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EXERCISE- AND DEHYDRATION-ASSOCIATED RHABDOMYOLYSIS: PATHOPHYSIOLOGY, CLINICAL FEATURES, MANAGEMENT, AND PEDIATRIC CONSIDERATIONS.

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ABSTRACT

Introduction: *Rhabdomyolysis is a clinical syndrome characterized by skeletal muscle breakdown with subsequent release of intracellular components, including myoglobin and creatine kinase, into the circulation. Strenuous physical exercise associated with dehydration represents an important trigger and may lead to severe complications such as acute kidney injury.*
Objective: *To review current evidence on rhabdomyolysis and its association with physical exercise, dehydration, and related clinical complications, including considerations in pediatric populations.*
Methodology: *A narrative literature review was conducted using PubMed, Cochrane Library, and Google Scholar databases. A total of 32 relevant articles published in English, Spanish, and Portuguese were selected based on relevance, methodological quality, and clinical applicability. The analyzed literature addressed pathophysiological, clinical, diagnostic, therapeutic, and preventive aspects of rhabdomyolysis associated with strenuous physical exercise, dehydration, and pediatric considerations.*
Results: *The review demonstrated a consistent association between strenuous physical exercise, dehydration, and the development of rhabdomyolysis, characterized by significant elevations in creatine phosphokinase levels and an increased risk of acute kidney injury. Hypovolemia and reduced renal perfusion were identified as key factors influencing clinical severity. Pediatric data also indicate that children and adolescents are susceptible to rhabdomyolysis, particularly in the presence of dehydration and intense physical activity, with early recognition and adequate hydration being essential to prevent complications.*
Conclusions: *Exercise-induced rhabdomyolysis is significantly exacerbated by dehydration, increasing muscle damage and the risk of renal complications. Early recognition, adequate hydration, and preventive strategies remain essential to reduce morbidity and improve clinical outcomes in both adult and pediatric populations.*
KEYWORDS: *Rhabdomyolysis; Exercise-Induced Rhabdomyolysis; Dehydration; Acute Kidney Injury; Pediatric; Skeletal Muscle.*

INTRODUCTION

Rhabdomyolysis is defined as the breakdown of skeletal muscle, characterized by the leakage of intracellular contents—including myoglobin, creatine kinase (CK), sarcoplasmic proteins, and electrolytes—into the extracellular fluid and systemic circulation. Etymologically, the term rhabdomyolysis derives from the Greek words *rhabdos* (striated), *mys* (muscle), and *lysis* (breakdown), reflecting the underlying process of muscle destruction. This clinical syndrome has a multifactorial origin, including traumatic, toxic, and metabolic causes; however, extreme physical exertion has become one of the most relevant etiologies in current clinical practice. In particular, rhabdomyolysis associated with intense exercise and dehydration has gained increasing importance in various sports settings and during prolonged physical activity. The massive release of muscle components into the bloodstream can lead to systemic complications, most notably acute kidney injury, which is of special concern in the context of growing participation in endurance events and its potential impact on renal function and health (1–3).

This study initially provides an overview of rhabdomyolysis and subsequently analyzes the role of dehydration and strenuous exercise in its development and in the occurrence of renal complications.

METHODOLOGY

A narrative literature review was conducted to analyze current evidence regarding rhabdomyolysis associated with

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physical exercise and dehydration. A total of 40 articles were initially identified, including original studies, systematic and narrative reviews, case reports, and clinical trials. After screening for relevance and methodological quality, 32 studies were selected for inclusion, while studies that did not significantly contribute to the objectives of the present review were excluded.

The information sources consulted included the PubMed, Cochrane Library, and Google Scholar databases. The bibliographic search was performed in English, Spanish, and Portuguese using the following keywords: rhabdomyolysis, exercise-induced rhabdomyolysis, physical exercise, dehydration, acute kidney injury, skeletal muscle, and pediatric rhabdomyolysis, combined using Boolean operators such as AND and OR.

Articles were selected based on relevance to the objectives of the study, scientific quality, and clinical applicability. The selected literature allowed for the analysis of the pathophysiological, clinical, diagnostic, therapeutic, and preventive aspects of rhabdomyolysis, with emphasis on its association with strenuous physical exercise, dehydration, and pediatric considerations.

