BMC Nephrology

RESEARCH

Open Access

Association between perioperative platelet distribution width changes and postoperative acute kidney injury in patients with renal insufficiency: a retrospective study

Yiqi Su[1,2,3], Xialian Xu[1,4], Zhe Luo[5], Yi Fang[1,4], Shaomin Gong[1], Jie Teng[1,2,3,4], Xiaoqiang Ding[1,2,3,4*], Jiarui Xu[1*] and Wuhua Jiang[1*]

Abstract

Background Acute kidney injury (AKI) is a major complication following cardiac surgery with a high incidence in those with existing kidney dysfunction. Platelet distribution width (PDW) reflects variability in platelet size and serves as an indicator of platelet activation. Recent investigations linked PDW changes to kidney pathology, suggesting its utility in identifying individuals at risk for AKI, thus necessitating exploration of its predictive value.

Methods Patients with preoperative renal dysfunction [15 ≤ estimated glomerular filtration rate (eGFR) < 60 ml/min/1.73m^2] who underwent cardiac surgery from January 2018 to December 2021 were retrospectively enrolled. PDW values were measured preoperatively and again upon admission to the ICU immediately after cardiac surgery, with the change in PDW (dPDW) defined as the difference between these two measurements. The primary outcome was postoperative AKI, defined base on the Kidney Disease: Improving Global Outcomes (KDIGO) definition and staging criteria. Multivariate regression models were performed to identify the association between dPDW and AKI and its potential trend. Restricted cubic spline analysis assessed non-linear associations between dPDW and AKI. The Youden index identified an optimal dPDW cut-off for AKI prediction. Subgroup analysis was performed to elucidate the consistency of these associations across the various subgroups.

Results AKI occurred in 53.10% (513/966) of patients, accompanied by significant PDW increases in cases of AKI ($P < 0.001$). After adjusting confounders, dPDW was identified as a significant risk factor for AKI [odds ratio (OR) = 1.09, 95% confidence interval (CI): (1.02 ~ 1.16), $P = 0.012$]. Patients in the highest dPDW quartile (Q4) had a 195% higher AKI risk compared to those in the lowest quartile (Q1) (OR = 2.95, 95% CI:1.78 ~ 4.90, $P < 0.001$). Trend analysis indicates that the risk of AKI increased with higher dPDW quartiles (P for trend < 0.001). Youden index showed that dRDW = 1.1

*Correspondence:
Xiaoqiang Ding
ding.xiaoqiang@zs-hospital.sh.cn
Jiarui Xu
xujiarui1984@protonmail.com
Wuhua Jiang
jiang.wuhua@zs-hospital.sh.cn

Full list of author information is available at the end of the article

was identified as the optimal diagnostic cut-off value for AKI. Subgroup analyses and interaction tests showed a robust association between dPDW and AKI in all subgroups (P for interaction > 0.05).

Conclusions This study underscored perioperative PDW changes as a significant predictor of postoperative AKI in patients with renal insufficiency, highlighting its potential in refining risk stratification and management strategies.

Clinical trial number Not applicable for this observational retrospective study.

Keywords Cardiac surgery, Acute kidney injury, Risk factors, Platelet distribution width, Renal function

Introduction

Acute kidney injury (AKI) following cardiac surgery represents a significant clinical concern, affecting approximately 30% of patients undergoing these procedures [1, 2]. The incidence of AKI varies based on the definition and clinical settings, but is notably higher in patients with pre-existing kidney dysfunction. AKI is associated with a marked increase in morbidity and mortality, emphasizing the need for early identification and intervention. The development of AKI following cardiac surgery is multifactorial, involving perioperative hemodynamic instability, ischemia-reperfusion injury, and inflammatory responses [3, 4]. In patients with pre-existing renal impairment, the risk is further exacerbated by their reduced renal reserve and vulnerability to hemodynamic changes [5]. This population, therefore, represents a critical cohort for the application of predictive markers for AKI.

Platelet distribution width (PDW) is an emerging biomarker reflecting platelet size variability and serving as a measure of platelet activation. Recently, PDW has undergone exploration in the context of cardiovascular diseases and inflammatory states [6–10]. A recent study has begun to elucidate its association with renal function, suggesting that changes in PDW may indicate underlying kidney pathology [11]. Elevated PDW levels have been associated with chronic kidney disease (CKD) and are considered indicative of increased platelet activation and inflammation, which are common pathways in renal injury. The potential link between PDW and abnormal kidney function represents a novel area for exploration, particularly in the context of AKI following cardiac surgery.

