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Original Research Article

ORIGINAL RESEARCH ARTICLE

Postoperative acute kidney injury is associated with persistent renal dysfunction: a multicentre propensity-matched cohort study

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Abstract

Background: The risk of developing a persistent reduction in renal function after postoperative acute kidney injury (pAKI) is not well established. The goal of this investigation was to evaluate whether patients who develop pAKI have a greater decline in long-term renal function than patients who do not.

Methods: In this multicentre retrospective propensity-matched study, anaesthesia data warehouses at three tertiary care hospitals were queried. Adult patients undergoing surgery with available preoperative and postoperative creatinine results and without baseline haemodialysis requirements were included. Patients were stratified by occurrence of pAKI as defined by the Acute Kidney Injury Network classification. The primary outcome was a decline in follow-up glomerular filtration rate (GFR) of 40% relative to baseline, based on follow-up outpatient visits from 0 to 36 months after hospital discharge. A propensity score-matched sample was used in Kaplan–Meier analysis and a piecewise Cox model to compare the time to reach a 40% decline in GFR for patients with and without pAKI.

Results: In 95 213 patients, the rate of pAKI ranged from 9.9% to 13.7%. In the piecewise Cox model, pAKI was associated with a significantly increased hazard of a 40% decline in GFR. The common-effect hazard ratio was 13.35 (95% confidence interval [CI] 10.79–16.51, $P<0.001$) for 0–6 months, 7.07 (5.52–9.05, $P<0.001$) for 6–12 months, 6.02 (4.69–7.74, $P<0.001$) for 12–24 months, and 4.32 (2.65–7.05, $P<0.001$) for 24–36 months.

Conclusions: pAKI is associated with a significantly increased hazard of a 40% decline in GFR up to 36 months after surgery across three institutions.

Keywords: acute kidney injury; anaesthesia; chronic kidney disease; perioperative; postoperative outcomes; renal recovery; surgery

Acute kidney injury (AKI) is a known complication of surgery through mechanisms including hypoperfusion and direct tubular injury.[1] Postoperative AKI (pAKI) has been associated with substantial consequences such as impaired cardiac

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function, pulmonary complications, volume overload, higher rates of systemic and wound infection, and delirium.[1–6] Overall, pAKI has been associated with a three-to five-fold increased risk of postoperative mortality,[7,8] longer length of stay, and increased rates of readmission.[8,9]

Despite many studies of the short-term complications of pAKI, little work has been done regarding progression to long-term renal dysfunction. In nonsurgical patients, significant associations between AKI and the new onset or progression of existing chronic kidney disease (CKD) have been observed in the inpatient and intensive care unit (ICU) settings.[10–12] However, the pathophysiology of pAKI in patients undergoing often elective surgical procedures may differ from causative factors in medical patients (e.g. acute illness such as sepsis). Thus, the risk of developing a persistent reduction in renal function after pAKI cannot be directly extrapolated from nonsurgical populations. The studies that did look at the progression of pAKI to CKD have been smaller single-centre studies, thus limiting generalisability.[13,14] Although AKI itself is associated with significant short-term morbidity and mortality, an association with an increased incidence of CKD would substantially increase its long-term implications.

To better understand the association between pAKI and progression to CKD, we performed a multicentre retrospective propensity-matched examination of the association between pAKI and longer-term CKD. The primary outcome was a decline in glomerular filtration rate (GFR) of at least 40% (relative to preoperative baseline),[15] as measured at outpatient follow-up visits up to 36 months after discharge.

Methods

Data were collected from departmental anaesthesia data warehouses at Mount Sinai Hospital (MSH), the University of Miami (UM), and the University of California Los Angeles (UCLA). Institutional Review Board approval was obtained at each institution with a waiver of informed consent. All three institutions use a perioperative data warehouse from Extrico Health, which has been described previously.[16]

Inclusion and exclusion criteria

Patients were included if they were ≥18 yr old and underwent surgery at one of the three sites. Data were collected between 2016 and 2021 at MSH, between 2020 and 2024 at UCLA, and between 2017 and 2023 at UM. No surgical specialties were excluded in the data extraction. Patients were included regardless of the baseline CKD stage, which was controlled for in the analysis. However, patients undergoing haemodialysis (as identified by haemodialysis events in each database or by dialysis-related procedures) before the index surgery were excluded because further progression of their renal dysfunction (i.e. GFR) cannot be determined from their serum creatinine values. Patients were also excluded if they did not have at least one creatinine result from the baseline and postoperative periods, which are described below. Because of this criterion, ambulatory surgical patients who did not have postoperative blood samples drawn before discharge were excluded from the cohort. For patients with multiple operations during one hospitalisation, only the first operation was included in the analysis. If patients had multiple separate hospitalisations during the study period, each of these encounters was included. However, once a patient experienced an AKI event, subsequent encounters were excluded.

