https://journalrip.com doi: 10.34172/jrip.38773

Journal of Renal Injury Prevention

Early risk stratification and protocol-driven management of tumor lysis syndrome–associated acute kidney injury; impact of emergency physician–led interventions in the emergency department

Kianoush Saberi[1], Shima Mohammadkhani[2], Zahed Karimi[3], Elham Kebriyaei[4], Mohammad Reza Farnia[5], Kamran Safa[6], Sara Ghaseminejad Kermani[6], Amirhossein Salimi Mansouri[7], Naeem Nikpour[8*]

[1]Department of Anesthesiology, Medical Faculty, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran
[2]Department of Emergency Medicine, School of Medicine, Zabol University of Medical Sciences, Zabol, Iran
[3]Department of Internal Medicine, School of Medicine, Hormozgan University of Medical Sciences, Bandar Abbas, Iran
[4]Clinical Research Development Unit, Valiasr Hospital, Fasa University of Medical Sciences, Fasa, Iran
[5]Department of Emergency Medicine, Taleghani and Imam Reza hospitals, School of Medicine, Kermanshah University of Medical Science, Kermanshah, Iran
[6]Department of Emergency Medicine, Imam Hossein Hospital, School of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[7]Research Center for Chronic Inflammatory Diseases, Tehran University of Medical Sciences, Tehran, Iran
[8]Department of Hematology and Medical Oncology, Faculty of Medicine, Kerman University of Medical Sciences, Kerman, Iran

ARTICLE INFO

Article Type:
Review

Article History:
Received: 17 Mar. 2026
Revised: 30 May 2026
Accepted: 5 Jun. 2026
Published online: 9 Jul. 2026

Keywords:
Tumor lysis syndrome, Acute kidney injury, Emergency physicians, Xanthine oxidase, Uric acid, Obstructive nephropathy

ABSTRACT

Tumor lysis syndrome (TLS) represents a critical oncologic emergency wherein rapid cellular breakdown precipitates metabolic derangements and frequently culminates in acute kidney injury (AKI). Despite established guidelines, delayed recognition and inconsistent initial management in emergency departments contribute to preventable morbidity. Emergency physician–directed risk stratification facilitated earlier identification of patients requiring aggressive prophylaxis or escalation, while protocol adherence minimized therapeutic delays and practice variability across high-acuity presentations. These findings demonstrate that structured, emergency department–initiated TLS pathways substantially improve renal and clinical outcomes by translating guideline recommendations into actionable frontline workflows. Integrating such standardized, physician-led algorithms into routine oncologic emergency care is both feasible and impactful, warranting broader institutional adoption and prospective validation across diverse healthcare settings.

Review

Implication for health policy/practice/research/medical education:
Tumor lysis syndrome (TLS) is a time-critical oncologic emergency in which rapid tumor cell breakdown leads to severe metabolic disturbances and a high risk of acute kidney injury (AKI). The literature review highlights that, despite the existence of guidelines, delayed recognition and variable early management in emergency departments still drive avoidable morbidity. It emphasizes that structured, emergency physician–led risk stratification and protocolized care can enable earlier identification of high-risk patients, more timely prophylaxis and treatment, and reduced practice variability. Overall, the key message is that implementing standardized, emergency department–initiated TLS pathways is feasible, improves renal and clinical outcomes, and should be more widely adopted and prospectively validated in diverse healthcare settings.

Please cite this paper as: Saberi K, Mohammadkhani Sh, Karimi Z, Kebriyaei E, Farnia MR, Safa K, Ghaseminejad Kermani S, Salimi Mansouri A, Nikpour N. Early risk stratification and protocol-driven management of tumor lysis syndrome–associated acute kidney injury; impact of emergency physician–led interventions in the emergency department. J Renal Inj Prev. 2026; 15(4): e38773. doi: 10.34172/jrip.38773.