Cardiac surgery involves a complex interplay of factors influencing PDW. These factors include the use of cardiopulmonary bypass (CPB), known to induce a systemic inflammatory response, alter hemodynamics, and necessitate potential blood product transfusions. Such factors significantly impact platelet function and size distribution, as evidenced by PDW changes [12, 13]. Understanding the relationship between these perioperative factors and PDW alterations is crucial for elucidating the pathophysiology of AKI in this setting.

The predictive value of PDW in the development of AKI following cardiac surgery remains relatively unexplored. Given PDW's association with inflammatory states and its alterations in response to cardiac surgery, it presents as a promising biomarker for early identification of patients at risk for AKI. Early prediction of AKI is essential for implementing strategies to mitigate kidney injury and enhance patient outcomes. This pilot study aimed to investigate PDW's potential role as a predictive marker for AKI in patients undergoing cardiac surgery, especially those with pre-existing kidney dysfunction, due to their diminished renal reserve and increased susceptibility to perioperative hemodynamic changes and inflammatory responses.

Methods

Patients and inclusion/exclusion criteria

This study targeted adult patients with preoperative renal dysfunction [15 ≤ estimated glomerular filtration rate (eGFR) < 60 ml/min/1.73m^2] who underwent elective valve/coronary artery bypass or combined surgery at our hospital between January 2018 and December 2021. Exclusions included patients with prior renal replacement therapy or transplant, preoperative AKI by KDIGO criteria [14], incomplete medical records, death within 48 h of intensive care unit (ICU) admission, and emergent surgeries. The study was approved by the Zhongshan Hospital's Ethics Committee, and written informed consent was obtained from participants to participate in the study.

Design

This retrospective study extracted clinical data from electronic medical records, which included demographics, existing health conditions, cardiac function, including left ventricular ejection fraction (LVEF) and N-terminal pro-B-type natriuretic peptid (NT-proBNP), laboratory results, surgery parameters, CPB duration, bleeding severity (according to Bleeding Academic Research Consortium grades), postoperative medications, including vasopressors, blood transfusion, urine volume, length of hospital stay, and mortality rates. The eGFR calculation utilized the CKD-EPI equation, using preoperative serum creatinine (SCr) levels—the most recent measurement before the surgery. SCr was monitored daily in the postoperative ICU, and renal function tests were conducted during the initial three days post-ICU, then on alternate

days, up to discharge. All patients were routinely admitted to the ICU immediately after cardiac surgery for postoperative monitoring and care.

The primary outcome of the present study was AKI. Participants were divided into two groups based on the development of postoperative AKI, according to the criteria defined in the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines. This classification enabled an in-depth investigation of the various risk factors contributing to the incidence of AKI following surgery.

PDW is routinely included as part of the common term complete blood count tests conducted in our hospital. In our study, common term complete blood counts were obtained from BD EDTA-K2 samples and analyzed using a Sysmex XN9000 electronic counter. PDW values were collected preoperatively and time of admission to the ICU after cardiac surgery, with the change in PDW (dPDW) defined as the difference between these two measurements. The standard reference range for PDW in our hospital is established between 9.0% and 21.0%. All blood samples were processed within 30 min of collection to minimize potential changes due to storage effects.

Data analysis was performed using R version 4.3.0. Normally distributed data were depicted as mean ± standard deviation, non-normally distributed continuous variables as medians with interquartile ranges, and categorical variables as counts and percentages. Normality and variance homogeneity assessment employed the Kolmogorov-Smirnov test. The Student t-test and non-parametric tests were used to determine differences in continuous data, while analysis of categorical variables utilized Fisher's exact or chi-square tests. Multivariable logistic regression models, ranging from crude to Model 3, examined the association between dPDW and AKI incidence, adjusting for various confounders. Model 1 was an unadjusted analysis, while Model 2 adjusted for age and gender, while Model 3 also adjusted for surgical type, preoperative eGFR, CPB duration, Body Mass Index (BMI), hypertension, diabetes, preoperative hemoglobin, preoperative platelet count, preoperative albumin, platelet transfusion, preoperative antiplatelet medicine and post-op major bleeding. Trend analyisis was performed to observe potential trending association between dPDW and AKI. Subgroup analyses were conducted to explore the associations between dPDW and AKI among individuals of different sexes, ages, diabetes status, hypertension status, surgical type, CPB duration, baseline renal function, preoperative antiplatelet medication, platelet transfusion or major bleeding. Interaction tests determined the consistency of these associations across the various subgroups. Possible nonlinear relationships between the change in dPDW and AKI were examined by a logistic regression model with restricted cubic splines (RCS). We conducted RCS with 3 knots at the 10th, 50th and 90th centiles to flexibly model the association. The significance threshold was established at $P<0.05$.

