Intraoperative Hemodynamic Parameters Related to Acute Kidney Injury in Non-ruptured Infrarenal Abdominal Aortic Aneurysm

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Abstract

Background: Postoperative acute kidney injury (AKI) affects up to 30% of patients after infrarenal abdominal aortic aneurysm (AAA) repair, increasing mortality, extending hospitalization, and causing long-term renal dysfunction. The specific intraoperative hemodynamic factors that influence perioperative AKI risk remain unclear. Clarifying these modifiable factors is key to developing targeted interventions to improve outcomes and reduce costs.

Objective: To determine the association between intraoperative hemodynamic parameters and perioperative acute kidney injury (AKI) in patients with non-ruptured infrarenal AAA repair.

Material and Methods: Patients underwent non-ruptured infrarenal AAA repair by open aneurysmorrhaphy and endovascular aneurysm repair (EVAR) at Thammasat University Hospital between January 2016 and October 2021. Intraoperative hemodynamic parameters and AKI by raising of creatinine (Cr) correlation were analysed by Spearman's rank correlation test. The multivariate regression analysis determined the perioperative risk factors of AKI.

Results: 32 patients were included, with 14 patients (43.75%) in open aneurysmorrhaphy and 18 patients (56.25%) in EVAR. Peri-operative urine output < 0.5 ml/kg/min was associated with AKI in patients with non-ruptured infrarenal AAA repair (RR 11.5, 95%CI 1.49-88.27; *p*-value = 0.019). Immediate de-clamping or deployment of diastolic blood pressure (DBP) and mean arterial pressure (MAP) were significantly related with Cr raising by moderated correlation (DBP: ρ = 0.434, P = 0.013; MAP: ρ = 0.414, P = 0.018, respectively). De-clamping hypotension by SBP, DBP, and MAP was significantly related with Cr raising by moderated correlation (SBP: ρ = 0.471, P = 0.006; DBP: ρ = 0.609, p < 0.001; MAP: ρ = 0.612, 95%, p < 0.001), respectively).

Conclusion: Peri-operative urine output < 0.5 ml/kg/min was a risk factor of perioperative AKI. Immediate de-clamping/deployment DBP, MAP, and de-clamping hypotension were significantly correlated with peri-operative urine output and AKI in non-ruptured infrarenal AAA repair procedures. This result is a pilot data for further large multicenter prospective cohort to confirm this result.

Keywords: Abdominal aortic aneurysm, Acute kidney injury, Perioperative

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Introduction

Abdominal aortic aneurysm (AAA) is a highly fatal vascular disease leading to a high morbidity and mortality incidence, incredibly complicated ruptured AAA. Acute kidney injury (AKI) is a common postoperative complication associated with poor outcomes after AAA repair surgery[1-2]. Around one-third of AAA patients developed AKI after the repair procedure[3-4]. Many perioperative factors may have influenced the development of AKI after AAA repair. Evidence shows an incidence of renal complications after endovascular and open repair of AAA with predictors of AKI, including elevated baseline glomerular filtration rate (GFR), open approach, transfusion, and prolonged operative time[5]. Perioperative AKI may be associated with in-hospital mortality [4,6], even though few studies reported that there were no worsening outcomes following AKI[7-8]. Regarding intraoperative arterial blood pressure, Michael Walsh [9] studied the relationship between intraoperative mean arterial pressure (MAP) and clinical outcomes after noncardiac surgery. They concluded that even short durations of an intraoperative MAP less than 55 mmHg were associated with AKI and myocardial injury. However, there are few studies investigating intraoperative hemodynamic parameters during AAA repair related to AKI. Thus, we aimed to estimate the effects of correlation between the raising of perioperative creatinine (Cr) level and intraoperative hemodynamic parameters in patients with non-ruptured infrarenal AAA repair and determine the perioperative factors associated with AKI.

Materials and methods

We conducted a retrospective cohort study from the electronic medical record (E-phis) at Thammasat University between January 2016 and October 2021, with a total of 32 patients underwent non-ruptured infrarenal AAA repair by open aneurysmorrhaphy or endovascular aneurysm repair (EVAR). We excluded patients with thoracoabdominal aortic aneurysms or undergoing other operations. The Human Research Ethics Committee of Thammasat University (Medicine) reviewed and approved this study with waived written informed consent. (IRB No. MTU-EC-AN-0-345/64; 10th Feb 2023)

Data collection

The medical records of patients were reviewed, including age, gender, body mass index (BMI), The American Society of Anesthesiologists (ASA) physical status classification, medical history of cardiovascular diseases (hypertension, diabetic mellitus, congestive heart failure, arrhythmia, chronic kidney disease (defined by GFR<60)[10], peripheral arterial disease,

chronic obstructive pulmonary disease), smoking habits, preoperative medication, and aneurysm morphology.

