# Transient acute kidney injury after major abdominal surgery increases chronic kidney disease risk and 1-year mortality

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**Abbreviations:** AKI, acute kidney injury; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; SCr, serum creatinine; CI, confidence interval.

**Abstract**

**Purpose:** We conducted a retrospective cohort study to determine incidences of transient and persistent acute kidney injury (AKI) after major abdominal surgery and their impacts on long-term outcome.

**Materials and Methods:** We enrolled 3,751 patients undergoing major abdominal surgery. Postoperative AKI was classified as transient or persistent based on the return of serum creatinine to the non-AKI range within 7 days post-surgery. Primary outcome was mortality within 1 year. We used multivariable Cox proportional hazard regression analysis to assess independent associations between AKI type and mortality.

**Results:** Most patients with AKI were classified as transient (84%). Compared to patients without AKI, both patients with transient and persistent AKI demonstrated elevated 1-year mortality rates [adjusted hazard ratio (95% confidence interval): 2.01 (1.34–2.93); P = 0.001, and 6.20 (3.00–11.43); P < 0.001, respectively] and greater risk of chronic kidney disease progression at 1 year [adjusted odds ratio (95% confidence interval): 3.87 (2.12–7.08) and 23.70 (9.64–58.22), respectively; both P < 0.001].

**Conclusions:** Although most AKI cases after major abdominal surgery recover completely within 7 days, even these patients with transient AKI are at higher risk for 1-year mortality and chronic kidney disease progression compared to patients without AKI.

**Key words:** acute kidney injury, abdominal surgery, postoperative complication, chronic kidney disease

## 1. Introduction

Acute kidney injury (AKI) is a leading cause of perioperative morbidity and mortality. It occurs in 6%–13% of patients undergoing non-cardiac surgery [1–3] and is associated with higher mortality [4,5]. Moreover, AKI is associated with an increased risk of chronic kidney disease (CKD) development or CKD progression in various clinical settings [6–11].

In our clinical experience, postoperative AKI is often transient. It has been suggested that transient AKI represents a temporary reduction in glomerular filtration rate without structural kidney injury, whereas persistent AKI reflects structural renal tubular damage [12]. However, recent studies in hospitalized patients have found that even transient AKI is associated with long-term morbidity and mortality [13–15].

To date, few studies have evaluated the incidence of transient AKI after non-cardiac surgery [16], and the prognostic value of transient AKI in non-cardiac surgery patients is not well validated. Moreover, the impact of transient AKI on long-term kidney function is not known. Revealing the incidence of transient AKI and its impact on outcome in surgical populations may help identify patients with poor postoperative course and provide appropriate treatment alternatives.

Therefore, we conducted a retrospective cohort study to determine the incidence and impact on long-term outcomes of transient and persistent AKI after major abdominal surgery. We hypothesized that the majority of AKI after major abdominal surgery would be transient. In addition, we investigated the associations between AKI type and subsequent long-term outcomes including progression of CKD and mortality.

## 2. Materials and Methods

### *2.1 Study design, setting, and patients*

We conducted a single-center retrospective cohort study at Kyoto University Hospital, an 1,121-bed teaching hospital in Japan. The study protocol was approved by the Ethics Committee of Kyoto University Hospital (approval number: R0672, July 26$^{th}$, 2016), and the requirement for informed consent was waived. This article adheres to the Strengthening the Reporting of Observational Studies in Epidemiology guidelines [17]. We recruited patients using the database created for our previous study investigating the relationship between intraoperative oliguria and AKI after major abdominal surgery [18]. Patients 18 years or older who underwent major abdominal surgery (liver, colorectal, gastric, pancreatic, or esophageal resection) under general anesthesia at Kyoto University Hospital from March 2008 to April 2015 were eligible for inclusion. Exclusion criteria included concurrent cardiac or urological procedures and patients with end-stage renal disease [i.e., estimated glomerular filtration rate (eGFR) < 15 mL/min/1.73 $m^2$ as determined using a formula validated in Japan [19] or a receipt of hemodialysis]. We also excluded patients who died within 7 days of surgery because mortality in these cases appeared unrelated to AKI, and assessing the incidences of transient and persistent AKI or their impacts on subsequent outcomes are not relevant in this population.

### *2.2 Data collection*

The research database for our previous study [18] included age, sex, preoperative comorbidities, American Society of Anesthesiologists Physical Status, preoperative serum creatinine (SCr) (the most recent SCr level measured before surgery), type of surgery, SCr during the 7 days after surgery, postoperative length of hospital stay, and in-hospital mortality. In addition, data on survival time during the year after surgery and SCr at 3 months

and 1 year post-surgery were collected from the electronic medical record system.

