



Relationship Between Platelet-to-Lymphocyte Count Ratio and Acute Kidney Injury Following Surgery of Stanford Type A Aorta Dissection

Xiaoyan Ding^{1,2} · Yuanxiang Chen^{2,3} · Xiao'e Zheng^{1,2} · Lijuan Zeng^{1,2} · Xiaofen Zhou^{1,2}

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Abstract

Objective The aim of this study was to evaluate the correlation between the hematological index of platelet to lymphocyte ratio (PLR) and the occurrence of acute kidney injury (AKI) following surgery of Stanford acute type A aorta dissection (ATAAD).

Methods We analyzed the perioperative data of patients with ATAAD who were treated surgically in Fujian Provincial Hospital from December 2016 to December 2021.

Results Patients were divided into AKI (n = 66) and non-AKI (n = 36) groups based on the Kidney Disease: Improving Global Outcomes (KDIGO). There was no difference in preoperative PLR values (T0 PLR). In addition, the PLR within 24 h (T1 PLR) following surgery was higher in the AKI group than in the non-AKI group ($P = 0.001$). Reintubation rates, intensive care unit (ICU) stay, and 30-day mortality were significantly different in the AKI group ($P = 0.004$, $P = 0.002$ and $P = 0.002$). Multivariate analyses showed that higher body mass index (BMI) and reduced T1 PLR were the risk factors for postoperative AKI. Receiver operating characteristic (ROC) curve analysis revealed that the decline in the cut-off level of T1 PLR predicted AKI: 144.6, the area under the curve (AUC): [95% CI] 0.7146, 0.6112–0.8181, and $P = 0.0004$, with a sensitivity of 58.33% and a specificity of 77.27%.

Conclusions This retrospective study demonstrated that a decreased T1 PLR after surgery is a risk factor for the occurrence of postoperative AKI in ATAAD patients and has possible predictive value for AKI.

Keywords Acute aorta dissection · Acute kidney injury · PLR

1 Background

Stanford acute type A aortic dissection (ATAAD) is a life-threatening cardiovascular disease and the most frequent dissection with high mortality. Even with immediate surgical intervention, ATAAD mortality remained high [1]. Moreover, the most common complication is acute kidney injury (AKI) which is one of Cardiac surgery-associated acute kidney injury (CSV-AKI). The reported incidence of AKI after ATAAD surgery is 18–65% [2, 3]. The pathophysiology of CSV-AKI is very complex and probably includes renal ischaemia–reperfusion injury, inflammation, oxidative stress, haemolysis and nephrotoxins [4]. As we know, ATAAD can induce a systemic inflammatory response, which leads to multiple organ damage and thus, influence prognosis [5]. After acute aortic dissection, mechanical damage to the aortic wall stimulates neutrophil, chemokine and granulocyte colony-stimulating factor expression, inducing neutrophil proliferation; a large number of neutrophils

✉ Xiaofen Zhou
zhouxiaofen888@126.com

Xiaoyan Ding
dingxy6601@163.com

Yuanxiang Chen
306697308@qq.com

Xiao'e Zheng
917928256@qq.com

Lijuan Zeng
zlj20220119@163.com

¹ The Fourth Department of Intensive Care Unit, Fujian Provincial Hospital, Fuzhou 350001, China

² Provincial Clinical Medical College of Fujian Medical University, Fuzhou 350001, China

³ Department of Cardiac Surgery, Fujian Provincial Hospital, Fuzhou 350001, China

are recruited to the dissected vascular wall, releasing IL-6 and MMP-9, thereby promoting the outer membrane inflammatory reaction process [6–8]. Platelet to lymphocyte ratio (PLR) as a novel inflammatory biomarker has been widely studied and considered one of the predictors of acute exacerbations of chronic obstructive pulmonary disease (AECOPD), arteriosclerotic heart disease (AHF), acute coronary syndromes (ACS), sepsis, and renal disease prognosis [9–13]. However, to the best of our knowledge, no research has examined the relationship between PLR and the incidence of AKI following ATAAD surgery. Therefore, we hypothesize that PLR can predict the incidence of AKI.

