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ORIGINAL RESEARCH **PERIOPERATIVE MEDICINE**

Renal perfusion pressure and capillary leaks index as risk factors for acute kidney injury after major abdominal surgery

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ABSTRACT

Background & Objective: Many factors can contribute to the development of acute kidney injury (AKI) following major abdominal surgery, including the effect of fluid extravasation into interstitial space due to capillary leakage. Microalbuminuria is also brought on by capillary leakage. Increased central venous pressure (CVP) or intra-abdominal pressure (IAP) will decrease renal filtration according to the degree of pressure transmission to the glomeruli. The objective of this study was to know the correlation of capillary leakage index (CLI), microalbuminuria [urine albumin to creatinine ratio (ACR)] and renal perfusion pressure (RPP) with the incidence of AKI.

Methodology: A prospective longitudinal cohort design with consecutive sampling was used in this study. The parameters capillary leakage index (CLI), microalbuminuria (ACR) and renal perfusion pressure (RPP) were examined preoperatively, and at 12 h and 36 h postoperatively, and the incidence of AKI was observed till the fourth postoperative day. CLI was defined as C-reactive protein (CRP mg/dL) over albumin (g/L) multiplied by 100. RPP as glomerular pressure was obtained from mean arterial pressure (MAP), IAP, and renal venous pressure estimated by CVP, by the formula RPP = MAP-(IAP+CVP).

Results: Following major abdominal surgery, 19 (25.68%) of the 74 subjects developed AKI. There was no significant difference in CLI and ACR between patients with and without AKI. Renal perfusion measurements examined at 0, 12, and 36 h showed significantly lower values in AKI patients. The relative risk (RR) of RPP was 9.125 with a 95 % CI of 1.141293−72.95725 (P = 0.037) after data analysis with Cox Regression to establish the correlation between CLI, ACR, and RPP, as well as covariate variables on the occurrence of AKI at 0 h. Compared to participants without AKI, those with AKI had a mortality risk of 2.384 times higher (P = 0.0351, 95 % CI = 1.133−5.018).

Conclusion: Renal perfusion pressure showed a significant correlation with acute kidney injury. Additionally, there is a strong connection between acute kidney injury and mortality.

Abbreviations: ACR- albumin to creatinine ratio; AKI- acute kidney injury; CLI- capillary leakage index; CVP- central venous pressure; IAP- intra-abdominal pressure; MAP- mean arterial pressure; RPP- renal perfusion pressure

Key word: AKI; Capillary Leaks Index; Microalbuminuria; Renal perfusion pressure; Major abdominal surgery; Systemic capillary leak syndrome

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1. INTRODUCTION

The pathophysiology of postoperative acute kidney injury (AKI) is multifactorial based on the interaction of various events, including renal hypoperfusion, intrarenal vasoconstriction, inflammation, oxidative stress, ischemic reperfusion injury, and administration of nephrotoxic drugs.[1] Several risk factors for AKI in patients with major abdominal surgery include old age, diabetes mellitus, hypertension, cardiovascular disease, cardiorespiratory disorders, type and duration of surgery, intra- and post-operative fluid overload, use of vasoactive drugs, and intra-operative erythrocyte transfusion.[2] Liberal administration of fluids could harm kidney function, resulting in alveolar capillary edema, endothelial and glycocalyx damage, and increased capillary permeability and leakage. Also, it causes a fluid shift from the circulation to the interstitium.[3] Capillary leakage causes albuminuria.[4] Central venous pressure (CVP) is also associated with AKI in critically ill and cardiac surgery patients, as it is associated with renal swelling, which significantly reduces renal perfusion pressure (RPP = MAP − CVP).[5]

It is currently well accepted that increased intra-abdominal pressure (IAP) is an independent predictor of AKI after major abdominal surgery.[6] Microalbuminuria is also brought on by capillary leakage. Following the level of pressure transmitted to the glomeruli, a rise in CVP and IAP will reduce renal filtration. Considering that the incidence of postoperative AKI is multifactorial, this study focused on the effect of the extravasation of fluid into the interstitium caused by capillary leakage. This study aimed to investigate the correlation between capillary leakage index (CLI), microalbuminuria [urine albumin to creatinine ratio (ACR)], and renal perfusion pressure (RPP) as predictors of AKI in patients undergoing major abdominal surgery.

