Predictors and clinical outcomes of acute kidney injury after thoracic surgery: a retrospective cohort study

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Background: Acute kidney injury (AKI), defined as a rapid decline of renal function, is a frequent complication observed after major surgical procedures. In patients undergoing thoracic surgery, the reported incidence of AKI ranges from 5% to 15%. The development of AKI is associated with an elevated risk of postoperative complications, including the need for reintubation, longer hospital admissions, and higher mortality rates. During one-lung ventilation (OLV), intraoperative hypoxemia and hypercarbia, along with systemic inflammation induced by alveolar over-distension, can contribute to decreased renal perfusion, which may ultimately result in AKI. This study aims to determine the incidence, identify predictive factors, and evaluate the clinical outcomes associated with AKI following thoracic surgery.

Methods: In retrospective cohort study, all consecutive patients aged 18 years and over undergoing non-cardiac thoracic surgery at a tertiary university hospital between 2012 and 2021 were enrolled. AKI was diagnosed by Kidney Disease Improving Global Outcomes (KDIGO)-2012. The univariable and multivariable logistic regression were analyzed and presented as odds ratio (OR) and 95% confidence interval (CI). A Kaplan-Meier curve and log-rank test were used for comparison of hospital mortality between patients with or without AKI.

Results: The incidence of AKI was 23.9% (318 of 1,329 patients), with cases classified as stage I (184, 13.8%), stage II (115, 8.7%), or stage III (19, 1.4%) according to the KDIGO criteria. In the multivariable logistic regression analysis, age ≥70 years (OR, 1.74; 95% CI: 1.21–2.50, P=0.003), body mass index (BMI) >25 kg/m^2 (OR, 2.39; 95% CI: 1.73–3.36; P<0.001), baseline estimated glomerular filtration rate (eGFR)

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<60 mL/min/1.73 m^2 (OR, 1.89; 95% CI: 1.28–2.79; P=0.001), open thoracotomy (OR, 1.54; 95% CI: 1.15–2.07; P=0.003), intraoperative hydroxyethyl starch administration >1,000 mL (OR, 3.18; 95% CI: 1.19–8.54; P=0.02) and intraoperative urine output <0.5 mL/kg/h (OR, 1.80; 95% CI: 1.30–2.52; P=0.001) were significantly associated with the development of AKI. Patients who developed AKI had a significantly higher incidence of postoperative delirium (15.5%), requirement for continuous renal replacement therapy (CRRT) (3.5%), prolonged intensive care unit (ICU) stay [0 (0–2) *vs.* 0 (0–1) days, P=0.001], longer hospital stays {14.5 [9–24] *vs.* 9 [6–18] days, P=0.005}, and an increased mortality rate (10.1% *vs.* 5%, P=0.001) compared to those without AKI. Patients with stage III AKI had a significantly higher 30-day mortality rate compared to other stages (P<0.001).

Conclusions: The findings of this study highlight important predictors for AKI in patients undergoing thoracic surgery. Recognizing these predictors early may support timely interventions and the development of strategies aimed at reducing the likelihood of AKI, thereby promoting better postoperative recovery.

Keywords: Acute kidney injury (AKI); thoracic surgery; predictors; outcomes

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Introduction

Acute kidney injury (AKI) is a frequent complication following surgery. The incidence of postoperative AKI varies, from 1% to 50%, depending on the type of surgery, patient population, and the definition of AKI (1-10). After thoracic surgery, the reported incidence is between 5% and 15% (11-16). Postoperative AKI can lead to short and long-term adverse outcomes, including re-intubation, respiratory complications, prolonged length of intensive care unit (ICU) and hospital stay, and increased hospital mortality (11,13-15).

The pathophysiology of AKI following thoracic surgery has distinctly different characteristics compared to other types of surgery. The development of AKI following thoracic surgery involves several mechanisms. High tidal volume ventilation leads to renal microvascular dysfunction by inducing renal cell apoptosis and disrupting endothelial integrity (17). Ventilator-induced lung injury (VILI) further exacerbates systemic inflammation mediators (biotrauma), thereby contributing to AKI (17). Additionally, one-lung ventilation (OLV) can cause hypoxia, hypercarbia, atelectasis and acute lung injury (ALI), resulting in renal hypoperfusion. Pulmonary resection can lead to postoperative right ventricular dysfunction, a decrease in cardiac output and subsequent reduction in renal perfusion pressures (18). Perioperative fluid restriction, widely recommended to reduce the risk of ALI during OLV, increases the risk of hypovolemia and impaired renal perfusion (11). The use of colloids, such as hydroxyethyl starch (HES), which is commonly administered to minimize intraoperative fluid requirements, has also been associated with renal impairment (11). Previous studies have reported that the combination of HES administration and fluid restriction increases the risk of AKI following thoracic surgery (11,13).

Several risk factors for postoperative AKI have been reported, including advanced age (2,4,5,7-9,15), hypertension (2,3,5,7,8,13), diabetes mellitus (DM) (2,3,5,7-9,11), pre-existing chronic kidney disease (CKD) or decreased renal function (2-5,7-9,11,13,15), congestive heart failure (2,3,5,8,9), the use of angiotensin-converting enzyme inhibitors (ACEIs) (2,5,7,8,11,15), angiotensin receptor blockers (ARBs) (2,5,7,8,11,13,15), or non-steroidal anti-inflammatory drugs (NSAIDs) (5,7,8,15), American Society of Anesthesiologists (ASA) physical status classification III–IV (14), intraoperative HES infusion (11,13), and open thoracotomy (11,15). While thoracic surgical procedures are associated with distinct pathophysiological and technical features in comparison to other surgical categories, research specifically examining the predictors of AKI in this setting remains limited. The purpose of this study was to identify the incidence, predictors, and clinical outcomes of AKI following thoracic surgery. We present this article in accordance with the STROBE reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-2025-aw-2045/rc).

