Journal of Education, Health and Sport. 2026;88:69592
eISSN 2391-8306.

https://doi.org/10.12775/JEHS.2026.88.69592

Journal of Education, Health and Sport. eISSN 2450-3118

Journal Home Page
https://apcz.umk.pl/JEHS/index

TYMIŃSKA, Paulina, FRĄCZEK, Julia, BORÓWKA, Karolina, KAWKA, Natalia, GÓRKA, Piotr Artur, KĄDZIOŁKA, Witold, and DOMAGAŁA, Szymon. The Kidney at the Limit: A Comprehensive Review of Exertional Rhabdomyolysis and Acute Kidney Injury in the Amateur Athlete. Journal of Education, Health and Sport. 2026;88:69592. eISSN 2391-8306.
https://doi.org/10.12775/JEHS.2026.88.69592

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 06.03.2026. Revised: 09.03.2026. Accepted: 13.03.2026. Published: 15.03.2026.

The Kidney at the Limit: A Comprehensive Review of Exertional Rhabdomyolysis and Acute Kidney Injury in the Amateur Athlete

Paulina Tymińska

Medical University of Silesia, Poland

s86287@365.sum.edu.pl

ORCID: 0009-0004-5624-1599

Julia Frączek
Medical University of Silesia, Poland
s82577@365.sum.edu.pl
ORCID: 0009-0005-0239-6824

Karolina Borówka
Medical University of Silesia, Poland
s856162@365.sum.edu.pl
ORCID: 0009-0001-5669-1491

Natalia Kawka
Medical University of Silesia, Poland
s858292@365.sum.edu.pl
ORCID: 0009-0008-8441-7052

Piotr Artur Górka
Medical University of Silesia, Poland
s82797@365.sum.edu.pl
ORCID: 0009-0002-1044-8952

Witold Kądziołka
Medical University of Silesia, Poland
witold.kadziolka1@gmail.com
ORCID: 0009-0007-6952-0054

Szymon Domagała
Medical University of Silesia, Poland
s82757@365.sum.edu.pl
ORCID: 0009-0000-7989-2942

Abstract

The modern fitness landscape has seen a significant rise in Exertional Rhabdomyolysis (ER), a potentially life-threatening condition traditionally associated with extreme endurance or military combat. As high-intensity functional training and aggressive indoor cycling become mainstream, recreational athletes are increasingly exposed to metabolic crises previously rare in the commercial gym sector.

This review examines the pathophysiology of ER, beginning with the ATP depletion-calcium dysregulation cascade that leads to myocyte necrosis and the systemic release of intracellular contents. Central to the clinical concern is the "Nephrotoxic Triad" - a synergistic mechanism involving renal vasoconstriction, oxidative stress via the Fenton Reaction, and physical tubular obstruction by myoglobin casts, which precipitates Acute Kidney Injury (AKI).

The article identifies a "perfect storm" of risk factors, including eccentric loading, heat strain, and the pervasive, unsupervised use of NSAIDs, which disable the kidney's autoregulatory mechanisms. Diagnosis remains challenging, as the classic triad of symptoms is present in less than 10% of cases; thus, the review highlights the importance of serum Creatine Kinase (CK) and the emerging utility of Cystatin C as a muscle-mass-independent biomarker for renal function.

Management focus is placed on aggressive, early fluid resuscitation using balanced crystalloids to induce forced diuresis. Finally, the review proposes a four-phase return-to-play protocol, emphasizing a risk-stratified approach to transition athletes safely from recovery to sport-specific activity while mitigating the long-term risks of chronic kidney disease and recurrence.

Keywords: Exertional Rhabdomyolysis, Acute Kidney Injury, Myoglobinuria, Creatine Kinase, Eccentric Exercise, Return-to-Play Protocol

1. Introduction

The fitness industry has experienced a significant paradigm shift over the past ten years. We have moved away from steady-state aerobic conditioning toward High-Intensity Functional Training (HIFT) and immersive indoor cycling studios. While this evolution has allowed the general public to access elite-level training intensities, it has also exposed recreational athletes

to medical risks previously confined to extreme endurance sports or military service. The most significant of these risks is Exertional Rhabdomyolysis (ER) [1].

Specific activities, particularly aggressive indoor cycling, have been identified as high-risk, leading to a phenomenon often termed "Spinning-induced Rhabdomyolysis" in neophytes [2]. Historically, ER was considered a pathology of the battlefield or ultra-marathons. However, current epidemiological trends reveal a migration of this diagnosis into the commercial gym sector. A longitudinal study utilizing data from the National Electronic Injury Surveillance System (NEISS) identified over 40,000 emergency department visits related to ER. Notably, the study highlighted a ten-fold increase in incidence over a recent decade, underscoring a growing public health concern [3].