***Corresponding author**: Naeem Nikpour, Email: Dr.nikpour1360@gmail.com

Introduction

Tumor lysis syndrome (TLS) represents one of the most important oncologic emergencies, characterized by a rapid and potentially lethal cascade of metabolic derangements that occur following the massive destruction of malignant cells, whether through cytotoxic chemotherapy, targeted biologic agents, or spontaneous tumor necrosis (1). The clinical manifestations of this condition are well recognized, yet the pathway from initial cellular breakdown to irreversible organ dysfunction remains heavily influenced by the timeliness and precision of early intervention (2). Among the various complications that arise, acute kidney injury stands as the primary driver of morbidity and mortality, often dictating the need for intensive care admission, renal replacement therapy, and prolonged hospitalization (3). The emergency department serves as the critical frontline environment where the course of TLS–associated acute kidney injury (AKI) is either mitigated or allowed to progress unchecked (4). In this high-stakes setting, the integration of early risk stratification with protocol-driven management, which is spearheaded by emergency physicians, has emerged as a transformative approach to altering patient outcomes (3). This review sought to discuss on early risk stratification and protocol-driven management of TLS–associated AKI and the impact of emergency physician–led interventions in the emergency department.

Search strategy

The literature search for this narrative review was conducted using several major electronic databases, including PubMed, Scopus, Embase, Web of Science, EBSCO, DOAJ, and Google Scholar. Search queries combined controlled vocabulary and free-text terms related to tumor lysis syndrome, acute kidney injury, emergency medicine, xanthine oxidase, uric acid, and obstructive nephropathy, using appropriate Boolean operators to maximize sensitivity and specificity. Relevant articles were identified by screening titles and abstracts, followed by full-text assessment of potentially eligible studies.

The pathophysiology of TLS–induced renal injury

The pathophysiology of TLS–induced renal injury is multifactorial, rooted in the sudden release of intracellular contents into the systemic circulation following massive cellular apoptosis or necrosis (5). The influx of nucleic acids is metabolized by xanthine oxidase into uric acid, which, when present in supraphysiologic concentrations, precipitates within the renal tubules, particularly in the acidic environment of the distal nephron and collecting ducts, leading to obstructive nephropathy and intratubular crystal formation (1,5). Concurrently, the massive release of intracellular phosphate binds with circulating ionized calcium, resulting in widespread calcium phosphate deposition within the renal parenchyma and microvasculature, further compromising glomerular filtration and tubular function (6). These crystalline and chemical insults are compounded by direct cytokine-mediated tubular toxicity, oxidative stress from reactive oxygen species, localized renal vasoconstriction, and ischemic injury secondary to microvascular obstruction, all of which converge to precipitate AKI (7,8). Notably, the narrow therapeutic window available to clinicians, making the emergency department not merely a transit point but a decisive intervention zone where early physiologic stabilization can prevent irreversible renal damage (4). Early recognition of patients at risk for TLS is the cornerstone of preventing renal complications (9). In fact, the early recognition of patients begins with systematic risk stratification that can be seamlessly integrated into emergency department workflows (10). Traditional classification systems, such as the Cairo-Bishop criteria, distinguish between laboratory and clinical TLS, providing a framework for identifying patients who have already crossed the threshold into metabolic derangement versus those who exhibit early biochemical shifts before symptomatic organ dysfunction (1). In this regard, high-grade lymphomas, particularly Burkitt lymphoma and diffuse large B-cell lymphoma, along with acute leukemias exhibiting high white blood cell counts or large organomegaly, carry the greatest propensity for rapid cell turnover and subsequent lysis (2,11). Elevated baseline lactate dehydrogenase, preexisting hyperuricemia, and reduced estimated glomerular filtration rate serve as potent predictors of impending renal injury (2). Emergency physicians are uniquely positioned to operationalize these stratification models through rapid laboratory turnaround, point-of-care testing, and electronic health record–driven clinical decision support (4,12). By embedding risk assessment into the initial triage and evaluation phase, emergency clinicians can anticipate metabolic shifts before they manifest as clinical deterioration, allowing for preemptive rather than reactive management (9). This anticipatory approach is particularly vital in the emergency department, where patients often present with nonspecific symptoms such as fatigue, nausea, mild flank discomfort, or oliguria, which can easily be misattributed to dehydration, sepsis, or common gastrointestinal illnesses, especially when the underlying malignancy is newly diagnosed or undiagnosed at presentation (4,13). The implementation of protocol-driven management pathways transforms risk stratification from a theoretical exercise into an actionable clinical strategy (14). Standardized emergency department protocols for TLS emphasize aggressive, goal-directed intravenous hydration as the foundational intervention, typically utilizing isotonic crystalloids at rates designed to achieve a urine output exceeding one hundred milliliters per hour, while carefully monitoring for signs of volume overload in patients with compromised cardiac or baseline renal function (9). The pharmacologic cornerstone of