Perioperative details were collected, as well as the type of operation, choice of anesthesia, type of AAA repair, perioperative laboratory examination, intraoperative hemodynamic parameters, use of vasopressor or inotropic drugs, intraoperative arterial blood gas, change of serum Cr level before and within 48 hours after surgery, operative time, intraoperative fluid, intraoperative urine output, blood loss, length of hospital stay, and perioperative mortality.

Outcomes

The main outcome of this study was perioperative AKI, defined as KDIGO Stage 1 criteria, which involved an abrupt rise in serum creatinine (Cr) of≥ 0.3 mg/dL within 48 hours after surgery[10-12], among patients undergoing non-ruptured infrarenal AAA repair.

Statistical analysis

Baseline characteristics and demographic data, operative details, length of hospital stay, and peri-operative mortality were assessed in AKI and non-AKI groups. Mean and standard deviation (SD) with independent t-test analysis was performed in normal distribution group. Non-parametric test was performed by Mann-Whitney test and reported in median with interquartile range (IQR). The relation between intraoperative hemodynamic parameters and creatinine change was planned to be assessed by Pearson correlation analysis. The non-parametric or non-bivariate normality was assessed by Spearman Rank correlation coefficient. The equality of distribution and bivariate normality was evaluated by the Shapiro-Wilk W test and Doornik–Hansen omnibus tests, respectively. In correlated data, the change in the magnitude of 1 variable is associated with a change in the magnitude of another variable, either in the same (positive correlation) or in the opposite (negative correlation) direction.

The linear relation between intraoperative hemodynamic parameters and creatinine change was demonstrated in scatter plots and Linear prediction with 95% confidence interval (CI) plots. All statistical analyses were performed using a STATA/SE 16.0 for Windows (Stata Corp LP, TX, USA), and p-values < 0.05 indicated statistical significance. The study process and report followed the strengthening of the reporting of observational studies in epidemiology (STROBE) statement in reports of cohort studies (Figure 1).

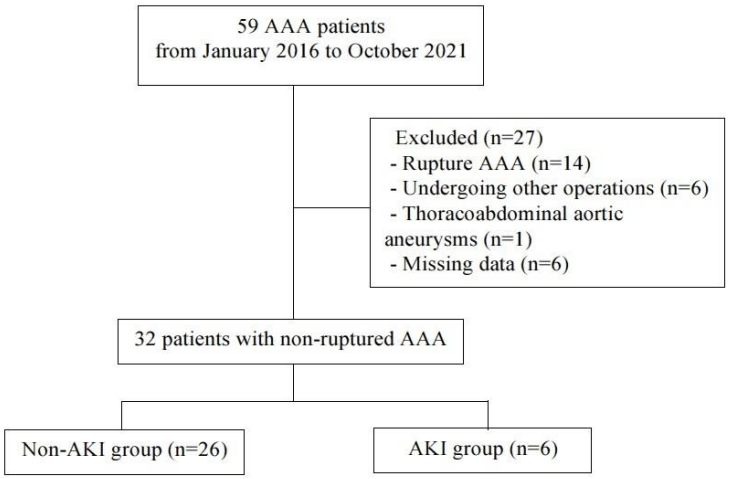

Figure 1 STROBE diagram

Sample size calculation

A type I error (α) and a type II error (β-error) determine at 0.2 and 0.05, respectively. The 80% of power calculation demonstrated the total number (N) of subjects by creatinine change at 30 with 0.25 of n2: n1[13]. Thus, the subject requires 24 patients in non-AKI and 6 in AKI group. Our study reviews the total number of patients who underwent non-ruptured infrarenal AAA repair at Thammasat University Hospital between January 2016 and October 2021 is 32 (26 patients in non-AKI and 6 in AKI group).

Results

Over five-year period, there were 59 patients diagnosed with AAA. After excluding 27 patients with ruptured AAA, undergoing other operations, thoracoabdominal aortic aneurysm, and missing data, a total of 32 eligible patients divided into 26 patients in non-AKI and six patients in AKI group were examined (Figure1).