ER is characterized by the rapid breakdown of skeletal muscle tissue, which results in the leakage of intracellular components, such as myoglobin and creatine kinase (CK) into the systemic circulation. From a sports management perspective, a "quality gap" exists: unlike professional athletes who benefit from periodized load management, amateurs often engage in unaccustomed, maximal eccentric exercise without adequate physiological preparation. This lack of conditioning leads to a metabolic crisis that overwhelms the renal system [4]. This review seeks to connect the disciplines of sports medicine and nephrology, offering a detailed examination of how amateur athletic exertion can precipitate acute kidney failure.

2. Pathophysiology

To comprehend how a standard workout can progress to renal failure, it is necessary to examine the cellular events that occur when metabolic demand exceeds the muscle's energy supply. The progression from myocyte damage to kidney injury follows a distinct and lethal pathway.

2.1 The Cellular Cascade: ATP Depletion and Calcium Dysregulation

The fundamental pathophysiology of Exertional Rhabdomyolysis is precipitated by the critical exhaustion of Adenosine Triphosphate (ATP) within the myocyte, a phenomenon frequently observed during high-volume eccentric loading in unconditioned athletes [5]. Beyond its role in contractile mechanics, ATP is essential for preserving cellular homeostasis via active transport channels, specifically the sodium-potassium (Na^+/K^+) and calcium (Ca^{2+}) ATPase pumps. In the absence of sufficient energy substrates, these transmembrane pumps are rendered non-functional, leading to an inability to extrude calcium from the cytosol.

This energetic failure results in a rapid, pathological sequestration of intracellular calcium (Ca^{2+}) [6]. This ionic dysregulation serves as a potent signaling mechanism, activating calcium-dependent proteolytic enzymes, notably calpains and phospholipases, which degrade the structural phospholipids of the cell membrane [7]. The ultimate sequela of this enzymatic cascade is the disintegration of the sarcolemma and the subsequent efflux of intracellular contents, including the 17.8 kDa hemoprotein myoglobin, into the systemic circulation [8].

2.2 The Nephrotoxic Triad: Mechanisms of Acute Kidney Injury

Upon entering circulation, myoglobin is filtered by the glomerulus. In a healthy physiological state, the plasma protein haptoglobin binds to free myoglobin to facilitate hepatic clearance. However, in cases of massive muscle breakdown, haptoglobin stores are rapidly depleted, leaving "free" myoglobin to precipitate acute renal failure through three synergistic mechanisms collectively known as the "Nephrotoxic Triad" [9].

The initial insult is hemodynamic, characterized by severe renal vasoconstriction. Myoglobin functions as a potent scavenger of Nitric Oxide (NO) within the renal vasculature. Since NO is the primary mediator of local vasodilation, its depletion results in unopposed vasoconstriction and ischemic injury. This effect is frequently exacerbated by the athlete's concurrent hypovolemic state, which triggers the Renin-Angiotensin-Aldosterone System (RAAS) to further restrict Renal Blood Flow (RBF) in a maladaptive attempt to preserve systemic pressure [10].

Simultaneously, the renal tubules are subjected to direct cytotoxic injury via oxidative stress. As myoglobin concentrates within the renal tubules, the heme moiety becomes chemically unstable. In the acidic environment of the proximal tubule, ferrous iron (Fe2+) is dissociated from the heme group. This free iron catalyzes the Fenton Reaction, generating hydroxyl radicals (•OH) and other Reactive Oxygen Species (ROS). These volatile radicals aggressively attack the lipid bilayers of the tubular epithelial cells, leading to lipid peroxidation and direct tubular necrosis [11].

This cascade culminates in physical obstruction within the distal nephron. Myoglobin interacts with Tamm-Horsfall Protein (uromodulin), a glycoprotein constitutively secreted in the Loop of Henle. Under conditions of low urinary pH (< 6.5), these proteins cross-link to precipitate solid, pigmented casts. These casts mechanically occlude the tubular lumen, halting urine flow and causing a precipitous, often anuric, decline in Glomerular Filtration Rate (GFR) [9].

3. Etiology and Risk Factors in the Amateur Athlete

The etiology of exertional rhabdomyolysis is rarely monofactorial, rather, it typically presents as a "perfect storm" of intrinsic physiological vulnerabilities and extrinsic environmental stressors. In the context of amateur sports, where physiological screening is limited, understanding these variables is critical for risk stratification.