2. METHODOLOGY

In this prospective longitudinal cohort study, patients between 18 and 65 y, who undergoing major abdominal surgery under general anesthesia from August to December 2021 and who stayed in the hospital for at least four days were sequentially sampled. After receiving approval from the ethics committee, the study was carried out in the operating theatre, intensive care unit (ICU), and surgical ward at West Nusa Tenggara General Hospital in Mataram, Indonesia. Informed consent was obtained from all study subjects. Patients with a diagnosis of idiopathic systemic capillary leak syndrome (SCLS), patients on renal replacement therapy, patients with burns, menstruating or pregnant women, anuria, macroscopic hematuria, patients with chronic kidney disease, and patients who required inotropes or vasopressors were excluded from the study. We used an Excel-based method to collect data that included demographic data, perioperative information, the Sequential Organ Failure Assessment (SOFA) score, and research information, such as AKI occurrence up to four days following surgery. The preoperative serum creatinine was utilized as the baseline serum creatinine, and AKI criteria based on The KDIGO were applied.

CLI, ACR, and RPP were measured preoperatively, and at 12 and 36 h postoperatively. The AKI incidence was observed until the fourth day after surgery. CLI was calculated by comparing albumin and CRP after being multiplied by 100. RPP as effective glomerular pressure was obtained from MAP, IAP, and renal venous pressure estimated by CVP (RPP = MAP−(IAP+CVP)). IAP measures were performed by general practitioners using a urine catheter according to the WSAC (The Abdominal Compartment Society) Guidelines. At expiration's end, CVP readings were done. Thus, all pressures are presented in mmHg (1 mmHg = 1.36 cmH_2O).

Statistical Analysis

After submitting the research form, the data were processed using Strata version 15.0. The data are reported as mean ± standard deviation (SD) if it has a normal distribution. Data that is categorical is presented as a percentage. Bivariate analysis using the chi-square test for categorical variables and homogeneity between the two groups if $P > 0.05$. The area under the curve (AUC), sensitivity, and specificity values for CLI were calculated using a receiver operating characteristic (ROC) curve. The ratio of urine albumin to creatinine (ACR) with excretion above 30 mg/g was used to identify microalbuminuria. Additionally, the RPP value of 40 mmHg from Kopitko's research was chosen as the cut-off RPP. Chi-square analysis for categorical data and Mann-Whitney for numerical variables were performed in bivariate analysis for independent and dependent variables. The difference was determined to be statistically significant if $P < 0.05$.

3. RESULTS

Seventy-six patients underwent major abdominal surgery during the study period. Two were not included in the analysis because one died before the 12 h record and one was discharged from the hospital before the four-day observation period. Tables 1−3 display demographic information, and perioperative information, and study results, including comparisons between the AKI and no AKI groups. During the perioperative period, AKI patients had a lower induction SpO_2 compared to those without AKI (97.84% ± 3.28 vs. 99.05% ± 0.45; P= 0.16), and a higher pulse rate during surgery (94.53 ± 20.90 vs. 86.96 ± 9.76; P= 0.026). In comparison to patients no AKI, subjects with AKI had

Table 1: Laboratory Parameters, Hemodynamics, and Incidence of AKI

Variable	AKI	Without AKI	P-value
Hemoglobin (g/dL)	12.06 ± 2.42	13.31 ± 13.71	NS
Urea (mg/dL)	26.72 ± 14.87	31.85 ± 19.63	NS
Creatinine (mg/dL)	0.87 ± 0.25	0.81 ± 0.26	NS
MAP-induction (mmHg)	95.19 ± 7.68	97.96 ± 8.26	NS
Pulse-induction (per min)	94.52 ± 20.63	88.13 ± 8.00	NS
SpO_2-induction (%)	97.84 ± 3.28	99.05 ± 0.49	**0.016**
Operating time (min)	106.84 ± 46.22	123.91 ± 61.93	NS
Anesthesia duration (min)	140.79 ± 55.23	146 ± 62.80	NS
Durante_BP (mmHg)	120.53 ± 12.93	125.73 ± 12.65	NS
Durante_MAP (mmHg)	93.35 ± 8.42	95.30 ± 9.39	NS
Durante_Pulse (mmHg)	94.53 ± 20.90	86.96 ± 7.96	**0.026**
Durante_ SpO_2(%)	98.95 ± 0.85	99.22 ± 2.73	NS
Urine output per h (ml)	164.34 ± 193.11	183.57 ± 179.83	NS
Total urine output (ml)	295.79 ± 358.08	346.36 ± 395.56	NS
RL (ml)	784.21 ± 505.81	816.36 ± 339.81	NS
NaCl (ml)	260.53 ± 335.23	222.73 ± 305.02	NS
Colloids (ml)	342.10 ± 238.78	374.55 ± 217.07	NS

