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Original Article

How Much Do We Know About Acute Kidney Injury Following Pneumonectomy? A Retrospective Study

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ABSTRACT

OBJECTIVE: Acute kidney injury (AKI) is a significant postoperative complication of thoracic surgery, but data on AKI after pneumonectomy remain scarce. This study aimed to determine the incidence, risk factors, and short-term outcomes of AKI, as defined by Kidney Disease Improving Global Outcomes 2012 criteria, occurring within one week after pneumonectomy.

MATERIAL AND METHODS: This retrospective single-center cohort included adults who underwent elective pneumonectomy between 2008–2018. Patients with preoperative chronic kidney disease or AKI, or with missing data, were excluded. Demographic, perioperative, and postoperative data were collected from hospital records. AKI was identified based on postoperative creatinine values measured within one week. Univariate and multivariate logistic regression analyses were performed to identify independent risk factors.

RESULTS: Of the 308 patients, 166 met the inclusion criteria. The incidence of AKI was 12% (19 stage 1, 1 stage 2); none required renal replacement therapy. In multivariate analysis, increased body mass index [odds ratio (OR): 1.10, 95% confidence interval (CI): 1.01–1.21, $P = 0.038$]; acetylsalicylic acid use (OR: 10.56, 95% CI: 1.58–70.60, $P = 0.015$); higher intraoperative fluid volume (OR: 1.00, 95% CI: 1.00–1.00, $P = 0.036$); and length of stay (OR: 1.07, 95% CI: 1.01–1.13, $P = 0.016$) were associated with increased AKI risk, while nonsteroidal anti-inflammatory drug use was independently protective (OR: 0.03, 95% CI: 0.00–0.13, $P < 0.001$), as was diuretic use (OR: 0.06, 95% CI: 0.01–0.50, $P = 0.009$). AKI was associated with longer hospitalization but not with increased mortality.

CONCLUSION: Reducing the incidence of AKI may improve patient outcomes, and AKI should be considered a key quality indicator in thoracic surgery. Identifying and understanding the risk factors for AKI may provide the foundation for predictive models and guide strategies to prevent this complication.

KEYWORDS: Acute kidney injury, pneumonectomy, anesthesia

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INTRODUCTION

Acute kidney injury (AKI) is a common and serious complication associated with substantial morbidity and mortality after major surgical procedures.[1] Thoracic surgery carries the third-highest risk of postoperative AKI, after cardiac and general surgery.[2] Patient characteristics, medical history, and surgical and anesthetic factors were identified as risk factors for AKI after thoracic surgery.[3,4] The incidence of AKI was 12.1% after bilobectomy or pneumonectomy and was associated with increased 30-day mortality and longer hospital stay.[3] Furthermore, AKI was associated with significantly higher rates of tracheal reintubation and postoperative mechanical ventilation following lung resection surgery.[5] The identification of patient and procedural risk factors may improve long-term outcomes by allowing for targeted perioperative management.

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This study was presented as a poster at the 18th World Congress of Anaesthesiologists (2024).

The main objective of our work was to determine the incidence and risk factors for postoperative AKI within the first 7 days after pneumonectomy, defined according to the serum creatinine (sCr) criteria of the Kidney Disease Improving Global Outcomes (KDIGO) 2012 guideline.[6] The secondary aims included assessment of postoperative length of stay in the intensive care unit and in hospital, complications, and mortality at 30 days, 90 days, and 1 year. We present this article in accordance with the STROBE reporting checklist.

MATERIAL AND METHODS

This retrospective, observational, single-center cohort study was conducted at a university hospital. The Ethics Committee of Ankara University Faculty of Medicine approval number is I8-568-21, date: 15/10/2021, and the clinical trial number is NCT05672238. The requirement to obtain informed consent was waived because the study was retrospective. Patients aged over 18 years who underwent elective pneumonectomy for primary lung cancer at Ankara University İbni Sina Hospital between January 2008 and December 2018 were included. Patients with chronic kidney disease, preoperative AKI, missing data, or who underwent pneumonectomy for trauma or other indications were excluded.

Patient demographics, American Society of Anesthesiologist (ASA) status, comorbidities, and intraoperative variables [surgery and anesthesia duration, fluid and red blood cell (RBC) administration, hemodynamic events, urine output, and furosemide use] were collected. The Charlson comorbidity index (CCI) and the estimated 10-year survival were calculated using patients' preoperative information.[7] Postoperative data included fluid therapy during the first 24 hours, sCr levels, RBC transfusions, medications, and complications occurring within 7 days. All data were collected from patients' records. Non-AKI complications were classified using the Clavien-Dindo classification (CDC),[8] and mortality was assessed at 30 days, 90 days, and 1 year.