2 Methods

All patients were diagnosed by preoperative thoracoabdominal aortic angiography (CTA) examination. Inclusion criteria were as follows: ① age ≥ 18 years; ② surgical treatment; ③ need for in-patient intensive care unit (ICU) monitoring and treatment; and ④ no chronic renal insufficiency or required renal replacement therapy (RRT) before this visit. Exclusion criteria were as follows: ① age < 18 years; ② intraoperative death. We conducted a retrospective study, which neither interfered with patients' treatment plans nor brought risks to patients' physiology and collected only clinical data. We protected patient confidentiality and applied for an exemption from informed consent. The flowchart of the research process is shown in Fig. 1.

2.1 Data Collection

The demographic data of the patients included: gender, age, body mass index (BMI), smoking status, underlying

diseases such as diabetes and hypertension. Preoperative clinical assessment included whether there was a combination of shock and renal artery tear, and whether the surgical status was emergency or elective. Intraoperative indices included duration of surgery, the duration of cardiopulmonary bypass (CBP) and aortic cross-clamping time (AOT) and the deep hypothermic circulatory arrest (DHCA) time. Laboratory indicators comprised of preoperative lymphocyte and platelet levels (T0 PLR) and the preoperative level of creatinine clearance (T0 CCR), Perioperative renal function insufficient, postoperative within 24 h lowest platelet to lymphocyte ratio value (T1 PLR), and serum albumin levels. Postoperative data comprised the length of ICU stay, reintubation rate, needs for RRT, and 30-day mortality. The worst value was selected if more than one outcome was available.

2.2 Definitions

This study classified patients by their highest sCr levels within seven days after surgery. Furthermore, AKI was diagnosed according to the 2012 Kidney Disease: Improving Global Outcomes (KDIGO) criteria as follows, stage 1: sCr increases by 0.3 mg/dL or 1.5–1.9 times the preoperative value within 48 h; stage 2: sCr increases by 2.0–2.9 times the preoperative value; stage 3: sCr increases by greater than or equal to 3.0 times the preoperative value or 4.0 mg/dL or RRT is initiated. The last highest sCr before surgery was used as a baseline value, and all AKI stages based on KDIGO criteria were analyzed.

Hypoalbumin refers to postoperative serum albumin levels below 30 g/L.

2.3 Statistical Analysis

The statistical software SPSS 23.0 and GraphPad Prism 9.3.0 were used to analyze the data. The mean \pm standard deviation was used for measurement data conforming to a normal distribution, and the independent sample t-test was used to compare groups. Furthermore, nonparametric continuous variables were expressed as median (interquartile range) [M(IQR)], and the rank sum test was used for comparison between groups; the count data were expressed as [n (%)], and the χ^2 test was used. The variables with statistically significant differences were analyzed by univariate analysis, and the risk factors for the occurrence of postoperative AKI were analyzed by multivariate logistic regression analysis. Receiver operating characteristic (ROC) curves were plotted to calculate the sensitivity and specificity of PLR in assessing patient prognosis and the value of PLR in evaluating the occurrence of AKI in ATAAD patients. $P < 0.05$ represented a statistical significant difference.