Measurement of baseline renal function, AKI, and CKD

Baseline renal function was determined by collecting the most recent creatinine value up to 6 months before surgery. Postoperative creatinine readings included results drawn after the 'anaesthesia stop' event in the anaesthetic record and before hospital discharge. Follow-up creatinine results were included from outpatient visits (excluding those obtained during subsequent hospitalisations) from 0 to 36 months after hospital discharge.

For each creatinine value, the GFR was calculated from each recorded creatinine using the 2021 CKD-EPI formula to maintain a consistent GFR definition.[17] The Acute Kidney Injury Network (AKIN) classification was used to compare postoperative creatinine measurements with baseline renal function to identify patients who experienced pAKI.[18,19] The pAKI group included patients that met criteria for AKIN stage 1 (an increase of ≥0.3 mg dl^{-1} or 1.5–2× from baseline), stage 2 (an increase of >2–3× from baseline), or stage 3 (an increase of >3× or creatinine of >4 mg dl^{-1} with an acute increase of ≥0.5 mg dl^{-1}). Persistent renal dysfunction at each endpoint was defined as a decline in follow-up GFR of 40% from baseline, which is in line with trials of kidney disease progression.[15]

Covariates

Data were also collected for various variables based on a review of published risk factors for AKI.[1,20–23] These included age, sex, body mass index (BMI), self-reported race, surgical specialty, American Society of Anesthesiologists physical status (ASA-PS) classification, history of hypertension, congestive heart failure (CHF), chronic pulmonary disease, diabetes mellitus, peripheral vascular disease, and liver disease. The comorbidity variables were determined from the International Classification of Diseases, Ninth and Tenth Revision codes using the *icd* package[24] in R version 4.2.3 (R Foundation for Statistical Computing, Vienna, Austria). This package implements coding algorithms published by Quan and colleagues.[25]

Statistical analysis

Data collection and analysis were performed separately at each site. Patients were grouped based on whether they experienced pAKI. Standardised mean differences between the two groups were calculated using the R package *tableone*.

To control for differences in the baseline characteristics of patients in the AKI *vs* non-AKI cohorts, we performed a propensity score matching analysis. For patients who experienced AKI, we included only the AKI encounter while accounting for the number of surgical encounters before the AKI encounter. We fitted a logistic regression propensity model to estimate the probability of having AKI for each individual, using the variables listed above, along with the number of surgical encounters before the current encounter. Next, we matched every AKI patient to non-AKI patients with a 1:3 ratio based on the propensity score using nearest-neighbour matching with replacement and a calliper width of 0.1 units (R package *MatchIt*). When the balance goal, defined as an absolute standardised mean difference of <0.1 for all propensity score model predictors, was achieved, we performed Kaplan–Meier analysis to estimate the association of AKI with time to reach a 40% decline in GFR, based on the outpatient follow-up laboratory data (R package *survminer* and *survival*). Furthermore, a stratified Cox proportional hazards

regression model was used to assess the association of AKI with time to reach a 40% decline in GFR for outpatient follow-up laboratory data while accounting for fluctuations in renal function during the follow-up period. The periods were stratified into 0–6, 6–12, 12–24, and 24–36 months.

To evaluate the overall effect of AKI across the three sites, a meta-analysis of the three centres' results was performed (R package *meta*). We used the inverse variance method to pool the hazard ratios (HRs) with 95% confidence intervals (CIs) separately for each period. Pooled results are reported as odds ratio with 95% CI using random-effects models where the medical centre is considered a fixed effect.

