International Journal of Advances in Medicine
Patel M et al. Int J Adv Med. 2026 Sep;13(5):309-315
https://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20262828

Original Research Article

Acute kidney injury in patients with malignancy: clinical characteristics, etiology and outcomes across KDIGO stages

Maulik Patel*, Akash Dholakia

Department of Nephrology, Shankus Hospital, Mehsana, Gujarat, India

Received: 03 June 2026
Revised: 08 July 2026
Accepted: 01 August 2026

***Correspondence:**
Dr. Maulik Patel,
E-mail: maulikpatel88.md@gmail.com

ABSTRACT

Background: Acute kidney injury (AKI) is a frequent and serious complication in patients with malignancy, contributing to increased morbidity, mortality and adverse renal outcomes. This study evaluated the clinical profile, primary etiologies, laboratory characteristics and outcomes of oncology patients with AKI according to kidney disease. Improving global outcomes (KDIGO) staging.
Methods: This retrospective observational study included 100 adult oncology patients with KDIGO-defined AKI admitted between May 2021 and April 2026. Patients were classified as KDIGO stage 1 (n=30), stage 2 (n=35) or stage 3 (n=35). Demographic characteristics, comorbidities, primary etiologies, laboratory parameters and clinical outcomes were compared across AKI stages.
Results: Increasing AKI severity was associated with older age and a higher prevalence of diabetes mellitus, hypertension and hematological malignancies. Sepsis was the most common primary etiology of AKI across all stages. Progressive worsening of hematological, inflammatory, metabolic and biochemical parameters was observed with increasing KDIGO stage (all $p<0.001$). Complete renal recovery at discharge declined significantly from 83.3% in stage 1 to 20.0% in stage 3, while in-hospital mortality increased from 10.0% to 51.4% ($p=0.001$). Among hospital survivors, complete recovery at 3 months decreased from 88.9% to 29.4%, whereas progression to chronic kidney disease increased from 7.4% to 52.9% across stages 1 to 3 (both $p<0.001$). Hospital readmission was more frequent in patients with severe AKI ($p=0.048$).
Conclusions: Higher KDIGO stage is associated with greater clinical and biochemical derangement, reduced renal recovery, increased CKD progression and higher mortality in oncology patients with AKI. Early recognition and timely intervention may improve renal and patient outcomes in this high-risk population.

Keywords: Acute kidney injury, KDIGO, Malignancy

INTRODUCTION

AKI is a common and clinically significant complication in patients with malignancy, often resulting in interruption of cancer-directed therapy, prolonged hospitalization, increased healthcare utilization and poorer renal and patient survival. KDIGO classification remains the standard framework for the diagnosis and staging of AKI, providing a clinically relevant severity-based approach in which even modest increases in serum creatinine may reflect substantial kidney injury in vulnerable individuals.[1] In oncology patients, AKI is frequently multifactorial and may result from sepsis, intravascular volume depletion, nephrotoxic medications, urinary tract obstruction or metabolic complications related to rapid tumor cell turnover. Infection remains one of the most common precipitating factors, highlighting the relevance of contemporary sepsis definitions and management strategies in this population.[2,3] The burden of AKI among patients with cancer is substantial. Population-based data

have demonstrated a higher incidence of AKI in patients with malignancy compared with the general population, particularly among older individuals, those with multiple comorbidities and patients receiving active anticancer treatment.[4] Onco-nephrology studies have further shown that the etiology and clinical presentation of AKI vary according to malignancy type, disease stage, treatment exposure and supportive care requirements.[5,6] Critically ill oncology patients who develop AKI constitute a particularly high-risk subgroup, with significantly increased risks of morbidity and mortality.[7]

The clinical complexity of cancer-associated AKI becomes even more evident when renal replacement therapy is required. The decision regarding initiation, modality selection and expected benefit of renal replacement therapy is often challenging, particularly in patients with advanced malignancy or multiorgan dysfunction.[8] Hematological malignancies deserve special consideration because intensive chemotherapy, tumor lysis syndrome, infection and marrow suppression frequently coexist and may precipitate severe AKI.[9,10] Importantly, AKI in cancer patients is not merely a transient biochemical abnormality; it may lead to treatment delays, dose modifications, progression to CKD and adverse long-term outcomes.[11]