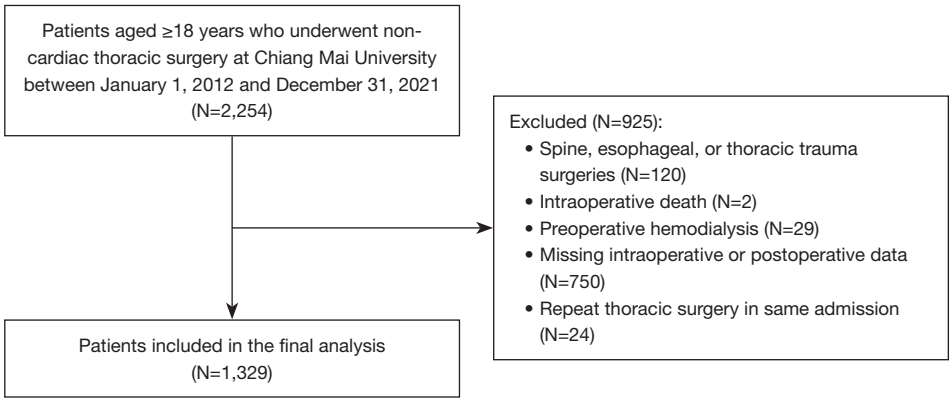

Figure 1 The study flow chart.

Highlight box

Key findings

- Age ≥70 years, body mass index (BMI) over 25 kg/m^2, reduced preoperative renal function [estimated glomerular filtration rate (eGFR) less than 60 mL/min/1.73 m^2], open thoracotomy, administration of more than 1,000 mL of intraoperative hydroxyethyl starch (HES) and intraoperative oliguria were significantly associated with the development of acute kidney injury (AKI) following thoracic surgery.
- AKI is associated with increased mechanical support, prolonged hospital stays and increased mortality.

What is known and what is new?

- Multiple preoperative factors (advanced age, hypertension, diabetes, pre-existing chronic kidney disease, heart failure, and use of angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or non-steroidal anti-inflammatory drugs) and intraoperative factors [American Society of Anesthesiologists physical status III–IV, HES infusion, and open thoracotomy] have been identified as predictors of postoperative AKI; however, evidence specific to thoracic surgery remains limited.
- In this study, AKI after thoracic surgery was independently predicted by age ≥70 years, BMI over 25 kg/m^2, baseline eGFR less than 60 mL/min/1.73 m^2, open thoracotomy, intraoperative HES administration more than 1,000 mL, and intraoperative urine output less than 0.5 mL/kg/h.
- This study demonstrates the incidence, staging of AKI and its clinical outcomes after thoracic surgery

What is the implication, and what should change now?

- Careful patient assessment, avoiding nephrotoxic agents and intraoperative HES especially in those with low preoperative renal function and monitoring intraoperative oliguria are recommended to reduce AKI risk and severity.

Methods

Ethics, study design, and population

This study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments. This retrospective cohort study was approved by the institutional ethics committee of the Faculty of Medicine, Chiang Mai University (Study Code: ANE: 2568-0155), and informed consent was waived because of the retrospective nature of the study. Data were retrospectively collected on all consecutive patients who underwent non-cardiac thoracic surgery at the Department of Anesthesiology, Faculty of Medicine, Chiang Mai University. between January 2012 and December 2021. Inclusion criteria were patients aged 18 years or older undergoing both elective and emergency open thoracotomy or video-assisted thoracoscopic surgery (VATS). Patients who required surgery for thoracic spine or thoracic injury, died during intraoperative period, received preoperative hemodialysis and those patients without postoperative serum creatinine and urine output within 7 days after operation were excluded.

Variables were collected from the electronic medical database. Data from 2,254 patients were included in the study. The study flow chart is presented in *Figure 1*. Electronic medical records were retrospectively reviewed by four trained research assistants. Patient characteristics collected included age, sex, ASA physical status, type of surgery (emergency or elective surgery), body mass index (BMI), and comorbidities such as hypertension, DM, dyslipidemia, CKD, chronic obstructive pulmonary disease,

coronary artery disease, and cerebrovascular disease. Current medications, including NSAIDs, ACEIs, ARBs, statins, and diuretics were also recorded. Preoperative laboratory data collected included hemoglobin (Hb), neutrophil-lymphocyte count ratio (NLR), albumin, serum creatinine, and estimated glomerular filtration rate (eGFR).

Intraoperative surgical and anesthetic data included diagnosis, types of surgical procedure (e.g., wedge resection, segmentectomy, lobectomy, pneumonectomy, decortication, or other), surgical approach (open thoracotomy or VATS), side of operation, and the duration of surgery, anesthesia, and OLV. Information regarding the type of analgesia used (thoracic epidural anesthesia, paravertebral block, or intravenous opioids) was also collected. Fluid management details included the type and volume of intraoperative fluids (crystalloids or colloids) and blood products administered, as well as intraoperative blood loss, urine output, and the use of inotropic agents such as ephedrine and norepinephrine. Intraoperative complications were recorded, including hypotension, defined as systolic blood pressure <90 mmHg for a cumulative duration of 10 minutes or more during surgery (19), hypoxemia, defined as an arterial oxygen saturation by pulse oximetry (SpO_2) <90% (13), and hypercarbia defined as end-tidal carbon dioxide ($ETCO_2$) >40 mmHg.

