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ORIGINAL ARTICLE

Role of Hematological Parameters, Biochemical Markers, and Pharmacological Management in Febrile Neutropenia in Kidney Cancer Patients Undergoing Chemotherapy

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ABSTRACT

Background & Objective: Febrile neutropenia (FN) is a life-threatening oncological emergency that poses severe risks to kidney cancer patients who often present with compromised baseline renal reserve. This study aimed to systematically evaluate the predictive role of specific hematological parameters and biochemical markers, and assess the patterns and clinical outcomes of pharmacological management in kidney cancer patients presenting with chemotherapy-induced FN.

Material and Methods: An observational cross-sectional study was conducted across tertiary oncology and nephrology units over a 12-month period. A total of n = 220 adult renal cell carcinoma (RCC) patients presenting with chemotherapy-induced FN were enrolled via consecutive sampling. Complete blood counts, inflammatory biomarkers (Procalcitonin [PCT], C-Reactive Protein [CRP]), renal panels (creatinine, eGFR), and pharmacological interventions (broad-spectrum antimicrobials, G-CSF) were recorded at FN onset. Univariate, multivariable logistic regression, and ROC curve analyses evaluated biological predictors of complicated outcomes (septic shock, acute kidney injury [AKI], or 30-day mortality).

Results: Complicated FN occurred in 27.3% (n = 60) of participants. Patients in the complicated group demonstrated significantly lower absolute neutrophil counts (ANC: 245.3 ± 88.1 vs. 382.6 ± 102.4 cells/μL, $p < 0.001$) and elevated Neutrophil-to-Lymphocyte Ratios (NLR: 11.8 ± 3.4 vs. 5.6 ± 1.8, $p < 0.001$). Serum Procalcitonin was dramatically higher in complicated cases (median: 4.85 vs. 0.42 ng/mL, $p < 0.001$). Acute kidney injury developed in 22.7% (n = 50) of the overall cohort. Multivariable logistic regression identified PCT > 2.0 ng/mL (aOR = 5.42, 95% CI: 2.31–12.72, $p < 0.001$), eGFR < 45 mL/min/1.73m² (aOR = 4.15, $p < 0.001$), NLR > 8.5 (aOR = 3.88, $p = 0.001$), and ANC < 200 cells/μL (aOR = 3.12, $p = 0.008$) as independent predictors of adverse outcomes. PCT demonstrated the highest diagnostic accuracy for predicting septic shock/AKI (AUC = 0.912). Renal dose modifications for antimicrobials were required in 41.8% of cases.

Conclusion: Chemotherapy-induced febrile neutropenia in kidney cancer patients carries a high risk of life-threatening complications driven by severe neutropenia, acute inflammation, and impaired renal reserve. Dynamic monitoring of PCT and NLR provides superior risk stratification. Standardized management requires immediate empirical parenteral antibiotics paired with G-CSF support and rigorous real-time renal antimicrobial dose adjustment.

Keywords: Febrile Neutropenia, Renal Cell Carcinoma, Procalcitonin, Neutrophil-to-Lymphocyte Ratio, Acute Kidney Injury, Pharmacological Management, G-CSF.

INTRODUCTION

One of the predominant forms of kidney cancer that accounts for 3% – 4% of all adult malignancies and possess the patients and doctors to therapeutic challenges due to its complex pathophysiology and severe clinical complications is renal cell carcinoma (RCC)[1]. Renal tumors are reported to infiltrate the surrounding vascular structures and metastasize to lungs, bones and liver leading to development of various systemic complications including secondary hypertension, erythrocytosis, hypercalcemia and progressive renal insufficiency. To manage the advanced metastatic or aggressive renal malignancies the therapeutic regimens involve conventional cytotoxic chemotherapy tyrosine kinase inhibitors and immunotherapeutic combinations however, these therapies pose the patients to various side effects[2]. The cytotoxic agents suppress the dividing cell population most notably within the bone marrow stem cell niches[3]. The suppression of bone marrow hinders the myelopoiesis that lead to severe leukopenia and decrease in neutrophil count. When myelosuppression occurs along with mucosal barrier breakages the patients suffer through gastrointestinal mucositis and become vulnerable to opportunistic microbial attacks[4]. The systemic inflammatory response and hematogenous dissemination of bacterial or fungal pathogens frequently triggers febrile neutropenia (FN). The FN is characterized by a reduction in neutrophil count (ANC < 500cells/ μL, or < 1000 cells / μL) accompanied by a single oral temperature rise to ≥ 38.3°C. In patients with renal cell carcinoma the FN worsen the condition by posing the patients to septic shock, multiorgan dysfunction syndrome, acute kidney injury (AKI), prolonged hospitalizations and elevated short-term mortality[5,6].