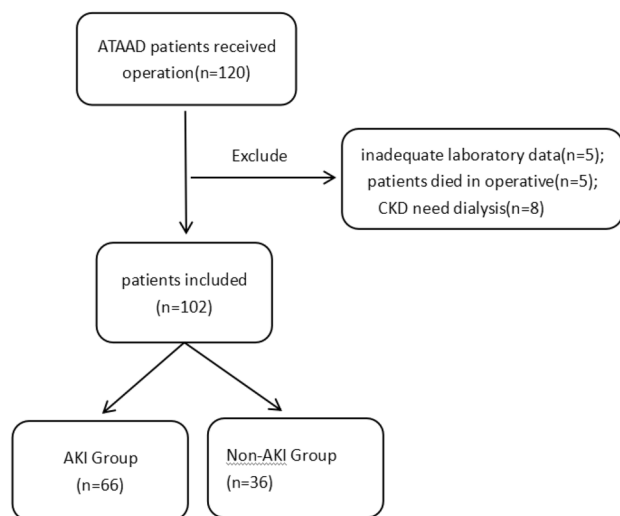


Fig. 1 Flowchart of patient inclusion

3 Results

A total of 120 ATAAD patients who underwent surgery in our center between December 2016 and December 2021 were included. However, 15 patients (12.5%) were excluded due to inadequate laboratory data, 5 patients (4.17%) died in operation, and 8 patients (6.67%) were excluded because of preoperative chronic kidney disease necessitating dialysis. Finally, 102 patients including 75 males were enrolled in this study. The demographic features and preoperative blood parameters of the study subjects are reported in Table 1. Medical histories included hypertension (62.70%), diabetes (5.88%), and Marfan syndrome (11.77%). Overall, 59 patients (57.84%) underwent emergency surgery. Of the 102 patients, 66 (64.71%) exhibited AKI, and 19 (18.63%) AKI patients required RRT. Univariate analysis of the preoperative platelet counts (T0 platelet), lymphocyte counts (T0 lymphocyte), and PLR (T0 PLR) revealed no statistical difference ($P > 0.05$), and the intraoperative duration of the CPB, AOT, duration of surgery time, DHCA time and red blood cell infusion between the two groups were not different ($P > 0.05$). The PLR within 24 h (T1 PLR) and BMI were different ($P < 0.05$).

The length of ICU stays, reintubation rates, and need for RRT in the AKI group were significantly higher than that of the non-AKI group ($P < 0.05$). Myasthenia, postoperative coma, and total hospital stay were not statistically significant ($P > 0.05$) (Table 2).

Multivariable regression analysis was utilized to assess the risk factors of AKI in ATAAD patients after surgery. Indicators with $P < 0.2$ from the univariate analysis were included in the multifactorial regression analysis. Analysis showed that BMI (odds ratio 1.187; 95% CI 1.009–1.396, $P = 0.038$) and T1 PLR (odds ratio 0.996; 95% CI 0.993–1.000, $P = 0.035$) were risk factors for postoperative AKI in ATAAD patients after surgery (Fig. 2).

ROC curves were utilized to evaluate the predictive efficacy of T1 PLR for postoperative AKI in ATAAD patients. Results showed that the area under the curve (AUC) of AKI following ATAAD predicted by T1 PLR was 0.7146 (95% CI 0.6112–0.8181, $P = 0.0004$) with a sensitivity of 58.33% and a specificity of 77.27% (Fig. 3).

Perioperative renal function insufficient: The preoperative serum creatinine exceeded the upper limit

BMI body mass index, *T0* preoperative time, *T0 PLR* preoperative platelet to lymphocyte ratio, *AKI* acute kidney injury, *Non-AKI* none acute kidney injury, *T0 CCR* the preoperative level of creatinine clearance

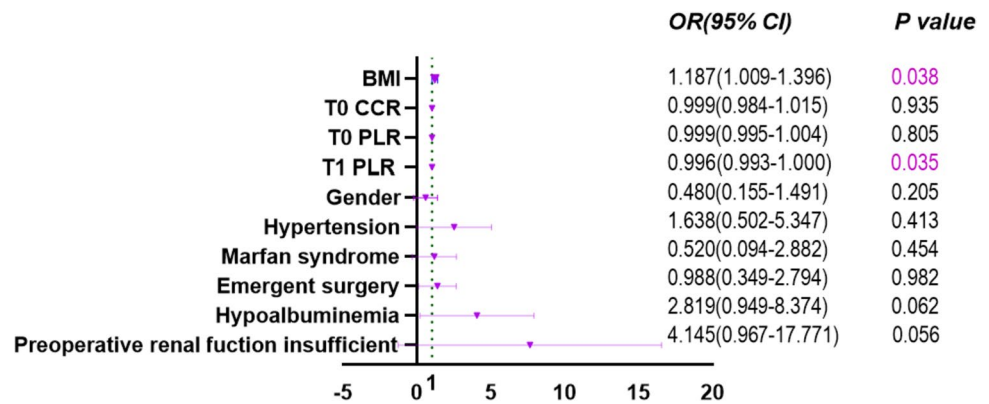
Table 1 Baseline characteristics of study population