The baseline sCr was defined as the most recent measurement obtained within 7 days before surgery. Patients without a sCr measurement during this period were excluded. All available sCr values measured within the first postoperative week were evaluated. Blood sampling, performed as part of routine clinical follow-up, was not conducted at fixed intervals, resulting in variable sampling frequency among patients. AKI was defined and staged according to the KDIGO criteria.[6] Since urinary catheters were not routinely used at our center during the one-week postoperative period, AKI was defined based on sCr levels. The maximum AKI stage was calculated for patients who developed AKI.

Main Points

- Acute kidney injury (AKI) developed in 12% of patients following pneumonectomy in this single-center, 10-year retrospective cohort study.
- Multivariate analysis identified higher body mass index, greater intraoperative fluid administration, and preoperative acetylsalicylic acid use as independent risk factors for AKI development.
- AKI was associated with a significantly longer hospital stay but not with increased short-term mortality.
- Recognizing these risk factors underscores the need for individualized perioperative management and targeted preventive strategies in patients undergoing pneumonectomy.

Statistical Analysis

Statistical analyses were performed using SPSS version 11.5. Quantitative variables were summarized using mean ± standard deviation and median (minimum–maximum), while qualitative variables were presented as counts and percentages. The normality of quantitative variables was assessed using the Shapiro-Wilk test. Differences between two groups were assessed using the Student's t-test or Mann-Whitney U test, as appropriate. The chi-square test or Fisher's exact test was used to evaluate associations between qualitative variables. Univariate and multivariate logistic regression analyses were performed to identify independent risk factors for AKI development. Variables with a P value <0.10 in univariate analysis, as well as those deemed clinically relevant, were included in the multivariate model. Odds ratios (OR) and 95% confidence intervals (CI) were reported. A P value <0.05 was considered statistically significant.

RESULTS

From January 2008 to December 2018, 308 patients underwent pneumonectomy at our center. Seven patients with preoperative chronic kidney disease and 135 patients with missing data were excluded from the statistical analysis (Figure 1). AKI was observed in 20 patients (12%); 19 patients (95.0%) were classified as stage 1 and one patient (5.0%) was classified as stage 2. None of the patients required renal replacement therapy. At discharge, 6 (30.0%) patients still had AKI: 5 (83.3%) were classified as stage 1 and 1 (16.7%) as stage 2. Among the patients, hypertension (HT) was present in 49 patients (29.5%), diabetes mellitus (DM) in 24 patients (14.5%), obstructive lung disease in 23 patients (13.9%), atherosclerotic heart disease in 20 patients (12.0%), hypothyroidism in 6 patients (3.6%), and other comorbidities in 11 patients (6.6%). Patient demographics are summarized in Table 1.

Compared with patients without AKI, patients with AKI had a higher body mass index (BMI), more comorbidities, and a higher CCI score. AKI occurred in 20.4% of patients with HT and in 25% of patients with DM (Table 2). In univariate analysis, increased BMI (OR: 1.12, 95% CI: 1.03–1.21, P = 0.006), higher CCI score (OR: 1.62, 95% CI: 1.11–2.36, P = 0.013), presence of comorbidities (OR: 2.92, 95% CI: 1.01–8.45, P = 0.048), HT (OR: 2.74, 95% CI: 1.06–7.09, P = 0.037), DM (OR: 3.05, 95% CI: 1.04–8.94, P = 0.042), drug use (OR: 3.49, 95% CI: 1.11–10.94, P = 0.032), non-steroidal anti-inflammatory drug (NSAID) use (OR: 4.98, 95% CI: 1.09–22.69, P = 0.038), oral antidiabetic drug use (OR: 3.25, 95% CI: 1.10–9.59, P = 0.033), and acetylsalicylic acid use (OR: 5.83, 95% CI: 1.84–18.44, P = 0.003) were significantly associated with development of AKI. In the multivariate logistic regression analysis, increased BMI (OR: 1.10, 95% CI: 1.01–1.21, P = 0.038), acetylsalicylic acid use (OR: 10.56, 95% CI: 1.58–70.60, P = 0.015), and diuretic use (OR: 0.06, 95% CI: 0.01–0.50, P = 0.009) were found to be independent predictors of AKI. Although diuretic use was