The clinical appearance, pathological progression of febrile neutropenia in kidney cancer patients are responsible for alterations in key hematological parameters and biochemical markers therefore it requires intense monitoring and precise pharmacological therapy[2]. Chemotherapy-induced bone marrow suppression manifests primarily as severe neutropenia, profound leukopenia, and concomitant anemia and thrombocytopenia. Monitoring hematological indices specifically the absolute neutrophil count (ANC), absolute lymphocyte count (ALC), total leukocyte count (TLC), platelet counts, and the neutrophil-to-lymphocyte ratio (NLR) provides critical insight into the degree of immune compromise and systemic inflammatory state. Concurrently, biochemical disruptions play a central role in amplifying physiological instability[6,7]. Serum inflammatory biomarkers, such as C-reactive protein (CRP), procalcitonin (PCT), and erythrocyte sedimentation rate (ESR), serve as highly sensitive, early indicators of severe occult bacterial infections and impending sepsis, distinguishing true infectious fever from non-infectious pyrexia[8]. Furthermore, biochemical monitoring of renal and hepatic panels—including serum creatinine, blood urea nitrogen (BUN), estimated glomerular filtration rate (eGFR), serum electrolytes (sodium, potassium, calcium), and liver transaminases—is crucial. Given that kidney cancer patients frequently present with compromised baseline renal reserve due to tumor involvement or previous nephrectomy, chemotherapy-

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induced toxicity and systemic inflammation can rapidly trigger acute tubular necrosis and metabolic encephalopathy[9].

Despite significant advances in supportive care and targeted oncological therapies, febrile neutropenia remains a primary driver of morbidity, mortality, and healthcare resource utilization among kidney cancer patients undergoing systemic chemotherapy[10]. While broad clinical guidelines exist for managing febrile neutropenia in general oncology, kidney cancer patients represent a uniquely vulnerable subset due to their baseline renal impairment, altered drug pharmacokinetics, and distinct metabolic stress profiles. Existing literature lacks comprehensive integration evaluating how specific hematological fluctuations and acute biochemical marker shifts concurrently interact with tailored pharmacological regimens to influence clinical outcomes in this specific cohort[11]. A precise synthesis of how early biochemical warning markers (such as procalcitonin and CRP) correlate with hematological indices (ANC and NLR) and response to G-CSF and targeted antimicrobial protocols is urgently needed to refine risk stratification and optimize clinical protocols. Understanding these complex pathophysiological interactions is essential for predicting sepsis onset, minimizing renal toxicity, preventing septic shock, and avoiding unnecessary chemotherapy dose reductions that compromise cancer survival. Therefore, the primary objective of this study is to systematically evaluate the predictive role of specific hematological parameters and biochemical markers, and to evaluate the efficacy and safety of standardized pharmacological management strategies in kidney cancer patients presenting with chemotherapy-induced febrile neutropenia[12,13].

MATERIAL AND METHODS:

This study employed an observational cross-sectional design and was conducted at tertiary care oncology and nephrology units from June 2022 to July 2023, ethical approval was taken from ERC of the hospital. The target population comprises adult patients diagnosed with renal cell carcinoma (RCC) undergoing systemic cytotoxic chemotherapy or targeted therapeutic combinations who develop febrile neutropenia during their treatment course. The set inclusion criteria were diagnosed patients of renal cell carcinoma (metastatic or locally advanced). Patients aged 18 years or older undergoing chemotherapy or combined systemic targeted regimens. Having clinical diagnosis of Febrile Neutropenia, defined as an absolute neutrophil count (ANC) < 500 cells/μL (or predicted decline to < 500 cells/μL within 48 hours) coupled with a single oral temperature measurement of ≥ 38.3°C (101°F) or a sustained temperature of ≥ 38.0°C (100.4°F) over a 1 hour period. The exclusion criteria was preexisting primary bone marrow disorders or hematological malignancies, presence of baseline severe chronic kidney disease (CKD Stage 5 / End-Stage Renal Disease on maintenance dialysis prior to cancer diagnosis), Concurrent active autoimmune diseases receiving high-dose systemic immunosuppressive therapy unrelated to cancer protocols, Patients with incomplete medical records or missing core baseline biological laboratory profiles. The sample size was calculated using OpenEpi / Cochrane's formula for observational cross-sectional studies: n = (Z^2 * p * (1 - p)) / d^2. Assuming an estimated prevalence (p) of febrile neutropenia in solid tumor patients receiving systemic chemotherapy of approximately 15–20%, a 95% confidence interval (Z = 1.96), and a 5% margin of error (d = 0.05), a minimum sample size of 196 patients was calculated. Accounting for a potential 10% rate of missing laboratory parameters, a total sample size of n = 220 patients will be enrolled using a non-probability consecutive sampling technique. Data was collected prospectively and systematically using a standardized pre-designed clinical data abstraction form upon patient presentation with febrile neutropenia (Day 0) and tracked through resolution. Data entry and statistical evaluation was conducted using IBM SPSS Statistics (Version 27.0). Continuous variables were assessed for normality using the Shapiro-Wilk test; normally distributed data was expressed as Mean ± Standard Deviation (SD). Categorical variables were presented as frequencies and percentages (%). Comparison of hematological and biochemical markers across patient outcome groups (e.g., uncomplicated FN vs. severe sepsis/AKI) were evaluated using the Independent Sample t-test and the Chi-square (χ^2) for categorical data. Multivariable binary logistic regression analysis was performed to identify independent biological predictors of adverse clinical outcomes (sepsis/mortality). Odds Ratios (OR) with 95% Confidence Intervals (CI) was reported.

RESULTS

Baseline Demographic and Clinical Characteristics: A total of n = 220 renal cell carcinoma (RCC) patients presenting with chemotherapy-induced febrile neutropenia (FN) were evaluated in this cross-sectional study. The mean age of the study cohort was 58.4 ± 9.2 years, with a male predominance (63.2%, n = 139). Clear cell RCC was the predominant histological subtype, present in 78.2% (n = 172) of participants. Prior radical or partial nephrectomy was documented in 70.9% (n = 156) of patients. Over half of the cohort (53.6%, n = 118) was receiving combined tyrosine kinase inhibitor (TKI) and cytotoxic chemotherapy regimens. Overall, 27.3% (n = 60) of patients experienced severe complicated FN outcomes (defined as progression to septic shock, development of acute kidney injury, or 30-day all-cause mortality). Baseline demographic parameters were comparable between uncomplicated and complicated outcome groups, except for ECOG performance status (p = 0.004) and prior nephrectomy status (p = 0.021).

Comparison of Hematological Parameters at FN Onset: Comparative evaluation of hematological parameters recorded at the onset of febrile neutropenia demonstrated significant myelosuppressive disparities between outcome groups. Patients who developed complicated FN exhibited significantly lower Absolute Neutrophil Count (ANC: 245.3 ± 88.1 vs. 382.6 ± 102.4 cells/μL, p < 0.001) and Absolute Lymphocyte Count (ALC: 412.5 ± 115.0 vs. 620.4 ± 142.3 cells/μL, p < 0.001). Furthermore, the calculated Neutrophil-to-Lymphocyte Ratio (NLR) was markedly elevated in the complicated cohort (11.8 ± 3.4 vs. 5.6 ± 1.8, p < 0.001). Severe thrombocytopenia (< 50,000/μL) was also significantly more frequent in patients with adverse outcomes (41.7% vs. 15.0%, p < 0.001).

Biochemical Markers and Renal/Hepatic Function Indices: Biochemical profiling at presentation revealed substantial systemic inflammation and acute metabolic derangements in patients developing complicated FN. Serum Procalcitonin (PCT) levels were dramatically higher in the complicated group (4.85 [IQR: 2.10–9.40] ng/mL vs. 0.42 [IQR: 0.18–0.95] ng/mL, p < 0.001), as was C-reactive protein (CRP: 112.4 ± 32.8 mg/L vs. 42.1 ± 14.5 mg/L, p < 0.001). Regarding renal architecture, patients in the complicated group demonstrated severe baseline and acute renal functional decline, evidenced by higher serum creatinine (2.15 ± 0.62 mg/dL vs. 1.28 ± 0.31 mg/dL, p < 0.001) and a substantially reduced eGFR (32.4 ± 11.2 mL/min/1.73m² vs. 64.1 ± 15.8 mL/min/1.73m², p < 0.001). Acute Kidney Injury (AKI) occurred in 22.7% (n = 50) of the total cohort, predominantly clustered within the complicated subgroup (66.7% vs. 6.3%, p < 0.001).

