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# Nephrostomy-Associated Sepsis in Cancer Patients: What Are the Risk Factors? A Retrospective Cohort Study

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#### Abstract

**Objective:** To evaluate sepsis and mortality following nephrostomy tube due to malignant etiology. Material and Methods: Patients who underwent nephrostomy tube at our center were retrospectively evaluated. Only those with malignancy-related indications were included in the study. Patients were initially categorized into two main groups: those with urological malignancies and those with non-urological malignancies. Subsequently, they were further divided into subgroups based on the development of sepsis and survival status. Predictive factors associated with sepsis and mortality were analyzed.

Results: A total of 517 patients were identified, of whom 173 met the inclusion criteria. The mean age was 62.53 years, with a male-to-female ratio of 112:61. Among patients who developed sepsis, post-operative (post-op) platelet counts, post-op creatinine, as well as pre-operative (pre-op) and post-op neutrophil and lymphocyte counts and neutrophil-to-lymphocyte ratio (NLR) were significantly lower, whereas procalcitonin and C-reactive protein (CRP) levels were significantly higher (p<0.05). The presence of perirenal fat stranding and intensive care unit (ICU) admission were also significantly associated with sepsis development(p<0.05). Regarding mortality, lower pre-op and post-op lymphocyte counts and higher procalcitonin levels were statistically significant (p < 0.05). Postoperative NLR, creatinine and CRP were also significantly associated with mortality. Furthermore, the presence of diabetes mellitus (DM), immunosuppressive drug use (ISDU), ICU admission, and non-urological malignancies were found to be statistically significant factors associated with mortality.

**Conclusion:** Our findings indicate that NLR, procalcitonin, CRP, as well as pre-and post-op platelet, lymphocyte and neutrophil counts, along with the presence of perirenal fat stranding, DM, ISDU, and ICU admission and non-urological malignancies play significant roles in the development of sepsis and mortality. These findings emphasize the importance of early risk stratification and targeted management in patients undergoing nephrostomy for malignant obstruction.

Keywords: malignancy, mortality, percutaneous nephrostomy, sepsis

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## INTRODUCTION

Percutaneous nephrostomy (PN) and ureteral catheterization are essential interventions commonly utilized prior to definitive management in cases of renal or supravesical urinary tract obstruction [1,2]. Although these procedures are generally effective, they are not without risks. Potential complications include bleeding, injury to adjacent organs, ureteral perforation or avulsion, and long-term sequelae such as ureteral stricture or impaired renal function. Among these, sepsis stands out as a particularly critical complication, characterized by systemic inflammation and organ dysfunction, and is associated with a high mortality rate [3,4].

Percutaneous nephrostomy is frequently effective in relieving hydroureteronephrosis (HUN) and pyelonephritis secondary to urinary tract obstruction. However, in certain patient groups—particularly those with renal failure or pre-existing septic conditions—PN may aggravate the clinical status [3–6]. In routine urological practice, it has been observed that sepsis may develop in some patients following PN, while in others with an already septic state, the clinical course may deteriorate further, often requiring admission to the intensive care unit (ICU). In rare instances, such complications can even result in mortality.

This study aims to identify clinical factors associated with the development of sepsis and mortality in patients undergoing nephrostomy due to malignancy. The findings are intended to provide valuable insights to facilitate early diagnosis, risk stratification and the development of more effective management strategies for both groups.

## MATERIALS AND METHODS

This retrospective cohort study was conducted in accordance with the Declaration of Helsinki and received approval from the institutional ethics committee. Patient data were systematically utilized in strict compliance to confidentiality and privacy standards. In this study, the terms pre-operative(pre-op) and post-operative(post-op) refer to the clinical periods before and after nephrostomy tube insertion. Both pre-op and post-op clinical data were meticulously retrieved from hospital records and electronic medical systems. The study included all patients who underwent PN in our clinic, focusing exclusively on cases performed due to malignant etiologies. Patients with benign conditions—including stone-related HUN, surgical

complications, ureteral strictures, or ureteropelvic junction obstructions—were excluded. The decision to perform PN was predominantly based on computed tomography (CT) evaluations. In instances where CT was not feasible, magnetic resonance imaging or ultrasound was used to guide clinical decision-making. Patients were initially categorized into two primary groups: those with urological malignancies and those with non-urological malignancies. Subsequently, they were stratified based on the presence of sepsis and survival status to comprehensively evaluate potential predictive factors. Patients diagnosed with sepsis were included only if it was considered to be nephrostomy-associated urosepsis. Sepsis was defined using the systemic inflammatory response syndrome (SIRS) criteria.