For all thoracic patients, the standard techniques of general anesthesia patients involved propofol for intravenous anesthesia and either cisatracurium or rocuronium as the neuromuscular blocking agent. Anesthesia was maintained using a volatile anesthetic agent with sevoflurane or desflurane, and intermittent boluses of intravenous fentanyl. Lung isolation was achieved using a double-lumen endotracheal tube (DLT), a bronchial blocker, or an endotracheal tube was managed with volume-controlled ventilation (4–6 mL/kg tidal volume, peak pressure <30 cmH_2O), fraction of inspired oxygen (FiO_2) adjusted from 1.0 to maintain SpO_2 >94%, and respiratory rate adjusted to keep $ETCO_2$ <45 mmHg. Invasive monitoring, such as direct blood pressure monitoring or a central venous catheter, was performed when indicated. Crystalloids were used for routine fluid maintenance and initial resuscitation. Colloids, such as HES or gelatin, were reserved for specific indications like the management of hypovolemic shock. Thoracoscopic cases that were converted to mini-thoracotomy or open thoracotomy were classified as open cases. Intraoperative transfusions of red blood cells, fresh frozen plasma, or platelets were recorded. Postoperative analgesia consisted of intravenous opioid infusion, thoracic epidural or thoracic paravertebral nerve block depending on the attending anesthesiologists.

Assessment of primary and secondary outcomes

The primary outcome was to identify predictors of AKI following thoracic surgery. The secondary outcomes were the incidence of postoperative AKI within 7 days and evaluated postoperative clinical outcomes, including the length of ICU stay, length of hospital stay, need for continuous renal replacement therapy (CRRT), delirium, and in-hospital mortality. AKI was diagnosed based on the Kidney Disease Improving Global Outcomes (KDIGO) criteria, defined as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours, an increase in serum creatinine to ≥1.5 times the baseline within 7 days postoperatively, or a urine output of less than 0.5 mL/kg/h for 6 consecutive hours during the postoperative period (20). The severity of AKI was staged in accordance with the KDIGO criteria. Postoperative delirium was assessed daily, with diagnoses made using the Confusion Assessment Method (CAM) for non-ICU patients and the Confusion Assessment Method for the patients in the ICU (CAM-ICU) (21).

Missing data

Prior to analysis, we assessed the patterns of missing data, which appeared to be a missing completely at random (MCAR) mechanism. Given this finding, we employed listwise deletion, excluding any case with one or more missing values across the variables included in the analysis. This resulted in a final sample size of 1,329 patients for subsequent analysis.

Statistical analysis

Statistical analyses were performed using STATA, version 18.0 (StataCorp LP, College Station, TX, USA). Sample size was calculated based on predictive factors affecting the incidence of AKI from previous studies (11,13,15). A sample size of at least 113 patients in AKI and 565 in non-AKI was required (α=0.05 and power =80%) for binary outcome. Categorical data were presented as frequencies and percentages, whereas continuous data were presented as mean ± standard deviation or median with interquartile range (25^{th} to 75^{th} percentile), depending on data distribution. The difference in categorical and continuous data between patients with or without AKI was

analyzed using the exact probability test and independent *t*-test or Wilcoxon rank-sum test as appropriate. The Kruskal-Wallis test was used to compare the differences in continuous variables across AKI stages. To assess potential multicollinearity among the risk factors, we first examined the pairwise correlation matrix and then we calculated the variance inflation factor (VIF) for each risk factor using ordinary least squares regression.

Potential predictors were analyzed using logistic regression and presented as odds ratios (ORs) with 95% confidence interval (CI). Any risk factor with a P value <0.20 in univariable logistic regression, along with other potential confounders associated with AKI, was included in a multivariable logistic regression analysis using a cluster-robust variance-corrected model with backward elimination. A Kaplan-Meier curve and log-rank test were used for comparison of hospital mortality between patients with any stage of AKI and those without AKI. Cox proportional hazard model was used to obtain hazard ratio (HR) and 95% CI. Multivariable Cox regression model was used to determine the effect of AKI stage after adjusting for all potential confounders. A P value of less than 0.05 was considered statistically significant.

Results

A total of 2,254 patients underwent non-cardiac thoracic surgery at a single-center university hospital over a 10-year period. Following exclusion, 1,329 patients were included in the final analysis. The incidence of any severity of AKI was 23.9% (318 of 1,329 patients). Classification of the AKI cases was stage I (184, 13.8%), stage II (115, 8.7%), or stage III (19, 1.4%).

Patients in the AKI group were significantly older (60.0±14.2 *vs.* 57.3±14.3 years, P=0.003), had a higher BMI (22.9±4.4 *vs.* 21.4±3.8 kg/m^2, P<0.001), and higher incidence of preoperative comorbidities such as hypertension (46.8% *vs.* 33.6%, P<0.001) and CKD (52.5% *vs.* 41.9%, P<0.001), compared to patients without AKI. They were also more likely to have been taking ARBs (10.7% *vs.* 5.7%, P=0.002) and diuretics (9.4% *vs.* 5.7%, P=0.02) preoperatively. Moreover, patients with AKI had higher preoperative serum creatinine levels [0.9 (0.7–1.1) *vs.* 0.8 (0.6–1.0) mg/dL, P=0.002] (*Table 1*). Intraoperatively, there was a higher proportion of open thoracotomies in the AKI group (59.7% *vs.* 50.1%, P=0.003), a longer duration of one lung ventilation was required {135 [70–190] *vs.* 110 [40–170] minutes, P=0.01}, and received a greater cumulative dose of intraoperative norepinephrine [0 (0–0) *vs.* 0 (0–0) μg/kg, P=0.006] (*Table 2*). There was no significant difference in intraoperative crystalloid administration between patients with and without AKI {700 [400–1,050] *vs.* 735 [450–1,070] mL, respectively; P=0.12}. Patients who received more than 1,000 mL of HES had a significantly higher median blood loss {1,500 [700–2,100] mL} compared to those who received 1,000 mL or less {100 [50–300] mL, P<0.001}.