Results

Basic characteristics

The flow chart of patient enrollment is depicted in Fig. 1. Our study enrolled 966 individuals with preoperative renal dysfunction, among whom 53.10% ($n=513$) developed AKI subsequent to their surgeries. The mean time to onset of AKI was 2.6 days postoperatively, with 346 (67.4%) cases occurring within the first 3 days after surgery. Forty-nine (5.07%) patients required dialysis postoperatively. Those with AKI exhibited a significantly higher mortality rate than their non-AKI counterparts (4.87% vs. 0.66%, $P<0.001$) and experienced longer hospital stays [15 (12–21) vs. 14 (11–18) days, $P<0.001$]. The AKI subset featured a higher proportion of males, more instances of hypertension, more use of preoperative antiplatelet medication, and more frequent erythrocyte and platelet transfusions. Laboratory evaluations revealed more severe renal dysfunction and lower preoperative hemoglobin and albumin levels in AKI patients. In contrast, pre- and postoperative platelet distribution width did not differ significantly. However, the dPDW was significantly greater in the AKI subset. AKI occurrences were more prevalent among those undergoing combined surgery, with the AKI group also experiencing longer CPB durations (refer to Table 1).

Identifying predictors of AKI: the role of preoperative factors and regression analysis findings

Despite the lack of significant differences in pre- and postoperative PDW values between the AKI and non-AKI groups, the dPDW demonstrated its potential as a predictive marker. The results of the multivariate logistic regressions showed that dPDW was significantly associated with AKI (Table 2). The first model was unadjusted, the second was partially adjusted, and the third was fully adjusted. The fully adjusted model demonstrated an association between dPDW and AKI. With full adjustment, the effect size between dPDW as a continuous variable and AKI was odds ratio (OR) of 1.09 [95% confidence interval (CI): 1.02 ~ 1.16, $P=0.012$]. Then we classified dPDW into quartiles: Q1 [-20.4, -0.2); Q2 [-0.2,0.9); Q3 [0.9,2); Q4 [2,13.4]. Comparing the higher quartile (Quartile 2–4) to the reference (Quartile 1), Model 3 showed a significant association between dPDW and AKI. This trend was consistent across quartiles (P for trend <0.001), with each increase in quartile showing an elevated risk of AKI. (refer to Table 2).

Based on the Youden index for each coordinate of the ROC curve, dRDW = 1.1 was identified as the optimal diagnostic cut-off value for AKI, with a sensitivity