Collected data encompassed a comprehensive range of demographic and clinical parameters, including age, sex, body mass index (BMI), diabetes mellitus (DM), immunosuppressive conditions, immunosuppressive drug use (ISDU), perirenal fat stranding, solitary kidney status, urine test results (nitrite positivity, pyuria), urine culture findings, ICU admission, and both pre-op and post-op biochemical parameters (creatinine, platelet count, white blood cell count, neutrophil count, lymphocyte count, C-reactive protein [CRP], and procalcitonin). Additionally, inflammatory markers such as the neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR), and systemic immuneinflammatory index (SII) were calculated and assessed (Table 1). These comprehensive data sets were utilized to identify and analyze factors associated with the development of sepsis and mortality.

### **Statistical Analysis**

Data were analyzed using SPSS version 27.0. The normality of distribution was assessed using the Kolmogorov-Smirnov test. Descriptive statistics were presented as mean ± standard deviation (SD) for normally distributed variables and as median with interquartile range (IQR) for non-normally distributed variables. Group comparisons were performed using the Student's t-test for parametric data and the Mann-Whitney U test for non-parametric data, based on the distribution characteristics. Categorical variables were analyzed using the Pearson Chi-square test. Independent factors associated with sepsis and mortality were identified through univariate and multivariable logistic regression

analyses. A p-value of <0.05 was considered statistically significant.

### **RESULTS**

A total of 517 patients who underwent PN were initially identified, among whom 173 met the predefined inclusion and exclusion criteria. The mean age of the study population was 62.53 years, with a male-to-female ratio of 112:61. Sepsis and mortality rates were observed at 21% (37) and 26% (45), respectively. In the sepsis group, the male-to-female distribution was 22:15, while in the mortality group it was 27:18. (Table 1). No statistically significant difference was found between the groups regarding these distributions (Table 2).

Among the sepsis group, post-op platelet count and creatinine were significantly lower (p<0.05). Both pre-op and post-op neutrophil and lymphocyte counts, along with the neutrophil-to-lymphocyte ratio (NLR), were significantly lower, whereas

procalcitonin and CRP levels were markedly higher (p<0.05). The presence of perirenal fat stranding on CT and ICU admission were both significantly associated (p<0.05) with the development of sepsis. Logistic regression analysis identified low pre-op platelet count, perirenal fat stranding, and post-op ICU admission as independent predictors of sepsis in both univariate and multivariable analyses (p<0.05, Table 3).

Regarding mortality, low pre-op and post-op lymphocyte counts, as well as elevated procalcitonin levels, were significantly associated with adverse outcomes (p<0.05). Postoperative NLR, creatinine and CRP were also significantly associated with mortality. Furthermore, the presence of DM, ISDU, ICU admission, and the presence of non-urological malignancies were significantly correlated with increased mortality (Table 2). Multivariable logistic regression analysis revealed that post-op NLR and ICU admission were independent predictors of mortality (Table 4).

Table 1. Demographic datas, Pre-operative and Post-operative Characteristics

		Mean ± SD	Min-Max				
Age (Years)		62.5 ± 13. 8	25-92				
BMI (kg/m²)		25.9 ± 3.9	18-34				
		% (n)					
Gender	Male	65 (112)					
Gender	Female	35 (61)					
DM		29 (51)					
IS		21 (36)					
ISDU		42 (72)					
Perirenal fat stranding		24 (41)					
N. 1	Unilateral	49 (84)					
Nephrostomy	Bilateral	51 (89)					
Solitary Kidney		6 (11)					
Nitrite (+)		9 (16)					
Pyuria		21 (37)					
Pre-op Urine Culture Growth	1	21 (36)					
ICU	(-)	80 (139)	80 (139)				
100	(+)	20(34)					
Malignancy	Urological	52 (90)					
171011gildlicy	Non-urological	48 (83)					
Sepsis	Male	20 (22)					
ocpoio .	Female	25 (15)					