Characteristics	AKI Group (N=66)	Non-AKI Group (N=36)	<i>t/z/x</i> ²	<i>P</i>
Age (years) (mean ± sd)	54.45 ± 11.8	53.22 ± 13.09	<i>t</i> = 0.485	0.629
Male/female, n/n	53/13	22/14	<i>x</i> ² = 4.408	0.036
Height (m) (mean ± sd)	1.69 ± 0.07	1.69 ± 0.08	<i>t</i> = -0.207	0.836
Weight (kg) (mean ± sd)	72.97 ± 14.65	67.61 ± 12.33	<i>t</i> = 1.864	0.065
BMI (kg/m ²) (mean ± sd)	25.52 ± 4.45	23.52 ± 3.20	<i>t</i> = 2.377	0.019
Smoking, n (%)	14 (21.2%)	7 (19.4%)	<i>x</i> ² = 0.045	0.833
Hypertension, n (%)	46 (69.7%)	18 (50%)	<i>x</i> ² = 3.866	0.049
Diabetes mellitus, n (%)	4 (6.06%)	2 (5.56%)	<i>x</i> ² = 0.000	1.000
Marfan syndrome, n (%)	5 (7.6%)	7 (19.44%)	<i>x</i> ² = 2.121	0.145
T0 Lymphocyte (10 ⁹ /L) (M(IQR))	1.5 (1.5)	1.50 (1.2)	<i>z</i> = -1.143	0.253
T0 Platelet (10 ⁹ /L) (M(IQR))	198.50 (81.00)	186.50 (53.00)	<i>z</i> = -0.144	0.886
T0 PLR (10 ⁹ /L) (M(IQR))	127.89 (112.97)	144.14 (132.49)	<i>z</i> = -1.372	0.170
AOT(min)(mean ± sd)	167.17 ± 49.10	178.72 ± 45.45	<i>t</i> = -1.165	0.247
Duration of surgery (min) (mean ± sd)	583.69 ± 216.143	516.67 ± 90.738	<i>t</i> = 0.504	0.628
CPB (min) (M(IQR))	282.50 (91.00)	285.00 (85.00)	<i>t</i> = 0.508	0.785
Perioperative renal function insufficient, n (%)	27 (40.91%)	4 (11.1%)	<i>x</i> ² = 9.777	0.002
T0 CCR (ml/min) (M(IQR))	98.86 (49.42)	74.15 (46.70)	<i>z</i> = -2.339	0.019
Shock preoperative, n (%)	9 (13.6%)	2 (5.6%)	<i>x</i> ² = 1.581	0.209
Renal artery tear, n (%)	19 (28.8%)	11 (30.6%)	<i>x</i> ² = 0.035	0.851
Emergent surgery, n (%)	42 (63.6%)	17 (47.2%)	<i>x</i> ² = 2.574	0.109

Table 2 Intraoperative and postoperative outcomes of patients with and without AKI after surgery for ATAAD

Characteristics	AKI group (N=66)	Non-AKI Group (N=36)	$t/z/x^2$	<i>P</i>
T1 Lymphocyte ($10^9/L$) (M(IQR))	0.8 (0.7)	0.60 (0.40)	$z = -2.966$	0.003
T1 Platelet ($10^9/L$) (M(IQR))	69.5 (59.00)	105.50 (74.00)	$z = -3.235$	0.001
T1 PLR ($10^9/L$) (M(IQR))	87.35 (101.13)	184.00 (222.28)	$z = -3.571$	0.001
Hypoalbumin, n (%)	16 (24.24)	45 (68.18)	$\chi^2 = 5.460$	0.019
RBC infusion intraoperative (ml) (M(IQR))	480 (470)	480 (480)	$z = -0.741$	0.459
DHCA (min) (M(IQR))	35 (26)	38 (22)	$z = -0.084$	0.933
30 day-mortality, n (%)	22 (33.3%)	2 (5.6%)	$\chi^2 = 9.989$	0.002
Coma postoperative, n (%)	15 (23.1)	1 (2.8)	$\chi^2 = 7.161$	0.007
Reintubation, n (%)	13 (20%)	0 (0)	$\chi^2 = 8.264$	0.004
Myasthenia, n (%)	18 (27.3%)	4 (11.1%)	$\chi^2 = 3.597$	0.058
ICU-stay (h) (M(IQR))	176.63 (228.21)	88.79 (81.46)	$z = -3.053$	0.002
RRT needs, n (%)	19 (28.8%)	0 (0)	$\chi^2 = 10.908$	0.001
Hospital-stay(hour) (M(IQR))	19.5 (21)	23.5 (15)	$z = -1.734$	0.083