In addition, a secondary analysis evaluating the interaction between AKI, race, and time was performed. A three-way interaction variable was included in the stratified Cox proportional hazard regression model to assess the association of AKI with time to reach a 40% decline in GFR for different racial groups for each follow-up period. Race was stratified into White or non-White to reduce the complexity of the interaction model, and time was stratified into less than or greater than 6 months. The Kaplan–Meyer analysis was also repeated, with patients stratified into White or non-White race.

Given the likelihood of patients not having follow-up creatinine results within each health system, a sensitivity analysis was performed to assess for factors that may predispose patients to lack of any follow-up creatinine determinations post discharge. A weight of inverse probability of having any follow-up data was applied to all analyses. Specifically, we fitted a logistic model to estimate the probability of having any follow-up using all baseline variables and their interaction with pAKI. All outcome analyses were then weighed using the inverse of this probability and matching weight.

An additional sensitivity analysis was performed using standard multivariable modelling rather than propensity score matching in the stratified Cox proportional hazards regression model for the association of AKI with a 40% decline in GFR over the follow-up periods.

A priori statistical significance was set at $P<0.05$. All statistical analysis was performed using R version 4.2.3.

Secondary endpoints

As a secondary endpoint, each site's database was also queried for postoperative consultations or referrals to the nephrology service. Inpatient and outpatient consultations up to 6 months after surgery were recorded. The rates of nephrology consultations were calculated for patients stratified by severity of AKI (as reported by the AKIN score).

Results

The study included 95 213 patients: 38 061 patients from MSH, 35 730 patients from UM, and 21 422 patients from UCLA. The flowchart of the number of patients included and excluded at each step is shown in Supplementary Figure S1. The number of patients who experienced pAKI was 5211 (13.7%) from MSH, 3520 (9.9%) from UM, and 2297 (10.7%) from UCLA. A full comparison of patient characteristics between the unmatched AKI and non-AKI groups is included in Table 1. At all three sites, the AKI group was older and had a higher proportion of male and ASA-PS 4 and 5 patients, lower baseline GFR, and higher rates of certain comorbidities, such as CHF, hypertension, diabetes, liver disease, and peripheral

Table 1 Case series characteristics. AKI, acute kidney injury; ASA-PS, American Society of Anesthesiologists physical status; BMI, body mass index; CHF, congestive heart failure; CKD, chronic kidney disease; IQR, interquartile range; MSH, Mount Sinai Hospital; SMD, standardised mean difference; UCLA, University of Southern California; UM, University of Miami. *Postoperative period includes readings drawn after the 'anaesthesia stop' event in the anaesthetic record and before hospital discharge.

	MSH				UM				UCLA			
	No AKI	AKI	SMD	Overall	No AKI	AKI	SMD	Overall	No AKI	AKI	SMD	Overall
Patients (*n*)	32 850	5211		38 061	32 210	3520		35 730	19 125	2297		21 422
Age (yr), mean (range)	58.8 (18-102)	60.8 (18-97)	0.12	59.1 (18-102)	61.3 (18-90)	65.2 (18-90)	0.28	61.7 (18-90)	61.5 (18-103)	64.1 (18-99)	0.17	61.7 (18-103)
Male sex, *n* (%)	16 467 (50.1)	3078 (59.1)	0.18	19 545 (51.4)	17 514 (54.4)	2245 (63.8)	0.19	19 759 (55.3)	9034 (47.2)	1348 (58.7)	0.23	10 382 (48.5)
Race			0.13				0.13				0.17	
White	15 432 (47.0)	2241 (43.0)		17 673 (46.4)	24 847 (77.1)	2567 (72.9)		27 414 (76.7)	8244 (43.1)	986 (42.9)		9230 (43.1)
Black	5374 (16.4)	1097 (21.1)		6471 (17.0)	4216 (13.1)	626 (17.8)		4842 (13.6)	1203 (6.3)	187 (8.1)		1390 (6.5)
Asian	1647 (5.0)	207 (4.0)		1854 (4.9)	403 (1.3)	42 (1.2)		445 (1.2)	2024 (10.6)	242 (10.5)		2266 (10.6)
Other	10 397 (31.7)	1666 (32.0)		12 063 (31.7)	2744 (8.5)	285 (8.1)		3029 (8.5)	7654 (40.0)	882 (38.4)		8536 (39.8)
BMI, median (IQR)	27.0 (23.4–31.9)	27.6 (24.0–32.0)	0.04	27.1 (23.4–31.9)	27.4 (24.0–31.3)	28.5 (24.8–32.9)	0.002	27.4 (24.1–31.5)	26.5 (23.0–31.0)	26.4 (22.8–30.5)	0.07	26.5 (22.9–30.9)
ASA-PS			0.73				0.38				0.68	
1	393 (1.2)	291 (5.6)		684 (1.8)	326 (1.0)	10 (0.3)		336 (0.9)	144 (0.8)	5 (0.2)		149 (0.7)
2	9623 (29.3)	743 (14.3)		10 366 (27.2)	7983 (24.8)	497 (14.1)		8480 (23.7)	4487 (23.5)	181 (7.9)		4668 (21.8)
3	17 534 (53.4)	1947 (37.4)		19 481 (51.2)	20 080 (62.3)	2217 (63.0)		22 297 (62.4)	11 954 (62.5)	1247 (54.3)		13 201 (61.6)
4	5176 (15.8)	2052 (39.4)		7228 (19.0)	3713 (11.5)	755 (21.4)		4468 (12.5)	2512 (13.1)	829 (36.1)		3341 (15.6)

