriction, and cast formation — helps explain why severity is not fully captured by CK alone: hydration status, urine pH, and the pace of fluid resuscitation modulate how much filtered myoglobin actually precipitates within the tubule, independent of the total muscle burden released into the circulation [1,2].

Clinical Presentation

Classic presentation includes myalgia, muscle swelling or weakness, and dark ("tea-colored") urine following unaccustomed or extreme exertion, though this triad is frequently incomplete. Published cases illustrate the phenotypic range: a healthy 28-year-old developed CK of 97,472 U/L after a novel weight-training circuit yet never developed AKI [17], while a 24-year-old with sickle cell trait developed severe AKI (creatinine 6.03 mg/dL, potassium 8.6 mmol/L) requiring emergent dialysis after a week of poorly hydrated training [16]. A 27-year-old developed stage 3 AKI after prolonged swimming in hot, humid conditions despite normokalemia [18], and a 61-year-old developed AKI after an unsupported ~30 km walk of ~44 hours without food or water [19]. Compartment syndrome, disseminated intravascular

coagulation, and multidrug-resistant bacteremia complicated one severe case requiring fasciotomy [20]. Rare presentations include posterior reversible encephalopathy syndrome with transient bilateral vision loss [21] and dual-hit injury from concurrent viral infection and exertion in an adolescent [22]. Underlying metabolic myopathy, such as CPT II deficiency diagnosed only after an adult presentation of exertional rhabdomyolysis, is a further recognized contributor and should prompt evaluation when episodes are recurrent or disproportionate to the trigger [15].

Table 1: Published cases of exertional rhabdomyolysis with renal involvement reviewed here [17,16,18,19,20,22,15,21]; N = 8 individual cases.					
Report	Age/sex	Trigger	Peak CK	AKI stage/diagnosis	Outcome
Berry 2014	28/M	Novel weight-training circuit	97,472 U/L	No AKI	Full recovery
Vincent 2026	24/M, sickle cell trait	Week of exertion, poor hydration	51,100 U/L	Severe AKI, hemodialysis	Outpatient HD, recovering
Güven 2023	27/M	Prolonged swimming, heat	14,851 U/L	Stage 3 AKI, hemodialysis	Residual impairment
Zhang 2024	61/M	44-h unsupported walk	3,917 U/L	AKI, no dialysis	Full recovery
Al Badi 2020	19/M	Unaccustomed aerobic exercise	Not stated	AKI, hemodialysis	Foot drop, eventual recovery
Benaini 2026	17/M	Influenza A plus exertion	>60,000 U/L	AKI, CVVH then HD	Progressive recovery
Cucchiari 2018	41/M, CPT II deficiency	Strenuous effort	Not stated	AKI, no dialysis	Complete recovery
Dai 2022	Not stated	Intense exercise	Not stated	AKI, blood purification	Full recovery; PRES

Diagnostic Approach

Diagnosis rests on markedly elevated serum CK (typically several-fold to over 100-fold the upper limit of normal) together with clinical context, supported by myoglobinuria, hyperkalemia, hyperphosphatemia,

hypocalcemia, and rising creatinine [1,2]. However, no verified published dataset supports a single CK threshold that reliably predicts AKI: counter-examples include CK of 97,472 U/L without AKI [17] and CK of 14,851 U/L with stage 3 AKI [18], and a large hospital cohort found no association between high CK and AKI risk [6]. Risk-prediction tools perform better than CK alone: the McMahon score, derived from 2,371 patients with CK >5,000 U/L, achieved a C-statistic of 0.82-0.83 for predicting RRT or death, with predicted risk ranging from 2.3% (score <5) to 61.2% (score >10) [23], and was subsequently validated in an independent 10-year cohort [24]. In a large dataset of 73,036 adults with CK ≥1,000 U/L, dialysis risk rose from 1.84% in the lowest CK stratum to 8.79-8.81% above 50,000 U/L, plateauing at higher CK levels [25]. Myoglobin has also been proposed as a complementary marker, with levels above 15,000 µg/L associated with substantially higher AKI incidence in some series [3]. Reported ranges for the proportion of all-cause rhabdomyolysis complicated by AKI span roughly 13-60%, depending on the population and AKI definition applied [26,2,3], reinforcing that case definition and cohort selection, not CK alone, drive much of the variability across the literature. Baseline laboratory evaluation should also include serum potassium, phosphate, calcium, and urinalysis for heme-positive, sediment-poor urine, which distinguishes myoglobinuria from hematuria.

