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Recent Advances and Updates in Rhabdomyolysis: A Comprehensive Review

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Abstract

Rhabdomyolysis (RML) is a complex clinical syndrome marked by the breakdown of skeletal muscle cells, with historical recognition dating back to biblical times and modern understanding evolving since World War II. This comprehensive review explores the diverse landscape of RML, encompassing its epidemiology, etiology, pathogenesis, clinical manifestations, diagnosis, treatment, and prognosis. As a result of trauma or non-traumatic muscle injuries, pathogenesis involves the release of intracellular components into the bloodstream. Epidemiologically, RML exhibits demographic nuances, emphasizing the importance of recognizing risk factors for prevention. Clinical manifestations range from the classic triad of muscle pain, weakness, and dark urine to diverse presentations. Diagnosis involves evaluating elevated creatine kinase levels and myoglobinuria, with management strategies focusing on fluid balance, AKI prevention, and addressing underlying causes. The prognosis is diverse, influenced by complications such as AKI, with various scoring systems aiding risk assessment. Prevention and treatment strategies include a multifaceted approach, integrating tailored warm-ups, mindful exercise, education, and dietary modifications. Overall, the prognosis for RML remains favorable, underscoring the significance of meticulous management. This review provides a comprehensive understanding of RML, bridging historical insights with contemporary medical strategies and guiding ongoing research and advancements in patient care for this intricate condition. RML serves as a paradigm for integrating nuanced historical perspectives and modern medical approaches, highlighting the intricate balance required for effective management in clinical practice.

Keywords: Rhabdomyolysis; Skeletal Muscle; Muscle Breakdown

Abbreviations: RML: Rhabdomyolysis; CK: Creatine Kinase; AKI: Acute Kidney Injury; ULN: Upper Limit of Normal; ATP: Adenosine Triphosphate; Na/K-ATPase: Sodium-Potassium Adenosine Triphosphatase; Ca2+ ATPase: Calcium Adenosine Triphosphatase; DIC: Disseminated Intravascular Coagulation; BMI: Body Mass Index; ARF: Acute Renal Failure; eGFR: Estimated Glomerular Filtration Rate; CKD: Chronic Kidney Disease; IV: Intravenous; NSAIDs: Nonsteroidal Anti-Inflammatory Drugs

Introduction

Rhabdomyolysis (RML) refers to the lysis of skeletal muscle cells. It is a relatively rare condition characterized by myalgia, weakness, and dark urine, often absent, which can result in respiratory failure and altered mental status [1]. Its clinical

consequences are frequently dramatic in morbidity and mortality [2]. RML is believed to have first been reported in the Old Testament, when Jews, following consumption of quail during the Exodus, developed symptoms similar to those of RML (3). This condition has multiple and diverse causes, but central to the pathophysiology is the destruction of the sarcolemmal membrane and the release of intracellular components into the systemic circulation. The clinical presentation may vary, ranging from an asymptomatic increase in serum levels of enzymes released from damaged muscles to problematic conditions such as volume depletion, metabolic and electrolyte abnormalities, and acute kidney injury (AKI) [4]. A systematic review of the definition of RML recommends these clinical symptoms combined with a CK cut-off value of >1000 IU/L or CK > 5 times the upper limit of normal (ULN) as mild RML. Additionally, measured myoglobinuria and acute kidney injury (AKI) indicate a severe RML [1]. Treatment of the underlying cause of the muscle insult is the first component of rhabdomyolysis management [4]. The true incidence of RML is unknown. Approximately 26,000 cases of rhabdomyolysis are reported annually in the United States. African Americans, males, obese patients, patients younger than ten years of age, and patients older than 60 years old all have a higher incidence of RML [1]. AKI has been reported to be associated with 10-67% of patients with rhabdomyolysis, while 5-10% of the AKIs are caused by rhabdomyolysis. Of patients with rhabdomyolysis, 4-8% were reported to require hemodialysis [3]. As we delve into this comprehensive review, we aim to unfold the complex story of rhabdomyolysis. This narrative detailedly intertwines historical elements, epidemiology, pathophysiology, and the latest advancements in diagnosis and management as we seek to understand and navigate its enigmatic course.