*2.3 Outcomes and exposure*

The primary outcome was mortality during 1-year follow-up. Secondary outcomes included in-hospital mortality, hospital length of stay, and CKD progression at 3 months and 1 year after surgery. CKD progression was defined as worsening of eGFR category (stage 1, ≥ 90 ml/min/1.73 $m^2$; stage 2, 60–89 ml/min/1.73 $m^2$; stage 3a, 45–59 ml/min/1.73 $m^2$; stage 3b, 30–44 ml/ min/1.73 $m^2$; stage 4, 15–29 ml/min/1.73 $m^2$; and stage 5, <15 ml/min/1.73 $m^2$) coupled with a ≥25% reduction in eGFR from baseline according to the Kidney Disease: Improving Global Outcomes 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease [20]. We calculated eGFR at 3 months and 1 year using the lowest SCr measured at postoperative days 8–90 and postoperative days 276–365, respectively. If SCr was not measured during these periods, the first value measured after the indicated period was used.

The exposure of interest was AKI type (transient or persistent). AKI was defined as any stage of AKI according to the Kidney Disease: Improving Global Outcomes guidelines [21]. The AKI stages were based on the SCr concentration measured during the 7 days after surgery. Preoperative SCr was considered as the baseline. Transient AKI was defined as SCr values returning to below the AKI range within the first 7 days after surgery and persistent AKI as incomplete reduction of SCr at 7 days after surgery. This assessment window of 7 days was chosen because SCr levels fall below the AKI range within 7 days in the majority of patients with AKI and it may allow for comparison with previous studies [16,22].

*2.4 Statistical analyses*

Continuous variables are presented as median [interquartile range] and compared using the Mann–Whitney U test. Categorical variables are presented as numbers (percentage) and

compared using the Pearson chi-square test or Fisher exact test as appropriate. Competing risk analysis was used to compare length of hospital stay between groups. Median length of hospital stay and 95% confidence intervals (CIs) were obtained using the cumulative incidence estimates of discharge while alive accounting for death as a competing risk, and Gray's test was used to assess differences between groups. Survival was plotted on Kaplan–Meier curves and compared between groups by the log-rank test. Moreover, Cox proportional hazard regression analysis was performed to investigate the independent relationship between AKI type and risk of death during 1-year follow-up. The following variables were included in the multivariable model based on clinical relevance: age, sex, preoperative comorbidities (hypertension, diabetes mellitus, CKD, and active congestive heart failure), type of surgery, emergency status, duration of surgery, intraoperative blood loss, and the need for intraoperative vasopressor infusion. CKD was defined as a preoperative eGFR level of <60 ml/min/1.73 $m^2$. Multivariable logistic regression analysis was performed to assess the independent relationship between AKI type and CKD progression among patients with 1-year survival. On the basis of clinical relevance and a literature search for factors associated with CKD and its progression [23], seven variables [age, sex, body mass index, preoperative comorbidities (hypertension, diabetes mellitus, and active congestive heart failure), and preoperative eGFR] were included in the multivariable model. Moreover, the adjusted hazard ratio for mortality and adjusted odds ratio for CKD progression at 1 year was assessed after stratifying based on the maximal AKI stage.

All eligible patients in the study database were included to maximize statistical power. Our study database included approximately 3,800 eligible surgeries, and we assumed 1-year mortality after abdominal surgery to be 5%–6% on the basis of a published report [24]. We estimated that approximately 20 variables could be included in the Cox proportional hazard regression analysis using our dataset. We planned to conduct a complete patient analysis

including only patients with complete data on all variables required for multivariable analyses. Such an analysis is feasible only if <5% of patients have missing data [25]. Our data set fulfilled this criterion.

All statistical tests were two tailed, and a P value of $< 0.05$ was considered statistically significant. All statistical analyses were performed using the statistical program R (http://cran.r-project.org).

## 3. Results

### *3.1 Baseline characteristics*

Figure 1 shows the flow diagram of this study. A total of 3,804 index major abdominal surgeries were identified in the study database. After excluding 39 patients with end-stage renal disease preoperatively and 6 patients who died within 7 days postoperatively, 3,759 patients were eligible for the study. Among these patients, 8 (0.2%) had missing data on variables required for multivariable analyses. Patients with missing values accounted for less than 5%, so we conducted complete patient analysis including 3,751 patients.

The median age of the study participants was 66 years [interquartile range: 56–73] and 2,323 patients (61.9%) were male. AKI occurred in 258 patients (6.9%) of which 216 (5.8%) were classified as transient and 42 (1.1%) as persistent. Table 1 shows the preoperative patient characteristics and operative variables stratified by AKI type. Patients who developed AKI had a higher AKI risk index, more blood loss, and were more likely to receive intraoperative red blood cell transfusion and intraoperative vasopressor infusion. There were no statistically significant differences in clinical variables between patients with transient and persistent AKI except that patients with persistent AKI were younger than those with transient AKI. Table 2 shows maximal AKI stage stratified by AKI type. The maximal AKI stage was lower in patients with transient AKI (of which 89.4% were in stage 1).