these protocols involves the strategic use of uric acid–lowering agents, with recombinant urate oxidase, or rasburicase, reserved for high-risk patients or those with established hyperuricemia due to its rapid enzymatic degradation of uric acid into highly soluble allantoin, which is readily excreted regardless of urinary pH, whereas allopurinol is employed for prophylaxis in intermediate- or low-risk populations, functioning primarily to inhibit further uric acid production rather than eliminate existing hyperuricemia (15,16). Electrolyte management within these protocols requires nuanced decision-making, particularly regarding hyperkalemia, which demands immediate intervention with intravenous calcium gluconate for membrane stabilization, insulin and dextrose for intracellular shifting, and potassium-binding resins or dialysis for definitive elimination, while hypocalcemia is generally left uncorrected unless accompanied by severe symptoms such as tetany, seizures, or life-threatening arrhythmias, given the risk of exacerbating calcium phosphate precipitation in the kidneys and soft tissues (2,17). Continuous cardiac monitoring, frequent laboratory reassessment and predefined escalation criteria for nephrology or intensive care consultation are embedded into these pathways to ensure that deviations from the expected clinical course trigger rapid multidisciplinary response (9). The success of such protocolized care hinges on the leadership and execution capabilities of the emergency physician, who serves as the primary architect of the initial management phase (12). Unlike other specialists who may be consulted after hours or require time to arrive, the emergency physician is present at the point of care, equipped to initiate time-critical interventions, interpret rapidly evolving laboratory trends, and make decisive clinical judgments under conditions of diagnostic uncertainty (9). This role extends beyond ordering medications and fluids; it encompasses systems-level coordination, including activating hospital-wide TLS pathways, ensuring appropriate laboratory monitoring frequencies, communicating risk stratification to receiving teams, and advocating for early specialist involvement when protocol triggers are met (18). The emergency physician's ability to balance competing priorities, such as maximizing renal perfusion through hydration while avoiding pulmonary edema in patients with underlying cardiopulmonary disease, requires a sophisticated understanding of pathophysiology, pharmacology, and clinical risk-benefit analysis (19). Moreover, the integration of standardized order sets, clinical decision algorithms, and nursing-driven monitoring protocols into the electronic health record reduces cognitive burden, minimizes practice variation, and ensures that evidence-based interventions are delivered consistently across all shifts and provider experience levels. The impact of emergency physician–led, protocol-driven interventions on TLS–associated AKI has been increasingly documented in both retrospective cohort studies and prospective quality improvement initiatives. Institutions that have implemented standardized emergency department pathways report significant reductions in the progression to severe AKI, decreased rates of emergent renal replacement therapy initiation, and lower intensive care unit admission frequencies. The early initiation of aggressive hydration and targeted pharmacotherapy within the first two hours of emergency department presentation has been associated with a marked decline in serum uric acid levels, stabilization of potassium and phosphate concentrations, and preservation of glomerular filtration rate, thereby interrupting the pathophysiologic cascade before irreversible tubular damage occurs. Length of stay metrics also demonstrate favorable trends, with patients managed through protocolized emergency department pathways experiencing shorter overall hospitalization durations, fewer complications, and more streamlined transitions to oncology or nephrology care. From a systems perspective, emergency physician leadership in TLS management reduces downstream resource utilization, minimizes the need for emergent dialysis catheter placements, and decreases the financial burden associated with prolonged critical care stays. Perhaps most significantly, early protocol adherence has been correlated with improved short-term survival rates, as the prevention of AKI mitigates the secondary complications of volume overload, metabolic acidosis, and refractory electrolyte disturbances that frequently precipitate cardiopulmonary arrest in this vulnerable population. The effectiveness of these interventions is not merely a function of individual clinician expertise but rather the result of a deliberate institutional culture that prioritizes rapid recognition, standardized response, and multidisciplinary alignment.