Etiology and Pathogenesis

Rhabdomyolysis directly translates to the "dissolution of striated muscle" (rhabd/o, my/o, -lysis). It is brought on by the disintegration and necrosis of muscle tissue and the discharge of intracellular material into the circulation [4]. It typically happens when skeletal muscle is injured, either traumatically or non-traumatically [4]. About 40% of the body is made up of skeletal muscle. Thus, an insult of this kind may cause the cellular contents to build up and eventually overwhelm the body's natural elimination system [4]. Rhabdomyolysis can be anything from a benign sickness with elevated creatine kinase levels to a potentially fatal disorder marked by extremely high levels of the enzyme, electrolyte imbalances, abrupt renal failure, and disseminated intravascular coagulation. It is most frequently caused by traumatic muscle damage [5]. Less often occurring causes include endocrinopathies, medications, toxins, viral causes, muscle enzyme deficits, and aberrant electrolyte levels [5]. Despite the wide variety of causes of rhabdomyolysis, there seems to be a final common pathway that the pathogenesis follows, which results in the destruction of myocytes and the release of muscle components into the bloodstream [6]. The sarcolemma, a thin membrane enclosing striated muscle fibers, is home to many pumps in a typical myocyte that control cellular electrochemical gradients [6]. The sodium-potassium adenosine triphosphatase (Na/K-ATPase) pump in the sarcolemma keeps the intercellular sodium concentration constant at 10 mEq/l [6]. From the inside of the cell to the outside, sodium is actively transported by the Na/K-ATPase pump because positive charges are transferred across the membrane; the inside of the cell is, therefore, more negatively charged than the exterior [6]. The gradient attracts sodium to the inside of the cell through a different ion exchange channel in return for calcium [3].

Furthermore, an active calcium exchanger (Ca2+ ATPase pump) that facilitates calcium entry into the sarcoplasmic reticulum and mitochondria maintains low intracellular calcium levels [3]. The functions mentioned above rely on ATP as their energy source, and most rhabdomyolysis causes appear to culminate in ATP depletion, which causes dysfunction in the Na/K-ATPase and Ca2+ ATPase pump [6]. Consequently, cellular permeability to sodium ions increases due to either disruption of the plasma membrane or a decrease in the generation of cellular energy (ATP) [1]. The intracellular calcium concentration rises due to sodium buildup in the cytoplasm (which is generally relatively low relative to the extracellular concentration) [1]. The internal proteolytic enzymes that break down the muscle cell are subsequently more active as a result of this extra calcium, and large amounts of potassium, aldolase, phosphate, myoglobin, CK, lactate dehydrogenase, aspartate transaminase, and urate seep into the blood when the myocyte degenerates [1]. Under healthy settings, the plasma concentration of myoglobin is deficient, ranging from 0.003 to 0.03 mg/dl; if the quantity of myoglobin in circulation exceeds the plasma's capacity to bind proteins, it may precipitate in the glomerular filtrate once more than 100 g of skeletal muscle is lost. Therefore, an excess of myoglobin may cause renal tubular blockage, acute renal failure, and direct nephrotoxicity [1].

Epidemiology and Risk Factors

The term "rhabdomyolysis" originates from the Greek words rhabdo ("rod") + mus ("muscle") + lusis ("loosening") [7]. It denotes the rapid skeletal (striated) muscle breakdown, releasing myoglobin into the bloodstream, potentially leading to kidney failure. The earliest documented case of rhabdomyolysis is found in the book of Exodus, describing poisoning among the Hebrews after consuming quail [8]. In the modern era, its first documentation dates to World War II, when four cases with crush injuries and renal impairment exhibited notable clinical similarities [9].

The frequency of rhabdomyolysis needs to be precisely understood due to the lack of prospective studies, potentially resulting in the under-recognition of mild cases [10]. This condition can affect individuals of any age and gender [11], with approximately 26,000 cases reported annually in the United States. It occurs more frequently in specific demographics,

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including males, African Americans, individuals under 10 or over 60, and those with a body mass index exceeding 40 kg/m^2 [12,13]. Childhood rhabdomyolysis, a rare clinical disorder, demonstrated an incidence of 0.26% within the pediatric study group over three years at the University of California, San Diego (UCSD) [14].