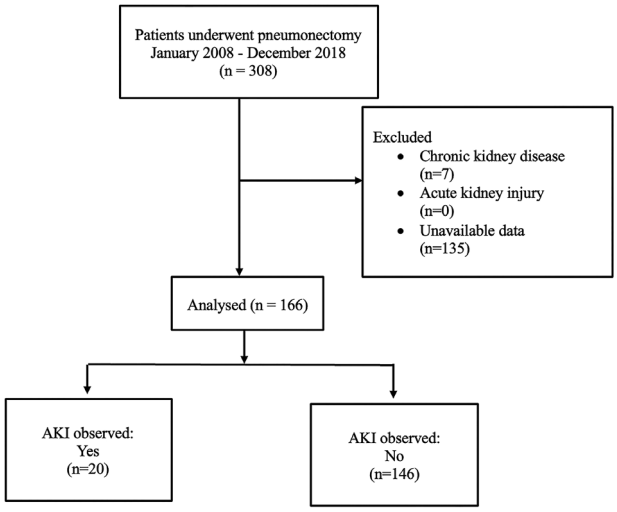

Figure 1. Flow chart

AKI: acute kidney injury

not significantly associated with AKI in univariate analysis, it emerged as an independent protective factor in the multivariate logistic regression model.

AKI occurred in seven (28%) of the patients who received colloids, compared with 13 (9.2%) of the patients who did not receive colloids; this difference was statistically significant (P = 0.015). Moreover, the mean fluid volume administered was 2525.00±1312.64 mL in patients with AKI and 1882.53±768.11 mL in patients without AKI, representing a statistically significant difference between the two groups (P = 0.006). Other intraoperative variables did not differ significantly between groups (Table 3). Univariate logistic regression analysis indicated that higher total intraoperative fluid volume was significantly associated with an increased risk of AKI [OR: 1.00 (95% CI: 1.00–1.00), P = 0.008], whereas surgical duration exhibited only a borderline association [OR: 1.01 (95% CI: 1.00–1.01), P = 0.092]. In the multivariate analysis, total fluid volume remained an independent predictor of AKI (OR: 1.00; 95% CI: 1.00–1.00; P = 0.036), whereas surgical duration was not statistically significant (P = 0.761).

In the postoperative period, AKI incidence was significantly higher in patients who received RBC transfusion, NSAIDs, or furosemide, as well as in those with complications and CDC grade ≥ 3. The length of hospital stay (LOS) was significantly longer among patients with AKI than among those without AKI. However, there was no significant difference in mortality rates between groups (Table 4). Univariate logistic regression analysis revealed that postoperative AKI was significantly associated with RBC transfusion during the first week (OR: 4.10, 95% CI: 1.18–14.21, P = 0.026), NSAID use (OR: 0.03, 95% CI: 0.01–0.13, P < 0.001), and LOS (OR: 1.06, 95% CI: 1.02–1.11, P = 0.001), while furosemide use (OR: 3.45, 95% CI: 0.89–13.33, P = 0.073), fluoroquinolone use (OR: 2.79, 95% CI: 0.85–9.13, P = 0.090), and CDC class ≥3 (OR: 0.95, 95% CI: 0.35–2.56, P = 0.922) were not significantly associated. In the multivariate logistic regression analysis, only postoperative NSAID use (OR: 0.03, 95% CI: 0.00–0.13, P < 0.001) and LOS (OR: 1.07, 95% CI: 1.01–1.13, P = 0.016) were identified as independent predictors.

Table 1. Demographic data of all patients

Parameters		
Age (years)	Mean ± SD	58.96±8.50
	Median (min-max)	59.00 (26.00–80.00)
Gender, n (%)	Female	14 (8.4)
	Male	152 (91.6)
Diagnosis, n (%)	Squamous epithelial cell carcinoma	107 (64.5)
	Adenocarcinoma	29 (17.5)
	Non-small cell lung cancer	15 (9.0)
	Pleomorphic carcinoma	5 (3.0)
	Carcinoid tumor	2 (1.2)
	Others	8 (4.8)
BMI (kg/m²)	Mean ± SD	26.53±5.18
	Median (min-max)	25.86 (15.28–48.33)
ASA class, n (%)	1	76 (45.8)
	2	78 (47.0)
	3	12 (7.2)
Comorbidity, n (%)	No	77 (46.4)
	Yes	89 (53.6)
CCI point	Mean ± SD	3.59±1.21
	Median (min-max)	4.00 (1.00–9.00)
Diuretic usage, n (%)	No	131 (78.9)
	Yes	35 (21.1)
AKI stage, n (%)	1	19 (95.0)
	2	1 (5.0)
Presence of AKI at discharge, n (%)	No	14 (70.0)
	Yes	6 (30.0)
AKI stage at discharge, n (%)	1	5 (83.3)
	2	1 (16.7)
Complication, n (%)	No	117 (70.5%)
	Yes	49 (29.5%)
	Wound infection	14 (8.4)
	Arrhythmia	12 (7.2)
	Pneumonia	11 (6.6)
	Bleeding	10 (6.0)
	Bronchopleural fistula	5 (3.0)
	Elevated cardiac enzymes	4 (2.4)
	Respiratory failure	2 (0.12)
	Empyema	2 (0.12)
	Chylothorax	2 (0.12)
	Pulmonary thromboembolism	1 (0.6)
	Pericarditis	1 (0.6)
	Recurrent nerve damage	1 (0.6)
	Esophageal rupture	1 (0.6)