Continued

Table 1 Continued

	MSH				UM				UCLA			
	No AKI	AKI	SMD	Overall	No AKI	AKI	SMD	Overall	No AKI	AKI	SMD	Overall
5	124 (0.4)	178 (3.4)		302 (0.8)	34 (0.1)	31 (0.9)		65 (0.2)	28 (0.1)	35 (1.5)		63 (0.3)
CHF, *n* (%)	2358 (7.2)	795 (15.3)	0.26	3153 (8.3)	757 (2.4)	206 (5.9)	0.18	963 (2.7)	1274 (6.7)	382 (16.6)	0.32	1656 (7.7)
Hypertension, *n* (%)	16 611 (50.6)	3140 (60.3)	0.20	19 751 (51.9)	16 364 (50.8)	2392 (68.0)	0.36	18 756 (52.5)	9703 (50.7)	1439 (62.6)	0.24	11 142 (52.0)
Chronic pulmonary disease, *n* (%)	5420 (16.5)	877 (16.8)	0.01	6297 (16.5)	4104 (12.7)	497 (14.1)	0.04	4601 (12.9)	3483 (18.2)	479 (20.9)	0.07	3962 (18.5)
Diabetes	6982 (21.3)	1608 (30.9)	0.22	8590 (22.6)	6476 (20.1)	1193 (33.9)	0.31	7669 (21.5)	3895 (20.4)	651 (28.3)	0.19	4546 (21.2)
Liver disease, *n* (%)	2377 (7.2)	895 (17.2)	0.31	3272 (8.6)	1283 (4.0)	162 (4.6)	0.03	1445 (4.0)	1607 (8.4)	385 (16.8)	0.25	1992 (9.3)
Peripheral vascular disease, *n* (%)	3506 (10.7)	675 (13.0)	0.07	4181 (11.0)	611 (1.9)	110 (3.1)	0.08	721 (2.0)	1493 (7.8)	300 (13.1)	0.17	1793 (8.4)
Baseline creatinine, median (IQR)	0.87 (0.72–1.08)	1.01 (0.79–1.41)	0.29	0.89 (0.73–1.11)	0.89 (0.73–1.08)	1.01 (0.80–1.30)	0.43	0.90 (0.73–1.10)	0.85 (0.70–1.05)	0.99 (0.78–1.34)	0.21	0.86 (0.71–1.07)
Peak postoperative* creatinine, median (IQR)	0.86 (0.70–1.06)	1.76 (1.35–2.83)	0.91	0.91 (0.72–1.20)	0.89 (0.73–1.09)	1.58 (1.28–2.17)	0.91	0.93 (0.75–1.16)	0.83 (0.68–1.03)	1.67 (1.30–2.37)	0.86	0.87 (0.69–1.13)
Day of peak postoperative creatinine, median (IQR)	1 (1–2)	2 (1–5)	0.36	1 (1–2)	1 (1–2)	2 (1–4)	0.36	1 (1–2)	1 (1–2)	2 (1–5)	0.39	1 (1–2)
Baseline GFR, median (IQR)	88.9 (69.1–103)	75.2 (48.6–97.5)	0.43	87.7 (66.6–102)	83.8 (70.2–95.0)	73.5 (55.0–86.7)	0.56	82.9 (68.9–94.3)	88.7 (69.9–101.0)	76.5 (52.2–5.4)	0.39	87.8 (63.5–101.0)
Baseline CKD stage, *n* (%)			0.45				0.53				0.43	
1	15 856 (48.3)	1814 (34.8)		17 670 (46.4)	11 547 (35.9)	672 (19.1)		12 219 (34.2)	9066 (47.4)	748 (32.6)		9814 (45.8)
2	11 436 (34.8)	1569 (30.1)		13 005 (34.2)	16 316 (50.7)	1763 (50.1)		18 079 (50.6)	6941 (36.3)	801 (34.9)		7742 (36.1)