Common risk factors for rhabdomyolysis include high body temperature, seen in individuals working in outdoor and hot environments like forge and smelter workers, roofers, and asphalt pavers. Other risk factors encompass muscle overuse during high levels of physical exertion or intense workouts leading to muscle necrosis, physical damage to muscles from crush injuries or fall from heights, illicit drug use (cocaine and methamphetamine), excessive alcohol consumption, infections (flu, cytomegalovirus, Epstein-Barr virus, HIV, bacterial, fungal, and parasitic infections), inflammatory and autoimmune muscle conditions (e.g., polymyositis), and certain underlying medical conditions (uncontrolled diabetes, sickle cell disease, thyroid disorders) [15-17]. Medications such as antibiotics, antidepressants, cholesterol-lowering agents (statins), and cold and allergy medications are also associated with an increased risk. Bariatric surgery introduces an elevated risk of rhabdomyolysis [18]. Men, individuals with an elevated body mass index (BMI), and those undergoing prolonged surgical procedures are recognized as having increased susceptibility to rhabdomyolysis (RML) [18]. The use of statins in elderly patients is linked to a slight increase in the likelihood of developing rhabdomyolysis [19]. Defects in various metabolic pathways, including glycolysis, fatty acid oxidation, Krebs cycle, pentose phosphate pathway, purine nucleotide cycle, and mitochondrial respiratory chain, contribute to an elevated risk of rhabdomyolysis [20]. Concurrently, metabolic myopathies are increasingly recognized as significant risk factors for this condition [21]. Various factors contribute to an increased risk of rhabdomyolysis, including exercise, crush injuries, prolonged immobility, exaggerated lithotomy and surgical positions, child abuse, torture victims, extremes of body temperature, and the use of certain medications during anesthesia and the preoperative periods [21].

Clinical Manifestations

The clinical presentation of rhabdomyolysis varies widely depending on the extent and severity of muscle damage, ranging from an asymptomatic increase in serum levels of enzymes released from muscle cells to problematic conditions associated with severe intravascular volume depletion, metabolic acidosis, multiple electrolyte abnormalities, including hyperkalemia, hyperphosphatemia, and hypocalcemia, and AKI [22]. The classic triad of symptoms of rhabdomyolysis includes muscle pain, weakness, and dark urine. The most frequently involved muscles are those in the proximal muscle groups, such as the thighs, calves, and lower back. These muscles may appear tense and swollen, sometimes associated with bed sores. In some patients, pain and swelling only manifest after repletion of volume status. However, this classic triad is only observed in 10% of patients, and more than 50% do not complain of muscle pain or weakness. Skin changes, such as discoloration or blisters, indicative of pressure necrosis, may also be seen but only in less than 10% of patients [23]. In these cases, the first sign may be the appearance of dark urine (from pink to brown to black). Nonetheless, it is observed in approximately half of the cases, and its absence does not exclude the syndrome. The clinician must have a high suspicion index based primarily on the event's history [24]. Systemic manifestations include fever, general malaise, tachycardia, nausea, and vomiting. Cardiovascular symptoms can stem from associated electrolyte abnormalities (i.e., potassium, calcium, phosphate). The clinical manifestations of ARF, DIC, and multiorgan failure may subsequently appear [23].

Diagnosis and Laboratory Evaluation

Evaluating and diagnosing rhabdomyolysis primarily involves detecting a significant elevation in the serum CK levels or by the findings of myoglobin in the urine [25]. After muscle injury, the CK levels rise in just 2 to 12 hours. This peaks at 3 to 5 days and then declines over 6 to 10 days. Also, post-injury plasma and myoglobin levels rise quickly and are really clear. Myoglobin levels quickly return to baseline after 24 hours. CK levels are the most reliable laboratory indicator of rhabdomyolysis and indicate its severity. Several case series demonstrate that a CK level at least 5 times the upper limit of normal is characteristic of rhabdomyolysis [26,27]. However, many other conditions, such as inflammatory myopathies, may present with similar CK levels, but the clinical presentation will help differentiate these conditions [25]. However, CK levels are the most sensitive laboratory evaluation for muscle damage [28]. It is also essential to evaluate acute kidney injury by obtaining a complete metabolic panel, including serum creatinine and electrolytes [29]. A urine dipstick would also be positive for erythrocytes as the dipstick becomes blue in the presence of myoglobin due to the orthotolidine portion [30].