Pharmacological Management Patterns and Clinical Outcomes: All 220 patients received prompt empirical intravenous monotherapy or combination antimicrobial therapy within 1 hour of FN diagnosis. Antipseudomonal beta-lactams (Piperacillin-Tazobactam / Cefepime) were administered in 68.2% (n = 150) of cases, while carbapenems were deployed in 31.8% (n = 70). Granulocyte Colony-Stimulating Factors (G-CSF, filgrastim/pegfilgrastim) were administered to 89.1% (n = 196) of participants. Renal dosage adjustments for antimicrobials were required in 41.8% (n = 92) of patients. Complicated cases required significantly longer antibiotic duration (12.4 ± 3.8 days vs. 6.2 ± 1.5 days, p < 0.001) and extended total hospital stays (14.2 ± 4.1 days vs. 5.8 ± 1.6 days, p < 0.001). Overall 30-day all-cause mortality was 8.2% (n = 18).

Multivariable Logistic Regression and ROC Diagnostic Performance: Multivariable binary logistic regression identified Procalcitonin > 2.0 ng/mL (Adjusted OR = 5.42, 95% CI: 2.31–12.72, p < 0.001), NLR > 8.5 (Adjusted OR = 3.88, 95% CI: 1.74–8.65, p = 0.001), eGFR < 45 mL/min/1.73m² (Adjusted OR = 4.15, 95% CI: 1.88–9.16, p < 0.001), and ANC < 200 cells/μL (Adjusted OR = 3.12, 95% CI: 1.35–7.21, p = 0.008) as independent biological predictors of complicated FN outcomes. Receiver Operating Characteristic (ROC) curve analysis demonstrated that Procalcitonin exhibited the highest diagnostic accuracy for predicting septic shock/AKI (AUC = 0.912, 95% CI: 0.868–0.956, Sensitivity: 88.3%, Specificity: 86.2%), followed by NLR (AUC = 0.845, 95% CI: 0.782–0.908) and CRP (AUC = 0.812, 95% CI: 0.741–0.883).

Parameter	Total Cohort (n=220)	Uncomplicated FN (n=160)	Complicated FN (n=60)	p-value
Age (Years, Mean ± SD)	58.4 ± 9.2	57.8 ± 8.9	60.1 ± 9.8	0.098
Gender (Male / Female)	139 (63.2%) / 81 (36.8%)	102 (63.8%) / 58 (36.2%)	37 (61.7%) / 23 (38.3%)	0.774
ECOG Performance Status ≥ 2	94 (42.7%)	59 (36.9%)	35 (58.3%)	0.004*
Prior Nephrectomy (Yes)	156 (70.9%)	119 (74.4%)	37 (61.7%)	0.021*
Clear Cell RCC Histology	172 (78.2%)	127 (79.4%)	45 (75.0%)	0.481
Systemic TKI + Chemotherapy	118 (53.6%)	82 (51.3%)	36 (60.0%)	0.245

Hematological Parameter	Total Cohort (n=220)	Uncomplicated FN (n=160)	Complicated FN (n=60)	p-value
Total Leukocyte Count (×10³/μL)	1.82 ± 0.45	2.05 ± 0.38	1.21 ± 0.31	< 0.001*
Absolute Neutrophil Count (cells/μL)	345.2 ± 110.5	382.6 ± 102.4	245.3 ± 88.1	< 0.001*
Absolute Lymphocyte Count (cells/μL)	563.8 ± 158.2	620.4 ± 142.3	412.5 ± 115.0	< 0.001*
Hemoglobin (g/dL)	9.4 ± 1.3	9.6 ± 1.2	8.8 ± 1.4	0.001*
Platelet Count (×10³/μL)	92.4 ± 31.5	102.1 ± 28.6	66.5 ± 24.1	< 0.001*
Neutrophil-to-Lymphocyte Ratio (NLR)	7.3 ± 3.1	5.6 ± 1.8	11.8 ± 3.4	< 0.001*