The results of the univariable logistic regression analysis for AKI are presented in *Table 3*. Six factors were independently associated with AKI, as identified by multivariable logistic regression analysis: age ≥70 years (OR, 1.74; 95% CI: 1.21–2.50; P=0.003), BMI >25 kg/m^2 (OR, 2.39; 95% CI: 1.73–3.36; P<0.001), preoperative eGFR <60 mL/min/1.73 m^2 (OR, 1.89; 95% CI: 1.28–2.79; P=0.001), open thoracotomy (OR, 1.54; 95% CI: 1.15–2.07; P=0.003), intraoperative HES administration >1,000 mL (OR, 3.18; 95% CI: 1.19–8.54; P=0.02), and intraoperative urine output <0.5 mL/kg/h (OR, 1.80; 95% CI: 1.30–2.52; P=0.001) (*Table 3*).

Patients with AKI experienced higher rates of postoperative adverse events, including an increased need for postoperative mechanical ventilation (31% *vs.* 23.3%, P=0.004), a higher incidence of postoperative delirium (15.5% *vs.* 8%, P<0.001), longer duration of ICU stay [0 (0–2) *vs.* 0 (0–1) days, P=0.001], greater requirement of CRRT (3.5% *vs.* 0.3%, P<0.001), prolonged hospital stay {14.5 [9–24] *vs.* 9 [6–18] days, P=0.005}, and increased in-hospital mortality (10.1% *vs.* 5%, P=0.001) (*Table 4*). Postoperative clinical outcomes stratified by AKI stage are presented in Table S1. Secondary outcomes were further delineated according to the surgical approach, comparing patients who underwent open thoracotomy with those who had VATS. Compared to VATS patients, those who underwent open thoracotomies had a significantly higher incidence of postoperative delirium, AKI, and death, and experienced prolonged ICU and hospital stays (Table S2).

Figure 2 illustrates the probability of 30-day mortality across AKI stages, showing that patients with stage III AKI had a significantly higher 30-day mortality rate compared to other stages (P<0.001). After adjusting for potential confounders—including age, sex, BMI, baseline eGFR, anemia, hypoalbuminemia, type of surgical procedure, thoracotomy, intraoperative HES and vasopressor use, and intraoperative oliguria—patients with AKI stage III had significantly higher mortality rates compared to those without AKI (HR, 2.62; 95% CI: 1.01–6.83; P=0.048).

Table 1 Patient characteristics

Variables	Missing	AKI (n=318)	Non-AKI (n=1,011)	P value
Age (years)	0	60.0±14.2	57.3±14.3	0.003†
Male	0	197 (61.9)	608 (60.1)	0.56‡
Body mass index (kg/m^2)	0	22.9±4.4	21.4±3.8	<0.001†
ASA physical status	0			0.10‡
ASA I		40 (12.6)	156 (15.4)	
ASA II		188 (59.2)	606 (59.9)	
ASA III		83 (26.2)	241 (23.8)	
ASA IV		7 (2.0)	8 (0.8)	
Comorbidity				
Hypertension	3 (0.2)	149 (46.8)	339 (33.6)	<0.001‡
Diabetes mellitus	4 (0.3)	53 (16.7)	143 (14.2)	0.27‡
COPD	4 (0.3)	30 (9.4)	76 (7.5)	0.27‡
Coronary artery disease	5 (0.4)	14 (11.2)	150 (14.9)	0.27‡
Cerebrovascular disease	3 (0.2)	9 (3.8)	35 (3.5)	0.58‡
Chronic kidney disease	3 (0.2)	167 (52.5)	415 (41.9)	<0.001‡
Preoperative medications				
NSAIDs	5 (0.4)	32 (10.1)	88 (8.7)	0.46‡
ACE inhibitors	3 (0.2)	27 (8.5)	65 (6.4)	0.21‡
ARBs	3 (0.2)	34 (10.7)	58 (5.7)	0.002‡
Diuretics	4 (0.3)	30 (9.4)	58 (5.7)	0.02‡
Preoperative laboratory				
Hemoglobin (g/dL)	0	11.8±2.0	11.9±2.0	0.44†
NLR	22 (1.6)	3.0 (2.0–3.9)	5.3 (2.6–7.9)	0.08§
Albumin (g/dL)	93 (7.0)	3.8 (2.9–4.2)	3.8 (3.1–4.3)	0.10§
Serum creatinine (g/dL)	4 (0.3)	0.9 (0.7–1.1)	0.8 (0.6–1.0)	0.002§
eGFR (mL/min/1.73 m^2)	4 (0.3)	86.9 (67.4–105.5)	93.0 (76.0–107)	0.004§
Diagnosis	5 (0.3)			0.67‡
Benign lung lesion		61 (19.2)	217 (21.5)	
Malignant lung lesion		163 (51.2)	503 (49.9)	
Infection of lung		94 (29.6)	288 (28.6)	

Values are mean ± standard deviation, median and interquartile range (25th–75th percentile) or n (%). †, Student's *t*-test; ‡, Chi-squared test; §, Mann-Whitney *U* test. ACE, angiotensin-converting enzyme; AKI, acute kidney injury; ARBs, angiotensin receptor blockers; ASA, American Society of Anesthesiologists; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate; NLR, neutrophil-lymphocyte count ratio; NSAIDs, non-steroidal anti-inflammatory drugs.