AKI: acute kidney injury, ASA: American Society of Anesthesiologist, BMI: body mass index, CCI: Charlson comorbidity index, SD: standard deviation, min: minimum, max: maximum

Table 2. Evaluation of demographic characteristics and risk factors of patients with and without AKI

		AKI		*P* value
		No	Yes	
Gender, n (%)	Female	11 (78.6)	3 (21.4)	*P* = 0.38[b]
	Male	135 (88.8)	17 (11.2)	
Age (year)	Mean ± SD	58.72±8.47	60.75±8.75	*P* = 0.32[c]
	Median (min-max)	58.00 (26.00–80.00)	61.50 (42.00±78.00)	
BMI	Mean ± SD	26.17±5.02	29.25±5.77	***P* = 0.016**[d]
	Median (min-max)	25.51 (15.28–48.33)	30.69 (18.34–42.61)	
ASA, class, n (%)	1	71 (93.4)	5 (6.6)	*P* = 0.14[a]
	2	65 (88.3)	13 (16.7)	
	3	10 (83.3)	2 (16.7)	
Comorbidity n, (%)	No	72 (93.5)	5 (6.5)	***P* = 0.041**[a]
	Yes	74 (83.1)	15 (16.9)	
CCI point	Mean ± SD	3.50±1.19	4.25±1.16	***P* = 0.005**[d]
	Median (min-max)	3.00 (1.00–9.00)	4.00 (2.00–7.00)	
Hypertension, n (%)	No	107 (91.5)	10 (8.5)	***P* = 0.032**[a]
	Yes	39 (79.6)	10 (20.4)	
Diabetes mellitus, n (%)	No	128 (90.1)	14 (9.9)	***P* = 0.046**[b]
	Yes	18 (75.0)	6 (25.0)	
Drug usage, n (%)	No	68 (94.4)	4 (5.6)	***P* = 0.025**[a]
	Yes	78 (83.0)	16 (17.0)	
Diuretic usage, n (%)	No	114 (87.0)	17 (13.0)	*P* = 0.57[b]
	Yes	32 (91.4)	3 (8.6)	
NSAID, n (%)	No	141 (89.2)	17 (10.8)	*P* = 0.057[b]
	Yes	5 (62.5)	3 (37.5)	
Paracetamol, n (%)	No	145 (87.9)	20 (12.1)	*P* > 0.99[b]
	Yes	1 (100.0)	0 (0.0)	
ACEI, n (%)	No	128 (87.7)	18 (12.3)	*P* > 0.99[b]
	Yes	18 (90.0)	2 (10.0)	
Furosemide, n (%)	No	143 (87.7)	20 (12.3)	*P* > 0.99[b]
	Yes	3 (100.0)	0 (0.0)	
Oral antidiabetic, n (%)	No	129 (90.2)	14 (9.8)	***P* = 0.038**[b]
	Yes	17 (73.9)	6 (26.1)	
Acetylsalicylic acid, n (%)	No	136 (90.7)	14 (9.3)	***P* = 0.005**[b]
	Yes	10 (62.5)	6 (37.5)	

[a]: chi-square test, [b]: Fisher's exact test, [c]: Student's t-test, [d]: Mann-Whitney U test

ACEI: angiotensin-converting enzyme inhibitors, AKI: acute kidney injury, ASA: American Society of Anesthesiologist, BMI: body mass index, CCI: Charlson comorbidity index, SD: standard deviation, NSAID: non-steroidal anti-inflammatory drug, min: minimum, max: maximum

DISCUSSION

We assessed the incidence of AKI and its risk factors following pneumonectomy. AKI, as defined by KDIGO criteria, occurred in 12% of patients within the first postoperative week. In multivariable analysis, increased BMI, acetylsalicylic acid use, and diuretic use were independently associated with AKI. AKI was also significantly associated with higher complication rates and a prolonged hospital stay, but not with increased mortality.