3a	2817 (8.6)	689 (13.2)		3506 (9.2)	2842 (8.8)	536 (15.2)		3378 (9.5)	1747 (9.1)	326 (14.2)		2073 (9.7)
3b	1468 (4.5)	502 (9.6)		1970 (5.2)	1033 (3.2)	263 (7.5)		1296 (3.6)	770 (4.0)	222 (9.7)		992 (4.6)
4	677 (2.1)	327 (6.3)		1004 (2.6)	467 (1.5)	286 (8.1)		753 (2.1)	274 (1.4)	120 (5.2)		394 (1.8)
5	596 (1.8)	310 (5.9)		906 (2.4)	–	–		–	325 (1.7)	80 (3.5)		405 (1.9)
Surgical specialty, *n* (%)			0.60				0.59				0.47	
Cardiac	979 (3.0)	530 (10.2)		1509 (4.0)	2268 (7.0)	364 (10.3)		2632 (7.4)	1546 (8.1)	447 (19.5)		1993 (9.3)
Otolaryngology	987 (3.0)	90 (1.7)		1077 (2.8)	1838 (5.7)	157 (4.5)		1995 (5.6)	–	–		–
General	7617 (23.2)	1047 (20.1)		8664 (22.8)	3087 (9.6)	341 (8.9)		3428 (9.6)	–	–		–
Gastrointestinal	2174 (6.6)	293 (5.6)		2467 (6.5)	3593 (11.2)	374 (10.6)		3967 (11.1)	2108 (11.0)	288 (12.5)		2396 (11.2)
Neurosurgery	2406 (7.3)	109 (2.1)		2515 (6.6)	6191 (19.2)	173 (4.9)		6364 (17.8)	1803 (9.4)	68 (3.0)		1871 (8.7)
Orthopaedic	2985 (9.1)	253 (4.9)		3238 (8.5)	3891 (12.1)	343 (9.7)		4234 (11.9)	3553 (18.6)	243 (10.6)		3796 (17.7)
Other	3774 (11.5)	403 (7.7)		4177 (11.0)	3667 (11.4)	427 (12.1)		4094 (11.6)	10 115 (52.9)	1251 (54.5)		11 366 (53.1)
Spine	931 (2.8)	59 (1.1)		990 (2.6)	–	–		–	–	–		–
Thoracic	1982 (6.0)	215 (4.1)		2197 (5.8)	1674 (5.2)	126 (3.6)		1800 (5.0)	–	–		–
Transplant	1124 (3.4)	543 (10.4)		1667 (4.4)	–	–		–	–	–		–
Urology	3860 (11.8)	937 (18.0)		4797 (12.6)	5237 (16.3)	1066 (30.3)		6303 (17.6)	–	–		–
Vascular	2259 (6.9)	405 (7.8)		2664 (7.0)	764 (2.4)	149 (4.2)		913 (2.6)	–	–		–
Patients included after propensity score matching (*n*)	8963	4830		13 793	7615	3475		11 090	4703	2265		6968

vascular disease. A higher proportion of patients in the AKI group underwent cardiac, urology, and transplant procedures.

Propensity score matching for AKI

Propensity score matching for pAKI was performed. Supplementary Figure S1 shows the standardised mean differences for included variables before and after matching and demonstrates a good matching balance (<0.1 for all variables).