Table 2 Surgical and anesthetic details

Variables	Missing	AKI (n=318)	Non-AKI (n=1,011)	P value
Operative approach	3 (0.2)			0.003†
Open thoracotomy		190 (59.7)	507 (50.1)	
VATS		128 (30.3)	504 (49.9)	
Types of operation	3 (0.2)			0.27†
Wedge resection		47 (14.8)	169 (16.7)	
Segmentectomy		7 (2.2)	43 (4.3)	
Lobectomy		124 (39.0)	344 (34.1)	
Pneumonectomy		6 (1.9)	15 (1.5)	
Decortication		89 (28.0)	269 (26.6)	
Other procedures		45 (14.1)	110 (16.8)	
Side of operation	3 (0.2)			0.95†
Left		132 (41.5)	421 (41.6)	
Right		171 (53.8)	545 (54.0)	
Both		16 (4.7)	45 (4.4)	
Emergency surgery	0	117 (36.8)	325 (32.1)	0.12†
Duration of surgery (min)	4 (0.3)	180 [125–230]	180 [135–240]	0.68‡
Duration of anesthesia (min)	4 (0.3)	180 [125–230]	180 [135–240]	0.34‡
Requirement of OLV	4 (0.3)	274 (86.2)	904 (89.6)	0.09†
Duration of OLV (min)		135 [70–190]	110 [40–170]	0.01‡
Receiving TEA/TPB	9 (0.7)	102 (32.2)	338 (33.6)	0.63†
Total fluid intake (mL)	3 (0.2)	800 [450–1,250]	800 [500–1,070]	0.26‡
Total crystalloid (mL)	3 (0.2)	700 [400–1,050]	735 [450–1,070]	0.12‡
Total colloid (mL)	3 (0.2)	0 [0–0]	0 [0–0]	0.89‡
Total HES (mL)		0 [0–0]	0 [0–0]	0.95‡
≤1,000		309 (97.2)	1,001 (99.1)	0.02†
>1,000		9 (2.8)	10 (0.9)	
Total blood loss (mL)		100 [50–300]	100 [50–300]	0.12‡
Total PRC transfusion (mL)	0	0 [0–0]	0 [0–0]	0.19‡
Hypercarbia	3 (0.2)	68 (24.7)	250 (21.4)	0.22†
Hypoxemia	3 (0.2)	17 (5.3)	39 (3.8)	0.25†
Hypotension	5 (0.4)	67 (21.1)	244 (24.2)	0.25†
Lowest MAP (mmHg)	6 (0.4)	61.3 [55–68.3]	60 [56–68.3]	0.79‡
Duration of hypotension (min)	4 (0.3)	5 [0–5]	5 [0–5]	0.42‡
Intraoperative vasopressor				
Epinephrine (mg)	5 (0.4)	6 [0–18]	6 [0–15]	0.58‡
Norepinephrine (μg)	5 (0.4)	0 [0–0]	0 [0–0]	0.006‡
Intraoperative urine output (mL/kg/h)	31 (2.3)	1.0 [0.5–1.8]	1.3 [0.7–2.2]	<0.001‡

Categorical data are presented as n (%). Non-normal data are presented as median and interquartile range [25th–75th percentile]. †, Chi-squared test; ‡, Mann-Whitney *U* test. AKI, acute kidney injury; HES, hydroxyethyl starch; MAP, mean arterial pressure; OLV, one lung ventilation; PRC, pack red cell; TEA, thoracic epidural anesthesia; TPB, thoracic paravertebral block; VATS, video-assisted thoracoscopic surgery.

Table 3 Univariable and multivariable logistic regression for predictors of acute kidney injury after thoracic surgery (N=1,329)

Predictors	Univariable analysis		Multivariable analysis	
	OR (95% CI)	P value	OR (95% CI)	P value
Age ≥70 years	1.88 (1.38–2.56)	<0.001	1.74 (1.21–2.50)	0.003
Male	1.08 (0.83–1.40)	0.56		
Body mass index >25 kg/m^2	2.33 (1.73–3.15)	<0.001	2.39 (1.73–3.36)	<0.001
ASA physical status				
2	1.21 (0.82–1.77)	0.33	0.87 (0.56–1.34)	0.52
≥3	1.41 (0.92–2.15)	0.11	0.78 (0.47–1.29)	0.33
Comorbidity				
Hypertension	1.74 (1.35–2.25)	<0.001	1.36 (0.98–1.87)	0.06
Diabetes mellitus	1.21 (0.86–1.71)	0.27		
COPD	1.28 (0.82–1.99)	0.27		
CKD	1.58 (1.23–2.04)	<0.001		
Medications				
NSAIDs	1.17 (0.77–1.80)	0.46		
ACE inhibitors	1.35 (0.84–2.15)	0.21		
Angiotensin receptor blocker	1.96 (1.26–3.06)	0.003	1.38 (0.84–2.29)	0.20
Diuretics	1.71 (1.08–2.70)	0.02	1.19 (0.71–2.00)	0.49
Preoperative hemoglobin <10 g/dL	1.36 (1.00–1.86)	0.052	1.22 (0.87–1.73)	0.25
Serum albumin <3.5 g/dL	1.18 (0.90–1.55)	0.22		
NLR >10	1.23 (0.83–1.84)	0.30		
Preoperative eGFR <60 mL/min/1.73 m^2	2.28 (1.61–3.22)	<0.001	1.89 (1.28–2.79)	0.001
Emergency surgery	1.23 (0.94–1.60)	0.12	1.32 (0.90–1.93)	0.15
Open thoracotomy	1.47 (1.14–1.90)	0.003	1.54 (1.15–2.07)	0.003
Types of operation				
Other	Reference			
Wedge resection/segmentectomy	0.96 (0.62–1.50)	0.86		
Lobectomy/pneumonectomy	1.37 (0.93–2.01)	0.11		
Decortication	1.25 (0.83–1.88)	0.28		
Hydroxyethyl starch >1,000 mL	2.91 (1.17–7.23)	0.02	3.18 (1.19–8.54)	0.02
Intraoperative blood loss >2,000 mL	3.21 (0.92–11.15)	0.07	1.32 (0.37–4.78)	0.67
Intraoperative norepinephrine >1 μg/kg	1.05 (1.00–1.10)	0.03	1.03 (0.98–1.07)	0.21
Intraoperative urine <0.5 mL/kg/h	1.84 (1.36–2.51)	<0.001	1.80 (1.30–2.52)	0.001