The incidence of AKI following thoracic surgery was found to be 15.1% in the study conducted by Naruka et al.[9] We identified a lower AKI rate; however, our results pertain to a one-week period. In a meta-analysis in which 34,826 patients were evaluated, the incidence of AKI following thoracic surgery was 8.8%.[10] We may have observed a different incidence due to multiple factors related to the patient, surgery, and anesthesia that contribute to the development of AKI, as well as to the methods used in our center.

Table 3. Intraoperative data of patients with and without AKI

		AKI		*P* value
		No	Yes	
Hypotension, n (%)	No	131 (90.3)	14 (9.7)	***P* = 0.024**[b]
	Yes	15 (71.4)	6 (28.6)	
Hypertension, n (%)	No	126 (88.7)	16 (11.3)	*P* = 0.50[b]
	Yes	20 (83.3)	4 (16.7)	
Red blood cell transfusion, n (%)	No	132 (88.6)	17 (11.4)	*P* = 0.50[b]
	Yes	14 (82.4)	3 (17.6)	
Colloid, n (%)	No	128 (90.8)	13 (9.2)	***P* = 0.015**[b]
	Yes	18 (72.0)	7 (28.0)	
Total fluid	Mean ± SD	1882.53±768.11	2525.00±1312.64	***P* = 0.006**[c]
	Median (min-max)	1500.00 (750–6000)	2000.00 (1000–7000)	
Urine output (kg/hour)	Mean ± SD	1.53±1.06	1.45±1.29	*P* = 0.42[c]
	Median (min-max)	1.20 (0.19–5.70)	1.05 (0.17–5.80)	
Anuria, n (%)	No	130 (89.7)	15 (10.3)	*P* = 0.14[b]
	Yes	16 (76.2)	5 (23.8)	
Oliguria, n (%)	No	125 (89.3)	15 (10.7)	*P* = 0.32[b]
	Yes	21 (80.8)	5 (19.2)	
Furosemide, n (%)	No	143 (87.7)	20 (12.3)	*P* > 0.99[b]
	Yes	3 (100.0)	0 (0.0)	
Duration of anesthesia	Mean ± SD	219.66±63.03	250.25±80.42	*P* = 0.22[c]
	Median (min-max)	210.00 (75.00–540.00)	210.00 (150.00–390.00)	
Duration of surgery	Mean ± SD	187.10±57.31	210.50±75.57	*P* = 0.52[c]
	Median (min-max)	180.00 (75.00–480.00)	172.50 (120.00–360.00)	

[b]: Fisher's exact test, [c]: Mann-Whitney U test
AKI: acute kidney injury, SD: standard deviation, min: minimum, max: maximum

The identification of risk factors for postoperative AKI following thoracic surgery remains a challenge. In the study conducted by Licker et al.[11], anesthesia duration, forced expiratory volume in 1 second, and ASA classification were significantly associated with postoperative AKI. Ishikawa et al.[5] identified HT, peripheral vascular disease, angiotensin II receptor blocker (ARB) use, and hydroxyethyl starch use as independent risk factors. Zhao et al.[12] found that older age, HT, DM, ARB use and angiotensin-converting enzyme inhibitor use, preoperative albumin and sCr levels, intraoperative blood loss, and hypotension were significantly associated with postoperative AKI. In the univariate analysis, increased BMI, higher CCI score, HT, DM, and presence of comorbidity were each significantly associated with AKI following pneumonectomy. Similarly, in a large cohort study of 161,185 patients undergoing major surgery, older age, male sex, higher BMI, DM, HT, malignancy, lung disease, a low glomerular filtration rate, use of ACEI/ARB, use of diuretics, and surgery later during hospitalization were associated with postoperative AKI following thoracic surgery.[2] The development of AKI during the perioperative period is influenced by a number of patient-, anesthetic-, and surgery-related factors. We identified increased BMI, acetylsalicylic acid use, and diuretic use as independent predictors of postoperative AKI. Our results highlight BMI as a strong risk factor for AKI, consistent with previous studies reporting increased susceptibility to renal complications among overweight and obese patients. The observed association between acetylsalicylic acid use and AKI is notable, as acetylsalicylic acid can impair renal autoregulation and may exacerbate perioperative renal hypoperfusion, especially in patients with multiple comorbidities or in those exposed to other nephrotoxic agents. However, the strong association between acetylsalicylic acid use and AKI in our analysis should be interpreted with caution due to the relatively small number of AKI events and potential residual confounding. Interestingly, diuretic use was associated with a markedly reduced risk of AKI. While this finding may suggest a protective effect, it is possible that patients receiving diuretics were more closely monitored and managed in the perioperative period, or that diuretic use reflects other aspects of patient care not fully captured in the present dataset. Traditional risk factors such as HT and DM were not independent predictors of AKI in multivariate analysis, likely due to overlap with BMI and medications and to the limited statistical power of our cohort. Our results are consistent with the findings reported in previous studies. However, the heterogeneity of thoracic surgery patient populations and varying treatment protocols across centers may affect the identification of AKI-related risk factors. Further