### *3.2 Clinical events according to AKI type*

Clinical events during the 1-year follow-up are listed in Table 3. Patients with AKI (both transient and persistent) showed higher in-hospital mortality, longer length of hospital stay, higher mortality at 3 months and 1 year post-surgery, and more frequent CKD progression at 3 months and 1 year post-surgery than patients without AKI. Kaplan−Meier curve analysis revealed significantly higher death rates in both transient and persistent AKI groups

compared to patients without AKI ($P < 0.001$ for both comparisons; Figure 2). Survival probabilities at 1 year were 94.6% (93.8%–95.4%) for patients without AKI, 83.7% (77.8%–88.1%) for those with transient AKI, and 74.5% (57.6%–85.5%) for those with persistent AKI. Multivariable Cox proportional hazard regression analysis revealed that both transient and persistent AKI independently associated with higher mortality rate during 1-year follow-up even after adjustment for confounders [adjusted hazard ratio (95% CI) for transient AKI: 2.01 (1.34–2.93), $P = 0.001$; adjusted hazard ratio (95% CI) for persistent AKI: 6.20 (3.00–11.43), $P < 0.001$] (Table 4). The association between transient/persistent AKI and mortality was found to be qualitatively preserved when stratified by the maximal AKI stage (Supplementary Table 1).

*3.3 Associations between AKI type and CKD progression at 1 year*

Among the 1-year survivors, 3.2% exhibited CKD progression at 1 year (Table 3). Most (77.8%) of the patients with CKD progression demonstrated reduced eGFR level by one eGFR category. Four patients developed CKD stage 5 at 1 year; all these patients had CKD stage 4 preoperatively (Supplementary Table 2). Multivariable logistic regression analysis revealed that both transient and persistent AKI were independently associated with CKD progression at 1 year after adjustment for known risk factors [adjusted odds ratio (95% CI) for transient AKI: 3.87 (2.12–7.08), $P < 0.001$; adjusted odds ratio (95% CI) for persistent AKI: 23.70 (9.64–58.22), $P < 0.001$] (Table 5). The association between transient/persistent AKI and CKD progression at 1 year was qualitatively preserved when stratified by the maximal AKI stage (Supplementary Table 1).

## 4. Discussion

Of the 258 patients exhibiting AKI after major abdominal surgery, 84% were classified as transient according to the return of SCr value to the non-AKI range within the first 7 days post-surgery. However, like persistent AKI patients, these transient AKI patients demonstrated higher in-hospital mortality, longer hospital stay, and higher mortality rate during 1-year follow-up than non-AKI patients. Moreover, both transient and persistent AKI were associated with higher risk of CKD progression at 1 year independent of known risk factors for CKD and its progression. These data suggest that even temporary AKI after major abdominal surgery can increase the risk of mortality and CKD progression. Therefore, patients with AKI require careful follow-up even if SCr values return to the non-AKI range shortly after surgery.

Of those patients exhibiting postoperative AKI following major abdominal surgery, the vast majority (84%) recovered completely within 7 days of surgery. This result is consistent with a previous study in which 90% of patients with AKI after gastric resection exhibited full renal recovery within 7 days [16]. In contrast, full renal recovery within 7 days has been reported in only 43%–65% of hospitalized patients who develop AKI [13–15]. This discrepancy may be explained by differences in the pathophysiology of AKI between hospitalized patients and postoperative patients. For instance, the most common causes of AKI in hospitalized patients include sepsis and heart failure [26, 27]. In contrast, the intraoperative period is unique in that both anesthesia and surgery combine to affect renal function [28].

To the best of our knowledge, this is the first study demonstrating that postoperative transient AKI is associated with CKD progression in patients undergoing non-cardiac surgery. A recent meta-analysis reported the association between the duration of AKI and outcomes including long-term mortality and incident CKD [29]. However, only two studies included in this systematic review reported on the risk for CKD in association with the duration of AKI; of

these, one study reported that even transient AKI is associated with CKD development in hospitalized patients [30], whereas the other study targeted patients undergoing cardiac surgery but was underpowered to assess the impact of transient AKI on CKD development [31]. Kim et al. [16] reported that postoperative transient AKI after gastric surgery was associated with higher long-term mortality; however, postoperative transient AKI was not associated with CKD development in their study, in contrast to the results of our study. There are several possible explanations for this apparent inconsistency. First, our study included patients with preoperative CKD, whereas the study by Kim et al. included only patients with normal preoperative renal function. The impact of transient AKI on CKD progression may be less severe in patients with normal preoperative renal function compared to those with preoperative renal dysfunction. Second, we used CKD progression to assess deterioration of renal function, whereas the study by Kim et al. used new CKD development. These difference in outcome definitions may contribute to the discrepancy in results.