Demographic data

Of all patients, 16 patients were male (61.54%). The mean age was 71.81, and BMI was 21.85 kg/m^2. 71.88% and 25.0% of patients were categorized in ASA physical status class III and IV, respectively. Most patients had hypertension (81.25%), while forty percent of patients had chronic kidney disease and were smoking. More than half took beta blockers (59.38%), followed by 40.62% of patients who took calcium channel blockers and statin. The majority of all patients had infrarenal AAA (75%); however, most patients in AKI group had juxtarenal AAA (83.33%). (Table 1)

Table 1 Baseline demographic data between AKI and non-AKI groups

	Non-AKI (n=26)	AKI (n=6)	Total (n=32)	*p*-value
Male (N(%))	16 (61.54%)	5 (83.33%)	21 (65.62%)	0.311
Female (N(%))	10 (38.46%)	1 (16.67%)	11 (34.38%)	0.115
Age (years) (mean ± SD)	72.84 ± 6.58	67.33 ± 11.00	71.81 ± 7.69	0.971
BMI (kg/m^2) (mean ± SD)	21.83 ± 3.66	21.89 ± 2.49	21.85 ± 3.43	
***ASA physical status* (N(%))**				
ASA class 1	0	0	0	0.793
ASA class 2	1 (3.85%)	0	1 (3.12%)	
ASA class 3	19 (73.08%)	4 (66.67%)	23 (71.88%)	
ASA class 4	6 (23.08%)	2 (33.33%)	8 (25.00%)	
ASA class 5	0	0	0	
***Underlying disease* (N(%))**				
HTN	22 (84.62%)	4 (66.67%)	26 (81.25%)	0.310
DM	3 (11.54%)	2 (33.33%)	5 (15.62%)	0.185
CHF	1 (3.85%)	0	1 (3.12%)	0.625
CKD	8 (30.7%)	5 (83.3%)	13 (40.6%)	0.018*
CAD	5 (19.23%)	1 (16.67%)	6 (18.75%)	0.885
Arrhythmia	3 (11.54%)	0	3 (9.38%)	0.382
PAD	1 (3.85%)	0	1 (3.12%)	0.625
COPD	6 (23.08%)	0	6 (18.75%)	0.192
Smoking	9 (34.62%)	4 (66.67%)	13 (40.62%)	0.150
***Pre-operative medication*(N(%))**				
B-blocker	15 (57.69%)	4 (66.67%)	19 (59.38%)	0.687
Calcium channel blocker	9 (34.62%)	4 (66.67%)	13 (40.62%)	0.150
Statin	12 (46.15%)	1 (16.67%)	13 (40.62%)	0.185
Aspirin	7 (26.92%)	2 (33.33%)	9 (28.12%)	0.753
Clopidogrel	0	1 (16.6%)	1 (3.1%)	0.034*
Anticoagulant	2 (7.69%)	0	2 (6.25%)	0.483
Aneurysm morphology				
Aortic neck				
- Infrarenal (N(%))	23 (88.46%)	3 (11.54%)	24 (75%)	< 0.001*
- Juxtarenal (N(%))	1 (16.67%)	5 (83.33%)	8 (25%)	0.874
Maximal diameter of aortic aneurysm (mm) (mean ± SD)	5.83 ± 1.10	5.75 ± 1.47	5.81 ± 1.15	

* Statistically significant at P < 0.05; *AKI, acute kidney injury; BMI body mass index; HTN, hypertension; DM, diabetes mellitus; CHF, congestive heart failure; CKD, chronic kidney disease; CAD, coronary artery disease; PAD, peripheral arterial disease; COP, chronic obstructive pulmonary disease*