3.1 Intrinsic Factors: The Physiological Baseline

The hydration status of the athlete functions as a primary determinant of renal resilience. Pre-existing hypovolemia significantly exacerbates the risk of renal failure by reducing renal blood flow before the insult of myoglobinuria even begins. However, hydration is a delicate balance; over-consumption of hypotonic fluids can lead to Exercise-Associated Hyponatremia (EAH), a condition that disrupts cellular osmolality and complicates renal filtration dynamics [12]. Furthermore, specific electrolyte imbalances, particularly hypokalemia (potassium deficiency), can precipitate muscle necrosis by impairing the vasodilation of arterioles within the skeletal muscle during exercise, leading to local ischemia [13].

Beyond transient states, latent genetic variances play a significant role. While specific pathologies like Sickle Cell Trait pose a known high risk [14], there is also significant variability in the "healthy" population. Research indicates that certain individuals are "high responders" to eccentric stress due to inter-individual variability in CK clearance and membrane stability, meaning they may experience massive CK elevations from workloads that would be benign for others, even in the absence of metabolic disease [15].

3.2 Extrinsic Factors: Biomechanics and Environment

Among the extrinsic variables, the biomechanical nature of the muscle contraction is paramount. Eccentric loading - where the muscle lengthens under tension, such as during downhill running or plyometric box jumps - generates the highest degree of mechanical tension on the sarcolemma. This "braking" force causes physical shear stress on the membrane. Studies correlating exercise intensity with muscle damage markers confirm that repetitive eccentric contractions produce significantly higher peak CK levels compared to concentric-only movements, making this the primary driver in "cluster" outbreaks of rhabdomyolysis in gym settings [16, 4]. This mechanical stress is often compounded by thermal strain. Training in high ambient temperatures increases the rate of ATP hydrolysis and promotes sweating, which cycles back to exacerbate the intrinsic risk of dehydration and electrolyte loss [17].

3.3 Pharmacological Risks

A growing concern in amateur sports nephrology is the unsupervised use of pharmaceuticals and supplements. Non-steroidal anti-inflammatory drugs (NSAIDs) are ubiquitous, in fact, studies of ultramarathon participants have shown that up to 75% of runners ingest NSAIDs during competition, a behavior directly correlated with higher incidence rates of Acute Kidney Injury (AKI) [18]. NSAIDs inhibit the synthesis of prostaglandins necessary for maintaining afferent arteriolar dilation. When combined with the vasoconstrictive effects of myoglobin, NSAIDs effectively remove the kidney's autoregulatory safety mechanism [19].

Additionally, the management of chronic conditions in the "Masters Athlete" demographic introduces risk. HMG-CoA reductase inhibitors (statins) are widely prescribed for dyslipidemia, but they are known to potentiate exercise-induced muscle injury. Evidence suggests that vigorous exercise can unmask statin-associated muscle symptoms (SAMS) in otherwise asymptomatic patients, lowering the threshold for clinically significant rhabdomyolysis [20].

4. Clinical Presentation And Diagnosis

The traditional textbook description of rhabdomyolysis relies on a specific triad of symptoms consisting of muscle pain, weakness, and dark urine. However, relying on this presentation is a significant clinical pitfall because the full triad is present in less than 10% of confirmed cases [21]. The condition is frequently "silent," with over 50% of patients reporting no muscle pain or weakness at all, and visible pigmenturia (tea-colored urine) often appearing only in advanced stages [22].

For athletes and casual exercisers, the presentation is deceptive because it closely mimics the natural recovery process of a workout. Symptoms typically display a delayed onset, manifesting 24 to 72 hours after the precipitating event [23]. This delay frequently leads individuals to mistake the early signs for Delayed Onset Muscle Soreness (DOMS). A critical differentiation is that while DOMS generally peaks at 24 to 48 hours and improves with light movement, rhabdomyolysis pain worsens over time and is often accompanied by swelling or edema that disproportionately restricts range of motion [23].

4.1 Diagnostic Biomarkers

Serum CK is widely regarded as the gold standard for diagnosis due to its high sensitivity for muscle injury. A diagnosis is typically confirmed when CK levels exceed five times the Upper Limit of Normal (ULN), which generally corresponds to levels greater than 1000 IU/L [9].

Clinicians should note that CK levels rise within 2 to 12 hours of injury but may not peak until 24 to 72 hours post-exertion [23]. Consequently, a single normal CK test drawn immediately after an event does not conclusively rule out the condition.