ACEIs, angiotensin converting enzyme inhibitors; ASA, American Society of Anesthesiologists; CI, confidence interval; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate; NLR, neutrophil lymphocyte count ratio; NSAIDs, nonsteroidal anti-inflammatory drugs; OR, odds ratio.

Table 4 Postoperative clinical outcomes between patients with and without AKI

Clinical outcomes	AKI (n=318)	Non-AKI (n=1,011)	P value
Mechanical ventilation requirement	99 (31.0)	235 (23.3)	0.004†
Duration of mechanical ventilation (days)	0 [0–1]	0 [0–0]	<0.001‡
Delirium	49 (15.5)	80 (8.0)	<0.001†
Postoperative CRRT	11 (3.5)	3 (0.3)	<0.001†
Length of ICU stay (days)	0 [0–2]	0 [0–1]	0.001‡
Length of hospital stay (days)	14.5 [9–24]	9 [6–18]	0.005‡
Mortality	32 (10.1)	50 (5.0)	0.001†

Categorical data are presented as number (%). Non-normal data are presented as median and interquartile range [25th–75th percentile]. †, Chi-squared test; ‡, Mann-Whitney *U* test. AKI, acute kidney injury; CRRT, continuous renal replacement therapy; ICU, intensive care unit.

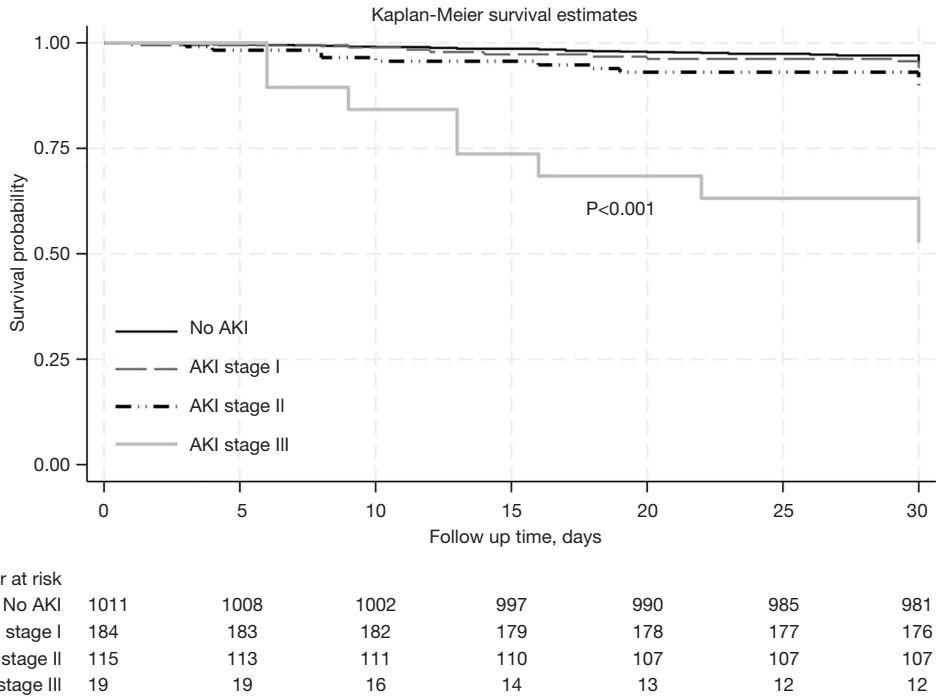

Figure 2 Kaplan-Meier survival curve compares the 30-day survival probability among different patient groups: AKI stage I, AKI stage II, AKI stage III, and patients without AKI. Patients with AKI stage III had the lowest 30-day survival probability, which was significantly less than patients with other AKI stages and those without AKI (P<0.001, by log-rank test). AKI, acute kidney injury.