DEVELOPMENT

Brief History

The recognition of rhabdomyolysis dates back to antiquity, with compatible descriptions found in biblical accounts, in which the Israelites developed symptoms of myolysis after consuming quail during the Exodus from Egypt—birds that ingested toxic plants without being affected during their spring migration. Similar phenomena were later described in the Mediterranean region following the consumption of other migratory birds, such as robins, finches, and larks, which are resistant to toxic alkaloids present in herbs such as hemlock. In the modern era, rhabdomyolysis gained clinical relevance in the context of wars, earthquakes, and natural or anthropogenic disasters associated with crush injuries. A major milestone occurred in 1943, when Bywaters and Stead identified myoglobin as the main agent responsible for acute tubular necrosis observed in patients with dark-colored urine, thereby establishing the pathophysiological basis of renal injury associated with rhabdomyolysis. With the development of modern sports and high-demand physical activities, strenuous exercise and dehydration became key contemporary factors in the occurrence of rhabdomyolysis, extending its relevance beyond classic traumatic contexts (2,4,5).

Etiology

The etiology of rhabdomyolysis is broad and multifactorial and can be generally classified into traumatic or physical causes and non-traumatic causes. Among the most frequently associated factors are strenuous physical exercise, trauma, and excessive alcohol consumption. Physical causes include crush injuries, polytrauma, burns, electrocution, prolonged immobilization, muscle ischemia secondary to vascular compression, as well as states of hyperthermia or hypothermia. Within this group, intense exercise—especially in untrained individuals or those exposed to extreme environmental conditions—represents one of the most relevant causes in current clinical practice, as sustained muscle activity combined with dehydration and electrolyte loss through sweating promotes disruption of the muscle membrane and the release of intracellular components. Exercise-induced rhabdomyolysis has been described mainly in marathon runners, military personnel, and weightlifters, although isolated cases following low-intensity activities have also been reported (3,6–8).

Non-traumatic causes include metabolic and electrolyte disorders, infections, endocrine diseases, inherited myopathies, and the use of medications or toxic substances. Among drugs, statins are notable, as statin-associated rhabdomyolysis is rare but clinically relevant, particularly in the presence of risk factors such as hypothyroidism, liver disease, or concomitant use of fibrates. Likewise, the use of substances such as cocaine, amphetamines, alcohol, and opioids may induce rhabdomyolysis through mechanisms including hyperthermia, vasoconstriction, direct muscle toxicity, and prolonged immobilization. Viral and bacterial infections, sepsis, and admission to intensive care units also represent high-risk scenarios due to the combination of tissue hypoxia, electrolyte disturbances, and polypharmacy. In many cases, rhabdomyolysis results from the interaction between individual susceptibility—including genetic predisposition—and one or more external triggers, with strenuous exercise and dehydration being key elements that potentiate muscle damage and the risk of systemic complications, particularly acute kidney injury (9–13).

Pathophysiology

Rhabdomyolysis shares a common final pathophysiological pathway characterized by direct myocyte injury or failure of intracellular energy supply, leading to disruption of sarcoplasmic membrane integrity and ATP-dependent ionic homeostasis. Factors such as trauma, strenuous physical exercise, hyperthermia, ischemia, electrolyte disturbances, or drugs induce massive influx of sodium and calcium into the cell, resulting in edema, structural dysfunction, activation of proteolytic enzymes, energy depletion, and muscle necrosis. The subsequent inflammatory and reperfusion responses intensify myolysis and release myoglobin, creatine kinase, potassium, phosphate, and uric acid into the circulation, producing characteristic metabolic disturbances. Circulating myoglobin, particularly in settings of hypovolemia and dehydration associated with strenuous exercise, is filtered and precipitates within the renal tubules, where it induces oxidative toxicity, cast formation, and vasoconstriction, contributing to the development of acute kidney injury. In addition, muscle edema may trigger compartment syndrome, while the pro-inflammatory and thrombogenic effects of heme promote disseminated intravascular coagulation, consolidating rhabdomyolysis as a potentially severe multisystem disorder (3,14–18).