Prevention and Treatment Strategies

Effective management of rhabdomyolysis involves addressing fluid balance, preventing acute kidney injury (AKI), and considering etiology-specific interventions. Aggressive hydration is crucial, with a recommended rate of 1.5 L/h or alternation between saline and glucose solutions. Urinary output goals, urine pH, and plasma pH targets should be achieved. Discontinuation of drugs like statins, fasciotomy for compartment syndrome, and cautious use of alkalizing agents are essential [31].

Traumatic Rhabdomyolysis

Early initiation of intravenous (IV) hydration in crush injuries, even pre-relief, is vital to prevent worsening hypovolemia. Fluid resuscitation up to 10-20 L is recommended, including

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mannitol for improved urine output, while cautious monitoring and avoidance of hyperkalemia are emphasized. If necessary, Foley catheter placement, loop diuretics, and hemodialysis are considered for fluid management and AKI prevention [32].

Nontraumatic Rhabdomyolysis

Nontraumatic rhabdomyolysis follows similar principles to fluid resuscitation, addressing underlying causes and monitoring CPK levels. Mannitol usage is infrequent, and loop diuretics may be considered for volume overload. Hemodialysis is a consideration for patients with persistent AKI despite aggressive fluid resuscitation. Management of Electrolyte Abnormalities in Rhabdomyolysis: Management of electrolyte abnormalities involves different strategies based on potassium levels. Potassium binders and avoiding potassium-containing fluids are recommended for hyperkalemia. Calcium is used for symptomatic hypocalcemia. Allopurinol addresses hyperuricemia. Hemodialysis is considered for severe electrolyte imbalances [32].

Other Supportive Care

Concomitant conditions, such as sepsis or malignant hyperthermia, require appropriate antibiotics, vasopressors, or dantrolene sodium. Steroids are utilized in inflammatory myopathies. Orthopedic consultation is crucial for compartment syndrome, and DIC is managed with blood products [32].

Diet in Metabolic Myopathies

Dietary modifications may alleviate symptoms in hereditary myopathies, but their impact varies based on the specific metabolic disorder. The role of diet is highlighted in managing symptoms associated with specific metabolic myopathies [32].

Anesthetic Considerations

Limited data exist on anesthetic considerations for rhabdomyolysis, especially in muscular dystrophies. Total intraisous anesthesia, avoiding succinylcholine, and careful muscle relaxant monitoring are suggested. While no specific anesthetic agent is risk-free, evidence on triggering agents is inconclusive, emphasizing the importance of cautious anesthetic choices in this patient population [31]. Effective prevention of rhabdomyolysis involves tailored warm-ups, mindful exercise selection, and education about symptoms. Individuals, especially those with limited strength, should avoid high-intensity activities. Cautious exercise during illnesses, environmental considerations like hydration, and suitable clothing are crucial. Post-exercise balanced nutrient intake, and electrolyte-rich fluids aid muscle recovery. Integrating antioxidants may protect against oxidative stress, reducing the risk of kidney damage. This holistic approach ensures a proactive stance in preventing rhabdomyolysis [33].

Prognosis

Rhabdomyolysis poses significant complications, such as acute renal failure (ARF) and hyperkalemia [34]. The prognosis for rhabdomyolysis is diverse, with mortality rates reaching approximately 20%, particularly in cases of rhabdomyolysis induced ARF from severe injuries, which further increases in the presence of multiple organ failure syndrome. The need for dialysis is common, occurring in about 85% of patients with oliguric ARF and 30% with non-oliguric ARF, carrying a mortality rate between 50% and 80%. These statistics emphasize the gravity of complications, underscoring the need for meticulous management and intervention [34]. In emergency medicine, McMahon et al. have introduced an admission prognostic score for Rhabdomyolysis (RML), incorporating critical factors like age, sex, injury type, and clinical lab values, as detailed in [35]. This score demonstrates superior sensitivity and specificity, particularly in predicting AKI requiring dialysis. Another risk score, tailored for severe RML cases, considers lab values and pre-event conditions, revealing minimal AKI risk with prompt fluid treatment, irrespective of initial CK levels [35]. A recent multicenter study, detailed in, underscores the link between invasive ventilation, RML severity, and stage 2-3 AKI risk. Long-term considerations include the correlation between declining estimated glomerular filtration rate (eGFR) and admission serum phosphate and myoglobin levels exceeding 8000U/L. These scoring systems provide valuable insights for clinicians, aiding in high-risk AKI patient identification, guiding triage for aggressive medical management, and extending considerations for transitioning from AKI to Chronic Kidney Disease (CKD) [35]. Despite these challenges, the overall prognosis for rhabdomyolysis, even in the presence of AKI, remains favorable, with most patients achieving complete kidney function recovery [32].