Association of pAKI with CKD

Table 2 shows the results of the piecewise Cox model for estimating the hazard of AKI for developing CKD (defined as a 40% decline in GFR) over a series of postoperative time intervals using the propensity score-matched sample. The HR for the effect of pAKI on a 40% decline in GFR was significant throughout the entire 36-month follow-up period at all three sites. In the meta-analysis, the 0–6-month interval had the highest common-effect HR of 13.35 (95% CI 10.79–16.51, $P<0.001$). The common-effect HR was 7.07 (5.52–9.05, $P<0.001$) for 6–12 months, 6.02 (4.69–7.74, $P<0.001$) for 12–24 months, and 4.32 (2.65–7.05, $P<0.001$) for 24–36 months. The results of the Kaplan–Meier analysis for the time to reach a 40% decline in GFR are shown in Fig 1. The curves show significantly greater time free from a 40% decline in GFR for the non-AKI group for all three sites (MSH: HR 5.38, 95% CI 4.70–6.16, $P<0.001$; UM: HR 4.01, 95% CI 3.19–5.04, $P<0.001$; UCLA: HR 5.06, 95% CI 4.21–6.29, $P<0.001$).

Sensitivity analyses

The percentage of patients with follow-up outpatient creatinine results in each database was 59% for MSH, 43% for UM, and 72% for UCLA. The rates of follow-up laboratory results for patients with and without pAKI at each time point are shown in Supplementary Table S1a. Because a percentage of patients were lost to follow-up (i.e. did not have serum creatinine values in our database after hospital discharge), a sensitivity analysis was performed using inverse probability weighting for factors that may be associated with patients having any available follow-up laboratory data. After applying this to the model for the effect of pAKI on a 40% decline in GFR, similar associations and HRs were observed. The results of this model are shown in Supplementary Table S1b.

When our main stratified Cox proportional hazards regression model was repeated using standard multivariable modelling rather than the propensity score matching, a similarly strong association between pAKI and a 40% decline in GFR was observed across all follow-up intervals. These results are shown in Supplementary Table S2.

Influence of race on CKD after AKI

As reported in Supplementary Table S3, a three-way interaction variable between AKI, race, and time was included in the stratified Cox proportional hazard regression model. The three-way interaction variable was not significant for the MSH ($P=0.902$) or UM data ($P=0.344$) but was significant for the UCLA data ($P=0.015$). The magnitude of the effect of AKI on the risk of GFR decline decreased over time for all racial groups, with more of a decrease in effect for non-White patients than for White patients within the UCLA dataset. Supplementary Figure S3 shows the Kaplan–Meier analysis for the time to reach a 40% decline in GFR stratified by race.

Post-discharge nephrology follow-up

The number of patients with consultation notes from the nephrology service up to 6 months after surgery was recorded, and the rates were stratified by severity of AKI (as reported by the AKIN score). The percentage of patients with postoperative nephrology visits increased with higher AKIN scores. Less than 10% of AKIN 1 and 2 patients had nephrology visits across all three sites. For AKIN 3 patients, the rate ranged from 9.7% at MSH to 21.6% at UCLA. The full rates and times to consultation are shown in Supplementary Table S4.

Discussion

In this study, AKI in the immediate postoperative period was associated with renal dysfunction up to 36 months after surgery. This finding was found across three institutions with different population procedural characteristics. The association was maintained after matching for established risk factors for AKI and during a sensitivity analysis accounting for missing data. These results demonstrate that even mild pAKI is often not a transient, benign phenomenon: a clinically significant fraction of such patients do not experience renal recovery to baseline

Table 2 Piecewise Cox model for estimating the hazard of AKI on developing a 40% decline in GFR using a propensity score-matched sample with meta-analysis. The inverse variance meta-analytical method was applied. AKI, acute kidney injury; CI, confidence interval; MSH, Mount Sinai Hospital; UCLA, University of Southern California; UM, University of Miami.