Figure 1. Pathophysiology of Exercise- and Dehydration-Associated Rhabdomyolysis

Strenuous physical exercise and dehydration promote skeletal muscle injury, leading to the release of intracellular components such as myoglobin and creatine phosphokinase (CPK). These changes contribute to hypovolemia, electrolyte

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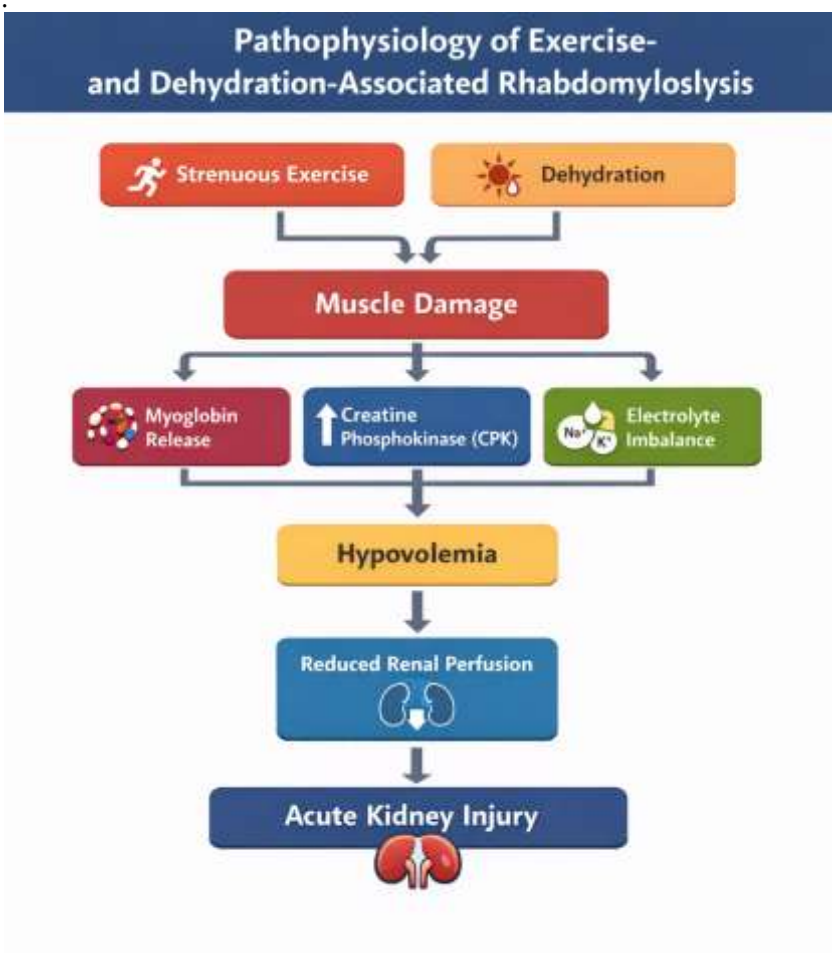
imbalance, reduced renal perfusion, and ultimately increase the risk of acute kidney injury.



Source: The Authors.

Histopathology

Muscle biopsy is useful when metabolic myopathies are suspected; however, its diagnostic yield depends on the timing of the procedure, as extensive necrosis during the acute phase of rhabdomyolysis may obscure an underlying pathology. Therefore, biopsy is recommended after clinical recovery. According to the European Federation of Neurological Societies, it is indicated in patients with muscle pain or weakness, moderate elevation of creatine phosphokinase, myoglobinuria, alterations in muscle trophism, or suggestive electromyographic findings. Histochemical staining allows differentiation of metabolic and mitochondrial myopathies as well as enzymatic deficiencies, whereas renal biopsy in rhabdomyolysis-associated acute kidney injury demonstrates tubular necrosis and pigmented casts consistent with myoglobin toxicity.

Evaluation and Diagnosis

Although the classic triad of rhabdomyolysis—muscle pain, weakness, and dark urine—is characteristic, it is present in fewer than half of cases. Muscle pain is the most frequent symptom, followed by proximal weakness and myoglobinuria, while systemic symptoms are often nonspecific. Diagnosis requires a high index of suspicion, particularly in patients with a history of intense exercise, dehydration, trauma, medication use, or substance abuse. Physical examination should assess local signs of muscle injury, hydration status, and, in traumatic cases, neurovascular compromise or compartment syndrome.

Serum creatine phosphokinase (CPK) elevation is the main diagnostic criterion, with rhabdomyolysis generally defined as levels exceeding five times the upper limit of normal. The CK-MM isoenzyme reflects skeletal muscle injury, showing early elevation and peaking between the first and fifth day. Levels above 5,000 IU/L are associated with a higher risk of acute kidney injury. Myoglobinuria can be detected by urine dipstick testing positive for blood in the absence of erythrocytes on microscopic examination. Laboratory studies commonly reveal electrolyte disturbances such as hyperkalemia, hyperphosphatemia, initial hypocalcemia, and metabolic acidosis.