AOT aortic cross-clamping time, CPB cardiopulmonary bypass, DHCA deep hypothermia circulatory arrest, Hypoalbumin postoperative albumin level < 30 g/L, RBC red blood cell, RRT renal replacement therapy, T1 postoperative within 24 h, T1 PLR postoperative within 24 h lowest platelet to lymphocyte ratio value

Fig. 2 Forest plot for multivariable analysis of patient related risk factors for AKI following surgery for ATAAD

4 Discussion

AKI is one of the most common complications after cardiac extracorporeal circulation procedures resulting in prolonged hospitalization and increased mortality. The occurrence of AKI after cardiac surgery is 40.6–52.7% [3, 14, 15]. In this study, the incidence of AKI after ATAAD operation was 64.7%, but the number of patients who needed RRT treatment was 19 (18.6%), consistent with Tian Yu Zhou's study [16]. However, AKI prevalence was 64.7% in our study, which is relatively high because we included patients of all stages based on the KIDGO criteria, including those with renal failure and requiring RRT.

Platelets have a pro-inflammatory effect and can release self-stored pro-inflammatory factors after platelet activation. In recent years, a lot of studies have confirmed that platelets participate in inflammatory reactions by the

release of IL-6 and IL-1 after platelet activation. They induce the expression of monocytes, promote their adhesion to endothelial cells, and synthesize TNF- and IL-6. This suggests that platelet activation is involved in the systemic inflammatory response process of aortic dissection [17, 18].

Numerous studies have explored the pathophysiology of cardiac surgery-associated acute kidney injury, which include inflammation [19], oxidative stress, ischemia-reperfusion injury, surgical trauma, blood exposed to the artificial surface of CPB circuit, etc. [20, 21]. A reduction in platelet counts is observed after CPB [22]. Studies have shown that decreased platelet counts increases the risk of AKI [23, 24]. In our study, we found that the postoperative early PLR were significantly low in patients with AKI. Multivariate logistic regression analysis showed that the postoperative reduced PLR within 24 h is a risk factor for the occurrence of AKI in ATAAD patients, which could be used as a predictor of AKI

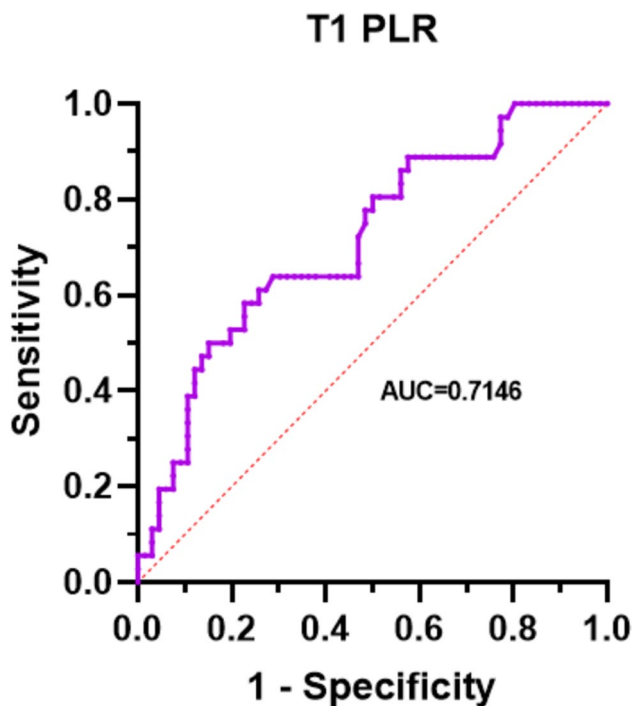


Fig. 3 ROC curve and AUC (area under the curve) for T1 PLR to predict AKI (T1 PLR cut-off level: 0.3257, AUC 0.7146, 95% CI 0.6112–0.8181, $P=0.0004$, sensitivity 58.33%, specificity 77.27%)