Early detection of CKD progression is extremely important because CKD-associated complications and progression to kidney failure can be delayed or even prevented through early introduction of appropriate interventions, including blood pressure and glycemic control [20]. Our data that postoperative transient AKI is associated with CKD progression suggest that even patients with transient AKI are at higher risk of CKD progression, and therefore require careful follow-up and may benefit from interventions to prevent CKD progression even after complete recovery of renal function. One possible explanation for this association with CKD progression is that many patients with transient AKI have structural kidney injury despite complete recovery of SCr. Alternatively, AKI may enhance the vulnerability to subsequent CKD due to other factors, such as hypertension and hyperglycemia.

As expected, persistent AKI was more strongly associated with outcomes including CKD progression and long-term survival than transient AKI. These data suggest that early

identification of AKI and intensive treatment aimed at early recovery of renal function may benefit patients with postoperative AKI, although verification by future studies is required. Limitations of our study are as follows. This was a single-center study, which may reduce the generalizability of our results. Our study included patients undergoing major abdominal surgery, so it is unclear whether our findings can be extrapolated to patients undergoing other surgeries. Considering the study design, effects of unmeasured confounding variables on the observed associations cannot be ruled out, despite of our best efforts to adjust for confounding factors. For example, data on albuminuria/proteinuria, the use of nephrotoxins, the use of angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, and the etiology of AKI for the enrolled patients were not available. Serum creatinine was not measured every day during the 7 days after surgery in all patients, so the incidence of transient AKI may have been underestimated. Nevertheless, the incidence of transient AKI in our study was as high as 84%. Finally, although an assessment window of 7 days was selected to distinguish between transient and persistent AKI, previous studies have applied different thresholds for determining the duration of AKI. Recently, the Acute Disease Quality Initiative proposed a definition of persistent AKI as the continuance of AKI beyond 48 h [32]. The optimal duration of AKI to define transient and persistent AKI remains to be elucidated.

## 5. Conclusions

Although most patients with postoperative AKI after major abdominal surgery completely recover within the first 7 days, even patients with transient AKI are at higher risk for long-term mortality and CKD progression compared to those without AKI. Patients with AKI require careful follow-up even if SCr values return quickly to the non-AKI range after surgery.

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**Table 1.** Patient characteristics and operative variables

| | All patients (n = 3,751) | No AKI (n = 3,493) | Transient AKI (n = 216) | Persistent AKI (n = 42) | P-value |
|---|---|---|---|---|---|
| Age (years) | 66 [56–73] | 66 [56–73] | 67 [61–74]* | 62 [57–69]† | 0.010 |
| Male sex | 2,323 (61.9%) | 2,112 (60.5%) | 176 (81.5%)* | 35 (83.3%)* | <0.001 |
| Hypertension | 1,163 (31.0%) | 1,026 (29.4%) | 112 (51.9%)* | 25 (59.5%)* | <0.001 |
| Diabetes mellitus | 617 (16.4%) | 547 (15.7%) | 54 (25.0%)* | 16 (38.1%)* | <0.001 |
| Active congestive heart failure | 63 (1.7%) | 53 (1.5%) | 9 (4.2%)* | 1 (2.4%) | 0.012 |
| Ascites | 311 (8.3%) | 282 (8.1%) | 21 (9.7%) | 8 (19.1%)* | 0.028 |
| ASA-PS (1/2/3/4/missing) | 1026/2432/248/6/39 | 1010/2224/219/3/37 | 14/172/25/3/2* | 2/36/4/0/0* | <0.001 |
| Preoperative SCr (mg/dL) | 0.7 [0.6–0.9] | 0.7 [0.6–0.9] | 0.8 [0.7–1.0]* | 0.8 [0.6–0.9] | <0.001 |
| Preoperative eGFR (mL/min/1.73 $m^2$) | 75.1 [63.8–87.7] | 75.4 [64.1–87.9] | 71.6 [56.0–85.4]* | 75.4 [66.5–88.2] | <0.001 |
| AKI risk index | | | * | * | <0.001 |
| Class 1 | 1,319 (35.2%) | 1,286 (36.8%) | 29 (13.4%) | 4 (9.5%) | |
| Class 2 | 1,229 (32.8%) | 1,158 (33.2%) | 62 (28.7%) | 9 (21.4%) | |
| Class 3 | 804 (21.4%) | 713 (20.4%) | 75 (34.7%) | 16 (38.1%) | |