research is warranted to definitively determine the risk factors for AKI following pneumonectomy.

During the perioperative phase, patients are at increased risk of developing AKI because of surgical stress, systemic inflammation, renal hypoperfusion induced by hypovolemia, and the vasodilatory and cardiodepressant effects of anesthesia.[1] In this cohort, the principal intraoperative risk factors for postoperative AKI were hypotension and greater total intraoperative fluid administration. In thoracic surgery,

Table 4. Evaluation of postoperative data of patients with and without AKI

		AKI		*P* value
		No	Yes	
RBC-tx in 7 days, n (%)	No	127 (91.4)	12 (8.6)	***P* = 0.006**[b]
	Yes	19 (70.4)	8 (29.6)	
Total fluid in 24 hours	Mean ± SD	1949.47±325.34	2026.25±415.67	*P* = 0.66[c]
	Median (min-max)	1970.00 (1260–3600)	1975.00 (1350–3090)	
Patient controlled analgesia, n (%)	IV fentanyl	2 (66.7)	1 (33.3)	*P* = 0.47[b]
	IV contromal	135 (87.7)	19 (12.3)	
	Epidural bupivacaine	7 (100.0)	0 (0.0)	
Postoperative drug usage				
NSAID, n (%)	No	4 (33.3)	8 (66.7)	***P* < 0.001**[b]
	Yes	142 (92.2)	12 (7.8)	
Paracetamol, n (%)	No	113 (90.4)	12 (9.6)	*P* = 0.10[b]
	Yes	33 (80.5)	8 (19.5)	
Opioid, n (%)	No	104 (88.9)	13 (11.1)	*P* = 0.57[a]
	Yes	42 (85.7)	7 (14.3)	
ARB, n (%)	No	138 (87.9)	19 (12.1)	*P* > 0.99[b]
	Yes	8 (88.9)	1 (11.1)	
ACEI, n (%)	No	128 (86.5)	20 (13.5)	*P* = 0.13[b]
	Yes	18 (100.0)	0 (0.0)	
Furosemide, n (%)	No	128 (90.8)	13 (9.2)	***P* = 0.015**[b]
	Yes	18 (72.0)	7 (28.0)	
Cephalosporin, n (%)	No	5 (100.0)	0 (0.0)	*P* > 0.99[b]
	Yes	141 (87.6)	20 (12.4)	
Fluoroquinolones, n (%)	No	103 (92.0)	9 (8.0)	***P* = 0.022**[a]
	Yes	43 (79.6)	11 (20.4)	
LOS in hospital	Mean ± SD	13.23±8.22	23.90±20.21	***P* = 0.011**[c]
	Median (min-max)	12.00 (4.00–63.00)	19.50 (6.00–85.00)	
Complication, n (%)	No	110 (94.0)	7 (6.0)	***P* < 0.001**[a]
	Yes	36 (73.5)	13 (26.5)	
CDC, n (%)	<3	137 (90.1)	15 (9.9)	***P* = 0.015**[b]
	≥3	9 (64.3)	5 (35.7)	
Hospital mortality, n (%)	No	145 (88.4)	19 (11.6)	*P* = 0.23[b]
	Yes	1 (50.0)	1 (50.0)	
30-day mortality, n (%)	No	144 (87.8)	20 (12.2)	*P* > 0.99[b]
	Yes	2 (100.0)	0 (0.0)	
90-day mortality, n (%)	No	140 (88.1)	19 (11.9)	*P* > 0.99[b]
	Yes	6 (85.7)	1 (14.3)	
1-year mortality, n (%)	No	136 (88.9)	17 (11.1)	*P* = 0.16[b]
	Yes	9 (75.0)	3 (25.0)	

ACEI: angiotensin-converting enzyme inhibitors, AKI: acute kidney injury, ARB: angiotensin receptor blockers, CDC: Clavien-Dindo classification, RBC-tx: red blood cell transfusion, SD: standard deviation, LOS: length of stay, NSAID: non-steroidal anti-inflammatory drug