A stage-based evaluation of AKI is therefore of considerable clinical importance. KDIGO stage 1, stage 2 and stage 3 AKI represent progressively severe degrees of renal injury and are associated with increasing physiological stress, greater risk of complications and lower likelihood of complete renal recovery. The clinical impact of AKI may vary according to baseline kidney function, age, comorbidity burden, malignancy subtype and treatment intensity. Therefore, a comprehensive assessment of clinical characteristics, primary etiologies, laboratory abnormalities and outcomes across KDIGO stages may improve understanding of disease severity and facilitate early recognition, risk stratification and targeted preventive strategies. Accordingly, the present study aimed to evaluate the clinical profile, primary etiologies, laboratory characteristics and short-term outcomes of oncology patients with AKI according to KDIGO stage.[1,4,5,11]

METHODS

This retrospective observational study was conducted at Shankus Hospital, Mehsana, a tertiary care oncology referral center and included a total of 100 adult patients with malignancy-associated AKI admitted between May 2021 and April 2026. AKI was defined and staged according to the KDIGO criteria. Patients were categorized into KDIGO stage 1 (n=30), stage 2 (n=35) and stage 3 (n=35).

Patients were excluded from the study if they had a pre-existing diagnosis of end-stage renal disease requiring maintenance dialysis prior to the current hospital admission or a history of prior kidney transplantation. Additionally, pregnant patients, individuals who developed acute kidney injury secondary to causes clearly unrelated to their underlying malignancy or its treatment and patients with incomplete clinical or laboratory records that precluded accurate KDIGO staging or assessment of primary outcomes were also excluded from the analysis.

Demographic characteristics, comorbidities, malignancy type, primary etiology of AKI, laboratory parameters and clinical outcomes were extracted from hospital medical records. Baseline variables included age, sex, diabetes mellitus, hypertension and underlying malignancy. Primary etiologies of AKI were classified as sepsis, drug-induced nephrotoxicity, obstructive uropathy, volume depletion, tumor lysis syndrome, renal infiltration and other causes. Laboratory parameters evaluated included hemoglobin, total leukocyte count, platelet count, serum creatinine, blood urea, serum sodium, potassium, bicarbonate, albumin, C-reactive protein, uric acid, corrected calcium and phosphorus.

Clinical outcomes assessed at discharge included complete renal recovery, partial renal recovery, in-hospital mortality and duration of hospital stay. Three-month follow-up outcomes among hospital survivors included complete renal recovery, progression to CKD, mortality after discharge and hospital readmission.

Continuous variables are presented as mean±standard deviation (SD), whereas categorical variables are expressed as frequency and percentage. Normality of continuous variables was assessed using the Shapiro–Wilk test. Comparisons among KDIGO stages were performed using one-way analysis of variance (ANOVA) for continuous variables and Pearson's chi-square test or Fisher's exact test, as appropriate, for categorical variables. All statistical tests were two-tailed and a p value <0.05 was considered statistically significant. This study was conducted in accordance with the ethical principles outlined in the declaration of helsinki and reported following the strengthening the reporting of observational studies in epidemiology (STROBE) guidelines.

RESULTS

A total of 100 adult patients with malignancy-associated AKI admitted between May 2021 and April 2026 were included in the study. According to KDIGO criteria, 30 patients were classified as stage 1, 35 as stage 2 and 35 as stage 3 AKI. Baseline demographic, clinical, laboratory and outcome characteristics according to AKI stage are summarized in Tables 1–4.

Increasing AKI severity was associated with significantly older age and a higher prevalence of diabetes mellitus, hypertension and hematological malignancies (Table 1). Sepsis was the most common primary etiology of AKI across all KDIGO stages, followed by drug-induced nephrotoxicity. Although obstructive uropathy and tumor

lysis syndrome were more frequently observed in patients with advanced AKI, the distribution of primary etiologies did not differ significantly across KDIGO stages (all p>0.05) (Table 2). Laboratory parameters demonstrated a progressive worsening with increasing AKI severity (Table 3). Patients with KDIGO stage 3 AKI had significantly lower hemoglobin, platelet count, serum sodium, bicarbonate, albumin and corrected calcium levels, along with significantly higher total leukocyte count, serum creatinine, blood urea, potassium, C-reactive protein, uric acid and serum phosphorus concentrations compared with patients in stages 1 and 2 (all p<0.001). These findings indicate increasing hematological, inflammatory, metabolic and biochemical derangement with advancing AKI severity.