| | | | | | |
|---|---|---|---|---|---|
| Class 4 | 309 (8.2%) | 263 (7.5%) | 34 (15.7%) | 12 (28.6%) | |
| Class 5 | 90 (2.4%) | 73 (2.1%) | 16 (7.4%) | 1 (2.4%) | |
| Type of surgery | | | * | | <0.001 |
| Liver | 1,189 (31.7%) | 1,079 (30.9%) | 93 (43.1%) | 17 (40.5%) | |
| Colorectal | 1,105 (29.5%) | 1,053 (30.2%) | 42 (19.4%) | 10 (23.8%) | |
| Gastric | 665 (17.7%) | 627 (18.0%) | 27 (12.5%) | 11 (26.2%) | |
| Pancreatic | 554 (14.8%) | 510 (14.6%) | 41 (19.0%) | 3 (7.1%) | |
| Oesophageal | 207 (5.5%) | 197 (5.6%) | 9 (4.2%) | 1 (2.4%) | |
| Complex | 31 (0.8%) | 27 (0.8%) | 4 (1.9%) | 0 (0.0%) | |
| Laparoscopic surgery | 1,965 (52.4%) | 1,888 (54.1%) | 59 (27.3%)* | 18 (42.9%) | <0.001 |
| Emergency surgery | 44 (1.2%) | 39 (1.1%) | 5 (2.3%) | 0 (0.0%) | 0.221 |
| Epidural anaesthesia | 1,798 (47.9%) | 1,657 (47.4%) | 122 (56.5%)* | 19 (45.2%) | 0.034 |
| Duration of surgery (min) | 353 [259–473] | 347 [256–465] | 444 [340–595]* | 446 [315–544]* | <0.001 |
| Intraoperative blood loss (mL) | 150 [30–515] | 132 [28–460] | 783 [216–1575]* | 621 [86–1602]* | <0.001 |
| Net fluid balance during the surgery (mL) | 2543 [1831–3487] | 2505 [1815–3449] | 3131 [2259–4319]* | 3274 [2402–4053]* | <0.001 |
| Intraoperative red blood cell transfusion | 328 (8.7%) | 256 (7.3%) | 63 (29.2%)* | 9 (21.4%)* | <0.001 |

| | | | | | |
|---|---|---|---|---|---|
| Intraoperative vasopressor infusion | 343 (9.1%) | 289 (8.3%) | 44 (20.4%)* | 10 (23.8%)* | <0.001 |

Data are presented as median [interquartile range] or numbers (percentages). *, P < 0.017 (significantly different with Bonferroni correction), compared with patients without AKI; †, P < 0.017 (significantly different with Bonferroni correction), compared with patients with transient AKI; AKI, acute kidney injury; ASA-PS, the American Society of Anaesthesiologists physical status; SCr, serum creatinine; eGFR, estimated glomerular filtration rate.

**Table 2.** Maximal AKI stage stratified by AKI type

| Maximal AKI stage | Transient AKI (n = 216) | Persistent AKI (n = 42) |
|---|---|---|
| Stage 1 | 193 (89.4%) | 22 (52.4%) |
| Stage 2 | 18 (8.3%) | 12 (28.6%) |
| Stage 3 | 5 (2.3%) | 8 (19.1%) |

Data are presented as numbers (percentages). AKI, acute kidney injury

**Table 3.** Clinical outcomes according to AKI type

| | All patients | No AKI | Transient AKI | Persistent AKI | P-value |
|---|---|---|---|---|---|
| In-hospital mortality | 42/3,750 (1.1%) | 27/3,492 (0.8%) | 9/216 (4.2%)* | 6/42 (14.3%)*† | <0.001 |
| Hospital length of stay (days) | 16 (16–16) | 16 (15–16) | 27 (23–30)* | 38 (21–74)* | <0.001 |
| 3-month mortality | 41/3,751 (1.1%) | 25/3,493 (0.7%) | 9/216 (4.2%)* | 7/42 (16.7%)*† | <0.001 |
| CKD progression at 3-month | 13/3,665 (0.4%) | 4/3,423 (0.1%) | 3/207 (1.4%)* | 6/35 (17.1%)*† | <0.001 |
| 1-year mortality | 217/3,751 (5.8%) | 174/3,493 (5.0%) | 33/216 (15.3%)* | 10/42 (23.8%)* | <0.001 |
| CKD progression at 1-year | 99/3,087 (3.2%) | 74/2,902 (2.6%) | 15/160 (9.4%)* | 10/25 (40.0%)*† | <0.001 |