Myoglobin is the direct nephrotoxin responsible for renal damage, yet it is a notoriously fickle diagnostic marker. Because it has a short half-life of approximately 2 to 3 hours, myoglobin is rapidly cleared from the bloodstream by the kidneys [9]. This rapid clearance can lead to false negatives; if blood work is delayed by even a day or two, plasma myoglobin levels may normalize even while significant renal damage is ongoing [22].

While serum creatinine is the standard metric for assessing kidney function, it is significantly flawed in athletic populations. Creatinine is a breakdown product of muscle; therefore, individuals with high muscle mass often have elevated baseline creatinine levels ("false positives"), while those experiencing acute muscle wasting may have misleadingly low levels. Cystatin C is emerging as a superior alternative biomarker. It is produced at a constant rate by all nucleated cells and, crucially, is independent of muscle mass. This makes it a far more accurate gauge of glomerular filtration rate (GFR) in muscular patients during acute rhabdomyolysis [35].

A definitive diagnostic clue often appears in the discrepancy between urine dipstick results and microscopic analysis. The orthotolidine reaction on a standard urine dipstick cannot distinguish between hemoglobin (from red blood cells) and myoglobin (from muscle tissue), as both react as "heme" [22]. This leads to the hallmark finding of rhabdomyolysis: a urinalysis that is dipstick positive for "blood" but microscopic analysis negative for red blood cells [9].

Biomarker/Test	Role	Diagnostic Findings	Clinical Implications
Serum Creatine Kinase	Gold Standard Indicator	>5 Times Upper Limit of Normal	Delayed Peak
Myoglobin	Direct Nephrotoxin	Elevated Plasma Levels	Rapid Clearance
Cystatin C	Renal Function Marker	Elevated Serum Levels	Muscle Mass Independent
Urinalysis	Screening Tool	Dipstick Dissociation	Non-Specific Reaction

Table 1. Diagnostic Markers in Rhabdomyolysis [9,22,23]

5. Management & Treatment Protocols

The immediate priority in the pre-hospital setting is the complete cessation of the inciting activity to prevent further muscle breakdown. Clinical practice guidelines emphasize that if the patient is conscious and able to tolerate liquids, aggressive oral hydration should be initiated immediately to maintain intravascular volume until intravenous access can be established [24]. Early intervention is critical, as delayed resuscitation is a primary predictor of acute kidney injury (AKI) and mortality [25].

Rapid and aggressive fluid replacement is universally accepted as the cornerstone of therapy to preserve renal function. The primary goal is to induce forced diuresis, with targets generally set at a urine output of 200–300 mL/h (or approximately 3 mL/kg/h) to flush myoglobin from the renal tubules [9]. While Isotonic Saline (0.9% NaCl) has historically been the standard, it carries the risk of hyperchloremic metabolic acidosis when used in large volumes. A pivotal trial comparing Lactated Ringer's (LR) to saline found that LR was superior in preventing metabolic acidosis, a condition that can paradoxically worsen renal injury by promoting myoglobin precipitation [26]. Systematic reviews support this, suggesting that balanced crystalloids may be the preferred initial fluid choice [7]. Recent meta-analyses further confirm that balanced crystalloids are associated with lower mortality and reduced AKI incidence compared to saline in critically ill patients [27].

The administration of sodium bicarbonate ($NaHCO_3$) to alkalinize the urine (target pH > 6.5) is theoretically sound, as an acidic environment promotes the precipitation of myoglobin into obstructive casts. However, clinical evidence supporting this practice is weak. Retrospective studies have repeatedly failed to show a significant difference in rates of renal failure between patients treated with bicarbonate and those treated with saline alone [28]. Comprehensive reviews indicate that the risks of alkalinization - such as worsening hypocalcemia and metabolic alkalosis - often outweigh the theoretical benefits [29]. Furthermore, propensity score-matched cohort studies have demonstrated that bicarbonate therapy does not reduce the need for dialysis or improve survival rates [30].

The routine use of diuretics like mannitol or loop diuretics is largely discouraged unless the patient is volume overloaded. While mannitol theoretically scavenges free radicals, it has not consistently demonstrated a clinical benefit over aggressive hydration alone and may be detrimental if the patient is hypovolemic [31]. Dialysis does not effectively remove myoglobin due to the molecule's size. Therefore, Renal Replacement Therapy (RRT) is indicated strictly for refractory complications, such as life-threatening hyperkalemia, severe metabolic acidosis, or volume overload [9, 31].