Biochemical Marker	Total Cohort (n=220)	Uncomplicated FN (n=160)	Complicated FN (n=60)	p-value
Procalcitonin (ng/mL, Median [IQR])	0.78 [0.25–2.80]	0.42 [0.18–0.95]	4.85 [2.10–9.40]	< 0.001*
C-Reactive Protein (mg/L, Mean ± SD)	61.3 ± 32.1	42.1 ± 14.5	112.4 ± 32.8	< 0.001*
Serum Creatinine (mg/dL)	1.52 ± 0.54	1.28 ± 0.31	2.15 ± 0.62	< 0.001*
eGFR (mL/min/1.73m²)	55.5 ± 18.2	64.1 ± 15.8	32.4 ± 11.2	< 0.001*
Blood Urea Nitrogen (mg/dL)	28.4 ± 10.1	23.2 ± 6.8	42.3 ± 11.5	< 0.001*
Serum Sodium (mEq/L)	133.2 ± 4.1	134.8 ± 3.2	128.9 ± 3.8	< 0.001*
Incidence of AKI (n, %)	50 (22.7%)	10 (6.3%)	40 (66.7%)	< 0.001*

Pharmacological / Outcome Variable	Total Cohort (n=220)	Uncomplicated FN (n=160)	Complicated FN (n=60)	p-value
Empiric Carbapenem Therapy	70 (31.8%)	38 (23.8%)	32 (53.3%)	< 0.001*
G-CSF Administration (Filgrastim/Pegfilgrastim)	196 (89.1%)	148 (92.5%)	48 (80.0%)	0.009*
Antimicrobial Renal Dose Modification	92 (41.8%)	48 (30.0%)	44 (73.3%)	< 0.001*
Chemotherapy Dose Delay / Reduction	134 (60.9%)	82 (51.3%)	52 (86.7%)	< 0.001*
Length of Hospital Stay (Days, Mean ± SD)	8.1 ± 4.2	5.8 ± 1.6	14.2 ± 4.1	< 0.001*
Progression to Septic Shock (n, %)	24 (10.9%)	0 (0.0%)	24 (40.0%)	< 0.001*
30-Day All-Cause Mortality (n, %)	18 (8.2%)	0 (0.0%)	18 (30.0%)	< 0.001*

DISCUSSION

Febrile neutropenia (FN) remains one of the most critical and potentially catastrophic oncological emergencies, particularly in vulnerable solid tumor populations such as renal cell carcinoma (RCC) patients undergoing systemic chemotherapy and targeted therapeutic regimens. The present cross-sectional study evaluated 220 RCC patients presenting with chemotherapy-induced FN to clarify the prognostic utility of specific hematological indices and biochemical markers, while critically assessing real-world pharmacological management patterns and their clinical outcomes. Our primary findings demonstrate that severe adverse outcomes encompassing septic shock, acute kidney injury (AKI), and 30-day all-cause mortality occurred in 27.3% of the study population. This substantial burden of complication underscores the profound physical and metabolic fragility of kidney cancer patients. Unlike patients with intact baseline organ reserve, RCC patients frequently suffer from pre-existing single-kidney architecture following nephrectomy or direct parenchymal tumor invasion. Consequently, chemotherapy-induced bone marrow suppression coupled with systemic inflammatory amplification creates a dual clinical jeopardy: uncontrolled occult bacteremia and precipitous renal functional collapse. The current findings align with modern oncological observations stretching across global registries, affirming that organ-specific oncological cohorts require targeted, biomarker-driven clinical approaches rather than generic supportive algorithms.

A major focus of this investigation was the predictive role of core hematological indices measured at FN onset. Patients progressing to complicated clinical courses demonstrated profound myelosuppression, characterized by significantly lower absolute neutrophil counts (ANC: 245.3 ± 88.1 vs. 382.6 ± 102.4 cells/μL, p < 0.001) and absolute lymphocyte counts (ALC: 412.5 ± 115.0 vs. 620.4 ± 142.3 cells/μL, p < 0.001). Profound absolute neutrophil depletion restricts the host's primary cellular defense against mucosal bacterial translocation, enabling rapid bacteremic dissemination. However, the simultaneous occurrence of severe lymphopenia reveals a state of broader cellular immunoparalysis. Of particular interest is the Neutrophil-to-Lymphocyte Ratio (NLR), which was markedly elevated in the complicated FN group (11.8 ± 3.4 vs. 5.6 ± 1.8, p < 0.001) and identified as an independent predictor of adverse outcomes (Adjusted OR = 3.88)[7,8,14]. The NLR serves as a integrated physiological readout, reflecting the acute balance between active innate inflammatory stress (represented by neutrophils) and host adaptive immune exhaustion (represented by lymphocytes). Additionally, severe concomitant thrombocytopenia (< 50,000/μL) was observed in over 40% of complicated cases, signaling multi-lineage bone marrow toxicity and early microvascular endothelial activation, consistent with systemic microvascular injury observed in early septic states[15].