One patient still hospitalized at the time of data collection was excluded from the analyses of in-hospital mortality and hospital length of stay. Forty-one patients who died within 3 months after surgery and 45 patients with missing SCr data at 3 months after surgery were excluded from the analysis of CKD progression at 3 months. Additionally, 217 patients who died within 1 year after surgery and 447 patients with missing SCr data at 1 year after surgery were excluded from the analysis of CKD progression at 1 year. The median time [interquartile range] from the surgery to the evaluation of CKD progression at 3-month and 1-year was 17 [10–40] and 335 [298–366] days, respectively. Denominators indicate the total number of patients. Hospital length of stay was presented as median (95% confidence interval). *, $P < 0.017$ (significantly different with Bonferroni correction), compared to patients without AKI. †, $P < 0.017$ (significantly different with Bonferroni correction), compared to patients with transient AKI. AKI, acute kidney injury; CKD, chronic kidney disease.

**Table 4.** Multivariable Cox proportional hazard regression analysis assessing independent association between AKI type and mortality

| | Adjusted hazard ratio (95% CI) | P-value |
|---|---|---|
| AKI type | | |
| No AKI | 1 (Reference) | |
| Transient AKI | 2.01 (1.34–2.93) | 0.001 |
| Persistent AKI | 6.20 (3.00–11.43) | <0.001 |
| Age (per 10 years) | 1.37 (1.20–1.58) | <0.001 |
| Male sex | 1.11 (0.82–1.52) | 0.502 |
| Hypertension | 0.96 (0.71–1.29) | 0.803 |
| Diabetes mellitus | 1.26 (0.91–1.73) | 0.154 |
| Chronic kidney disease | 1.11 (0.78–1.54) | 0.557 |
| Active congestive heart failure | 1.84 (0.76–3.73) | 0.160 |
| Type of surgery | | |
| Colorectal | 1 (Reference) | |
| Liver | 1.64 (1.02–2.64) | 0.039 |
| Gastric | 1.60 (0.96–2.67) | 0.070 |
| Pancreatic | 2.72 (1.70–4.34) | <0.001 |
| Esophageal | 3.70 (2.08–6.56) | <0.001 |
| Complex | 8.42 (3.91–18.12) | <0.001 |
| Emergency surgery | 0.70 (0.04–3.24) | 0.714 |
| Duration of surgery (per 1 h) | 1.11 (1.06–1.17) | <0.001 |
| Intraoperative blood loss (per 100 mL) | 1.01 (1.01–1.02) | <0.001 |
| The need for intraoperative vasopressor infusion | 1.14 (0.77–1.65) | 0.512 |

AKI, acute kidney injury.

**Table 5.** Multivariable logistic regression analysis assessing independent associations between AKI type and CKD progression at 1 year

| | Adjusted odds ratio (95% CI) | P-value |
|---|---|---|
| AKI type | | |
| No AKI | 1 (Reference) | |
| Transient AKI | 3.87 (2.12–7.08) | <0.001 |
| Persistent AKI | 23.70 (9.64–58.22) | <0.001 |
| Age (per 10 years) | 1.39 (1.14–1.70) | <0.001 |
| Male sex | 0.76 (0.49–1.18) | 0.223 |
| Body mass index | 0.96 (0.90–1.03) | 0.239 |
| Hypertension | 1.62 (1.02–2.56) | 0.039 |
| Diabetes mellitus | 1.54 (0.94–2.54) | 0.086 |
| Active congestive heart failure | 0.73 (0.16–3.30) | 0.681 |
| Preoperative eGFR (per 10 mL/min/1.73 $m^2$) | 1.20 (1.10–1.32) | <0.001 |

AKI, acute kidney injury; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate

**Figure captions**

**Figure 1.** Flow diagram of the study participants. AKI, acute kidney injury.

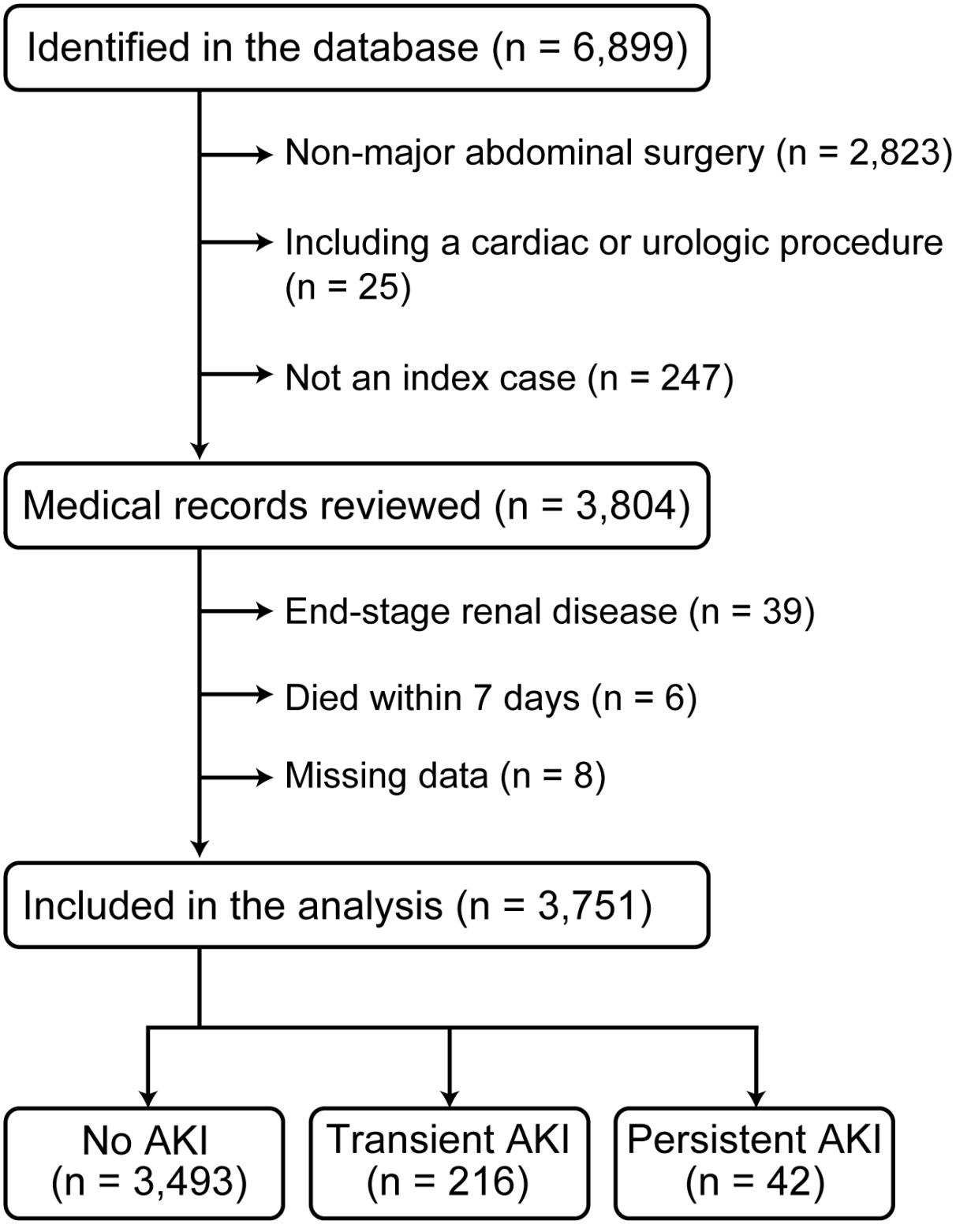
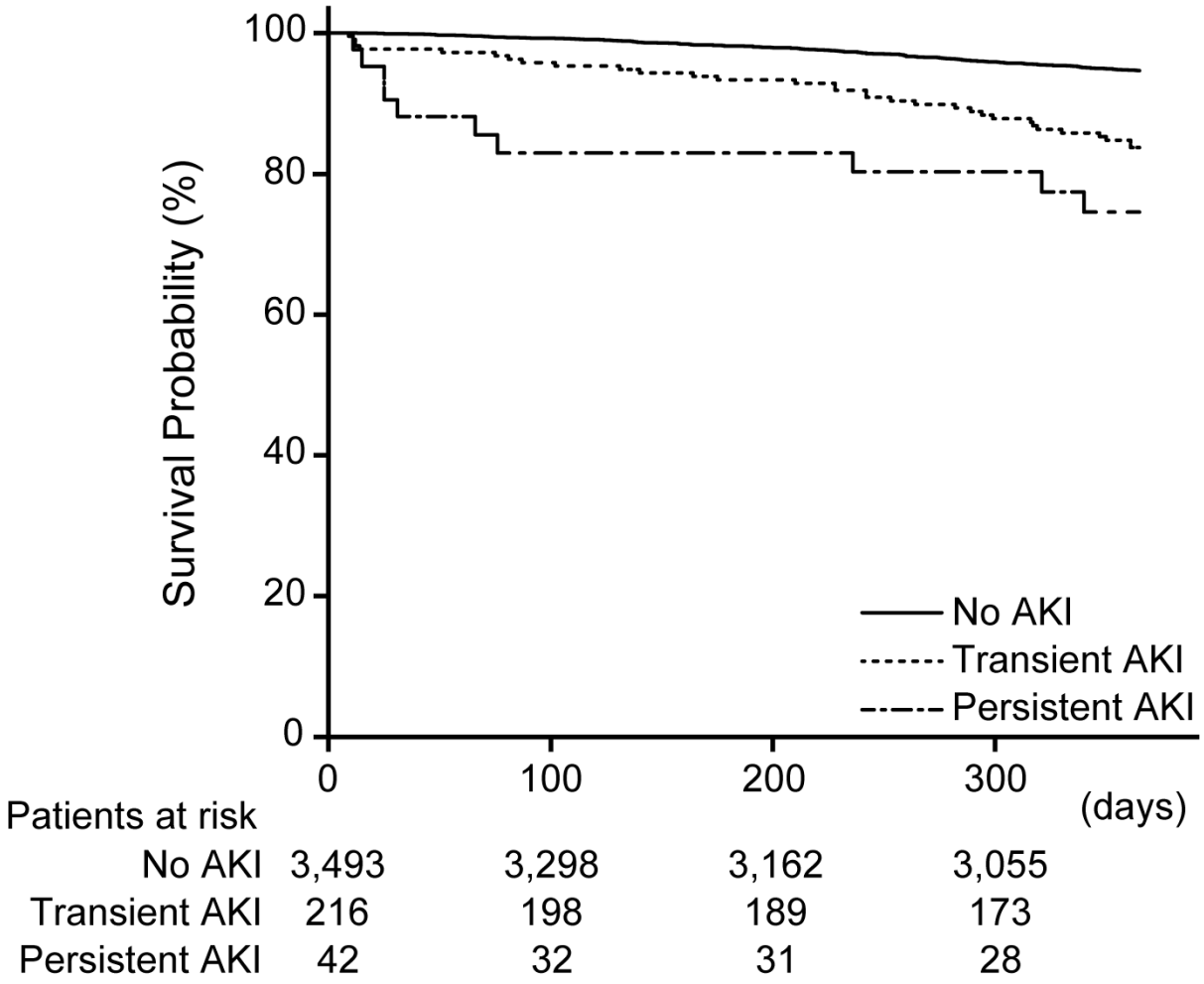