6. Outcomes

The overall long-term prognosis for exertional rhabdomyolysis is generally favorable, as the vast majority of athletes regain full renal function with appropriate management. However, the development of Acute Kidney Injury (AKI) during the acute phase is not a benign event. Longitudinal evidence suggests that even a solitary episode of rhabdomyolysis-associated AKI serves as a significant independent risk factor for the future development of Chronic Kidney Disease (CKD) and hypertension later in life [32].

Beyond the immediate threat of renal failure, clinicians must remain vigilant for two critical systemic complications that require immediate recognition. The first, Compartment Syndrome, represents a surgical emergency characterized by intramuscular edema within a closed fascial compartment that rises to pressures sufficient to compromise microvascular perfusion. This condition is disproportionately prevalent in the rhabdomyolysis population and frequently mandates urgent fasciotomy to prevent permanent ischemic damage or limb loss [25]. Additionally, severe cases may precipitate Disseminated Intravascular Coagulation (DIC). This life-threatening cascade is initiated by the release of thromboplastin from necrotic muscle tissue, triggering a paradoxical state of systemic coagulation and simultaneous hemorrhage, particularly in patients who experience delays in initial treatment [25].

Returning to sport is a structured process rather than a singular event. To ensure athlete safety, the Consortium for Health and Military Performance (CHAMP) and other sports medicine bodies recommend a risk-stratified approach that separates uncomplicated cases from those with underlying pathologies.

The Low Risk classification applies to athletes experiencing a single, isolated episode triggered by a clearly identifiable and substantial physical stressor, such as performing excessive volume without prior conditioning. Clinically, these patients demonstrate a rapid clearance of creatine kinase (CK) levels and show no evidence of renal injury. Consequently, they generally do not require advanced metabolic testing and may proceed directly to the graded return-to-play protocol once they are asymptomatic [15,16].

Conversely, the High Risk category encompasses cases that raise suspicion of an intrinsic metabolic myopathy or genetic disorder. This classification is assigned to athletes presenting with specific clinical "red flags," most notably massive CK elevations exceeding 20,000 to 30,000 IU/L or any evidence of Acute Kidney Injury (AKI). Furthermore, clinicians must flag patients who exhibit a disproportionate physiological response, where rhabdomyolysis occurs following only mild-to-moderate exertion that would not typically induce muscle failure. A history of recurrent episodes or the presence of known genetic

conditions, such as Sickle Cell Trait, McArdle Disease (Glycogen Storage Disease Type V), or Carnitine Palmitoyltransferase II (CPT II) deficiency, also mandates a high-risk designation. Individuals meeting these criteria are disqualified from immediately entering the return-to-play protocol and require a mandatory referral to a specialist for a comprehensive metabolic and genetic workup [33].

6.1 The Phased Return-to-Play Protocol

For standard uncomplicated cases, the return-to-play timeline is a structured, graded process that typically spans several weeks. The initial stage, designated as Phase I or the Recovery Phase, focuses on the restoration of baseline functional status. This period mandates a complete cessation of athletic activity for a minimum of two weeks following the normalization of mild creatine kinase elevations, with extended durations required for more severe presentations. The primary clinical objectives during this window are the complete resolution of myalgia and the sustained normalization of urine pigment to ensure renal recovery is absolute before placing any demand on the musculoskeletal system [24, 33].

Upon successful clearance, the athlete advances to Phase II, the Initiation Phase. This stage involves the introduction of light, non-impact aerobic conditioning, such as aquatic jogging or stationary cycling, designed to assess physiological tolerance without impact trauma. Strict monitoring for the recurrence of dark urine or soft tissue edema is mandatory during these initial efforts. Subsequently, Phase III, the Progression Phase, marks the gradual re-introduction of resistance training. Protocols dictate a high-repetition, low-load volume strategy to rebuild muscular endurance without imposing excessive mechanical shear [15,33].

Crucially, eccentric loading - the lengthening of muscle under tension - must be strictly limited during this phase, as it generates the highest mechanical stress on recovering muscle fibers and poses the greatest risk of injury recurrence. The final stage, Phase IV, permits a return to full sport-specific practice, authorized only after the athlete has successfully negotiated the resistance progression of Phase III without the recurrence of symptoms or biomarkers of muscle injury [16,33].

Phase	Designation	Duration and Entry Criteria	Activity Guidelines	Clinical Restrictions
I	**Recovery**	**Minimum 2 Weeks** Post Normalization of CK and Urine	Daily Living Activities Only	**Complete Rest.** No athletic activity.
II	**Initiation**	**Varies Based on Tolerance**	Light, Non-Impact, Aerobic Activity	**Heart Rate Monitoring.** Strict observation.
III	**Progression**	**Varies Based on Strength Gains**	Gradual Resistance Training	**Avoid Eccentric Loading.** High Mechanical Stress Must be Minimized.
IV	**Sport Specific**	**Indefinite**	Return to Full Practice	**Clearance required.** Must Complete Phase III symptom-free.