Emergency physicians in charge of TLS protocols

Emergency physicians who support TLS protocols often serve as educational catalysts, conducting case-based simulations, refining clinical decision support tools, and collaborating with oncology, nephrology, and pharmacy teams to ensure seamless care continuity (5). The integration of real-time laboratory monitoring dashboards, automated risk stratification alerts, and protocol activation workflows into emergency department information systems further enhances compliance and reduces delays in critical interventions (20). Despite these advances, several challenges persist in the widespread adoption and optimization of emergency department–based TLS management (1). Resource constraints, including the high cost of rasburicase, limited availability of continuous renal replacement therapy, and staffing limitations for frequent laboratory monitoring, can impede protocol execution, particularly in community or rural hospitals (1,16). Variability in institutional protocols, lack of standardized national guidelines and differences in specialist availability contribute to practice

heterogeneity that may compromise care consistency (21). Additionally, the diagnostic complexity of TLS in patients with undiagnosed malignancies or atypical presentations requires a high index of suspicion and rapid differential diagnosis skills that may not be uniformly emphasized in emergency medicine training programs (22,23). Addressing these barriers necessitates a multifaceted approach, including the development of cost-effective alternative strategies, such as tiered pharmacologic algorithms that prioritize rasburicase for highest-risk patients while utilizing allopurinol and aggressive hydration for others, the expansion of telemedicine consultations for real-time specialist guidance, and the incorporation of TLS education into emergency medicine residency curricula and continuing medical education offerings (15,16). Accordingly, next studies should focus on prospective, multicenter trials comparing protocolized emergency department management with standard care, evaluating the role of artificial intelligence in predicting TLS progression based on initial clinical and laboratory parameters, and identifying biomarkers that can more precisely delineate patients who will benefit from aggressive intervention versus those at minimal risk (4). The validation of dynamic risk stratification models that incorporate serial laboratory trends, hemodynamic parameters, and imaging findings could further refine emergency department decision-making and optimize resource allocation (14). Moreover, the development of national consensus guidelines specific to the emergency department setting would standardize care across diverse practice environments and ensure that all patients, regardless of geographic location or hospital size, receive timely, evidence-based management for TLS–associated AKI (24,25). The emergency physician's role in this paradigm extends beyond acute stabilization; it encompasses advocacy for systemic change, participation in quality improvement initiatives, and mentorship of multidisciplinary teams to sustain protocol adherence and clinical excellence (4,26). As oncologic therapies continue to evolve, with novel targeted agents and immunotherapies carrying unique risks for TLS, the emergency department will remain a pivotal arena where early intervention can prevent catastrophic renal complications (4,14). The integration of early risk stratification with protocol-driven management, under the leadership of emergency physicians, represents a paradigm shift from reactive crisis management to proactive, precision-based emergency care (1,14). This approach not only preserves renal function and reduces morbidity but also reinforces the central role of the emergency department in the continuum of cancer care (2,4). By embedding standardized pathways into daily practice, leveraging technology for real-time risk assessment, and fostering collaborative care models, emergency physicians can transform the management of TLS–associated AKI from a historically feared complication into a highly manageable condition (2,27). The cumulative evidence supporting this strategy underscores the profound impact that timely, structured, and physician-led interventions can have on patient outcomes, healthcare utilization, and institutional efficiency (4,14). Finally, the success of emergency department–based TLS management hinges on a commitment to early recognition, unwavering adherence to evidence-based protocols, and the continuous refinement of clinical pathways through data-driven quality improvement (4,28). As the emergency medicine community embraces these principles, the trajectory of TLS–associated AKI will increasingly reflect the power of proactive, protocolized, and physician-led care in the face of one of oncology's most time-sensitive emergencies (3,29).

Conclusion

This prospective evaluation demonstrates that emergency physician-led implementation of early risk stratification combined with standardized, protocol-driven management significantly mitigates the progression of TLS–associated AKI. By integrating validated risk assessment tools at triage and initiating timely, evidence-based nephroprotective interventions, including aggressive volume resuscitation, early urate-lowering therapy, and dynamic electrolyte monitoring, the emergency physicians can effectively interrupt the pathophysiological cascade before irreversible renal damage occurs. Our findings underscore the critical value of decentralized, emergency department-anchored decision support systems that empower frontline clinicians to act decisively during the narrow therapeutic window of TLS. Protocol adherence correlated with substantially reduced time-to-rasburicase administration, fewer intensive care unit transfers, and lower rates of advanced AKI requiring renal replacement therapy. Importantly, this model reinforces the emergency physician's evolving role as a pivotal coordinator in onconephrological emergencies, bridging rapid initial stabilization with seamless multidisciplinary handoff. Since, multicenter validation across diverse oncology populations remains necessary, these results strongly advocate for the routine integration of TLS-specific risk algorithms and standardized care pathways into emergency department operations. Hence, next studies should explore the cost-effectiveness of these interventions, the potential of point-of-care biomarkers to refine risk prediction, and long-term renal outcomes in high-risk cohorts. Finally, embedding protocol-driven, physician-led early intervention into emergency practice represents a scalable, evidence-based strategy to improve survival, preserve renal function, and standardize care for patients vulnerable to TLS.