**Independent T-test for normally distributed data; Mann Whitney for data that is not normally distributed*

lower post-operative fluid output (FO) at 24 and 48 h and higher fluid intake (FI) 24 h, fluid balance (FB) 24 and 48 h, and cumulative fluid balance (CFB) 48, 72, and 96 h. Between patients with AKI to those without, there was no significant difference in CLI and ACR.

In AKI patients the MAP at 36 h was significantly lower; central venous pressure (CVP) and intraabdominal pressure (IAP) were higher at 12 and 36 h. Renal perfusion pressure measured at 0, 12, and 36 h was significantly lower in AKI patients. Data analysis using cox-regression to determine the relation between CLI, ACR, and RPP as well as covariate variables on the incidence of AKI at 0 h obtained the relative risk (RR) of RPP was 9.125 with a 95% CI of 1.141293−72.95725 (P = 0.037). AKI patients had a mortality risk of 2.384 times greater (P = 0.0351; 95% CI 1.133−5.018).

4. DISCUSSION

The risk of fluid loss during major surgery is acknowledged at various stages, including pre-operative fasting, perioperative intravascular blood and fluid loss, extravasation of fluid from the vascular compartment (third space effect), insensible fluid loss, and fluid loss due to pathological abnormalities of the disease itself.[7] In any surgery, fluid administration is the most common intervention. Improper fluid administration can cause side effects and even contribute to death.[8] This fluid excess was independently associated with impaired organ function, intra-abdominal hypertension (IAH), capillary leakage, and worse outcome.[9] In this study, the CLI values preoperatively, and at 12 h, and 36 h postoperative had high values when compared to baseline in both subjects with and without AKI. The CLI values obtained were different from those of Cordemans and Moguel's studies, which both used critically ill patients, while this study used patients after major abdominal surgery with a dominant biliary malignancy (head of pancreas cancer). The CLI as an objective index of capillary leakage rate made by Cordemans is determined by the ratio of CRP in milligrams per deciliter, divided by albumin levels in grams per liter multiplied by 100. It is assumed that if there is an increase in CRP levels due to a systemic inflammatory process coupled with a decrease in albumin levels (hypoalbuminemia), there will be an increase in capillary permeability.[9] The subjects in this study had low albumin levels that were below average in both the AKI and without AKI groups, which resulted in a higher CLI value. The low albumin level in patients with malignancy occurs because the systemic inflammatory process suppresses albumin production in hepatocytes by producing cytokines.[10] Malignant conditions accompanied by the surgical process and the

Table 2. Laboratory and hemodynamic parameters per-hour measurement and the incidence of AKI