Table 2 Operative detail and outcomes between AKI and non-AKI groups

	Non-AKI (n=26)	AKI (n=6)	Total (n=32)	*p*-value
Operative detail				
Type of operation				0.732
Elective operation (N(%))	20 (76.92%)	5 (83.33%)	25 (78.12%)	
Emergency operation (N(%))	6 (23.08%)	1 (16.67%)	7 (21.88%)	
Choice of anesthesia				
General anesthesia (N(%))	19 (73.08%)	4 (66.67%)	23 (71.88%)	0.753
Spinal anesthesia (N(%))	7 (26.92%)	2 (33.33%)	9 (28.12%)	
Type of AAA repair				0.732
Open repair (N(%))	11 (42.31%)	3 (50.00%)	14 (43.75%)	
EVAR (N(%))	15 (57.69%)	3 (50.00%)	18 (56.25%)	
Operative time (minutes) (mean ± SD)	333.84 ± 162.29	439.83 ± 117.58	353.71 ± 158.76	0.143
Intraoperative fluid monitoring				
UOP per hour < 0.5 ml/kg/hr (N(%))	4 (15.3%)	5 (83.3%)	9 (28.1%)	0.001*
UOP / kg / hour (mean ± SD)	1.69 ± 1.16	0.29 ± 0.29	1.43 ± 1.19	0.007*
Estimated blood loss (mean ± SD)	878.07 ± 693.40	1778.33 ± 698.58	1046.87 ± 770.66	0.007*
Estimated blood loss (median, iqr)	800 (300, 1200)	1950 (1570, 2200)	900 (350, 1535)	0.010*,†
Positive fluid balance (mean ± SD)	2523.72 ± 2142.09	2809.3 ± 2795.15	2577.27 ± 2230.12	0.782
Positive fluid balance (median, iqr)	2317 (584, 4286)	2691 (2071, 3459)	2421 (714, 4235)	0.846†
Intraoperative vasopressor or inotropic drug	7 (26.92%)	1 (16.67%)	8 (25.00%)	0.601
Laboratory result				
Preoperative				
Hb (mean ± SD)	11.83 ± 1.72	10.76 ± 1.88	11.63 ± 1.77	0.186
Cr (mean ± SD)	1.29 ± 0.88	1.78 ± 1.83	1.38 ± 1.10	0.334
Cr (median, iqr)	1.08 (0.9, 1.4)	1.14 (0.83, 1.4)	1.08 (0.86, 1.4)	0.980†
K (mean ± SD)	3.90 ± 0.35	4.21 ± 0.47	3.96 ± 0.39	0.075
Postoperative				
Hb (mean ± SD)	10..83 ± 1.42	9.66 ± 2.41	10.61 ± 1.66	0.125
Cr‡ (mean ± SD)	1.19 ± 0.84	2.41 ± 2.17	1.41 ± 1.25	0.029*
Cr‡ (median, iqr)	1 (0.8, 1.3)	1.69 (1.4, 1.7)	1.11 (0.80, 1.43	0.004*,†
K (mean ± SD)	3.90 ± 0.45	4.46 ± 0.69	4.01 ± 0.54	0.020*
Intraoperative ABG				
pH (mean ± SD)	7.33 ± 0.06	7.25 ± 0.06	7.31 ± 0.07	0.014*
PaO_2 (mean ± SD)	223.59 ± 64.04	182.73 ± 86.53	215.93 ± 69.12	0.196
$PaCO_2$ (mean ± SD)	40.59 ± 4.84	37.83 ± 6.64	40.07 ± 5.22	0.248
HCO_3 (mean ± SD)	21.52 ± 2.59	16.41 ± 3.43	20.56 ± 3.38	<0.001*
Base excess (mean ± SD)	-4.76 ± 3.32	-13.36 ± 4.14	-6.37 ± 4.83	<0.001*
Base excess (median, iqr)	-6.64 (-6.64, -3)	-14.25 (-16.4, -8.9)	-6.64 (-7.85, -4)	<0.001*,†
Peri-op death (N(%))	0	1 (16.6%)	1 (3.1%)	0.034*
Length of hospital stay (days) (mean ± SD)	20.42 ± 19.24	20.50 ± 11.48	20.43 ± 17.88	0.992
Length of hospital stay (days) (median, iqr)	16.5 (7, 23)	21 (14, 30)	16.5 (7, 28)	0.628†

* Statistically significant at P < 0.05; † Mann Whitney U test for non-parametric data; ‡ Peak values collection within 48 hours

AKI, acute kidney injury; iqr, interquartile range; SD, standard deviation; ASA, American Society of Anesthesiologist; AAA, abdominal aortic aneurysm; EVAR, endovascular aneurysm repair; UOP, urine output; Hb, hemoglobin; Cr, creatinine; K, potassium; ABG, arterial blood gas

Table 3 Multivariate regression analysis demonstrated the association between AKI and peri-operative factors in patients with non-ruptured infrarenal AAA repair.

Factors	RR	95% CI	*p*-value
CKD	3.31	0.51 - 21.43	0.207
Clopidogrel	0.89	0.73 - 7.68	0.751
Juxtarenal AAA	3.21	0.55 - 18.60	0.193
UOP < 0.5 ml/kg/min	11.5	1.49 - 88.27	0.019*
Peri-op death	N/A	N/A	N/A
	Co-efficiency		
UOP (ml/kg/hr)	-0.605	-0.74 - 0.72	0.134
Estimate blood loss	0.06	-0.64 - 1.78	0.937
Post-operative Cr level	-0.32	-0.63 - 0.18	0.922
Post-operative K level	0.694	-0.05 - 8.49	0.775
pH	0.688	-2.56 - 4.92	0.447
HCO_3	0.721	-0.46 - 1.11	0.138
Base excess	-0.857	-0.99 - 0.98	0.986

RR, relative risk; CI, confidence interval; CKD, chronic kidney disease; AAA, abdominal aortic aneurysm; UOP, urine output; Cr, creatinine; K, potassium; N/A: not applicable due to low number of events,