	Common-effect hazard ratio (95% CI)	P-value	Site	Site-effect hazard ratio (95% CI)
0–6 months	13.35 (10.79–16.51)	<0.001	Miami	14.17 (9.53–21.06)
			Sinai	11.06 (7.80–15.67)
			UCLA	15.59 (10.83–22.46)
6–12 months	7.07 (5.52–9.05)	<0.001	Miami	7.12 (3.97–12.77)
			Sinai	6.56 (4.66–9.23)
			UCLA	8.03 (5.09–12.67)
12–24 months	6.02 (4.69–7.74)	<0.001	Miami	4.46 (2.21–8.98)
			Sinai	6.82 (4.77–9.77)
			UCLA	5.69 (3.81–8.51)
24–36 months	4.32 (2.65–7.05)	<0.001	Miami	3.89 (1.63–9.29)
			Sinai	6.05 (4.09–8.95)
			UCLA	4.32 (2.65–7.05)

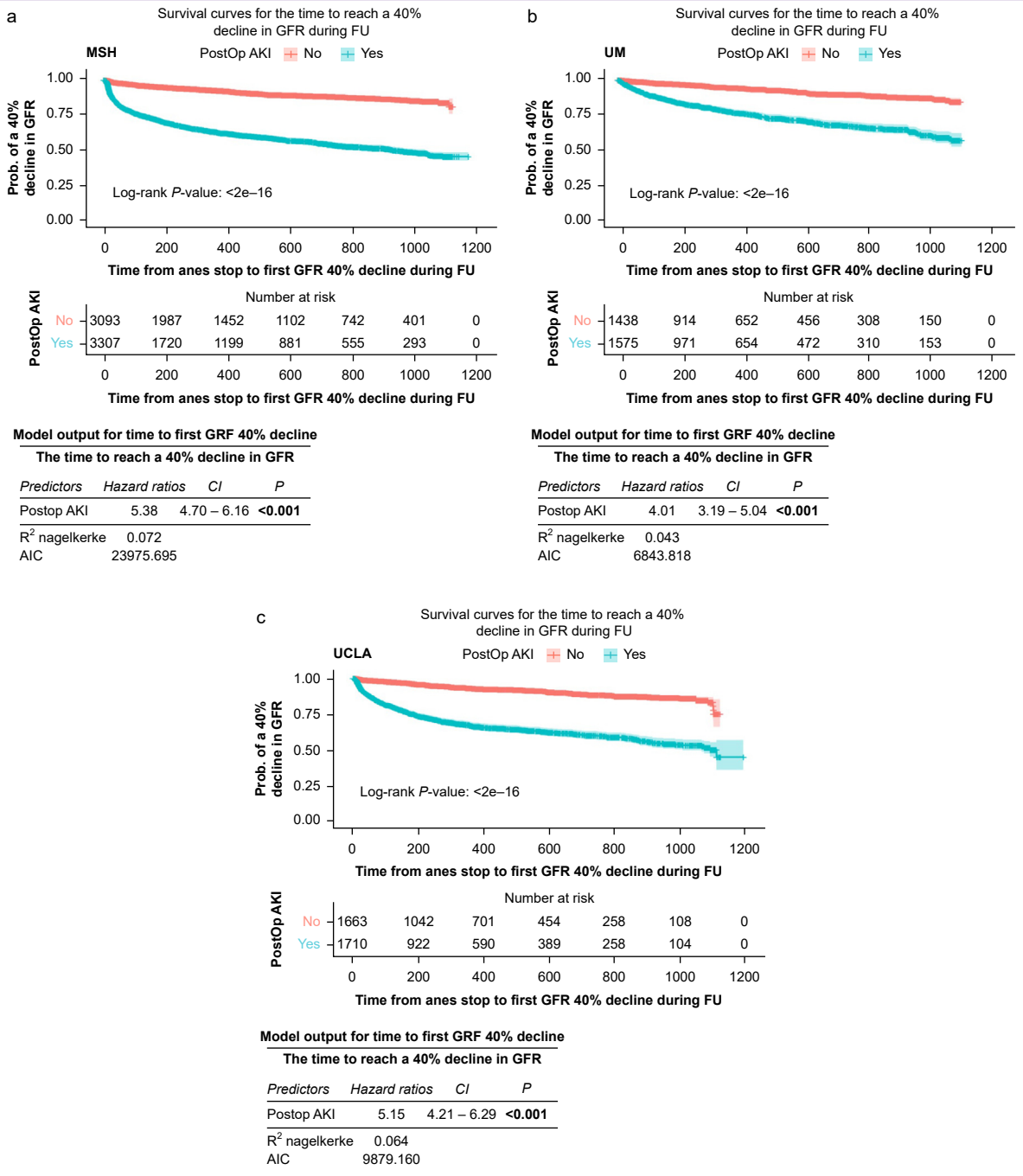

Figure 1. Kaplan–Meier analysis for the time to reach a 40% decline in GFR using propensity score-matched sample. AIC, Akaike information criterion; AKI, acute kidney injury; Anes stop, anaesthesia stop; CI, confidence interval; FU, follow-up; MSH, Mount Sinai Hospital; PostOp, postoperative; Prob., probability; UCLA, University of California Los Angeles; UM: University of Miami.

within several years after the index surgery. This study also showed that fewer than 20% of patients with pAKI saw a nephrologist after discharge, indicating that there may be opportunities for improved follow-up to decrease the progression to CKD.