Discussion

The present study found that the incidence of AKI following thoracic surgery was 23.9%, which is higher than previously reported rates which ranged from 5–15% (11-16). Most previous studies focused exclusively on lung resection surgery and employed varying diagnostic criteria for AKI, most commonly RIFLE (risk, injury failure, loss, end-stage renal disease), followed by KDIGO and acute kidney injury network (AKIN) criteria, respectively. We employed the KDIGO criteria to diagnose AKI, as they incorporate the most practical aspects of both the RIFLE and AKIN criteria. As shown in the previously cited studies, key differences among these diagnostic criteria

have directly influenced the reported incidence of AKI. First, all criteria for AKI diagnosis were based on changes in serum creatinine and/or urine output within 48 hours or 7 days. Unlike AKIN and KDIGO, RIFLE criteria include three severity stages (risk, injury, failure) and two outcome stages [loss and end-stage renal disease (ESRD)]. However, they do not account for minor changes in serum creatinine and do not include the initiation of renal replacement therapy (RRT) as a direct staging criterion (22). Our study identified several significant predictors of postoperative AKI, specifically age ≥70 years, BMI >25 kg/m^2, decreased baseline renal function (eGFR <60 mL/min/1.73 m^2), open thoracotomy, intraoperative HES administration >1,000 mL, and intraoperative oliguria. Patients who developed AKI, particularly those with stage III, experienced significantly higher rates of postoperative mechanical ventilation, delirium, a requirement for CRRT, longer hospital stays, and increased in-hospital mortality.

This study demonstrated that specific patient characteristics are significantly associated with the development of postoperative AKI. In particular, our findings revealed that advanced age (≥70 years) was an independent risk factor for postoperative AKI, a finding consistent with those from many previous studies (1,3-6,8,9,15,23). Renal aging is characterized by a progressive decline in nephron number and GFR, resulting in reduced renal reserve and impaired ability to adapt to surgical stress (24). In addition, elderly patients often have lower baseline serum creatinine levels due to decreased muscle mass, which can mask underlying renal impairment (11). Comorbidities commonly seen in older adults, including hypertension, diabetes, and heart disease, heighten the likelihood of postoperative AKI (25). Furthermore, factors such as the use of polypharmacy or nephrotoxic drugs, decreased renal autoregulation, greater vulnerability to hemodynamic instability, and an exaggerated systemic inflammatory response to surgery also contribute to increased risk (26).

Several mechanisms may contribute to a plausible account for an association between higher BMI and AKI, including obesity-induced hyperfiltration, chronic inflammation, increased surgical complexity, prolonged operative time, elevated blood loss, and significant hemodynamic instability and reduced renal perfusion (27-30). Obese patients (BMI of >30 kg/m^2) frequently exhibit altered drug metabolism and are more likely to develop diabetes and hypertension, factors that may adversely affect kidney function (31,32). Our study identified reduced preoperative baseline renal function (eGFR <60 mL/min/1.73 m^2) as a significant predictor of AKI following thoracic surgery, a finding consistent with previous studies (5,6,11,23). Patients with CKD have a reduced renal functional reserve and impaired autoregulation, increasing their susceptibility to intraoperative hypoperfusion and ischemic injury (25,33,34). Chronic inflammation, endothelial dysfunction, and impaired drug clearance further exacerbate renal vulnerability (35,36).

The present study did not find any significant relationship between preoperative hypertension, cerebrovascular peripheral disease, and preoperative ACEIs/ARBs and risk of postoperative AKI which was contrary to the findings of previous studies (11,13,16). There is, however, a potential for collinearity between these former predictors and postoperative AKI. As an observational study, we can only report associations and cannot infer causality.

Our study showed that open thoracotomy was a strong predictor of AKI which was consistent with a previous study (11). Open thoracotomy is associated with more extensive surgery, higher intraoperative blood loss, more significant hemodynamic fluctuations and a greater degree of tissue trauma, leading to an increased acute-phase inflammatory response in comparison to thoracoscopic surgery. Increased activation of acute-phase cytokines and mediators, such as interleukin-6 (IL-6) and C-reactive protein, leads to their release into systemic circulation, potentially contributing to the development of AKI (18,37).

We demonstrated that the use of intraoperative HES was a strong predictor of AKI following thoracic surgery which corresponded to previous retrospective studies by Ishikawa *et al.* (N=1,129) (13) and Ahn *et al.* (N=1,442) (11). The proposed mechanisms involve ischemic injury caused by hyperviscosity or hyperoncotic kidney failure, leading to decreased GFR due to reduction in the filtration fraction (38). Furthermore, renal toxicity may be exacerbated by the administration of higher molecular weight HES (39). The findings of Ahn *et al.* (11) indicated that AKI incidence was not significantly influenced by either the amount or the type of HES administered alone. However, HES should be administered with caution in patients with impaired renal function (11). Based on recent studies, the use of HES for various surgical procedures including thoracic surgery, has gradually declined in our institution due to concerns regarding its association with postoperative AKI.

Intraoperative oliguria often represents reduced intravascular volume as well as prolonged hypoperfusion,

resulting in compromised renal filtration and subsequently elevating the risk of postoperative AKI (40). Our study demonstrated that intraoperative oliguria (urine output <0.5 mL/kg/h) predicted an increased risk of postoperative AKI which corresponded to the findings of previous studies (40-42). A retrospective observational study by Shiba *et al.* (42) (N=5,894) reported that intraoperative oliguria (<0.5 mL/kg/h for at least 120 minutes) was consistently associated with a higher risk of postoperative AKI. Two meta-analytical studies (40,41) demonstrated that intraoperative oliguria was a strong predictor of postoperative AKI. Thoracic surgery patients often receive limited amount of intravenous fluids to reduce the risk of postoperative pulmonary complications. However, excessive fluid restriction may lead to oliguria, which is associated with an elevated risk of postoperative AKI (40).