34 . II.	Male	24 (27)	
Mortality	Female	30 (18)	
		Mean ± SD	Min - Max
Fever (°C)		37.4 ± 1.1	36.1-39.2
Respiratory rate (Breaths/minute)		21 ± 4.3	12-31
Heart rate (Beats/minute)		98 ± 9.7	61-142
Pre-op Cre (mg/dL)		2.9 ± 2.5	0-15
Pre-op PLT (x10 <sup>3</sup> /μL)		290.4 ± 143.8	8-726
Pre-op WBC (x10 <sup>3</sup> /μL)		14.6 ± 51.6	1-62.8
Pre-op Neutrophil (x10 <sup>3</sup> /µ	ıL)	8.7 ± 5.2	0-35
Pre-op Lymphocyte (x10 <sup>3</sup>	/μL)	1.3 ± 1.1	0-8
Pre-op CRP (x10 <sup>3</sup> /μL)		$107.4 \pm 88.1$	1-518
Pre-op Procalcitonin (mg	/L)	$9.2 \pm 20.7$	0-103
Post-op Cre (mg/dL)		2.5 ±5.9	0-7.7
Post-op WBC (x10 <sup>3</sup> /μL)		$10.3 \pm 5.0$	0-30
Post-op PLT (x10 <sup>3</sup> /μL)		290.3 ± 139.9	29-706
Post-op Neutrophil (x10 <sup>3</sup> /	/μL)	8.2 ± 4.6	0-28
Post-op Lymphocyte (x10	³/μL)	1.5 ± 2.3	0-25
Post-op CRP (x10 <sup>3</sup> /µL)		109.7 ± 126.8	1-1192
Post-op Procalcitonin (m	g/L)	6.9 ± 35. 4	0-426
NLR-Pre-op (Ratio)		9.9 ± 13.2	0.2-140
PLR-Pre-op (Ratio)		301.4 ± 232. 2	1-1288.2
SII-Pre-op (Score)		2975.2 ± 4239.6	10.8-39480
NLR-Post-op (Ratio)		8.5 ± 7.5	0.03-48.4
PLR-Post-op (Ratio)		276.9 ± 192	0.05-1084.8
SII-Post-op (Score)		2519.5± 2724.7	0.4-17346.7

**BMI:** Body Mass Index, **Cre:** Creatinine, **CRP:** C-reactive protein, **DM:** Diabetes mellitus, **ICU:** Intensive Care Unit, **IS:** Immunosuppression, **ISDU:** Immunosuppression drug use, **NLR:** Neutrophil-to-Lymphocyte Ratio, **PLR:** Platelet-to-Lymphocyte Ratio, **PLT:** Platelets, **Post-op:** Post-operative, **Pre-op:** Pre-operative, **SII:** Systemic Immune-Inflammation Index, **WBC:** White Blood Cells

Table 2. Biomarkers and Clinical Parameters Affecting Sepsis and Mortality Risk

	Sej	osis		Mort		
	(-)	(+)	P value	(-)	(+)	P value
	Mean	± SD		Mean		
BMI	25.5 ± 4	29.2 ± 0.8	0.216*	25.65 ± 4	28.51 ± 0.9	0.343*
Pre-op Plt(x10 <sup>3</sup> /μL)	301.5 ± 138	249.4 ± 158	0.051*	298 ± 144	266 ± 141	0.194*
Post-op Plt(x10³/μL)	304 ± 131	238 ± 158	0.013*	299 ± 134	262 ± 154	0.132*
Fever (°C)	36.9 ± 0.6	37.6 ± 0.9	0.089*	36.8 ± 0.7	37.5 ± 0.8	0.092*