* Statistically significant at $p < 0.0$

Table 4 Intraoperative blood pressure parameters between AKI and non-AKI groups

	Non-AKI (n=26)	AKI (n=6)	Total (n=32)	*p*-value
SBP before induction	135.80 ± 17.41	149.83 ± 20.84	138.43 ± 18.58	0.096
DBP before induction	71.84 ± 10.80	81.16 ± 12.48	73.59 ± 11.53	0.073
MAP before induction	93.16 ± 11.64	104.05 ± 14.89	95.20 ± 12.79	0.059
SBP before clampling/deployed	107.69 ± 8.48	109.5 ± 11.41	108.03 ± 8.92	0.662
DBP before clampling/deployed	59.80 ± 3.54	60.5 ± 3.20	59.93 ± 3.44	0.664
MAP before clampling/deployed	75.76 ± 4.39	76.83 ± 3.80	75.96 ± 4.25	0.589
SBP after clampling/deployed	109.92 ± 10.51	105.66 ± 9.47	109.12 ± 10.31	0.371
DBP after clampling/deployed	56.38 ± 3.82	55.5 ± 5.50	56.21 ± 4.10	0.641
MAP after clampling/deployed	74.23 ± 5.379	72.22 ± 5.93	73.85 ± 5.44	0.424
ΔSBP clamp	2.23 ± 15.95	-3.83 ± 19.31	1.09 ± 16.46	0.425
ΔDBP clamp	-3.42 ± 6.40	-5 ± 6.32	-3.71 ± 6.32	0.590
ΔMAP clamp	-1.53 ± 7.95	-4.61 ± 9.02	-2.11 ± 8.10	0.411
Lowest SBP	86.65 ± 10.59	83.33 ± 10.32	86.03 ± 10.46	0.492
	Non-AKI (n=26)	**AKI (n=6)**	**Total (n=32)**	***p*-value**
Lowest DBP	43 ± 8	42.5 ± 4.18	42.90 ± 7.38	0.884
Lowest MAP	57.55 ± 7.65	56.11 ± 4.17	57.28 ± 7.09	0.661
Highest SBP	158.84 ± 18.61	155 ± 16.43	158.12 ± 18.03	0.645
Highest DBP	85.07 ± 11.88	80.83 ± 14.28	84.28 ± 12.23	0.452
Highest MAP	109.66 ± 12.60	105.55 ± 14.24	108.89 ± 12.7	0.486
ΔSBP	72.19 ± 16.12	71.66 ± 25.62	72.09 ± 17.76	0.949
ΔDBP	42.07 ± 14.51	38.33 ± 12.9	41.37 ± 14.10	0.566
ΔMAP	52.11 ± 13.22	49.44 ± 16.58	51.61 ± 13.66	0.673
SBP after declamping/deployed	105.38 ± 6.31	83.33 ± 10.32	101.25 ± 11.21	<0.001*

DBP after declamping/deployed	56.38 ± 3.07	42.5 ± 4.18	53.78 ± 6.38	<0.001*
MAP after declamping/deployed	72.71 ± 3.53	56.11 ± 4.17	69.6 ± 7.49	<0.001*
SBP declamping/deployed	4.53 ± 6.83	22.33 ± 12.40	7.87 ± 10.59	< 0.001*,†
ΔDBP declamping/deployed	0 ± 2.28	13 ± 4.56	2.43 ± 5.84	< 0.001*,†
ΔMAP declamping/deployed	1.51 ± 2.75	16.1 ± 5.64	4.25 ± 6.69	< 0.001*,†
MAP decreased ≥ 20 mmHg‡	0	4 (66.67%)	4 (12.50%)	< 0.001*
MAP < 65 mmHg‡	0	6(100%)	6 (18.75%)	< 0.001*
ΔDeclamp MAP percentage (%)	1.84 ± 3.63	21.99 ± 7.03	5.62 ± 9.08	< 0.001*,†

All parameters presented as mean ± SD; except ‡ presented as N(%)

* Statistically significant at $p < 0.05$; † Mann Whitney U test for non-parametric data;

Perioperative details and outcomes

Most patients were scheduled for elective surgery (78.12%). There were 14 patients (43.75%) in open aneurysmorrhaphy and 18 (56.25%) in EVAR. 71.88% and 28.12% were general and spinal anesthesia, respectively. The mean (SD) operation time was 354 minutes (159), with a median (interquartile range, IQR) estimated blood loss of 900 mL (350, 1535) and a median (IQR) positive fluid balance was 2421 mL (714, 4235). A quarter of patients received intraoperative vasopressor or inotropic drugs. The median (IQR) of perioperative Cr change was 1.11 (0.80, 1.43). The median length of hospital stay was 16.5 days (7, 28). Perioperative death was reported in only one patient with AKI (3.1%).