**Figure 2.** Kaplan–Meier curve of patients without AKI, with transient AKI, or with persistent AKI. No AKI vs. Transient AKI: P < 0.001, No AKI vs. Persistent AKI: P < 0.001, Transient AKI vs. Persistent AKI: P = 0.066. AKI, acute kidney injury.



**Supplementary Table 1.** Association of AKI type and outcomes stratified by the maximal AKI stage

| | Transient AKI | | Persistent AKI | |
|---|---|---|---|---|
| | Adjusted association | P-value | Adjusted association | P-value |
| Mortality | | | | |
| Stage 1 AKI | 1.97 (1.32–2.96) | <0.001 | 1.13 (0.16–8.16) | 0.903 |
| Stage 2–3 AKI | 2.19 (0.69–6.96) | 0.183 | 11.38 (5.61–23.08) | <0.001 |
| CKD progression at 1 year | | | | |
| Stage 1 AKI | 3.59 (1.86–6.93) | <0.001 | 13.40 (4.21–42.70) | <0.001 |
| Stage 2–3 AKI | 5.37 (1.44–20.00) | 0.012 | 63.1 (13.6–293.0) | <0.001 |

Mortality was reported as hazard ratios and CKD progression at 1 year as odds ratios; AKI, acute kidney injury; CKD, chronic kidney disease

**Supplementary Table 2.** Preoperative eGFR category and eGFR category at 1 year of patients demonstrating CKD progression at 1 year

| | No AKI (n = 74) | | | | | Transient AKI (n = 15) | | | | | Persistent AKI (n = 10) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Preoperative eGFR category | 1 | 2 | 3a | 3b | 4 | 1 | 2 | 3a | 3b | 4 | 1 | 2 | 3a | 3b | 4 |
| eGFR category at 1 year | | | | | | | | | | | | | | | |
| 2 | 26 | - | - | - | - | 1 | - | - | - | - | 2 | - | - | - | - |
| 3a | 1 | 27 | - | - | - | 0 | 3 | - | - | - | 1 | 2 | - | - | - |
| 3b | 1 | 9 | 5 | - | - | 0 | 4 | 4 | - | - | 0 | 3 | 0 | - | - |
| 4 | 0 | 1 | 0 | 2 | - | 1 | 0 | 0 | 1 | - | 1 | 0 | 0 | 0 | - |
| 5 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |

Most patients with CKD progression demonstrated worsening of eGFR by one eGFR category. The proportions of patients with no AKI, transient AKI and persistent AKI who demonstrated reduced eGFR level by ≥2 eGFR categories were 16.2%, 33.3%, and 50.0%, respectively. Four patients developed CKD stage 5 at 1 year. eGFR, estimated; AKI, acute kidney injury.