Clinical outcomes worsened significantly with increasing AKI severity (Table 4). Complete renal recovery at discharge decreased progressively across KDIGO stages, whereas partial renal recovery and in-hospital mortality increased significantly.

Patients with KDIGO stage 3 AKI had the longest duration of hospitalization. Among patients discharged alive, complete renal recovery at 3 months declined markedly with increasing AKI severity, while progression to CKD and post-discharge mortality increased significantly. Hospital readmission rates were also higher among patients with advanced AKI. Overall, increasing KDIGO stage was strongly associated with poorer short-term renal recovery, greater risk of CKD progression, increased mortality and adverse clinical outcomes.

Table 1: Baseline characteristics according to KDIGO AKI stage.

Characteristics	Stage 1 (n=30)	Stage 2 (n=35)	Stage 3 (n=35)	P value
Age (in years), mean±SD	50±11	56±12	63±13	0.001
Male sex, N (%)	16 (53.3)	21 (60.0)	23 (65.7)	0.592
Diabetes mellitus, N (%)	5 (16.7)	10 (28.6)	17 (48.6)	0.014
Hypertension, N (%)	6 (20.0)	13 (37.1)	19 (54.3)	0.008
Hematological malignancy, N (%)	7 (23.3)	11 (31.4)	17 (48.6)	0.036
Gastrointestinal malignancy, N (%)	10 (33.3)	9 (25.7)	6 (17.1)	0.241
Lung malignancy, N (%)	5 (16.7)	5 (14.3)	5 (14.3)	0.951
Urological malignancy, N (%)	3 (10.0)	5 (14.3)	4 (11.4)	0.861
Gynecological malignancy, N (%)	2 (6.7)	3 (8.6)	3 (8.6)	0.954
Other malignancies*, N (%)	3 (10.0)	2 (5.7)	0 (0.0)	0.187

*Other malignancies include breast cancer, head and neck cancer, sarcoma, melanoma and miscellaneous solid organ malignancies not classified elsewhere.

Table 2: Primary etiology of AKI according to KDIGO stage.

Etiology	Stage 1 (n=30)	Stage 2 (n=35)	Stage 3 (n=35)	P value
	N (%)	N (%)	N (%)	
Sepsis	10 (33.3)	13 (37.1)	12 (34.3)	0.941
Drug-induced AKI	8 (26.7)	8 (22.9)	5 (14.3)	0.422
Obstructive uropathy	1 (3.3)	2 (5.7)	5 (14.3)	0.113
Volume depletion	4 (13.3)	3 (8.6)	2 (5.7)	0.528
Tumor lysis syndrome	2 (6.7)	4 (11.4)	6 (17.1)	0.338
Renal infiltration	1 (3.3)	1 (2.9)	2 (5.7)	0.811
Other etiologies*	4 (13.3)	4 (11.4)	3 (8.6)	0.784

*Other etiologies included contrast-induced nephropathy, hypercalcemia-associated nephropathy, cardiorenal syndrome, hepatorenal syndrome, chemotherapy-related thrombotic microangiopathy and other less common causes of AKI.

Table 3: Laboratory parameters according to KDIGO AKI stage.

Parameters	Stage 1 (n=30)	Stage 2 (n=35)	Stage 3 (n=35)	P value
Hemoglobin (g/dl)	11.2±1.8	10.1±1.9	8.8±1.7	<0.001
Total leukocyte count (×10⁹/l)	8.9±3.1	11.8±4.6	15.6±5.8	<0.001
Platelet count (×10⁹/l)	245±82	198±76	142±65	<0.001
Serum creatinine (mg/dl)	1.9±0.4	3.2±0.7	5.8±1.5	<0.001
Blood urea (mg/dl)	48±15	78±24	126±38	<0.001
Serum sodium (mEq/l)	136±4	133±5	129±6	<0.001
Serum potassium (mEq/l)	4.4±0.5	4.9±0.6	5.8±0.9	<0.001
Serum bicarbonate (mEq/l)	23.4±3.2	20.5±3.6	16.9±4.1	<0.001

Continued.