in ATAAD patients after surgery. The ROC curve analysis suggested that T1 PLR can predict the incidence of AKI in ATAAD patients after surgery. Chen-Fei Zheng observed that both low and high PLR were increased the 30-day and 90-day mortality [25]. As different design of the study protocol, we did not classify PLR into low and high PLR, and did not study their correlation with the occurrence of postoperative AKI. We found that the T1 PLR in the AKI group was lower than that in the non-AKI group, and T1 PLR had some predictive value for the occurrence of AKI. There are no differences in T0 PLR between the two groups. Hakan Parlar found that a greater inflammatory response is triggered in patients who will develop AKI in later days after surgery [23]. Maybe the inflammation reaction had not reached its peak, the inflammation indicators had no predictive value at preoperative. Moreover, the different surgical method selection and postoperative monitoring measures, can result in conflicting results.

Based on multivariate logistic regression analysis, Higher BMI was a contributor to AKI development. Higher BMI was associated with AKI. In fact, several studies have shown that obesity is associated with a high incidence and severity of AKI [26, 27]. However, the exact mechanism remains unclear. Some studies reported that the expression of inflammatory cytokines, including C-reactive protein (CRP), interleukin (IL)-1 β , IL-6, white blood count (WBC) and tumor

necrosis factor- α (TNF- α) in acute aorta dissection (AAD) patients remarkably increased in obese patients compared with non-obese AAD patients [28, 29]. Obesity play a role in the production of reactive oxygen species and oxidative stress [30]. It is reasonable to assume that patients with a high inflammatory state are more susceptible to AKI due to oxidative stress. In the future, more targeted and prospective studies such as dynamic monitoring of changes in inflammatory mediators in obese patients and changes in inflammatory mediators when AKI occurs, are needed to validate our hypothesis.

In the multivariate analysis, several variables which were associated with AKI in the univariate analysis were not significant. Gender, hypertension, T0 PLR, AND hypoalbumin were not risk factors. At the same time, we found that the duration of CPB and AOT is not an independent risk factors for AKI, which is in contrast to other studies [7, 12, 13, 31, 32]. The conflicting research results may be due to differences in cardiopulmonary bypass and surgical techniques. In our study, the duration of cardiopulmonary bypass time was 282.50 (91.00) min, and the aortic occlusion time was 167.17 \pm 49.10 min, which were longer than other study [33, 34]. Furthermore, different results may also be obtained due to the choice of population and sample size.

5 Limitations

There are some limitations to our study. First, due to the difference in individual medical treatment, we could not acquire the real baseline sCr values of the patients; instead, we used admission sCr as the baseline value, which might have underestimated the incidence of AKI. Secondly, this is a single-center retrospective study with small sample size. Consequently, a larger sample size and prospective randomized trials are required to confirm the association between the prevalence of AKI and PLR levels.

6 Conclusions

This retrospective study verified that decreased T1 PLR after surgery was associated with the occurrence of postoperative AKI in ATAAD patients. Therefore, it has some predictive value for AKI. Moreover, PLR could be a useful parameter for describing systemic inflammation.

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Author Contributions XD made a substantial contribution to the concept or design of the work and drafted the article; YC revised the article critically for important intellectual content; XZ and LZ contributed to

the data collection and data analysis; XZ approved the version to be published.

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Data Availability The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Conflict of Interests The Authors declare that there is no conflict of interest.

Ethical Approval The study was also approved by the Ethics Commission of Fujian provincial Hospital (K2021-03–072). The Ethics Commission of Fujian provincial Hospital waived the requirement for obtaining informed consent.

Consent to Participate The data are anonymous, and the requirement for informed consent was therefore waived.