Conclusion

Rhabdomyolysis (RML) presents a multifaceted clinical challenge, encompassing historical roots dating back to biblical times and evolving insights since its documented cases during World War II. The condition's clinical consequences range from mild cases with elevated creatine kinase (CK) levels to severe disorders associated with acute kidney injury (AKI) and disseminated intravascular coagulation. The pathogenesis involves the destruction of myocytes and the release of muscle components into the bloodstream, triggered by various traumatic and non-traumatic factors. Epidemiologically, RML exhibits a higher incidence in specific demographics, emphasizing the importance of recognizing risk factors for prevention and early intervention. Clinical manifestations vary widely, with the classic triad observed in a minority of cases. Diagnosis involves evaluating elevated CK levels and myoglobinuria, with management strategies focusing on fluid balance, AKI prevention, and addressing the underlying cause. Risk assessment is aided by various predictive scoring systems, influenced by the severity of complications such as AKI. Despite challenges, the overall prognosis for RML remains favorable, emphasizing the importance of meticulous management. Prevention and treatment strategies include a multifaceted approach involving tailored warm-ups, mindful exercise, education, and dietary modifications.

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Integrating antioxidants and adopting a proactive stance toward muscle recovery post-exercise contribute to a holistic strategy for preventing RML. A comprehensive understanding of RML, spanning historical context, epidemiology, etiology, pathogenesis, clinical manifestations, diagnosis, treatment, and prognosis, guides ongoing research and clinical advancements to refine knowledge and enhance patient care for this intricate condition. Overall, RML underscores the importance of a nuanced and integrated approach, emphasizing historical insights and contemporary medical strategies in comprehensively managing this complex syndrome.