Table 2. The Phased Return-to-Play Protocol [15,16,24,33].

7. Discussion: Implications for Sport Quality and Future Directions

The prevailing "culture of push" within commercial gyms and endurance events often obscures the critical warning signs of physiological failure, normalizing symptoms that should otherwise prompt immediate medical attention. To mitigate this, institutions have a responsibility to implement passive education strategies that function without active coaching intervention. A

simple yet effective measure is the installation of urine color charts in restrooms at training facilities and event venues. These visual aids empower athletes to self-monitor their hydration status and identify pigmenturia - the hallmark "tea-colored" urine of rhabdomyolysis - at its earliest onset [34].

Furthermore, educational efforts must target the pervasive misconception regarding prophylactic pain management. Athletes are frequently unaware that "pre-loading" with non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen or naproxen prior to competition significantly heightens renal risk. Physiologically, these medications constrict the afferent arterioles of the kidney. When this vasoconstriction is combined with the nephrotoxic effects of myoglobin and exercise-induced dehydration, the kidney's autoregulatory safety mechanisms are effectively disabled, compounding the risk of AKI [18].

A critical ethical distinction must also be drawn between training for mental resilience and ignoring the signs of physiological collapse. Rhabdomyolysis frequently occurs in environments where athletes are coached to override their body's innate safety signals under the guise of "mental toughness." Ethical coaching requires a paradigm shift that recognizes extreme localized swelling, disproportionate pain, and the acute loss of range of motion not as signs of weakness to be pushed through, but as objective clinical indicators of cellular necrosis.

From a diagnostic perspective, the current reliance on serum Creatine Kinase and creatinine has inherent limitations in athletic populations. Creatinine levels are naturally higher in individuals with significant muscle mass, leading to frequent "false positive" concerns regarding renal function, while simultaneously masking acute declines in filtration during injury. Future research is increasingly pivoting toward Cystatin C as a superior biomarker for renal assessment in athletes. Unlike creatinine, Cystatin C production is not influenced by muscle mass. This independence makes it a far more accurate gauge of glomerular filtration rate (GFR) in muscular populations, offering a clearer window into renal health during the management of rhabdomyolysis [35].

8. Disclosure

Authors contribution statement:

Conceptualization: Paulina Tymińska, Julia Frączek, Szymon Domagała, Karolina Borówka, Piotr Górka, Natalia Kawka, Witold Kądziołka
Methodology: Paulina Tymińska,Witold Kądziołka, Piotr Górka
Software check: Julia Frączek, Szymon Domagała, Piotr Górka
Formal analysis: Paulina Tymińska, Julia Frączek, Karolina Borówka
Investigation: Paulina Tymińska, Karolina Borówka, Natalia Kawka
Resources: Witold Kądziołka, Piotr Górka, Natalia Kawka
Data curation: Szymon Domagała, Karolina Borówka
Writing - rough preparation: Paulina Tymińska, Julia Frączek, Witold Kądziołka, Piotr Górka
Writing - review and editing: Paulina Tymińska, Julia Frączek, Karolina Borówka, Natalia Kawka, Szymon Domagała
Visualization: Witold Kądziołka, Piotr Górka, Natalia Kawka
Supervision: Paulina Tymińska, Karolina Borówka, Natalia Kawka
Project administration: Paulina Tymińska, Julia Frączek, Piotr Górka
Receiving funding, -

All authors have read and agreed with the published version of the manuscript.

Funding Statement
All authors state that the study did not receive any funding.

Conflict of Interest Statement
None of the authors declare any conflict of interest.