Respiratory rate (Breaths/							
minute)	18 ± 3.2	$25 \pm 4.1$	0.251*	17 ± 3.5	26 ± 4.4(22-31)	0.112*	
Heart rate (Beats/minute)	95 ± 7.5	112 ± 11.7	0.316*	92 ± 8.5	116 ± 11.3	0.117*	
	Median (IQR)			Media	n (IQR)		
Age (Years)	67 (19)	65 (11)	0.529	67 (19)	65 (10)	0.340**	
Pre-op Cre(mg/dL)	3.1 (7)	1.6 (3)	0.283 **	2.8 (7)	2.2 (3)	0.344 **	
Pre-op WBC (x10 <sup>3</sup> /μL)	11.4 (7)	7.7 (5)	0.963 **	10.9 (5)	7.7 (6)	0.999 **	
Pre-op Neutrophil (x10 <sup>3</sup> /μL)	9.1 (5)	5.9 (5)	0.020 **	9 (4)	5.9 (5)	0.814 **	
Pre-op Lymphocyte(x10 <sup>3</sup> /μL)	1.2 (1)	0.9 (1)	0.001 **	1.2 (1)	0.9 (1)	0.031 **	
Pre-op CRP(x10 <sup>3</sup> /μL)	146 (145)	164 (121)	0.001 **	146 (145)	164 (121)	0.054 **	
Pre-op Procalcitonin (mg/L)	0.5 (17)	5.3 (21)	0.001 **	0.5 (3)	5.8 (23)	0.002 **	
Post-op Cre(mg/dL)	2.9 (2)	1.7 (1)	0.032 **	2.3 (2)	1.9 (2)	0.004 **	
Post-op WBC(x10 <sup>3</sup> /μL)	10.5 (5)	6.2 (8)	0.638 **	10 (6)	6.7 (11)	0.275 **	
Post-op Neutrophil(x10³/μL)	8.6 (3)	4.5 (7)	0.024 **	8 (4)	5.7 (10)	0.311**	
Post-op Lymphocyte(x10³/μL)	1.2 (1)	0.8 (17.8)	0.001**	1.2 (1)	0.9 (1)	0.01**	
Post-op CRP(x10 <sup>3</sup> /μL)	109 (133)	152 (128)	0.001**	109 (133)	152 (128)	0.01**	
Post-op Procalcitonin(mg/L)	0.2 (2)	2.9 (4)	0.018 **	0.2 (2)	2.9 (4)	0.008 **	
NLR-Pre-op (Ratio)	7.5 (12)	5.8 (9.2)	0.007 **	7.5 (9)	5.8 (9.6)	0.08 **	
PLR-Pre-op (Ratio)	291 (241)	117 (114)	0.681 **	260 (278)	117 (138)	0.632 **	
SII-Pre-op (Score)	2226 (5848)	826 (532)	0.513 **	2165 (4406)	934 (621)	0.471**	
NLR-Post-op (Ratio)	7.5 (9.7)	3.5 (11.6)	0.009 **	6.9 (7.1)	5.5 (17.2)	0.043 **	
PLR-Post-op (Ratio)	294 (185)	178 (268)	0.508 **	300 (185)	178 (264)	0.854 **	
SII-Post-op (Score)	2891 (3846)	621 (1216)	0.530 **	2023 (3014)	984 (1655)	0.512 **	
	%	(n)		%	(n)		
C 1	Male	<b>Iale</b> 80 (90)		76 (85) 24 (27)			
Gender	Female	75 (46)	0.448 ***	70 (43)	30 (18)	0.402***	
DM	81 (41)	19 (10)	0.712 ***	63 (32)	27(19)	0.032***	
IS	89 (32)	11 (4)	0.091 ***	86 (31)	14 (5)	0.06***	
ISDU	72 (52)	28 (20)	0.083 ***	63 (45)	37 (27)	0.004***	
Perirenal fat stranding	66 (27)	34 (14)	0.023 ***	73 (29)	27 (11)	0.826***	
Nephrostomy	Unilateral	79 (66)	0.990 ***	70 (59)	30 (25)	0.294***	
- '	Billateral	79 (70)		78 (68)	22 (20)		
Solitary Kidney	73 (8)	27 (3)	0.631 ***	73 (8)	27 (3)	0.941***	
Nitrite (+)	81 (13)	19 (3)	0.923 ***	88 (14)	12 (2)	0.233***	
Pyuria	74(35)	26 (12)	0.559 ***	74 (31)	26 (16)	0.373***	
Pre-op Urine Culture Growth	75 (27)	25 (9)	0.170 ***	70 (26)	30 (11)	0.594***	
ICU	(-)	91 (127)	0.001***	90 (125)	10 (14)	0.001***	
	(+) Urological	26 (9) 82 (74)		9 (3) 82 (73)	91 (31) 18(17)		
Malignancy	Non-		0.228 ***				
Trunghuney	urological	75 (62)		75 (54)	25 (28)		