Parameters	Stage 1 (n=30)	Stage 2 (n=35)	Stage 3 (n=35)	P value
Serum albumin (g/dl)	3.8±0.5	3.3±0.6	2.8±0.5	<0.001
C-reactive protein (mg/l)	22±14	58±30	108±52	<0.001
Uric acid (mg/dl)	5.8±1.6	7.9±2.4	11.8±4.2	<0.001
Corrected calcium (mg/dl)	8.8±0.7	8.2±0.8	7.4±0.9	<0.001
Serum phosphorus (mg/dl)	3.9±1.1	5.2±1.5	7.3±2.0	<0.001

Table 4: Clinical outcomes according to KDIGO AKI stage.

Outcomes	Stage 1 (n=30)	Stage 2 (n=35)	Stage 3 (n=35)	P value
Complete renal recovery at discharge, N (%)	25 (83.3)	18 (51.4)	7 (20.0)	<0.001
Partial renal recovery at discharge, N (%)	2 (6.7)	8 (22.9)	10 (28.6)	<0.001
In-hospital mortality, N (%)	3 (10.0)	9 (25.7)	18 (51.4)	0.001
Hospital stay (days)	8.1±2.3	11.6±4.1	16.4±5.2	<0.001
Complete recovery at 3 months*, N (%)	24 (88.9)	16 (61.5)	5 (29.4)	<0.001
Progression to CKD at 3 months*, N (%)	2 (7.4)	8 (30.8)	9 (52.9)	<0.001
Mortality within 3 months after discharge*, N (%)	1 (3.7)	2 (7.7)	3 (17.6)	0.041
Readmission within 3 months*, N (%)	3 (11.1)	7 (26.9)	6 (35.3)	0.048

*Percentages at 3 months were calculated among patients discharged alive

DISCUSSION

The literature consistently demonstrates that AKI in patients with cancer is multifactorial, often resulting from the complex interplay between patient-related susceptibility factors and cancer-specific exposures. Cancer-associated inflammation, malnutrition, anemia and recurrent episodes of dehydration may diminish renal reserve, predisposing patients to kidney injury following additional insults. Several reviews and cohort studies have shown that the clinical presentation of AKI differs between patients with solid tumors and those with hematological malignancies; however, in both populations, AKI is strongly associated with increased mortality and reduced tolerance to anticancer therapy.[12-16] A single-center study from Palestine further highlighted the substantial burden of AKI among hospitalized cancer patients, emphasizing that kidney injury remains a common and clinically significant complication in routine oncology practice.[17]

Infection and sepsis continue to represent the predominant causes of AKI in oncologic populations. The Sepsis-3 criteria provide the contemporary framework for identifying infection-related organ dysfunction, while the Surviving Sepsis Campaign advocates early source control, prompt administration of appropriate antimicrobial therapy, hemodynamic optimization and judicious fluid management to minimize secondary renal injury.[2,3] These considerations are particularly relevant in oncology patients, who are frequently exposed to immunosuppression, indwelling vascular devices, mucosal barrier disruption and prolonged neutropenia. Consequently, multiple studies have consistently identified sepsis as a major contributor to AKI, particularly among critically ill cancer patients, in whom hypotension, vasoplegia and inflammatory tubular injury collectively exacerbate renal dysfunction.[6,7,14,17]

Drug-induced AKI has gained increasing importance with the evolution of modern cancer therapeutics. Contemporary evidence indicates that conventional cytotoxic agents, targeted therapies and immune checkpoint inhibitors can induce kidney injury through diverse mechanisms, including acute tubular injury, acute interstitial nephritis, thrombotic microangiopathy, glomerular disease and electrolyte disturbances.[11,1,8,19] Patients with hematological malignancies are especially vulnerable because they often require intensive chemotherapy regimens, repeated antimicrobial exposure and prolonged hospitalization, all of which contribute to cumulative nephrotoxic burden.[20,21] Furthermore, cancer-related sarcopenia may result in deceptively low serum creatinine levels, potentially delaying recognition of significant renal injury.[22]