References

1. Khan FY. “Rhabdomyolysis: a review of the literature”. *Neth J Med*. 2009 Oct;67(9):272-83. PMID: 19841484.
2. DeFilippis EM, Kleiman DA, Derman PB, DiFelice GS, Eachempati SR. “Spinning-induced Rhabdomyolysis and the Risk of Compartment Syndrome and Acute Kidney Injury: Two Cases and a Review of the Literature”. *Sports Health.* 2014 Jul;6(4):333-5. doi: 10.1177/1941738114522957. PMID: 24982706; PMCID: PMC4065558.
3. Boden BP, Isaacs DJ, Ahmed AE, Anderson SA. Epidemiology of Exertional Rhabdomyolysis in the United States: Analysis of NEISS Database 2000 to 2019. Phys Sportsmed. 2022 Dec;50(6):486-493. doi: 10.1080/00913847.2021.1956288. Epub 2021 Jul 30. PMID: 34278922.
4. Rawson, E. S., Clarkson, P. M., & Tarnopolsky, M. A. (2017). "Perspectives on Exertional Rhabdomyolysis." *Sports Medicine*, 47(Suppl 1), 33–49. DOI: 10.1007/s40279-017-0689-z
5. Giannoglou, G. D., Chatzizisis, Y. S., & Misirli, G. (2007). The syndrome of rhabdomyolysis: Pathophysiology and diagnosis. *European Journal of Internal Medicine*, 18(2), 90–100. DOI: 10.1016/j.ejim.2006.09.020
6. Torres, P. A., Helmstetter, J. A., Kaye, A. M., & Kaye, A. D. (2015). Rhabdomyolysis: pathogenesis, diagnosis, and treatment. *The Ochsner Journal*, 15(1), 58–69.
7. Chavez, L. O., Leon, M., Einav, S., & Varon, J. (2016). Beyond muscle destruction: A systematic review of rhabdomyolysis for clinical practice. *Critical Care*, 20(1), 135. DOI: 10.1186/s13054-016-1314-5
8. Petejova, N., & Martinek, A. (2014). Acute kidney injury due to rhabdomyolysis and renal replacement therapy: A critical review. *Critical Care*, 18(3), 224. DOI: 10.1186/cc13897
9. Bosch, X., Poch, E., & Grau, J. M. (2009). "Rhabdomyolysis and Acute Kidney Injury." *The New England Journal of Medicine*, 361(1), 62–72. DOI: 10.1056/NEJMra0801327
10. Holt, S. G., & Moore, K. P. (2001). "Pathogenesis and treatment of renal dysfunction in rhabdomyolysis." *Intensive Care Medicine*, 27(5), 803–811. DOI: 10.1007/s001340100878
11. Zager, R. A. (1996). "Rhabdomyolysis and myohemoglobinuric acute renal failure." *Kidney International*, 49(2), 314–326. DOI: 10.1038/ki.1996.48

12. Rosner, M. H., & Kirven, J. (2007). Exercise-associated hyponatremia. *Clinical Journal of the American Society of Nephrology*, 2(1), 151–161. DOI: 10.2215/CJN.02730806
13. Bo-Fan Yang, Duo Li, Chun-Li Liu, Yu Luo, Jie Shi, Xiao-Qin Guo, Hao-Jun Fan, Qi Lyu, "Advances in rhabdomyolysis: A review of pathogenesis, diagnosis, and treatment", *Chinese Journal of Traumatology*, Volume 29, Issue 1, 2026, Pages 21-31, ISSN 1008-1275, https://doi.org/10.1016/j.cjtee.2024.10.005.
14. Bäcker HC, Richards JT, Kienzle A, Cunningham J, Braun KF. Exertional Rhabdomyolysis in Athletes: Systematic Review and Current Perspectives. *Clin J Sport Med*. 2023 Mar 1;33(2):187-194. doi: 10.1097/JSM.0000000000001082. Epub 2022 Oct 20. PMID: 36877581.
15. Baird, M. F., Graham, S. M., Baker, J. S., & Bickerstaff, G. F. (2012). Creatine-kinase- and exercise-related muscle damage implications for muscle performance and recovery. *Journal of Nutrition and Metabolism*, 2012, 960363. DOI: 10.1155/2012/960363
16. Staniszewski M, Tkaczyk J, Kęska A, Zybko P, Mróz A. Effect of rest duration between sets on fatigue and recovery after short intense plyometric exercise. *Sci Rep*. 2024 Jul 2;14(1):15080. doi: 10.1038/s41598-024-66146-2. PMID: 38956280; PMCID: PMC11219752.
17. Garcia CK, Renteria LI, Leite-Santos G, Leon LR, Laitano O. Exertional heat stroke: pathophysiology and risk factors. *BMJ Med*. 2022 Oct 11;1(1):e000239. doi: 10.1136/bmjmed-2022-000239. PMID: 36936589; PMCID: PMC9978764.
18. Lipman GS, Shea K, Christensen M, et al. Ibuprofen versus placebo effect on acute kidney injury in ultramarathons: a randomised controlled trial. *Emerg Med J*. 2017;34(10):637-642. https://10.1136/emermed-2016-206353
19. Paoloni, J. A., et al. (2009). Non-steroidal anti-inflammatory drugs in sports medicine: guidelines for practical prescribing. *British Journal of Sports Medicine*, 43(11), 863–865. DOI: 10.1136/bjsm.2009.059980
20. Thompson, P. D., et al. (2016). Statin-Associated Side Effects. *Journal of the American College of Cardiology*, 67(20), 2395–2410. DOI: 10.1016/j.jacc.2016.02.071
21. McMahon GM, Zeng X, Waikar SS. A risk prediction score for kidney failure or mortality in rhabdomyolysis. *JAMA Intern Med*. 2013;173(18):1639–1646. doi:10.1001/jamainternmed.2013.9774
22. Cervellin G, Comelli I, Lippi G. Rhabdomyolysis: historical background, clinical, diagnostic and therapeutic features. *Clin Chem Lab Med*. 2010;48(6):749–756. doi:10.1515/CCLM.2010.151