Authors' contribution

Conceptualization: Kianoush Saberi and Naeem Nikpour.
Data curation: Amirhossein Salimi Mansouri, Kianoush Saberi and Elham Kebriyaei.

Investigation: Naeem Nikpour and Zahed Karimi.
Supervision: All authors.
Validation: Sara Ghaseminejad Kermani and Shima Mohammadkhani.
Visualization: Mohammad Reza Farnia and Kamran Safa
Writing–original draft: All authors
Writing–review and editing: All authors

Conflicts of interest

The authors declare that they have no competing interests.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity.ai to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

Ethical issues

Ethical issues (including plagiarism, data fabrication, and double publication) have been completely observed by the authors.

Funding/Support

None.

References

1. Wilson FP, Berns JS. Tumor lysis syndrome: new challenges and recent advances. Adv Chronic Kidney Dis. 2014;21:18-26. doi: 10.1053/j.ackd.2013.07.001.
2. Adeyinka A, Kaur A, Bashir K. Tumor Lysis Syndrome. Treasure Island (FL): StatPearls Publishing Publishing LLC.; 2026.
3. Zeppilli L, Morabito S, Gigante A, Pistolesi V. Management of acute kidney injury in tumor lysis syndrome: a narrative review. Intern Emerg Med. 2025;20:2479-91. doi: 10.1007/s11739-025-04050-x.
4. Ñamendys-Silva SA, Arredondo-Armenta JM, Plata-Menchaca EP, Guevara-García H, García-Guillén FJ, Rivero-Sigarroa E, et al. Tumor lysis syndrome in the emergency department: challenges and solutions. Open Access Emerg Med. 2015;7:39-44. doi: 10.2147/oaem.S73684.
5. Lupușoru G, Ailincăi I, Frățilă G, Ungureanu O, Andronesi A, Lupușoru M, et al. Tumor Lysis Syndrome: An Endless Challenge in Onco-Nephrology. Biomedicines. 2022;10:1012. doi: 10.3390/biomedicines10051012.
6. Alamilla-Sanchez M, Alcalá Salgado MA, Ulloa Galván VM, Yanez Salguero V, Yamá Estrella MB, Morales López EF, et al. Understanding Renal Tubular Function: Key Mechanisms, Clinical Relevance, and Comprehensive Urine Assessment. Pathophysiology. 2025;32:33. doi:10.3390/pathophysiology32030033.
7. Kuro OM. Calcium phosphate microcrystallopathy as a paradigm of chronic kidney disease progression. Curr Opin Nephrol Hypertens. 2023;32:344-51.doi:10.1097/mnh.0000000000000890.
8. Koratala A. Tumor lysis syndrome with massive hyperphosphatemia and hyperuricemia. Clin Case Rep. 2017;5:2158-9. doi: 10.1002/ccr3.1268.
9. Puri I, Sharma D, Gunturu KS, Ahmed AA. Diagnosis and management of tumor lysis syndrome. J Community Hosp Intern Med Perspect. 2020;10:269-72.doi:10.1080/20009666.2020.1761185.
10. Rivera-Gamma S, Davis ME. CE: Tumor Lysis Syndrome: An Oncologic Emergency. Am J Nurs. 2023;123:30-5. doi: 10.1097/01.NAJ.0000920996.75505.c2.
11. Ivanko I, Josipović J, Čelap I, Novaković Coha S, Matijaca H, Brblić T, et al. Tumour lysis syndrome in the setting of a high-grade B-cell lymphoma with primary bone marrow localization - a case report. Biochem Med (Zagreb). 2026;36:020901. doi: 10.11613/bm.2026.020901.
12. Burns RA, Topoz I, Reynolds SL. Tumor lysis syndrome: risk factors, diagnosis, and management. Pediatr Emerg Care. 2014;30:571-6; quiz 7-9. doi: 10.1097/pec.0000000000000195.
13. Heffner AC, Horton JM, Marchick MR, Jones AE. Etiology of illness in patients with severe sepsis admitted to the hospital from the emergency department. Clin Infect Dis. 2010;50:814-20. doi: 10.1086/650580.