\*: T-Test, \*\*: Mann Whitney U, \*\*\*: Pearson Chi-Square, BMI:Body Mass Index, Cre: Creatinine, CRP: C-reactive protein, DM: Diabetes mellitus, ICU: Intensive Care Unit, IS: Immunosuppression, ISDU Immunosuppression drug use, NLR: Neutrophil-to-Lymphocyte Ratio, PLR: Platelet-to-Lymphocyte Ratio, PLT: Platelets, Post-op: Post-operative, Pre-op: Pre-operative, SII: Systemic Immune-Inflammation Index, WBC: White Blood Cells,

**Table 3.** Univariate and multivariable logistic regression analysis to determine prognostic factors for sepsis after nephrostomy tube placement

	Univariate analysis							
	OR	95 % CI		P value	OR	95 % CI		P value
	OK	Lower	Upper	P value	OK	Lower	Upper	P value
Pre-op Plt(x10 <sup>3</sup> /μL)	0.981	0.966	0.996	0.012	0.993	0.988	0.998	0.010
Pre-op Lymphocyte(x10 <sup>3</sup> /μL)	1.715	0.544	5.404	0.357				
Pre-op CRP(x10 <sup>3</sup> /μL)	1.002	0.991	1.012	0.783				
<b>Pre-op Procalcitonin</b> (mg/L)	0.988	0.935	1.043	0.656				
Post-op Cre(mg/dL)	1.409	0.772	2.571	0.264				
Post-op CRP(x10 <sup>3</sup> /μL)	1.003	0.993	1.012	0.607				
Post-op procalcitonin(mg/L)	1.003	0.981	1.025	0.807				
Post-op Plt(x10³/μL)	1.011	0.998	1.023	0.102				
<b>Post-op: Lymphocyte</b> (x10 <sup>3</sup> /μL)	0.382	0.056	2.591	0.325				
NLR-Pre-op	0.992	0.872	1.129	0.905				
NLR-Post-op	1.082	0.980	1.195	0.118				
Perirenal fat stranding	6.058	1.008	36.399	0.049	5.216	1.082	25.144	0.040
ICU	52.48	6.989	94.118	0.001	38.17	7.905	184.34	0.001

Cre: Creatinine, CRP: C-reactive protein, ICU: Intensive Care Unit, NLR: Neutrophil-to-Lymphocyte Ratio, OR: Odds Ratio, PLT: Platelets, Post-op: Post-operative, Pre-op: Pre-operative,

**Table 4.** Univariate and multivariable logistic regression analysis to determine prognostic factors for mortality after nephrostomy tube placement

	Univariate analysis					Multivariable analysis			
	O.D.	95 % CI		D1	OP	95 % CI		D 1	
	OR	Lower	Upper	P value	OR	Lower	upper	P value	
<b>Pre-op Lymphocyte</b> (x10 <sup>3</sup> /μL)	0.384	0.025	5.958	0.494					
<b>Pre-op Procalcitonin</b> (mg/L)	1.013	0.964	1.064	0.606					
Post-op Cre(mg/dL)	2.646	1.086	6.449	0.032	1.829	0.990	3.382	0.54	
Post-op CRP(x10³/μL)	0.995	0.984	1.006	0.388					
Post-op procalcitonin(mg/L)	1.007	0.989	1.025	0.458					
Post-op: Lymphocyte (x10 <sup>3</sup> /µL)	2.393	0.424	13.492	0.323					
NLR-Post-op	1.159	1.022	1.314	0.021	1.121	1.023	1.228	0.014	
DM	4.997	0.710	35.155	0.106					