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References

- Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. *Ann Thorac Surg.* 1970;10(3):237–47.
- Palevsky PM, Liu KD, Brophy PD, Chawla LS, Parikh CR, Thakar CV, Tolwani AJ, Waikar SS, Weisbord SD. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for acute kidney injury. *Am J Kidney Dis.* 2013;61(5):649–72.
- Ko T, Higashitani M, Sato A, Uemura Y, Norimatsu T, Mahara K, Takamisawa I, Seki A, Shimizu J, Tobaru T, et al. Impact of acute kidney injury on early to long-term outcomes in patients who underwent surgery for Type A acute aortic dissection. *Am J Cardiol.* 2015;116(3):463–8.
- Wang Y, Bellomo R. Cardiac surgery-associated acute kidney injury: risk factors, pathophysiology and treatment. *Nat Rev Nephrol.* 2017;13(11):697–711.
- Arao K, Fujiwara T, Taniguchi Y, Jinnouchi H, Sasai H, Matsumoto M, Funayama H, Ako J, Momomura S-i: Implications of pentraxin 3 levels in patients with acute aortic dissection. *Heart Vessels.* 2015;30(2):211–7.
- Yuan Z, Lu Y, Wei J, Wu J, Yang J, Cai Z. Abdominal aortic aneurysm: roles of inflammatory cells. *Front Immunol.* 2020;11:609161.
- Li H, Bai S, Ao Q, Wang X, Tian X, Li X, Tong H, Hou W, Fan J. Modulation of immune-inflammatory responses in abdominal aortic aneurysm: emerging molecular targets. *J Immunol Res.* 2018;2018:7213760.
- Anzai A, Shimoda M, Endo J, Kohno T, Katsumata Y, Matsuhashi T, Yamamoto T, Ito K, Yan X, Shirakawa K, et al. Adventitial CXCL1/G-CSF expression in response to acute aortic dissection triggers local neutrophil recruitment and activation leading to aortic rupture. *Circ Res.* 2015;116(4):612–23.
- Goh BKP, Chok A-Y, Allen JC, Quek R, Teo MCC, Chow PKH, Chung AYP, Ong H-S, Wong W-K. Blood neutrophil-to-lymphocyte and platelet-to-lymphocyte ratios are independent prognostic factors for surgically resected gastrointestinal stromal tumors. *Surgery.* 2016;159(4):1146–56.
- Yao C, Liu X, Tang Z. Prognostic role of neutrophil-lymphocyte ratio and platelet-lymphocyte ratio for hospital mortality in patients with AECOPD. *Int J Chron Obstruct Pulmon Dis.* 2017;12:2285–90.
- Ye G-L, Chen Q, Chen X, Liu Y-Y, Yin T-T, Meng Q-H, Liu Y-C, Wei H-Q, Zhou Q-H. The prognostic role of platelet-to-lymphocyte ratio in patients with acute heart failure: a cohort study. *Sci Rep.* 2019;9(1):10639.
- Azab B, Shah N, Akerman M, McGinn JT. Value of platelet/lymphocyte ratio as a predictor of all-cause mortality after non-ST-elevation myocardial infarction. *J Thromb Thrombolysis.* 2012;34(3):326–34.
- Shen Y, Huang X, Zhang W. Platelet-to-lymphocyte ratio as a prognostic predictor of mortality for sepsis: interaction effect with disease severity—a retrospective study. *BMJ Open.* 2019;9(1):e022896.
- Tsai H-S, Tsai F-C, Chen Y-C, Wu L-S, Chen S-W, Chu J-J, Lin P-J, Chu P-H. Impact of acute kidney injury on one-year survival after surgery for aortic dissection. *Ann Thorac Surg.* 2012;94(5):1407–12.
- Helgason D, Helgadottir S, Ahlsson A, Gunn J, Hjortdal V, Hansson EC, Jeppsson A, Mennander A, Nozohoor S, Zindovic I, et al. Acute kidney injury after acute repair of Type A aortic dissection. *Ann Thorac Surg.* 2021;111(4):1292–8.
- Zhou T, Li J, Sun Y, Gu J, Zhu K, Wang Y, Lai H, Wang C. Surgical and early outcomes for Type A aortic dissection with preoperative renal dysfunction stratified by estimated glomerular filtration rate. *Eur J Cardiothorac Surg.* 2018;54(5):940–5.
- Zhang S, Qian H, Yang Q, Hu J, Gan C, Meng W. Relationship between the extent of dissection and platelet activation in acute aortic dissection. *J Cardiothorac Surg.* 2015;10:162.
- Qin C, Zhang H, Gu J, Xiao Z, Yang Q, Meng W. Dynamic monitoring of platelet activation and its role in post-dissection inflammation in a canine model of acute Type A aortic dissection. *J Cardiothorac Surg.* 2016;11(1):86.
- Moledina DG, Mansour SG, Jia Y, Obeid W, Thiessen-Philbrook H, Koyner JL, McArthur E, Garg AX, Wilson FP, Shlipak MG, et al. Association of T cell-derived inflammatory cytokines with acute kidney injury and mortality after cardiac surgery. *Kidney International Reports.* 2019;4(12):1689–97.
- O'Neal JB, Shaw AD, Billings FT. Acute kidney injury following cardiac surgery: current understanding and future directions. *Crit Care.* 2016;20(1):187.
- Kramer RS, Herron CR, Groom RC, Brown JR. Acute kidney injury subsequent to cardiac surgery. *J Extra Corpor Technol.* 2015;47(1):16–28.
- Epstein D, Vishnepolsky A, Bolotin G, Atweh N, Bonstein L, Lehavi A. Effect of prolonged hypothermic cardiopulmonary bypass, heparin, and protamine on platelet: a small-group study. *Thorac Cardiovasc Surg.* 2021;69(8):719–22.
- Parlar H, Arikian AA, Özmez A. Dynamic changes in perioperative cellular inflammation and acute kidney injury after coronary artery bypass grafting. *Braz J Cardiovasc Surg.* 2021;36(3):354–64.