23. Scalco RS, Snoeck M, Quinlivan R, Treves S, Laforét P, Jungbluth H, Voermans NC. Exertional rhabdomyolysis: physiological response or manifestation of an underlying myopathy? *BMJ Open Sport Exerc Med*. 2016 Sep 7;2(1):e000151. doi: 10.1136/bmjsem-2016-000151. PMID: 27900193; PMCID: PMC5117086.
24. Nye NS, Kasper K, Madsen CM, Szczepanik M, Covey CJ, Oh R, Kane S, Beutler AI, Leggit JC, Deuster PA, O'Connor FG. Clinical Practice Guidelines for Exertional Rhabdomyolysis: A Military Medicine Perspective. *Curr Sports Med Rep*. 2021 Mar 1;20(3):169-178. doi: 10.1249/JSR.0000000000000822. PMID: 33655999.
25. Rout P, Chippa V, Adigun R. Rhabdomyolysis. 2025 Jul 7. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 28846335.
26. Cho YS, Lim H, Kim SH. Comparison of lactated Ringer's solution and 0.9% saline in the treatment of rhabdomyolysis induced by doxylamine intoxication. *Emerg Med J*. 2007;24(4):276–280. doi:10.1136/emj.2006.043265
27. Chen Y, Gao Y. Comparison of Balanced Crystalloids versus Normal Saline in Critically Ill Patients: A Systematic Review with Meta-Analysis. *Ther Clin Risk Manag*. 2023;19:783-799. doi:10.2147/TCRM.S416785
28. Brown CV, Rhee P, Chan L, et al. Preventing renal failure in patients with rhabdomyolysis: do bicarbonate and mannitol make a difference?. *J Trauma*. 2004;56(6):1191–1196. doi:10.1097/01.ta.0000130761.78627.10
29. Somagutta MR, et al. Role of Bicarbonates and Mannitol in Rhabdomyolysis: A Comprehensive Review. *Cureus*. 2020;12(8):e9742. doi:10.7759/cureus.9742
30. Kim HW, et al. Role of bicarbonate and volume therapy in the prevention of acute kidney injury in rhabdomyolysis: a retrospective propensity score-matched cohort study. *Kidney Res Clin Pract*. 2022;41(3):310-321. doi:10.23876/j.krcp.21.144
31. Zimmerman JL, Shen MC. Rhabdomyolysis. *Chest*. 2013;144(3):1058-1065. doi:10.1378/chest.12-2016
32. Long B, Koyfman A, Gottlieb M. An evidence-based narrative review of the emergency department evaluation and management of rhabdomyolysis. *Am J Emerg Med*. 2019 Mar;37(3):518-523. doi: 10.1016/j.ajem.2018.12.061. Epub 2019 Jan 2. PMID: 30630682.
33. O'Connor FG, Brennan FH, Campbell W, Heled Y, Deuster P. Return to physical activity after exertional rhabdomyolysis. *Curr Sports Med Rep*. 2008;7(6):328–331. doi:10.1249/JSR.0b013e31818f0317

34. Cleary, Michelle & Ruiz, Daniel & Eberman, Lindsey & Mitchell, Israel & Binkley, Helen. (2007). Dehydration, Cramping, and Exertional Rhabdomyolysis: A Case Report with Suggestions for Recovery. *Journal of sport rehabilitation*. 16. 244-59. 10.1123/jsr.16.3.244.

35. Haines RW, Fowler AJ, Liang K, Pearse RM, Larsson AO, Puthucheary Z, Prowle JR. Comparison of Cystatin C and Creatinine in the Assessment of Measured Kidney Function during Critical Illness. *Clin J Am Soc Nephrol*. 2023 Aug 1;18(8):997-1005. doi: 10.2215/CJN.0000000000000203. Epub 2023 May 31. PMID: 37256861; PMCID: PMC10564373.