ISDU	9.039	0.628	130.194	0.106				
Post-op septic shock	13.538	0.714	256.812	0.083				
Malignancies (Urological/ Nonurological)	3.992	0.466	34.233	0.207				
ICU	185.99	9.705	3564.625	0.001	161.63	24.01	1087.957	0.001

Cre: Creatinine, CRP: C-reactive protein, DM: Diabetes mellitus, ISDU Immunosuppression drug use, ICU: Intensive Care Unit, NLR: Neutrophil-to-Lymphocyte Ratio, OR: Odds Ratio, Post-op: Post-operative, Pre-op: Pre-operative

## **DISCUSSION**

Obstruction caused by ureteral stones, malignancies, or other benign factors can lead to HUN, acute renal failure, and, when accompanied by infection, pyelonephritis. Acute renal failure is rarely observed in healthy individuals unless there is bilateral involvement; however, in patients with comorbidities, the disease progression may be significantly accelerated [7]. Among patients with malignancies, the reactive inflammatory response may be either exaggerated or suppressed compared to that in healthy individuals [8]. This variability can be attributed to tumor-related pathophysiology or to the effects of therapeutic agents, such as chemotherapeutics, hormonal treatments, and immunotherapy.

When sepsis-related findings are analyzed, low platelet levels—along with elevated procalcitonin and CRP levels emerge as significant markers in this patient group (Table 2). Platelets play a pivotal role in modulating the immune response during infection and maintaining vascular integrity by supporting endothelial function. In the context of sepsis, increased platelet consumption and destruction contribute to thrombocytopenia. This excessive consumption and activation of platelets may further amplify the dysregulated immune response, potentially leading to coagulopathy, multiorgan dysfunction, and increased mortality. Notably, when platelet counts fall to ≤50,000/µL, the risk of organ failure and mortality increases significantly [9-12]. Similarly, procalcitonin and CRP as essential biomarkers for assessing the severity of infection and evaluating the immune response. These markers have been consistently validated as reliable indicators across numerous studies [11]. Even though leukocytosis is commonly observed in sepsis, leukopenia may also occur, as highlighted in the SIRS criteria. In our patient cohort, neutropenia and lymphopenia were observed at statistically significant levels among those who developed

sepsis (Table 2,3). The severity of neutropenia was further supported by a decrease in post-op NLR compared to preop values. NLR is considered a reliable marker that reflects both the intensity of the inflammatory response and the functional state of the immune system. Neutrophils constitute the first line of defense against microorganisms during infection, whereas lymphocytes reflect the activity of the adaptive immune response. An increase in neutrophil counts accompanied by a decrease in lymphocyte counts during sepsis indicates a dysregulated immune response and the uncontrolled progression of inflammation [8,9,12-14]. Interestingly, although the existing literature generally reports an elevated NLR during inflammatory conditions, our study found that NLR was lower in the sepsis group, which consisted exclusively of patients with malignancies (Table 2-3). We believe this situation is attributable to immune system dysfunction resulting from the coexistence of malignancy and sepsis [15,16]. The fact that post-op NLR emerged as an independent predictor of mortality in both univariate and multivariable analyses further highlights the prognostic value of this parameter.

In our study, ICU admission emerged as an independent predictor of sepsis in both univariate and multivariable analyses. This finding indicates that patients requiring ICU-level care are at significantly higher risk for developing sepsis, even after adjusting for other clinical factors. ICU admission likely reflects early physiological deterioration and increased disease burden. Therefore, the presence of ICU-level needs should be regarded as an early warning sign, prompting heightened clinical vigilance and timely interventions to prevent or mitigate sepsis. Another independent predictor of sepsis identified in our study was the presence of perirenal fat stranding (Table 2–3). Inflammation within the perirenal fat tissue contributes to an increased microbial burden and a

heightened risk of bacterial infections. It is well known that invasive interventions under such inflammatory conditions can significantly elevate the risk of sepsis [5,17]. The Mayo Adhesive Probability (MAP) score is a valuable tool for evaluating the inflammatory burden of perirenal fat tissue and estimating the associated risks of infection and surgical complications. An elevated MAP score serves as a reliable indicator of severe perirenal inflammation and the potential progression of infection [18–20]. The integration of predictive scoring systems, such as the MAP score, into the clinical management of similar patient populations may enhance clinical decision-making and optimize treatment strategies.