Variable	Measurement	AKI	Without AKI	P-value
CRP (mg/dL)	0 h	58.315 ± 44.609	55.89 ± 48.879	NS
	12 h	74.894 ± 39.355	79.309 ± 43.036	NS
	36 h	105.33 ± 25.606	109.22 ± 22.46	NS
Albumin (g/L)	0 h	2.815 ± 0.522	2.898 ± 0.579	NS
	12 h	2.542 ± 0.732	2.774 ± 0.500	NS
	36 h	2.641 ± 0.638	2.725 ± 0.428	NS
CLI	0 h	154.83 (13.89-521.74)	136.11 (11.62 – 571.43)	NS
	12 h	320 (54.54-571.43)	342.31 (15.15-631.58)	NS
	36 h	428.57 (214.28-631.58)	444.44 (88.23-750)	NS
MAP (mmHg)	0 h	93.78 ± 7.697	97.23±9.04	NS
	12 h	90.754 ± 12.189	94.581 ± 6.582	NS
	36 h	89.67 ± 7.37	95.938 ± 6.528	**0.002***
IAP (mmHg)	0 h	10.50 ± 8.66	8.609 ± 4.16	NS
	12 h	13.499 ± 16.339	7.283 ± 9.511	**0.048***
	36 h	9.10 ± 9.04	5.659 ± 2.60	**0.019***
CVP (mmHg)	0 h	10.100 ± 5.47	8.843 ± 2.92	NS
	12 h	8.026 ± 6.131	5.474 ± 2.549	**0.015***
	36 h	8.964 ± 6.97	5.970 ± 2.276	**0.010***
RPP (mmHg)	0 h	72.293 ± 12.40	79.958 ± 11.12	**0.014***
	12 h	72.467 ± 14.743	80.964 ± 12.649	**0.019***
	36 h	77.597 ± 9.837	85.179 ± 8.252	**0.006***

Data presented as Mean ± SD or Median (min−Max)

Table 3: Postoperative fluid balance and AKI

Variable	Measurement	AKI	Without AKI	P-value
FI (ml)	24 h	2315.78 ± 448.78	2194.18 ± 1428.39	**0.031***
	48 h	2183.33 ± 382.50	2092.778 ± 344.32	NS
	72 h	1785.714 ± 694.048	2141.85 ± 411.12	NS
	96 h	2259.286 ± 278.801	2210.18 ± 451.65	NS
FO (ml)	Ke-24	1468.68 ± 797.47	1960.405 ± 450.739	**0.006***
	Ke-48	1388 ± 756.74	1907.13 ± 314.633	**0.007***
	Ke-72	1785.71 ± 694.04	1816.48 ± 1706.40	NS
	Ke-96	1803.57 ± 385.53	1849.07 ± 411.22	NS
FB (ml)	Ke-24	847.105 ± 835.55	71.636 ± 411.687	**< 0.001***
	Ke-48	795.33 ± 605.60	223.611 ± 276.98	**< 0.001***
	Ke-72	539.287 ± 755.283	334.629 ± 334.171	NS
	Ke-96	541.428 ± 445.85	331.48 ± 474.54	NS
CFB (ml)	Ke-48	1381.667 ± 922.68	293.429 ± 579.63	**< 0.001***
	Ke-72	1791.07 ± 1384.10	611.389 ± 747.49	**0.005***
	Ke-96	2421.07 ± 1469.58	926.20 ± 1046.64	**< 0.001***

**Wilcoxon test; Data presented as mean ± SD; *P is significant*

Table 4: CLI, ACR, and RPP, as well as covariate variable 0 h on the incidence of AKI

Variable	Coefficient	Standard Error	RR	CI 95%	P-value
RPP	3.052	1.461	21.156	1.206−371.13	**0.037**
CLI	1.332	0.792	3.790	0.801−17.935	0.093
ACR	0.679	0.823	1.972	0.392−9.906	0.410
Sex	−0.422	0.618	0.655	0.195−2.205	0.495
Age	0.086	0.305	1.090	0.598−1.984	0.778
Type of surgery	−0.131	0.557	0.876	0.293−2.617	0.814
Comorbids					
- Diabetes	−35.966	4.60	2.400	−	1.000
- Cancer	−1.353	0.868	0.258	0.047−1.416	0.119
- Liver disease	−35.968	4.770	2.390	−	1.000
Sepsis	0.935	0.611	2.549	0.768−8.459	0.126
Ketorolac	0.015	0.026	1.015	0.964−1.070	0.556
Paracetamol	−0.009	0.005	0.999	0.997−1.002	0.123
Tramadol	0.107	0.006	1.011	0.999−1.022	0.071
Transfusion	0.052	0.638	1.053	0.301−3.681	0.935

Cox Regression, P = 0.05

administration of excess fluids will aggravate the capillary leakage.