Among noninfectious causes, both hemodynamic and obstructive mechanisms play important roles. Volume depletion may result from vomiting, diarrhea, inadequate oral intake, fever, mucositis, diuretic therapy or increased insensible losses and remains a frequent precipitant of prerenal AKI in oncology patients.[5,11,17] Obstructive nephropathy is particularly relevant in patients with genitourinary, gynecological or advanced metastatic malignancies, where ureteric obstruction may cause rapid deterioration in kidney function. Available evidence supports early imaging and timely decompression because post-renal obstruction remains one of the most reversible causes of severe AKI when recognized promptly.[13,16]

AKI in cancer patients is also closely linked to the pathophysiology of acute kidney disease and CKD progression. The transition from AKI to CKD is increasingly recognized as a continuum rather than an isolated event. Chawla and colleagues described AKI and CKD as interconnected syndromes, while subsequent meta-analyses demonstrated that AKI significantly

increases the long-term risks of mortality, CKD and adverse renal outcomes.[23,24] Additional mechanistic and clinical studies have shown that incomplete renal recovery is particularly common following severe or recurrent episodes of AKI and in patients with underlying comorbid conditions.[25,26] In oncology populations, these risks may be further amplified by ongoing exposure to potentially nephrotoxic therapies during cancer treatment.[11,22]

Renal recovery should therefore be viewed as a dynamic and evolving process rather than a single endpoint assessed at hospital discharge. Current evidence highlights the importance of post-AKI surveillance, including serial assessment of kidney function, blood pressure control, medication review and monitoring for proteinuria and electrolyte abnormalities.[24-26] The work of Kellum remains particularly relevant because variability in recovery definitions across studies often complicates direct comparisons of outcomes.[26] Importantly, even patients who achieve apparent recovery at discharge may subsequently develop CKD, require rehospitalization or experience recurrent episodes of AKI if residual renal impairment persists.[23-26]

Tumor lysis syndrome (TLS) represents another well-recognized oncologic cause of AKI. Evidence-based guidelines and expert consensus statements consistently emphasize that TLS is largely predictable in patients with high tumor burden and highly chemosensitive malignancies and should be prevented through appropriate risk stratification, aggressive hydration, urate-lowering therapy and close biochemical monitoring.[27,28] The syndrome is characterized by rapid cellular breakdown leading to hyperkalemia, hyperphosphatemia, secondary hypocalcemia, hyperuricemia and intratubular crystal deposition, all of which contribute to acute renal injury.[28,29] Given its potentially life-threatening nature, prevention remains considerably more effective than treatment after AKI has already developed.[27-31]

Nephrotoxicity associated with anticancer therapy remains a major challenge in onco-nephrology. Cisplatin continues to be one of the most extensively studied causes of chemotherapy-related AKI and is associated with dose-dependent tubular injury and electrolyte wasting.[32-34] In addition, targeted therapies may induce hypertension, proteinuria, thrombotic microangiopathy or pseudo-AKI, whereas immune checkpoint inhibitors can trigger immune-mediated interstitial nephritis that often necessitates treatment interruption and corticosteroid therapy.[35-37] Consequently, the literature strongly supports comprehensive medication review, baseline renal assessment, serial monitoring of kidney function and close collaboration between oncologists and nephrologists when AKI develops during active cancer treatment.[11,18,35-37]

Taken together, the available evidence indicates that AKI in oncology should be regarded as a heterogeneous syndrome with multiple interacting etiologies rather than a single disease entity. The most consistent findings across studies include the dominant role of infection, the growing contribution of treatment-related nephrotoxicity and the adverse renal prognosis associated with severe AKI.[4-7,11,14,18,23-26,35-37] These observations underscore the importance of preventive strategies, early etiological evaluation, prompt intervention and structured post-discharge follow-up to reduce CKD progression, recurrent hospitalization and mortality.

Strengths

This study provides a comprehensive stage-wise evaluation of AKI in oncology patients using the KDIGO classification system. It integrates demographic characteristics, comorbidities, primary etiologies, laboratory parameters and clinically relevant outcomes across different stages of AKI. The assessment of both in-hospital and 3-month follow-up outcomes offers valuable insights into renal recovery CKD progression, mortality and hospital readmission. Furthermore, the use of standardized KDIGO diagnostic and staging criteria enhances the validity of the findings and facilitates comparison with previously published studies.