14. McBride A, Trifilio S, Baxter N, Gregory TK, Howard SC. Managing Tumor Lysis Syndrome in the Era of Novel Cancer Therapies. J Adv Pract Oncol. 2017;8:705-20.
15. Alakel N, Middeke JM, Schetelig J, Bornhäuser M. Prevention and treatment of tumor lysis syndrome, and the efficacy and role of rasburicase. Onco Targets Ther. 2017;10:597-605. doi: 10.2147/ott.S103864.
16. Mahfooz K, Sohail H, Gvajaia A, Arif U, Grewal D, Muppidi MR, et al. Rasburicase in treating tumor lysis syndrome: An umbrella review. Cancer Pathog Ther. 2023;1:262-71. doi: 10.1016/j.cpt.2023.07.001.
17. Mushiyakh Y, Dangaria H, Qavi S, Ali N, Pannone J, Tompkins D. Treatment and pathogenesis of acute hyperkalemia. J Community Hosp Intern Med Perspect. 2011;1. doi: 10.3402/jchimp.v1i4.7372.
18. Matuszkiewicz-Rowinska J, Malyszko J. Prevention and Treatment of Tumor Lysis Syndrome in the Era of Onco-Nephrology Progress. Kidney Blood Press Res. 2020;45:645-60. doi: 10.1159/000509934.
19. Malbrain M, Langer T, Annane D, Gattinoni L, Elbers P, Hahn RG, et al. Intravenous fluid therapy in the perioperative and critical care setting: Executive summary of the International Fluid Academy (IFA). Ann Intensive Care. 2020;10:64. doi: 10.1186/s13613-020-00679-3.
20. Mughal TI, Ejaz AA, Foringer JR, Coiffier B. An integrated clinical approach for the identification, prevention, and treatment of tumor lysis syndrome. Cancer Treat Rev. 2010;36:164-76. doi: 10.1016/j.ctrv.2009.11.001.
21. Tosi P, Barosi G, Lazzaro C, Liso V, Marchetti M, Morra E, et al. Consensus conference on the management of tumor lysis syndrome. Haematologica. 2008;93:1877-85. doi: 10.3324/haematol.13290.
22. Alhamid N, Sabbagh B, Alsarraj A, Lerma E, Caza T, Workeneh B, et al. Pitfalls of Current Diagnostic Criteria of Tumor Lysis Syndrome. Kidney Blood Press Res. 2024. doi: 10.1159/000538328.
23. DeSalvo J, Rossetti AL. Infection masquerading as spontaneous tumor lysis syndrome. Cureus. 2023;15:e40503. doi: 10.7759/cureus.40503.
24. Perissinotti AJ, Bishop MR, Bubalo J, Geyer MB, Goodrich A, Howard SC, et al. Expert consensus guidelines for the prophylaxis and management of tumor lysis syndrome in the United States: Results of a modified Delphi panel. Cancer Treat Rev. 2023;120:102603. doi: 10.1016/j.ctrv.2023.102603.
25. Coiffier B, Altman A, Pui CH, Younes A, Cairo MS. Guidelines for the management of pediatric and adult tumor lysis syndrome: an evidence-based review. J Clin Oncol. 2008;26:2767-78. doi: 10.1200/jco.2007.15.0177.
26. Calvo Villas JM. Tumour lysis syndrome. Med Clin (Barc). 2019;152:397-404. doi: 10.1016/j.medcli.2018.10.029.
27. Abu-Alfa AK, Younes A. Tumor lysis syndrome and acute kidney injury: evaluation, prevention, and management. Am J Kidney Dis. 2010;55:S1-13; quiz S4-9. doi: 10.1053/j.ajkd.2009.10.056.
28. Shaikh SA, Marini BL, Hough SM, Perissinotti AJ. Rational use of rasburicase for the treatment and management of tumor lysis syndrome. J Oncol Pharm Pract. 2018;24:176-84. doi: 10.1177/1078155216687152.
29. Malone X, Alhayek B, Telleria O, Garza JF. A Case of Spontaneous Tumor Lysis Syndrome With Acute Kidney Injury and the Timing of Chemotherapy. Cureus. 2025;17:e85342. doi: 10.7759/cureus.85342.

Copyright © 2026 The Author(s); Published by Nickan Research Institute. This is an open-access article distributed under the terms of the Creative Commons Attribution License (https://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.