References

1. Gupta A, Thorson P, Penmatsa KR, Gupta P(2021) Rhabdomyolysis: Revisited. Ulster Med J 90(2):61-69.
2. Cervellin G, Comelli, I, Lippi G (2010). Rhabdomyolysis: historical background, clinical, diagnostic and therapeutic features. Clin Chem Lab Med 48(6): 749-756.
3. Yang C, Li S, Dong Y, Paliwal N (2021) Epidemiology and the Impact of Acute Kidney Injury on Outcomes in Patients with Rhabdomyolysis. J Clin Med 10(9): 1950.
4. Cabral BMI, Edding SN, Portocarrero JP, Lerma EV (2020) Rhabdomyolysis. Dis Mon 66(8): 101015.
5. Huerta-Alardín AL, Varon J, Marik PE (2005) Bench-to-bedside review: Rhabdomyolysis -- An overview for clinicians. Crit Care 9(2): 158-169.
6. Khan FY (2009) Rhabdomyolysis: a review of the literature. Neth J Med 67(9): 272-283.
7. Henry R (2020) Etymologia: Rhabdomyolysis. Emerging Infectious Diseases 26(7): 1620.
8. Carneiro A, Viana-Gomes D, Macedo-da-Silva J, Humberto Oliveira Lima G, Simone Mitri, et al. (2021) Risk factors and future directions for preventing and diagnosing exertional rhabdomyolysis. Neuromuscular Disorders 31(7): 583-595.
9. Bywaters EG, Beall D (1941). Crush injuries with impairment of renal function. Br Med J 1(4185): 427-432.
10. Cabral BMI, Edding SN., Portocarrero JP, Lerma EV (2020) Rhabdomyolysis. Disease-a-Month: DM 66(8): 101015.
11. Sauret JM, Marinides G, Wang GK (2002) Rhabdomyolysis. American Family Physician 65(5): 907-912.
12. Chavez L O, Leon M, Einav S, Varon J (2016) Beyond muscle destruction: a systematic review of rhabdomyolysis for clinical practice. Critical Care (London, England) 20(1): 135.
13. Kupcsik L, Alini M, Stoddart M J (2009) Epsilon-aminocaproic acid is a useful fibrin degradation inhibitor for cartilage tissue engineering. Tissue Engineering. Part A 15(8): 2309-2313.
14. Chakravartty S, Sarma DR, Patel AG (2013). Rhabdomyolysis in bariatric surgery: a systematic review. Obesity Surgery 23(8): 1333-1340.
15. Who's at Risk? (2023) Rhabdomyolysis. NIOSH.
16. Heytens K, Bleecker DJ, Verbrugghe W, Baets J, Heytens L (2017) Exertional rhabdomyolysis and heat stroke: Beware of volatile anesthetic sedation. World Journal of Critical Care Medicine 6(1): 21-27.
17. Brown CVR, Rhee P, Chan L, Evans K, Demetriades D, et al. (2004). Preventing renal failure in patients with rhabdomyolysis: do bicarbonate and mannitol make a difference? The Journal of Trauma, 56(6): 1191-1196.
18. Iwere RB, Hewitt J (2015). Myopathy in older people receiving statin therapy: a systematic review and meta-analysis. Br J Clin Pharmacol 80(3): 363-371.
19. Andreu AL, Bruno C, Dunne TC, Tanji K, Shansk S, et al. (1999) A nonsense mutation (G15059A) in the cytochrome b gene in a patient with exercise intolerance and myoglobinuria. Ann Neurol 45(1): 127-130.
20. Warren JD, Blumbergs PC, Thompson PD (2002). Rhabdomyolysis: a review. Muscle & Nerve 25(3): 332-347.
21. Anderlini P, Buzaid AC, Legha SS (1995). Acute rhabdomyolysis after concurrent administration of interleukin-2, interferon-alfa, and chemotherapy for metastatic melanoma. Cancer 76(4): 678-679.
22. Cabral BMI, Edding SN, Portocarrero JP, Lerma EV, et al. (2020) Rhabdomyolysis, Disease-a-Month. 66(8):101015.
23. Cervellin, G, Comelli I, Lippi G (2010). Rhabdomyolysis: historical background, clinical, diagnostic and therapeutic features. Clin Chem Lab Med 48(6): 749-756.
24. Giannoglou, G. D., Chatzizisis, Y. S., & Misirli, G. (2007). The syndrome of rhabdomyolysis: pathophysiology and diagnosis. Eur J Intern Med 18(2): 90-100.
25. Nance JR, Mammen AL (2015). Diagnostic evaluation of rhabdomyolysis. Muscle nerve 51(6): 793-810.
26. Park JS, Se, MS, Gil HW, Yang JO, Lee EY, et al. (2013). Incidence, etiology, and outcomes of rhabdomyolysis in a single tertiary referral center. J Korean Med Sci 28(8): 1194-1199.
27. Chen CY, Lin YR, Zhao LL, Yang WC, Chang YJ, et al. 2013). Clinical spectrum of rhabdomyolysis presented to pediatric emergency department. BMC pediatrics 13: 134.
28. Stanley M, Chippa V, Aeddula NR, Quintanilla Rodriguez BS, Adigun R (2023) Rhabdomyolysis. In StatPearls. StatPearls Publishing.
29. Cabral BMI, Edding SN, Portocarrero JP, Lerma EV (2020) Rhabdomyolysis DM 66(8): 101015.
30. Torres PA, Helmstetter JA, Kaye AM, Kaye AD (2015). Rhabdomyolysis: pathogenesis, diagnosis, and treatment. Ochsner j 15(1): 58-69.
31. Torres PA, Helmstetter JA, Kaye AM, Kaye AD (2015) Rhabdomyolysis: pathogenesis, diagnosis, and treatment. The Ochsner J 15(1): 58-69.
32. Stanley M, Adigun R (2020) Rhabdomyolysis. StatPearls Publishing, Treasure Island (FL).
33. Kim J, Lee J, Kim S, Ryu HY, Cha KS, et al. (Exercise-induced rhabdomyolysis mechanisms and prevention: A literature review. J Sport Health Sci 5(3): 324-33.
34. Chatzizisis YS, Misirli G, Hatzitolios AI, Giannoglou GD (2008) "The syndrome of rhabdomyolysis: Complications and treatment." Eur J Intern Med 19(8): 572.
35. Gupta A, Thorson, Penmatsa KR, Gupta P (2021) "Rhabdomyolysis: Revisited." Ulster Medical Journal, 90(2): 61-69.

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