Initial evaluation should include a complete blood count, metabolic panel, CPK measurement, urinalysis, electrocardiography, and imaging studies as clinically indicated. The ECG helps identify abnormalities secondary to electrolyte disturbances, while computed tomography may be useful when compartment syndrome is suspected. Advanced diagnostic tests are not required to confirm rhabdomyolysis; however, muscle biopsy may be considered after complete recovery in recurrent cases or when underlying metabolic or inflammatory myopathies are suspected (3,6,19,20).

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Dehydration and rhabdomyolysis

Severe dehydration constitutes a key potentiating factor in the pathophysiology of rhabdomyolysis, as it contributes both to its onset and to greater clinical severity. Significant fluid loss reduces effective intravascular volume, compromises tissue and renal perfusion, and limits the kidney's ability to eliminate myoglobin and other muscle breakdown products released into the bloodstream after myocyte injury. In this context, reduced renal flow promotes tubular concentration of heme pigments, facilitating their precipitation and enhancing direct tubular damage, particularly in the presence of metabolic acidosis.

During prolonged or high-intensity physical exercise, especially in hot or highly humid environments, excessive sweating without adequate fluid replacement leads to a state of relative hypovolemia that exacerbates exercise-induced muscle stress. This combination of dehydration and strenuous exercise has been repeatedly described in clinical studies and case reports, particularly among young athletes, military personnel, and workers exposed to high physical workloads, in whom marked elevations of creatine kinase and myoglobinuria have been observed. In addition, dehydration disrupts electrolyte homeostasis, favoring imbalances such as hypokalemia or hypernatremia, which increase muscle membrane vulnerability and facilitate skeletal muscle fiber necrosis.

Although rhabdomyolysis may develop in the absence of dehydration, available evidence suggests that the coexistence of fluid loss and intense physical exertion significantly increases the risk of more severe disease forms and systemic complications, including acute kidney injury. Therefore, adequate hydration before, during, and after exercise represents a fundamental preventive measure, especially in individuals exposed to demanding training regimens or extreme environmental conditions, and constitutes an essential pillar in the prevention of exercise-associated rhabdomyolysis (21,22).

Physical exercise and rhabdomyolysis

Intense and unaccustomed physical exercise is a widely recognized trigger of rhabdomyolysis, particularly when it involves prolonged activities, high intensity, or a predominance of eccentric contractions for which the individual is not adequately adapted. This phenomenon is especially relevant in endurance sports and vigorous training programs such as marathons, ultradistance races, military training, high-intensity cross-training, and weightlifting. In these settings, structural damage to muscle fibers results from an imbalance between energy demand and the metabolic capacity of the myocyte, leading to disruption of the cell membrane and massive release of myoglobin, creatine kinase, and other intracellular components into the circulation.

The risk of exercise-induced rhabdomyolysis increases when physical effort exceeds the limits of physiological adaptation, particularly in untrained individuals or those exposed to abrupt increases in exercise intensity or duration. Moreover, environmental heat, high humidity, inappropriate clothing, and insufficient recovery between training sessions further potentiate muscle damage and accelerate myolytic mechanisms. The literature has shown that repeated eccentric contractions generate greater mechanical stress on the sarcolemma, promoting excessive calcium influx into the myocyte and activating proteolytic cascades responsible for muscle degradation.

From a clinical perspective, exercise-induced rhabdomyolysis encompasses a wide spectrum of severity, ranging from asymptomatic elevations of creatine kinase to severe presentations complicated by myoglobinuria, electrolyte disturbances, and acute kidney injury. The coexistence of factors such as dehydration, nonsteroidal anti-inflammatory drug use, intercurrent infections, or underlying metabolic diseases significantly increases the likelihood of systemic complications. In this context, early symptom recognition, identification of at-risk populations, and implementation of preventive strategies—such as gradual training progression, adequate hydration, and clinical monitoring—are essential to reduce the incidence and severity of exercise-associated rhabdomyolysis (23–25).