When evaluating the parameters associated with mortality, it becomes evident that, despite pathophysiological similarities with sepsis, certain distinctions are observed. Notably, neutropenia and thrombocytopenia do not emerge as significant predictive factors (Table 2,4). In contrast, low preop and post-op lymphocyte levels, reduced post-op NLR, elevated pre-op and post-op procalcitonin and post-op CRP levels appear as key predictors of mortality, consistent with their roles in sepsis. However, among these variables, only post-op NLR was found to be an independent predictor of mortality after nephrostomy in logistic regression analysis (Table 2,4). Lymphopenia reflects a weakened immune system in the context of infection and indicates an impaired adaptive immune response [16,21,22]. Additionally, creatinine levels following nephrostomy were identified as predictors of mortality in univariate analysis; however, this association did not remain significant in multivariable analysis (Table 2,4). Although mortality rates were statistically higher among patients with non-urological malignancies, logistic regression analysis revealed no significant difference in mortality between patients with urological and non-urological malignancies (Table 2,4). Diabetes mellitus and ISDU complicate management of infections, particularly in the context of immunosuppression induced by sepsis and malignancy. These conditions exacerbate septic progression, increase the likelihood of complications, and elevate the risk of mortality [15,23,24]. Interestingly, while a statistically significant association (p<0,05) was found between DM and ISDU with mortality in our cohort, logistic regression analysis showed that they were not independent predictors of mortality. These results suggest that although DM and ISDU may indicate disease severity, they do not necessarily translate into increased

mortality risk in all settings, highlighting the need for individualized patient assessment (Table 2,4). ICU admission emerged as a key predictor of both sepsis and mortality. ICU management strategies play a crucial role in directly influencing patient outcomes. The literature emphasizes that early ICU admission and timely implementation of supportive therapies can significantly reduce mortality rates [25]. In this context, the strong association between ICU admission and mortality observed in our study is consistent with findings in the literature. Furthermore, among the parameters variables included in multivariable logistic regression analysis, post-op ICU admission was identified as independent predictor of mortality (Table 4).

This study has several limitations, including its retrospective design, relatively small sample size, and single-center setting. Additionally, more detailed classification of malignancy types could have better illustrated the diversity of etiopathogenesis and inflammatory responses. The inclusion of data regarding chemotherapeutic agents, hormonal therapies or immunotherapies might have helped reduce heterogeneity and allowed for a more nuanced analysis. Due to the retrospective nature of our study, it was not possible to clearly differentiate between primary causes of mortality, which may have contributed to the high mortality rate observed. We believe that the actual mortality rate is likely lower, as our cohort consisted exclusively of cancer patients, and cancer-related deaths may have inflated the observed rate-constituting another important limitation of this study. Nevertheless, this study provides a foundation for future randomized prospective studies with larger cohorts, to evaluate dynamic changes in inflammatory markers. Including patients with benign causes of nephrostomy as a control group would enhance the understanding of malignancy-specific outcomes. Furthermore, assessing long-term results and quality of life would contribute the development of more comprehensive and evidence-based guidelines for the management of patients requiring nephrostomy.

#### CONCLUSION

In conclusion, this study has identified key predictors associated with sepsis and mortality following nephrostomy, offering important insights to inform clinical decision-making. Our findings emphasize the significance of inflammatory markers (NLR, procalcitonin, CRP), pre-op

and post-op blood counts (platelet, lymphocyte, neutrophil), creatinine, and clinical factors such as perirenal fat stranding, DM, ISDU, ICU admission. The early recognition of high-risk patients, combined with timely and targeted interventions, is essential for improving clinical outcomes and minimizing adverse events in this vulnerable patient population.

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**Informed Consent:** Informed consent was obtained from the all patient involved in this study.

**Ethical Approval:** The study was approved by the Ethics Committee of Başakşehir Çam and City Hospital (Approval No: 2023-183)

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