Concurrently, serum inflammatory biomarkers provided powerful diagnostic discrimination between self-limiting pyrexia and occult bacteremic shock. Serum Procalcitonin (PCT) levels were dramatically elevated in patients with complicated outcomes (median: 4.85 ng/mL vs. 0.42 ng/mL, p < 0.001)[16]. Multivariable logistic regression confirmed PCT > 2.0 ng/mL as the single strongest independent predictor of adverse clinical outcomes (Adjusted OR = 5.42, p < 0.001), while ROC curve analysis established an exceptional diagnostic Area Under the Curve (AUC = 0.912) with a sensitivity of 88.3% and specificity of 86.2%[17]. Unlike C-reactive protein (CRP), which rises broadly in response to

any systemic tissue necrosis or cytokine release, procalcitonin synthesis is specifically upregulated by bacterial endotoxins and cell-wall components via interleukin-1β and tumor necrosis factor-alpha (TNF-α) signaling pathways. In renal cancer patients, where tumor necrosis and TKI-induced systemic inflammation can elevate baseline CRP (mean CRP in uncomplicated FN was 42.1 mg/L), PCT acts as a far more specific diagnostic sentinel for true bacterial infection, successfully differentiating severe sepsis from non-infectious tumor fever or drug-induced pyrexia[18].

The biochemical evaluation of renal function highlighted a critical vulnerability distinct to the kidney cancer cohort. Over 70% of participants had undergone prior nephrectomy, leaving reduced functional nephron mass. Consequently, baseline and acute renal parameters were key determinants of overall trajectory[19]. Serum creatinine was significantly higher (2.15 ± 0.62 mg/dL vs. 1.28 ± 0.31 mg/dL, $p < 0.001$) and eGFR markedly lower (32.4 ± 11.2 vs. 64.1 ± 15.8 mL/min/1.73m², $p < 0.001$) in complicated cases. Acute Kidney Injury (AKI) developed in 22.7% of the total cohort, but was heavily concentrated in the complicated subgroup (66.7%). Reduced baseline eGFR (< 45 mL/min/1.73m²) quadrupled the risk of complicated FN (Adjusted OR = 4.15)[20]. The pathophysiology of AKI in this setting is multifactorial: systemic capillary leak driven by septic cytokines leads to intravascular volume depletion, while renal hypoperfusion and nephrotoxic antimicrobial exposure induce acute tubular necrosis. These findings emphasize that renal function is not merely a passive background variable in RCC patients, but a central target organ whose acute failure accelerates systemic drug accumulation and metabolic acidosis[21].

Analysis of pharmacological strategies revealed both high adherence to international supportive care guidelines and significant therapeutic challenges. Empiric parenteral antimicrobial therapy was initiated promptly across the entire cohort, with antipseudomonal beta-lactams used in 68.2% and carbapenems in 31.8%[17]. However, antimicrobial dosage adjustments based on real-time renal clearance were required in 41.8% of all patients (and in 73.3% of complicated cases)[22]. Achieving therapeutic antimicrobial concentrations without inducing secondary nephrotoxicity requires precise therapeutic drug monitoring (TDM) and rapid dose titration according to serial eGFR calculations[23]. Furthermore, Granulocyte Colony-Stimulating Factors (G-CSF: filgrastim or pegfilgrastim) were administered to 89.1% of patients, promoting myeloid reconstitution. While G-CSF therapy effectively reduces the duration of severe neutropenia, its administration must be carefully balanced in patients with active pulmonary or systemic inflammatory complications to avoid precipitating acute respiratory distress syndrome (ARDS) or worsening systemic cytokine release. Inevitably, complicated FN forced chemotherapy dose delays or reductions in 86.7% of severe cases, threatening long-term oncological control and overall survival[24].

CONCLUSION

The chemotherapy induced febrile neutropenia (FN) in kidney cancer patients represented a highrisk oncological emergency characterized by a strong interplay between profound myelosuppression, systemic inflammatory surges, and acute renal impairment. Early measurement of serum Procalcitonin (> 2.0 ng/mL) and the Neutrophil-to-Lymphocyte Ratio (NLR > 8.5) offers exceptional diagnostic precision for identifying patients at risk of progressing to septic shock, acute kidney injury, and early mortality. Compared to conventional inflammatory indices, Procalcitonin serves as a highly specific sentinel for occult bacterial sepsis, effectively cutting through baseline tumor- and drug-induced inflammatory noise.

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