24. Hui S, Yu C. Platelet counts, acute kidney injury, and mortality after coronary artery bypass grafting surgery. *Anesthesiology*. 2016;125(2):437–8.
25. Zheng C-F, Liu W-Y, Zeng F-F, Zheng M-H, Shi H-Y, Zhou Y, Pan J-Y. Prognostic value of platelet-to-lymphocyte ratios among critically ill patients with acute kidney injury. *Crit Care*. 2017;21(1):238.
26. Soto GJ, Frank AJ, Christiani DC, Gong MN. Body mass index and acute kidney injury in the acute respiratory distress syndrome. *Crit Care Med*. 2012;40(9):2601–8.
27. Liu T, Fu Y, Liu J, Liu Y, Zhu J, Sun L, Gong M, Dong R, Zhang H. Body mass index is an independent predictor of acute kidney injury after urgent aortic arch surgery for acute DeBakey Type I aortic dissection. *J Cardiothorac Surg*. 2021;16(1):145.
28. Shimizu T, Kimura N, Mieno M, Hori D, Shiraishi M, Tashima Y, Yuri K, Itagaki R, Aizawa K, Kawahito K, et al. Effects of obesity on outcomes of acute Type A aortic dissection repair in Japan. *Circ Rep*. 2020;2(11):639–47.
29. Wu Z, Wang Z, Wu H, Hu R, Ren W, Hu Z, Chang J. Obesity is a risk factor for preoperative hypoxemia in Stanford A acute aortic dissection. *Medicine (Baltimore)*. 2020;99(11): e19186.
30. Orrù G, Storari M, Scano A, Piras V, Taibi R, Viscuso D. Obstructive Sleep Apnea, oxidative stress, inflammation and endothelial dysfunction—an overview of predictive laboratory biomarkers. *Eur Rev Med Pharmacol Sci*. 2020;24(12):6939–48.
31. Vekstein AM, Yerokun BA, Jawitz OK, Doberne JW, Anand J, Karhausen J, Ranney DN, Benrashid E, Wang H, Keenan JE, et al. Does deeper hypothermia reduce the risk of acute kidney injury after circulatory arrest for aortic arch surgery? *Eur J Cardiothorac Surg*. 2021;60(2):314–21.
32. Wang Z, Ge M, Chen T, Chen C, Zong Q, Lu L, Wang D. Acute kidney injury in patients operated on for Type A acute aortic dissection: incidence, risk factors and short-term outcomes. *Interact Cardiovasc Thorac Surg*. 2020;31(5):697–703.
33. Sasabuchi Y, Kimura N, Shiotsuka J, Komuro T, Mouri H, Ohnuma T, Asaka K, Lefor AK, Yasunaga H, Yamaguchi A, et al. Long-term survival in patients with acute kidney injury after acute Type A aortic dissection repair. *Ann Thorac Surg*. 2016;102(6):2003–9.
34. Liu Y, Shang Y, Long D, Yu L. Intraoperative blood transfusion volume is an independent risk factor for postoperative acute kidney injury in Type A acute aortic dissection. *BMC Cardiovasc Disord*. 2020;20(1):446.

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