Treatment

The management of rhabdomyolysis is primarily aimed at preserving renal perfusion and preventing acute kidney injury, with early and adequate fluid resuscitation as the cornerstone of therapy. In cases associated with strenuous exercise and dehydration, immediate cessation of physical activity, correction of volume depletion, and normalization of the hydroelectrolytic state are key measures. Resuscitation with isotonic crystalloids should be initiated promptly and adjusted to maintain a target urine output of 200–300 mL/h, avoiding both hypovolemia and volume overload. Treatment includes identification and elimination of the triggering factor, close monitoring of urine output, serial surveillance of creatine phosphokinase levels, and correction of electrolyte disturbances, particularly hyperkalemia, hypocalcemia, and hyperphosphatemia. Urinary alkalinization with bicarbonate may be considered in selected severe cases, always with caution due to the risk of hypocalcemia and alkalosis. The use of mannitol or diuretics is not routine and should be individualized, being reserved for specific situations with adequate prior diuresis (26–29).

In patients who develop complications such as compartment syndrome, refractory hyperkalemia, fluid overload, or renal failure with oliguria or anuria, specific interventions are required, including hemodialysis. In exercise- and dehydration-induced rhabdomyolysis, early recognition and timely hydration are usually sufficient for a favorable outcome, significantly reducing the risk of systemic complications.

Pediatric Considerations

Rhabdomyolysis in pediatric populations presents distinct clinical considerations compared with adults. In children and adolescents, common etiologies include viral infections, trauma, metabolic disorders, and intense physical activity, particularly among young athletes involved in competitive sports or high-intensity training. Dehydration represents a relevant contributing factor in pediatric patients, as children are more susceptible to fluid imbalance due to physiological differences in thermoregulation and fluid distribution. Clinical manifestations may be nonspecific, and early symptoms such as muscle pain, fatigue, or dark-colored urine can be underrecognized, delaying diagnosis. Although diagnostic criteria and treatment principles are similar to those

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in adults, careful monitoring of fluid therapy is essential to prevent complications such as fluid overload or electrolyte imbalance. Early recognition and adequate hydration remain fundamental strategies to reduce morbidity and prevent acute kidney injury in pediatric patients(30-32).

RESULTS

The analysis of the selected literature shows that rhabdomyolysis associated with physical exercise and dehydration represents a frequent clinical entity in contexts of intense exertion, particularly among athletes, military personnel, and individuals exposed to adverse environmental conditions. The reviewed studies consistently demonstrate that unaccustomed strenuous exercise and significant fluid loss act synergistically, increasing skeletal muscle breakdown and promoting the release of myoglobin and creatine kinase into the circulation. Dehydration was also identified as a major factor associated with an increased risk of acute kidney injury by compromising renal perfusion and impairing the elimination of muscle breakdown products. Marked elevation of creatine phosphokinase (CPK) levels and myoglobinuria were consistent findings in the most severe cases, whereas early and adequate hydration was associated with a favorable clinical course and a lower incidence of renal complications.

Additionally, pediatric data included in the reviewed literature indicate that children and adolescents are also vulnerable to rhabdomyolysis, particularly in the presence of viral infections, trauma, or intense physical activity combined with dehydration. In these populations, nonspecific symptoms may delay diagnosis, reinforcing the importance of early clinical suspicion and timely fluid management to reduce the risk of complications such as acute kidney injury.

CONCLUSIONS

Rhabdomyolysis is a multisystem clinical syndrome of multifactorial etiology in which strenuous physical exercise and dehydration play a central role in its onset and severity in current clinical practice. The reviewed evidence demonstrates that intense physical exertion, particularly when it exceeds the individual's adaptive capacity and is associated with hypovolemia, promotes myocyte injury, massive release of intracellular components, and the development of systemic complications, with acute kidney injury representing the most clinically significant outcome.

Dehydration acts as a major potentiating factor by reducing renal perfusion, altering electrolyte balance, and facilitating myoglobin-mediated tubular toxicity, thereby increasing the severity of the clinical presentation. Early diagnosis, primarily based on elevated creatine kinase levels and the identification of predisposing risk factors, remains essential to initiate timely and effective treatment.

Early and adequate fluid replacement continues to represent the cornerstone of both therapeutic management and prevention, playing a decisive role in improving clinical outcomes and reducing associated morbidity. Preventive strategies—including adequate hydration, gradual progression of physical training, and early identification of high-risk individuals, including pediatric populations—are fundamental to reducing the incidence and severity of exercise- and dehydration-induced rhabdomyolysis, particularly in settings of intense or prolonged physical activity.

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