OLV, a specific strategy used during thoracic surgery, frequently induces a ventilation-perfusion mismatch, hypoxia and hypercarbia, all of which contribute to the development of AKI. Renal tissue is highly sensitive to even minimal changes in oxygen levels, and hypoxia has been shown to compromise renal perfusion, leading to dysfunction (43). Hypercarbia stimulates sympathetic activation, resulting in renal vasoconstriction and a reduction in glomerular filtration (43,44). Additionally, VILI caused by pulmonary overdistension and repetitive alveolar collapse, triggers systemic inflammatory responses, leading to the release of cytokines such as IL-6 and tumor necrosis factor-α (TNF-α), which exacerbate renal endothelial injury (18). Hemodynamic instability secondary to respiratory derangements further impairs renal perfusion, thereby increasing the risk of AKI. Intraoperative variables such as hypoxia (37), hypotension, and duration of OLV were associated with increased risk of postoperative AKI (5,16,23). However, our study did not detect any association between those variables and AKI. Intraoperative administration of vasopressors has been identified in a previous study (14) as an independent risk factor for the development of AKI, potentially due to recurrent arterial hypotension, inadequately corrected hypovolemia, and intrarenal vasoconstriction mediated by α-adrenergic effects. In the present study, intraoperative norepinephrine administration was not independently associated with the development of postoperative AKI after thoracic surgery. Although there were differences in the amount of intraoperative norepinephrine administered between patients with and without AKI in the univariable logistic regression analysis (OR, 1.05; 95% CI: 1.00–1.10, P=0.03), this association was not observed in the multivariable logistic regression model after adjusting for relevant confounders (OR, 1.03; 95% CI: 0.98–1.07, P=0.21). These findings suggest that intraoperative norepinephrine use >1 μg/kg observed in our cohort, may not contribute to increased AKI risk when other perioperative factors are considered. However, it is possible our study lacked the statistical power to detect a significant effect, especially if the true effect size of norepinephrine on postoperative AKI is small.

Clinical implication

This study provides useful information for the prevention of AKI after thoracic surgery. Our study provides important insights that either reinforce or extend previous recommendations for preventing postoperative AKI. Importantly, we observed that elderly and obese patients are at significantly increased risk, underscoring the need for tailored perioperative care in these populations. In particular, our findings strongly support withholding ACEIs and ARBs on the morning of surgery to reduce the incidence of intraoperative hypotension and subsequent renal complications. The study also adds further evidence against the use of HES for fluid management; HES should be avoided in critically ill adults, especially those with sepsis or preexisting renal dysfunction (11,45), and alternative intravenous fluids or blood transfusions, as clinically indicated, should be considered as indicated. Furthermore, our results highlight the value of continuous intraoperative urine output monitoring, which enables earlier detection of renal hypoperfusion and allows for prompt intervention to reduce the risk of postoperative AKI. While novel biomarkers such as neutrophil gelatinase—associated lipocalin and cystatin C hold promise for earlier detection of AKI, their clinical utility in thoracic surgery remains to be established (10). These focused recommendations, grounded in our study's findings, can help optimize perioperative management and improve renal outcomes in high-risk patients.

A major strength of this study was that the incidence and association of AKI stage with clinical outcomes in thoracic surgical patients were clearly demonstrated. However, there were a few limitations. First, this study was conducted at a single center. This limits the generalizability of the findings to other institutions because of differences in patient characteristics, clinical practices, and the availability of medical resources. Further multi-center studies are recommended to validate and extend these observations

across diverse settings. Second, the retrospective design may have affected the completeness of medical records, which could potentially lead to misclassification of AKI and exposure variables. To mitigate this, we used a standardized data extraction form with clear instructions and definitions for each variable. Research assistants also received training in standard techniques before the study began. Additionally, we performed multivariable logistic regression analysis to adjust for potential confounders and to determine the strength of associations between exposure variables and the occurrence of AKI. Third, in our study, patients who received higher amounts of HES generally experienced substantial intraoperative bleeding. This may have resulted in prolonged hypotension and increased requirements for norepinephrine. These factors could confound the association between high HES administration and postoperative AKI. To address these concerns, the multivariable logistic regression analysis was adjusted for intraoperative blood loss and norepinephrine dose. Multicollinearity among the variables was assessed, with all demonstrating VIF values between 1 and 3, well below the threshold of 10 and indicative of a low degree of multicollinearity. Nonetheless, the possibility of residual confounding remains, including factors such as the duration and severity of hypotension and other unmeasured variables. These limitations should be considered when interpreting our findings. In addition, the number of AKI patients who received HES in our study was limited. Further studies with larger patient populations are needed to more comprehensively address this issue. Finally, postoperative serum creatinine measurements were not routinely obtained for all patients, especially those undergoing minor thoracic procedures or those without significant comorbidities. These patients were unlikely to have risk factors for AKI, which may explain why our observed incidence is higher than that reported in other studies. As a result, the incidence in our cohort may be overestimated in comparison, since lower-risk cases were less likely to be included in the outcome assessment.

Conclusions

Postoperative AKI occurred in 23.9% of patients after non-cardiac thoracic surgery, as defined by KDIGO criteria. Independent risk factors included age ≥70 years, BMI >25 kg/m^2, reduced baseline renal function, open thoracotomy, intraoperative HES >1,000 mL, and oliguria. AKI was associated with increased rates of mechanical ventilation support, a greater need for CRRT, higher incidence of postoperative delirium, longer hospital stays, and increased mortality. Future predictive models are needed to enhance risk stratification, early detection, and optimal allocation of medical resources.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://jtd.amegroups.com/article/view/10.21037/jtd-2025-aw-2045/rc

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. This study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments. The study was approved by the institutional ethics committee of Faculty of Medicine, Chiang Mai University (Study Code: ANE: 2568-0155), and informed consent was waived because of the retrospective nature of the study.

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