Albumin is the main serum protein larger than the pores of the glomerular filtration membrane and is typically not filtered by healthy kidneys. The presence of albumin in the urine indicates glomerular pathology, and its presence in large amounts in the urine indicates impaired glomerular basement membrane integrity.[11] It is not surprising that early urine ACR was revealed as an independent prognostic factor for postoperative AKI as albuminuria occurs due to endothelial dysfunction.[12]

This study obtained ACR at 0 and 12 h, with AKI incidence not statistically significant. However, the RR values obtained were 1.261 (P = 0.104; 95% CI 1.003−1.586) and 1.211 (P = 0.102; 95% CI 1.017−1.444), indicating that the 0 and 12-h ACR were > 30, which were likely to be a risk factor for AKI. The findings are consistent with a prospective cohort study by Marouli et al. in adult patients undergoing elective major abdominal surgery who examined the ratio of urine albumin to creatinine (urine ACR) over a period and found that those with urine ACR > 30 mg/g had a fivefold greater risk of developing AKI.[12]

Acute circulatory failure in the ICU is the leading cause of renal failure because decreased cardiac output and mean arterial pressure (MAP) can reduce renal blood flow (RBF) and harm the kidneys.[13] This study utilized the formula RPP = MAP−(IAP+CVP). The RPP value at each measurement at 0, 12, and 36 h showed significant differences between subjects with AKI and without AKI, which was obtained in subjects with AKI having lower RPP values. The incidence of AKI and the RPP at every h of measurement were not found to be statistically significant in the bivariate analysis using chi-square. However, analyzing the relationship between CLI, ACR, and RPP, as well as the covariate variable on the incidence of AKI at h 0 in multivariate analysis using cox regression, showed the RR value of the RPP variable was 9.125, indicating that RPP < 40 is a risk factor for the occurrence of AKI. The results were nearly identical to those of Kopitko et al., who found lower MAP and RPP in the AKI group with decreased eRPP at 6 and 12 h. In the Kopitko study, the significant difference in 12-h eRPP was not due to changes in arterial blood supply because MAP remained unchanged but rather due to venous congestion factors (IAP, CVP, Pmean), which were shown to be independent of each other.[14] In this study, MAP, IAP, and CVP all impacted the RPP value. This study discovered a significant difference in the 36th h MAP, where subjects with AKI had a lower MAP. IAP and CVP at 12 and 36 h also showed significant differences, where subjects with AKI obtained higher IAP and CVP values than subjects without AKI. Numerous studies have demonstrated that elevated CVP adversely affects renal perfusion.[15] CVP is also associated with an increased incidence of AKI in critical illness and cardiac surgery patients because it produces significant renal edema and lowers perfusion pressure to the kidneys (RPP = MAP−CVP).[5] In the kidney, increased intra-abdominal pressure through the

accumulation of ascitic fluid can impede renal venous outflow, restricting renal blood supply, oxygen delivery, and general functional integrity of the nephron unit. Therefore, decreased perfusion and increased venous pressure, which may occur separately or act in synchrony and enhance each other's effects, may also contribute to poor renal perfusion and function rather than just heart pump failure.[6] Increased intra-abdominal pressure (IAH-Intraabdominal Hypertension) is also associated with organ dysfunction. The kidneys appear particularly susceptible to IAH, and renal failure is one of the organ dysfunctions consistently associated with increased IAH.[16] Those with increased IAP have decreased urine output, which is assumed that the decrease in urine output that occurs due to an increase in IAP is caused by a decrease in cardiac output due to reduced blood return.[17]

5. LIMITATIONS

One of the independent variables in this study, RPP used a cut-off from previous studies with slightly different characteristics of the subject.

6. CONCLUSION

There was a significant correlation between subjects with renal perfusion pressure values less than 40 of having AKI, and the risk of having AKI was 9.125 times. The risk of mortality was 2,384 times more in subjects with AKI than in those without AKI, but there was no correlation between capillary leakage index and urine albumin to creatinine ratio (ACR) to AKI.

7. Data availability

The numerical data related to the study is available with the authors on request.

8. Conflict of interest

The authors reported no potential conflicts of interest.

9. Funding

The study did not involve any external or industry funding, and it was completed with institutional help.

10. Authors' contribution

ASM, Study concept, prepare the manuscript and manuscript editing

DA: Manuscript editing, Final approval of the version to be published

AHM: Data collection, literature search, Manuscript editing

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