Published rates of AKI vary across studies, depending both on patient and surgical populations and on definitions of AKI. The rates of AKI in our study were relatively consistent across the three intuitions studied (13.7% at MSH, 9.9% at UM, and 11.0% at UCLA).

Our study also reaffirmed the patient and operative risk factors that have been previously shown to be associated with an increased risk of pAKI.[1,20–23]

Similar work focusing on cardiac surgery has demonstrated increased HRs of CKD after pAKI.[26] In noncardiac surgery, several studies have shown progression to CKD after pAKI.[13,14]

However, these studies were in smaller single-centre cohorts. Our study extends this work with a cohort six times larger than previously reported, uses a multicentre analysis, and adjusts risk for pAKI using propensity score matching. The fact that the progression to CKD remains common after these adjustments indicates that there may be a scientific basis for the assumption that pAKI is not only associated with CKD but may actually be an initiating factor in its development.

As part of our analysis, we also assessed the proportion of patients who received a nephrology consultation within 6 months after surgery: this was <10% for AKIN 1 and 2 patients and <22% for AKIN 3 patients. Although the current standard of care is not necessarily to refer patients with pAKI to a nephrologist for follow-up, we believe this may represent an unrecognised opportunity. Just as it is common to refer patients to a cardiologist after an acute cardiac event, referral to a nephrologist may be beneficial in optimising medication regimens (such as diuretics or antihypertensive medications) and ensuring the avoidance of nephrotoxic medications such as NSAIDs, which are often commonly used in the postoperative period.

Our study has several limitations. Certain pertinent variables such as postoperative urine output were not reliably available. Some patients did not have follow-up serum creatinine measurements in the institutional EHR database, and the retrospective, de-identified study design made obtaining missing data difficult for patients who may have had follow-up tests performed elsewhere. However, we performed a sensitivity analysis to assess the possible effect of differences between patients with and without documented follow-up of serum creatinine and did not find any significant changes to the results. Our study periods were defined to distinguish between inpatient acute changes in renal function and long-term outpatient renal function. However, because only patients with postoperative creatinine results before discharge were included, patients undergoing ambulatory surgery who did not have postoperative blood samples drawn before discharge were not captured. Thus, our cohort may be skewed toward patients with a higher comorbidity burden or undergoing more major operations (i.e. requiring inpatient postoperative care). However, the large number of patients and consistent findings across the three institutions support the generalisability of our findings. Lastly, the new initiation of renal replacement therapy after surgery was not included as an outcome because it is not delineated as an event in our perioperative data warehouse. We may thus have slightly underestimated the number of patients with new renal failure but still found a strong association between AKI and persistent renal dysfunction.

In conclusion, this multicentre investigation demonstrated that pAKI was associated with a significantly increased hazard of a persistent 40% decline in GFR up to 36 months after surgery. These findings underline the importance of further study into strategies to help prevent perioperative AKI given its potential for lasting renal dysfunction.

Authors' contributions

Study conception: BS, ISH.
Study design: BS, RHE, EG, GNN, YO, HML, ISH.
Data collection: BS, RHE, VS, ISH.
Data analysis: BS, RHE, EG, VS, ISH.
Data interpretation: BS, RHE, EG, GNN, ISH.
Statistical analysis and interpretation: YO, HML.
Manuscript preparation: BS.
Manuscript review: RHE, EG, GNN, YO, HML, VS, ISH.

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Declarations of interest

ISH is the founder and President of Extrico Health, a company that helps hospitals leverage data from their electronic health record for decision-making purposes. ISH receives research support and serves as a consultant for Merck.
EG has equity in Extrico Health. The Extrico platform was used to obtain data for this study from all sites.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bjao.2025.100384.

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