Association between perioperative factors and AKI

Peri-operative urine output < 0.5 ml/kg/min was associated with AKI in patients with non-ruptured infrarenal AAA repair (RR 11.5, 95%CI 1.49-88.27; *p*-value = 0.019). Other factors included CKD, clopidogrel, juxtarenal AAA, perioperative death, blood loss, postoperative Cr level, post-operative potassium level, pH, HCO_3, and base excess were not associated with AKI (Table 3).

Intraoperative Hemodynamic Parameters and AKI

After de-clamping or deployment, the mean (SD) of systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were significantly different between non-AKI and AKI groups, as shown in Table 4. Immediate de-clamping or deployment DBP and MAP were significantly related with perioperative Cr level raising by moderated negative correlation (Spearman's rho (ρ): *p*-value = -0.434: 0.013 and -0.414:0.018, respectively) (Figure 2). De-clamping hypotension by SBP, DBP, MAP, and decreasing MAP after de-clamping or deployment were significantly related with perioperative Cr level raising by moderated positive correlation (ρ: *p*-value = 0.471:0.006, 0.609:<0.001, 0.612:<0.001 and 0.615:<0.001, respectively) (Figure 3-4).

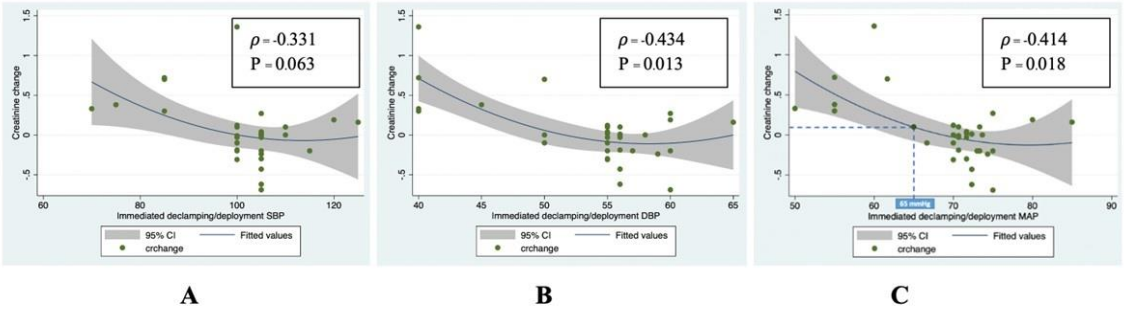

Figure 2 Spearman rank correlation graphs showing the relationship of negative effect (creatinine change) and immediate de-clamping/ deployment SBP (A), de-clamping/deployment DBP (B), and de-clamping/deployment MAP (C). Spearman's rho (ρ), SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; Cr, creatinine

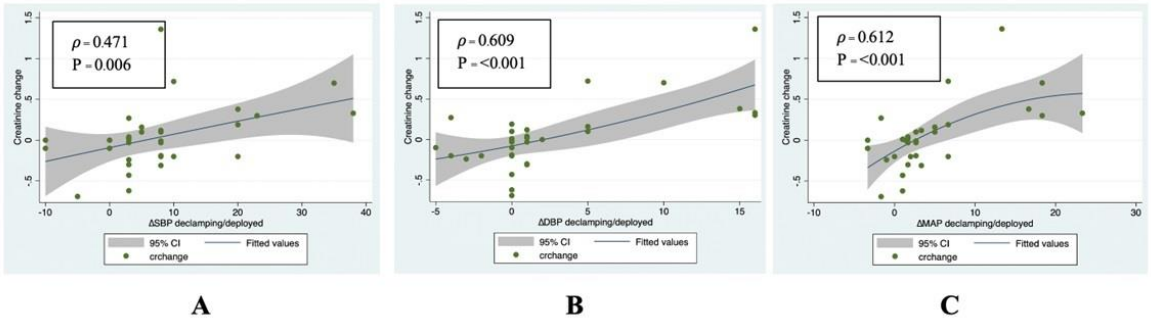

Figure 3 Spearman rank correlation graphs showing the relationship of positive effect (creatinine change) and Δ SBP de-clamping/ deployment (A), Δ DBP de-clamping/deployment (B), and Δ MAP de-clamping/deployment (C). Spearman's rho (ρ), SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; Cr, creatinine