Limitations

This study has several limitations. First, its retrospective, single-center design may limit the generalizability of the findings and is subject to inherent selection and information bias. Second, the relatively small sample size may have limited the statistical power to detect significant differences in less common etiologies and clinical outcomes. Third, detailed information regarding cancer stage, treatment modalities, exposure to specific nephrotoxic agents and baseline kidney function was not consistently available and therefore could not be incorporated into the analysis. Finally, the follow-up period was limited to three months, precluding evaluation of long-term renal outcomes, CKD progression beyond the study period and overall patient survival. Future large-scale, prospective, multicenter studies with longer follow-up are warranted to validate these findings and further clarify the long-term impact of AKI in patients with malignancy.

CONCLUSION

Acute kidney injury is a common and clinically significant complication among patients with malignancy and is associated with substantial renal and patient morbidity. In the present study, increasing KDIGO stage was associated with greater clinical and biochemical derangement, reduced rates of renal recovery, prolonged hospitalization, higher risk of CKD progression and increased mortality. Sepsis emerged as the most common primary etiology of AKI, highlighting the importance of infection prevention and early management in oncology patients. The marked decline in renal recovery and survival with advancing AKI severity underscores the need for timely recognition, risk stratification and multidisciplinary management. Early

intervention and structured post-discharge follow-up may improve renal outcomes and reduce the burden of CKD and mortality in this high-risk population.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl. 2012;2(1):1-138.
2. Singer M, Deutschman CS, Seymour CW. The third international consensus definitions for sepsis and septic shock (sepsis-3). JAMA. 2016;315(8):801-10.
3. Evans L, Rhodes A, Alhazzani W. Surviving sepsis campaign: international guidelines for management of sepsis and septic shock 2021. Intensive Care Med. 2021;47(11):1181-247.
4. Christiansen CF, Johansen MB, Langeberg WJ, Fryzek JP, Sørensen HT. Incidence of acute kidney injury in cancer patients: a Danish population-based cohort study. Eur J Intern Med. 2011;22(4):399-406.
5. Campbell GA, Hu D, Okusa MD. Acute kidney injury in the cancer patient. Adv Chronic Kidney Dis. 2014;21(1):64-71.
6. Benoit DD, Hoste EA. Acute kidney injury in critically ill patients with cancer. Crit Care Clin. 2010;26(1):151-79.
7. Soares M, Salluh JI, Carvalho MS, Darmon M, Rocco JR, Spector N. Prognosis of critically ill patients with cancer and acute renal dysfunction. J Clin Oncol. 2006;24(24):4003-10.
8. Lupușoru M, Lupușoru G, Ailincăi I, Frățilă G, Andronesi A, Micu E, et al. Renal replacement therapy in cancer patients with acute kidney injury (Review). Exp Ther Med. 2021;22(2):864.
9. Canet E, Zafrani L, Lambert J, Thieblemont C, Galicier L, Schnell D, et al. Acute kidney injury in patients with newly diagnosed high-grade hematological malignancies: impact on remission and survival. PLoS One. 2013;8(2):55870.
10. Darmon M, Vincent F, Canet E, Mokart D, Pène F, Kouatchet A, et al. Acute kidney injury in critically ill patients with haematological malignancies: results of a multicentre cohort study from the Groupe de Recherche en Réanimation Respiratoire en Onco-Hématologie. Nephrol Dial Transplant. 2015;30(12):2006-13.
11. Rosner MH, Perazella MA. Acute kidney injury in the patient with cancer. Kidney Res Clin Pract. 2019;38(3):295-308.
12. Kitchlu A, McArthur E, Amir E, Booth CM, Sutradhar R, Majeed H, et al. Acute kidney injury in patients receiving systemic treatment for cancer: a population-based cohort study. J Natl Cancer Inst. 2019;111(7):727-36.
13. Braet P, Re Sartò GV, Pirovano M. Treatment of acute kidney injury in cancer patients. Clin Kidney J. 2022;15(5):873-84.
14. Gupta S, Gudsoorkar P, Jhaveri KD. Acute kidney injury in critically ill patients with cancer. Clin J Am Soc Nephrol. 2022;17(9):1385-98.
15. Can W, Rong L, Lixia L. Incidence and risk factors of acute kidney injury in patients with malignant tumors: a systematic review and meta-analysis. BMC Cancer. 2023;23:1123.
16. Calças Marques R. Severe acute kidney injury in hospitalized cancer patients. Cancers (Basel). 2024;16(3):561.
17. Nazzal Z. The rate and risk factors of acute kidney injury among cancer patients' admissions in Palestine: a single-center study. Int J Nephrol. 2022;2:2972275.
18. Małyszko J, Kozłowska K, Kozłowski L. Nephrotoxicity of anticancer treatment. Nephrol Dial Transplant. 2017;32(6):924-36.
19. Sawinski D. The kidney effects of hematopoietic stem cell transplantation. Adv Chronic Kidney Dis. 2014;21(1):96-105.
20. Singh N, McNeely J, Parikh S, Bhinder A, Rovin BH, Shidham G. Kidney complications of hematopoietic stem cell transplantation. Am J Kidney Dis. 2013;61(5):809-21.
21. Abudayyeh A, Wanchoo R. Kidney Disease Following Hematopoietic Stem Cell Transplantation. Adv Chronic Kidney Dis. 2022;29(2):103-115.e1.
22. Chawla LS, Eggers PW, Star RA, Kimmel PL. Acute kidney injury and chronic kidney disease as interconnected syndromes. N Engl J Med. 2014;371(1):58-66.
23. Coca SG, Yusuf B, Shlipak MG, Garg AX, Parikh CR. Long-term risk of mortality and other adverse outcomes after acute kidney injury: a systematic review and meta-analysis. Am J Kidney Dis. 2009;53(6):961-73.
24. Forni LG, Darmon M, Ostermann M, Oudemans-van Straaten HM, Pettila V, Prowle JR, et al. Renal recovery after acute kidney injury. Intensive Care Med. 2017;43(6):855-66.
25. Macedo E, Mehta RL. Renal recovery after acute kidney injury. Contrib Nephrol. 2016;187:24-35.
26. Kellum JA. How can we define recovery after acute kidney injury? Considerations from epidemiology and clinical trial design. Nephron Clin Pract. 2014;127(1-4):81-8.
27. 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(16):2767-78.
28. Howard SC, Jones DP, Pui CH. The tumor lysis syndrome. N Engl J Med. 2011;364(19):1844-1854.
29. Puri I, Sharma D, Gunturu KS, Ahmed AA. Diagnosis and management of tumor lysis syndrome. J Community Hosp Intern Med Perspect. 2020;10(3):269-272.