[a]: chi-square test, [b]: Fisher's exact test, [c]: Mann-Whitney U test

perioperative fluid and hemodynamic management is a complex area. We hypothesized that intravascular volume restriction might be linked to AKI following pneumonectomies. However, fluid restriction alone did not appear to be a risk factor for AKI in our study population. Kaufmann et al.[13] found that fluid restriction to prevent pulmonary complications did not cause AKI when regulated to prevent relative hypovolemia. In our study, the association between increased intraoperative fluid administration and AKI development is noteworthy. While adequate fluid resuscitation is essential, excessive fluid administration may contribute to renal congestion and impaired oxygenation, thus paradoxically increasing the risk of AKI. Our findings highlight the importance of individualized fluid management strategies and careful intraoperative monitoring.

In contrast to crystalloids, we established a connection between colloid administration (we prefer albumin) and AKI following lung resection. There is no definitive conclusion regarding the use of albumin as a colloid in thoracic surgery. However, our results indicated that 28% of patients receiving colloids developed postoperative AKI. Hydroxyethyl starch use has previously been shown to be associated with AKI.[5,14] There is no evidence linking albumin use to AKI. In contrast, lower preoperative albumin levels have been shown to be a risk factor for AKI.[14,15] Low albumin levels negatively impact patient outcomes, which is not surprising given that albumin levels are influenced by numerous factors, including malnutrition and stress levels.

We observed that the incidence of AKI was significantly higher in patients with intraoperative hypotension. It was shown that intraoperative vasopressor use by Licker et al.[11], intraoperative hypotension by Ren and Meng[16], and lower intraoperative mean arterial pressure values by Zhao et al.[12] were related to the development of AKI. Cardinale et al.[17] found that AKI occurred in patients who experienced greater intraoperative blood loss and received more postoperative transfusions. Hypotension during surgery was more frequently observed among patients who developed AKI, underscoring the critical role of hemodynamic stability in renal protection. These findings are consistent with previous literature demonstrating the vulnerability of renal function to intraoperative hemodynamic disturbances. Although no association was found between intraoperative blood component transfusions and AKI in our study, it is important to note that our exposure level was very low. However, transfusions during the first postoperative week were associated with AKI.

Other intraoperative factors, such as HT, blood transfusion, altered urine output (anuria or oliguria), anesthesia, and surgical duration, did not independently predict AKI in this cohort. Although surgical duration showed a borderline association, it did not reach statistical significance in the multivariate analysis. Zhao et al.[12] found a moderate association but poor predictive value of urine output for AKI. Prolonged anesthesia duration[11] and longer surgical time[18] have been shown to be associated with AKI. Matesanz et al.[18] found that patients who developed AKI following thoracic surgery had a greater postoperative systemic inflammatory response, which was associated with an increased risk of complications and mortality.

Our study demonstrates that postoperative AKI was independently associated with prolonged LOS and the absence of NSAID use. While use of RBC-tx, furosemide, and fluoroquinolones was more common in patients who developed AKI, these associations did not remain statistically significant after adjustment for confounders in the multivariate model.

This study comprehensively evaluated postoperative risk factors for AKI following major surgery, with particular focus on clinical interventions and postoperative complications. Our findings demonstrate that patients with AKI had higher rates of RBC transfusion, greater use of NSAIDs, furosemide, and fluoroquinolones, more complications, and longer hospital stays. In a multivariate model, postoperative AKI was independently associated with prolonged hospital stay and the absence of NSAID therapy. This finding is counterintuitive, as NSAIDs are generally considered nephrotoxic and have been widely reported as risk factors for AKI, particularly in vulnerable populations.[19] The unexpected association between postoperative NSAID use and a reduced risk of AKI is most likely attributable to selection bias, as clinicians may have avoided NSAIDs in patients considered to be at higher risk (e.g., those perceived to have increased susceptibility to renal impairment). However, due to the retrospective design and incomplete documentation regarding the reasons for withholding NSAIDs, we could not exclude such cases from the analysis. As such, this finding should be interpreted with great caution and cannot be considered evidence of a true protective effect. Additionally, the relatively small number of AKI events may have influenced the statistical outcome. Therefore, while our results suggest a potential inverse association between NSAID use and postoperative AKI, these findings should be interpreted with caution and require validation in larger prospective studies.