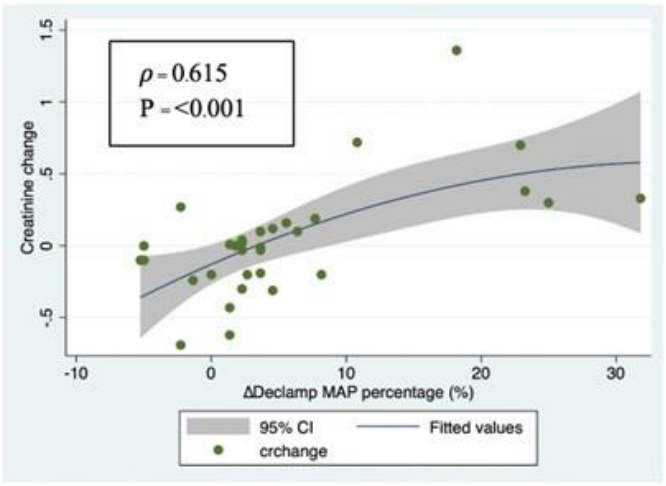

Figure 4 Spearman rank correlation graph showing the relationship of positive effect (creatinine change) and Δ de-clamping/ deployment MAP percentage (%). Spearman's rho (ρ), MAP, mean arterial pressure; Cr, creatinine

Discussion

Our study found two main results. First, changes in perioperative creatinine, reflecting kidney function, were closely linked to intraoperative blood pressure, especially after declamping or device deployment. Second, urine output below 0.5 ml/kg/hr during surgery was a strong risk factor for acute kidney injury, defined as a creatinine increase of at least 0.3 mg/dL. Lower diastolic and mean arterial pressures after declamping or deployment were moderately associated with higher creatinine levels, while low blood pressure episodes were also linked to greater increases in creatinine. These findings underscore the importance of monitoring and managing blood pressure after declamping or deployment to prevent creatinine increases and reduce the risk of acute kidney injury.

During aortic de-clamping, there is a hemodynamic response which is hypotension from reactive hyperemia in tissues and organs distal to the clamp and the resultant relative central hypovolemia, accumulation of vasoactive and cardio-depressant mediators from ischemic tissues, including myocardial-depressant factors, humoral factors, endotoxins, and cytokines[14-15]. Factors affected by decreasing renal blood flow, including hypotension and low cardiac output, were predictive factors for AKI[4,8,16]. In our study, de-clamping/ deployment hypotension significantly correlated with Cr level. The raising of Cr is significantly higher in AKI than non-AKI group and associated with perioperative AKI. In concordance with Rihan et al.[17] illustrated that elevation of serum Cr was associated with acute renal failure in patients undergoing percutaneous coronary intervention (PCI). As well as some studies reported that AKI was found in patients with postoperative increasing plasma Cr and decreased GFR [4,8,16]. Intraoperative urine output per hour was also less in AKI compared to non-AKI groups, but not associated with postoperative AKI in our study. Although, previous studies reported conflicting results regarding the association between intraoperative urine output and postoperative AKI. The reason why oliguria is not associated with postoperative AKI could be from physiologic response to surgery by reducing urine output [18,19]. In contrast, studies demonstrated that intraoperative oliguria could predict postoperative AKI, and factors that potentially impact those studies' results may be from the definition of intraoperative oliguria of less than 0.3 ml/kg/hr, longer duration of intraoperative oliguria for more than 120 minutes, which allow more

predictive ability for postoperative AKI and certain types of surgeries[14].

We found statistically significant correlations in hemodynamics ($p < 0.05$). These results may have implications for clinical practice. The correlation coefficients (ρ = 0.418 to 0.615) indicate moderate to strong associations. The AKI group was small, with a mean Cr of 2.41 ± 2.17 mg/dL. Only 6 patients (18.75%) met the KDIGO Stage 1 AKI criteria. It is still not clear if the hemodynamic thresholds we found actually lead to meaningful outcomes, such as needing dialysis, staying in the hospital longer, or an increased risk of death, based on our data. The value of targeting specific blood pressure levels during declamping or deployment needs to be confirmed, particularly by focusing on outcomes that matter to patients, rather than just changes in creatinine levels. Our findings suggest associations worth further study; however, these results do not have immediate clinical implications, as they do not yet provide sufficient evidence to warrant changes in practice.

Our studies results have shown benefits to the role of the anesthesiologist to prevent de-clamping hypotension, which is correlated with perioperative AKI and urine output by appropriate administration of intraoperative fluid and vasoactive drugs, correction of preoperative fluid deficits, maintenance of intraoperative fluid requirements, and replacement of blood loss be accomplished before de-clamping. Vasodilators, if used, should be gradually reduced and discontinued before unclamping[15].

This is the first study focusing on intraoperative hemodynamic parameters related to AKI in patients undergoing non-ruptured infrarenal AAA repair. Nonetheless, there are still some limitations. First, this was a retrospective study conducted at a single institution and limited the generalizability of the results with small sample size and insufficient events-per-variable ratio for stable regression modeling. Second, we did not follow up with patients for long-term renal dysfunction beyond the 30-day postoperative period. Lastly, the absence of contrast media data (volume, type, timing) in EVAR patients limits our ability to account for this potential confounder, as we cannot control for its possible impact on outcomes. Our small sample size precluded adequately powered subgroup analyses.