30. 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.
31. Cairo MS, Coiffier B, Reiter A, Younes A. Recommendations for the evaluation of risk and prophylaxis of tumour lysis syndrome (TLS) in adults and children with malignant diseases: an expert TLS panel consensus. Br J Haematol. 2010;149(4):578-86.
32. Yao X, Panichpisal K, Kurtzman N, Nugent K. Cisplatin nephrotoxicity: a review. Am J Med Sci. 2007;334(2):115-24.
33. Manohar S, Leung N. Cisplatin nephrotoxicity: a review of the literature. J Nephrol. 2018;31(1):15-25.
34. Tang C. Cisplatin nephrotoxicity: new insights and therapeutic implications. Nat Rev Nephrol. 2023;19(1):53-72.
35. Perazella MA, Shirali AC. Immune checkpoint inhibitor nephrotoxicity: what do we know and what should we do. Kidney Int. 2020;97(1):62-74.
36. Jhaveri KD, Wanchoo R, Sakhiya V, Ross DW, Fishbane S. Adverse renal effects of novel molecular oncologic targeted therapies: a narrative review. Kidney Int Rep. 2017;2(1):108-23.
37. Izzedine H, Perazella MA. Anticancer drug-induced acute kidney injury. Kidney Int Rep. 2017;2(4):504-14.

Cite this article as: Patel M, Dholakia A. Acute kidney injury in patients with malignancy: clinical characteristics, etiology and outcomes across KDIGO stages. Int J Adv Med 2026;13:xxx-xx.