The LOS stay after thoracic surgery is influenced by the type of surgery, patient comorbidities, and postoperative complications. Patients without complications after wedge resection are discharged earlier than those with CDC grade ≥3 complications after pneumonectomy. The LOS has been shown to be twice as long in patients with CDC grade ≥3 complications than in those with CDC grade <3 complications.[20] AKI following thoracic surgery has also been consistently associated with prolonged hospital stays, as demonstrated in multiple studies.[3,5] In our study, the incidence of AKI was higher among patients with CDC grade ≥3 complications than among those with CDC grade <3. Similarly, Murphy et al.[21] reported that AKI after thoracic surgery was significantly associated with CDC grade 3, the comprehensive complications index, pneumonia, and respiratory failure.

In contrast to much of the existing literature, we were unable to demonstrate a positive relationship between mortality and postoperative AKI.[3,18] Differences in patient characteristics and treatment protocols at our center may have contributed to this finding. Additionally, the relatively small number of AKI cases in our cohort may have limited the statistical power to detect a significant difference in mortality rates. Because of the correlation between preoperative comorbidities and the risk of AKI, perioperative management must be conducted more carefully to prevent AKI and improve patient outcomes. The incidence of AKI has been reported to increase with the extent

of lung resection.[17] Because of the distinctive nature of their condition, pneumonectomy patients require increased levels of care. Moreover, mortality in pneumonectomy patients is multifactorial and may be influenced by a range of perioperative and non-renal factors, potentially diluting the impact of AKI on overall outcomes. The lack of an established clinical prediction rule for AKI following pneumonectomy may negatively affect clinicians' approach to this population. Recognition of risk factors could help establish clinical prediction rules and enhance patient outcomes. Further studies with larger sample sizes are warranted to clarify the relationship between AKI and mortality after pneumonectomy.

The strengths of this study include its relatively large, single-center cohort spanning ten years, comprehensive perioperative data collection, and detailed evaluation of short-term outcomes. These features provide valuable and reliable information for identifying high-risk patients and improving perioperative management in clinical practice.

Study Limitations

This study has several limitations. First, it was designed as a single-center retrospective cohort study, which may limit the generalizability of the findings to other populations. The retrospective nature of the study may also introduce bias due to incomplete or missing data, as well as potential inaccuracies in the recorded information. Second, AKI was defined exclusively by changes in sCr levels because postoperative urine output data were not routinely collected. As a result, episodes of AKI identified solely by oliguria may have been missed, potentially leading to an underestimation of the true AKI incidence. Third, several relevant perioperative factors and comorbidities could not be fully assessed due to data limitations, which may have resulted in residual confounding. The preoperative pulmonary function test results (e.g., FEV) and tumor stage were not consistently available in the retrospective dataset; therefore, these variables could not be incorporated into the statistical analyses, although they are clinically relevant factors known to be associated with postoperative outcomes. Fourth, the relatively limited sample size may have reduced the statistical power of our analyses and the ability to detect weaker associations. Fifth, the findings regarding the potential protective effects of NSAID and diuretic use should be interpreted with caution, as these results may be subject to confounding by indication and selection bias—NSAIDs and diuretics may have been avoided in higher risk patients or those with impaired renal function, resulting in an apparent protective association in the remaining cohort. Furthermore, the small number of AKI events may have limited the ability to robustly evaluate the association between these drug exposures and AKI risk. Finally, long-term outcomes beyond hospital discharge, including persistent renal dysfunction and overall survival, were not systematically assessed, thereby preventing evaluation of the extended impact of postoperative AKI.

CONCLUSION

Reducing the incidence of AKI could significantly improve patient outcomes, and we propose that AKI may serve as a valuable quality metric in thoracic surgery. To our knowledge, no clinical prediction rules have yet been established for forecasting AKI following thoracic surgery. Recognition of risk factors, as outlined in this systematic review, could lay the groundwork for the development of such predictive models. Nevertheless, AKI should be regarded as a crucial outcome measure in thoracic surgery, necessitating further studies focused on early prediction, risk minimization, and the long-term consequences of this complication.

Ethics

Ethics Committee Approval: The Ethics Committee of Ankara University Faculty of Medicine approval number is I8-568-21, date: 15/10/2021.

Informed Consent: The need for obtaining informed consent was waived because of the retrospective nature of the study.

Footnotes

Authorship Contributions

Concept: Ç.Y.G., S.K.E., Design: Ç.Y.G., S.K.E., Data Collection or Processing: B.Ş., Y.K., S.G.G., Analysis or Interpretation: B.Ş., Y.K., S.G.G., Literature Search: Ç.Y.G., S.K.E., B.Ş., B.C.M., Writing: Ç.Y.G., B.Ş., B.C.M.

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