Conclusion

Peri-operative urine output < 0.5 ml/kg/min was a risk factor of perioperative AKI. Immediate post-declamping /deployment DBP and MAP were inversely

correlated with perioperative creatinine elevation, while greater post-declamping /deployment hypotension (SBP, DBP, MAP) and larger MAP decreases correlated positively with creatinine rise. These findings identify peri-declamping hemodynamic instability as a key correlate of renal dysfunction in non-ruptured infrarenal AAA repair. This result is a pilot data for further large multicenter prospective cohort to confirm this result.

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References

1. Reimerink JJ, van der Laan MJ, Koelemay MJ, Balm R, Legemate DA. Systematic review and meta-analysis of population-based mortality from ruptured abdominal aortic aneurysm. Br J Surg. 2013;100(11):1405-13.
2. Dang T, Dakour-Aridi H, Rizwan M, Nejim B, Malas MB. Predictors of acute kidney injury after infrarenal abdominal aortic aneurysm repair in octogenarians. J Vasc Surg. 2019;69(3):752-762.e1.
3. Abelha FJ, Botelho M, Fernandes V, Barros H. Determinants of postoperative acute kidney injury. Crit Care. 2009;13(3):R79.
4. Tang Y, Chen J, Huang K, Luo D, Liang P, Feng M, et al. The incidence, risk factors and in-hospital mortality of acute kidney injury in patients after abdominal aortic aneurysm repair surgery. BMC Nephrol. 2017;18(1):184.
5. Zettervall SL, Ultee KHJ, Soden PA, Deery SE, Shean KE, Pothof AB, et al. Predictors of renal dysfunction after endovascular and open repair of abdominal aortic aneurysms. J Vasc Surg. 2017;65(4):991-6.
6. Saratzis A, Melas N, Mahmood A, Sarafidis P. Incidence of Acute Kidney Injury (AKI) after Endovascular Abdominal Aortic Aneurysm Repair (EVAR) and Impact on Outcome. Eur J Vasc Endovasc Surg. 2015;49(5):534-40.
7. Nonaka T, Kimura N, Hori D, Sasabuchi Y, Nakano M, Yuri K, et al. Predictors of Acute Kidney Injury Following Elective Open and Endovascular Aortic Repair for Abdominal Aortic Aneurysm. Ann Vasc Dis. 2018;11(3):298-305.
8. Dariane C, Coscas R, Boulitrop C, Javerliat I, Vilaine E, Goeau-Brissonniere O, et al. Acute Kidney Injury after Open Repair of Intact Abdominal Aortic Aneurysms. Ann Vasc Surg. 2017;39:294-300.
9. Walsh M, Devereaux PJ, Garg AX, Kurz A, Turan A, Rodseth RN, et al. Relationship between intraoperative mean arterial pressure and clinical outcomes after noncardiac surgery: toward an empirical definition of hypotension. Anesthesiology. 2013;119(3):507-15.
10. Levey AS. Defining AKD: The Spectrum of AKI, AKD, and CKD. Nephron. 2022;146(3):302-5.

11. Makris K, Spanou L. Acute Kidney Injury: Definition, Pathophysiology and Clinical Phenotypes. Clin Biochem Rev. 2016;37(2):85-98.
12. Gelman S. The pathophysiology of aortic cross-clamping and unclamping. Anesthesiology. 1995;82(4):1026-60.
13. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney Int Suppl. 2012;2:1-138.
14. Kim JT, Kim MJ, Han Y, et al. A new risk-scoring model for predicting 30-day mortality after repair of abdominal aortic aneurysms in the era of endovascular procedures. Ann Surg Treat Res. 2016;90(2):95-100. doi:10.4174/astr.2016.90.2.95
15. Goel N, Jain D, Savlania A, Bansal A. Thoracoabdominal Aortic Aneurysm Repair: What Should the Anaesthetist Know? Turk. J Anaesthesiol Reanim. 2019;47(1):1-11.
16. Tallgren M, Niemi T, Pöyhiä R, Raininko E, Railo M, Salmenperä M, et al. Acute renal injury and dysfunction following elective abdominal aortic surgery. Eur J Vasc Endovasc Surg. 2007;33(5):550-5.
17. Rihal CS, Textor SC, Grill DE, Berger PB, Ting HH, Best PJ, et al. Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. Circulation. 2002;105(19):2259-64.
18. Goren O, Matot I. Perioperative acute kidney injury. Br J Anaesth. 2015;115 Suppl 2:ii3-14.
19. Mendes RS, Suassuna J. Perioperative oliguria: adequate physiological response or risk for acute kidney injury?. J Bras Nefrol. 2021;43(1):1-2.