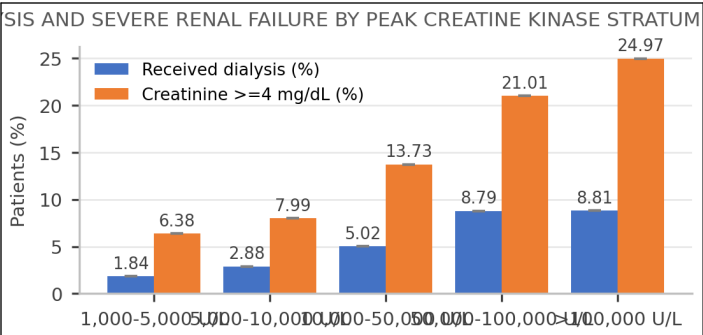


Figure 2: Dialysis and severe renal failure (creatinine ≥ 4 mg/dL) by peak creatine kinase stratum among 73,036 adults with rhabdomyolysis; risk rises with CK but plateaus above 50,000 U/L [25].

Management

Early, aggressive isotonic fluid resuscitation is the cornerstone of AKI prevention, with contemporary guidance recommending initial crystalloid infusion around 400 mL/h (range 200-1,000 mL/h) titrated to a urine output target of 1-3 mL/kg/h [27,2]. Sodium bicarbonate and mannitol are not recommended for routine AKI prevention; observational comparisons found no reduction in renal failure, RRT, or death with bicarbonate or mannitol added to crystalloid alone, and current guidelines from trauma and anesthesiology bodies converge on this position [27,28]. RRT is reserved for established AKI with refractory hyperkalemia, acidosis, fluid overload, or uremia, and has no established prophylactic role [27,3]. Individual cases required dialysis for severe hyperkalemia and volume overload [16,18], continuous veno-venous hemofiltration transitioning to intermittent hemodialysis for anuric AKI [22], or forced diuresis alone when renal function, though impaired, did not meet dialysis criteria [15]. Surgical decompression is required when compartment syndrome co-occurs [20]. Society guidelines from the American Association for the Surgery of Trauma and the Danish DASAIM/DSIT collaboration provide structured pathways for fluid dosing, monitoring, and RRT initiation [27,28]. Continuous reassessment of volume status, urine output, and electrolytes over the first 24-48 hours is emphasized

across guidelines, since both under-resuscitation (permitting ongoing tubular injury) and over-resuscitation (precipitating pulmonary edema, as required dialysis in one reviewed case after aggressive hydration) carry risk [18,27].

Outcomes and Prognosis

Renal recovery is generally favorable. In the largest pooled published-case series, 95.4% of patients achieved complete renal recovery with no deaths, though this figure likely reflects publication bias toward successfully treated cases [5]. In severe rhabdomyolysis of mixed etiology, mortality was 14% overall and rose to 19.2% among patients who developed AKI versus 3.6% without AKI [12]. Broader critical-care registry data report in-hospital mortality of 31.1% among rhabdomyolysis admissions, rising to 59% among those requiring RRT [26]. Not all published cases achieve full recovery: one patient remained with residual renal impairment at one-week discharge despite dialysis [18], and another required emergent dialysis before transitioning to outpatient hemodialysis with renal function only beginning to recover a week after discharge [16]. Long-term (>1 year) renal follow-up data are essentially absent from the reviewed literature, a gap explicitly noted in systematic reviews of endurance-sport ER, which highlight uncertainty about whether repeated ER-associated AKI episodes contribute cumulatively to chronic kidney disease risk [11].

Table 2: Incidence, renal outcome, and mortality across verified cohorts of exertional or severe rhabdomyolysis [7,8,10,12,23,5].						
Study	Population	N	AKI n (%)	RRT n (%)	Deaths	Incidence
Luetmer 2020	Olmsted County civilians, 2003-2015	20 exertional (of 431)	5 (25%)	Not reported	0	1.06 per 100,000 person-years
AFHSD 2025	US active-component military, 2020-2024	464 (2024)	Not reported	Not reported	Not reported	35.9 per 100,000 person-years

Study	Population	N	AKI n (%)	RRT n (%)	Deaths	Incidence
Lim 2023	Singapore spin-class admissions	93	1 (1.1%)	0 (0%)	0	Not applicable
Rodriguez 2013	Single-centre severe rhabdomyolysis	126	73 (58%)	12 (9.5%)	18 (14%)	Not applicable
McMahon 2013	Two Boston teaching hospitals, 2000-2011	2,371	Not reported separately	190 (8.0%)	334 (14.1%)	Not applicable
AlHumaidan 2026	Pooled published athlete cases	65	49 (75.4%)	14 (21.5%)	0	Not applicable

Conclusion

Exertional rhabdomyolysis produces AKI across a wide severity spectrum, from asymptomatic CK elevation to dialysis-dependent renal failure, and reported AKI incidence depends heavily on whether the source population is a consecutive clinical cohort or the published case literature. CK level alone is an unreliable predictor of renal outcome; validated composite risk scores, hydration status, and predisposing conditions such as sickle cell trait or underlying myopathy better inform risk stratification. Prompt fluid resuscitation remains the principal preventive intervention, while bicarbonate and mannitol lack supporting evidence. Clinicians should maintain a high index of suspicion in athletes, military trainees, and recreational exercisers presenting after unaccustomed exertion, and long-term renal outcome data are needed to clarify whether apparently complete recovery persists beyond hospital discharge.

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