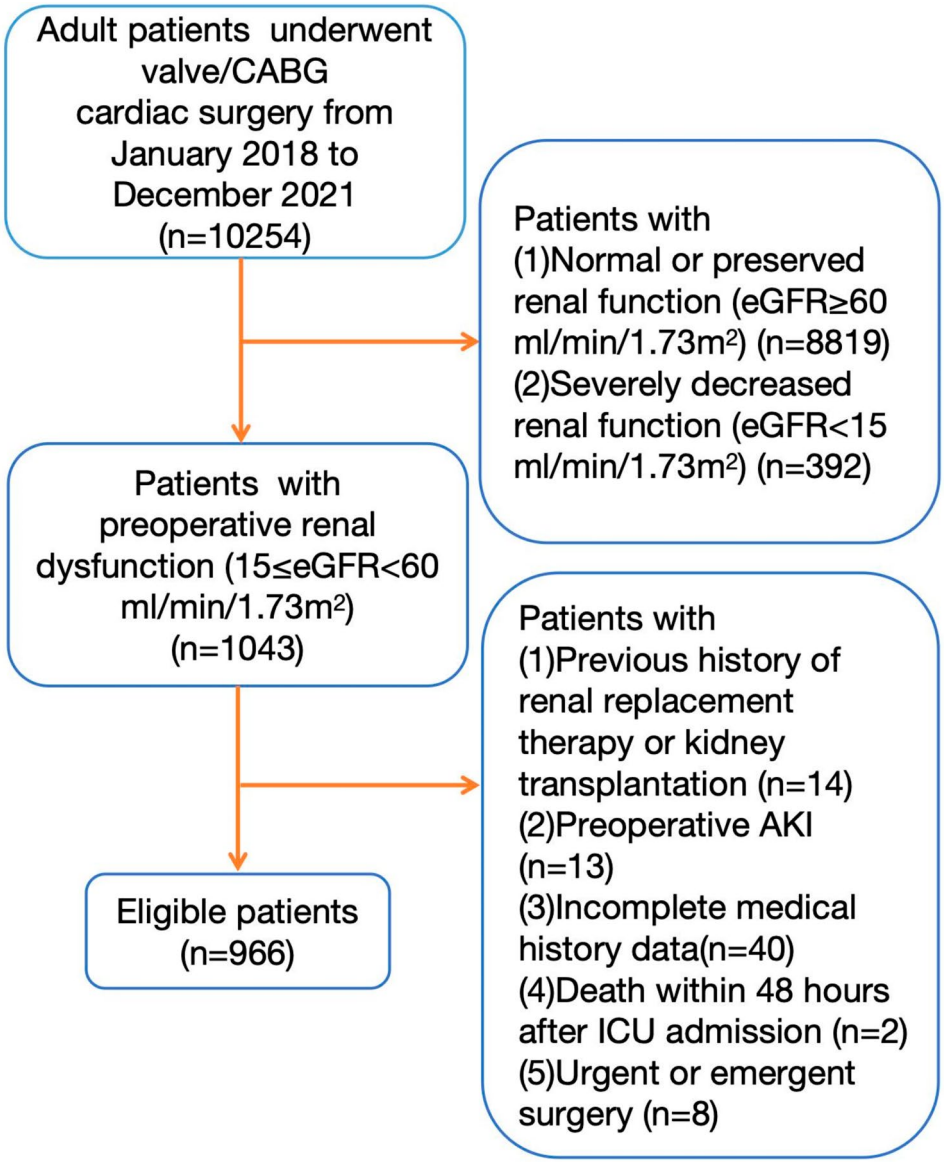

Fig. 1 The Flow chart of patient enrollment

of 0.526 and specificity of 0.627 (see Additional file 1). In addition to using the Youden index to determine the optimal cut-off for dPDW, we evaluated the AUC to assess the predictive performance of the models with and without dPDW. The AUC of the model including dPDW was 0.69, compared to 0.67 in the model without dPDW (see Additional file 2). This indicated that while the addition of dPDW slightly improved the model's predictive accuracy, the overall improvement was modest.

Subgroup analysis

Considering potential factors impacting PDW, our detailed examination revealed significant associations between the dPDW and postoperative AKI across various subgroups (refer to Fig. 2). The results showed a robust positive association between dPDW and AKI, independent of age, sex, surgical type, baseline renal function, hypertension, diabetes, preoperative antiplatelet medication, platelet transfusion, or major bleeding (P for interaction > 0.05).

Possible non-linear association between dPDW and AKI

The RCS analysis, adjusting for the effects of age, gender, surgical type, preoperative eGFR, CPB duration, hypertension, diabetes, preoperative hemoglobin, preoperative albumin, platelet transfusion, antiplatelet medication, and major bleeding suggested no non-linear association of dPDW with AKI (P-Nonlinear = 0.359) (Fig. 3).

Table 1 Perioperative characteristics of the Study Population

Characteristics	Total ($n=966$)	No-AKI ($n=453$)	AKI ($n=513$)	*P* value
Demographic data				
Male (%)	540 (55.90)	233 (51.43)	307 (59.84)	0.009
Age (years)	63.90 ± 10.35	63.49 ± 10.27	64.26 ± 10.41	0.252
BMI (kg/m^2)	23.56 ± 3.39	23.29 ± 3.24	23.79 ± 3.50	0.024
Pre-operative context				
Hypertension (%)	492 (50.93)	201 (44.37)	291 (56.73)	< 0.001
DM (%)	169 (17.49)	74 (16.34)	95 (18.52)	0.373
Anti-platelet administration (%)	626 (64.80)	274 (60.49)	352 (68.62)	0.008
LVEF (%)	60.00 (51.00–65.00)	60.00 (50.50–65.00)	60.00 (51.00–65.00)	0.607
COPD (%)	42 (4.34)	18 (3.97)	24 (4.67)	0.705
Cancer (%)	12 (1.24)	8 (1.77)	4 (0.78)	0.276
Cirrhosis (%)	13 (1.34)	6 (1.32)	7 (1.36)	0.998
Baseline laboratory indices				
Hemoglobin (g/L)	127.51 ± 17.87	129.59 ± 17.47	125.67 ± 18.03	< 0.001
Preoperative platelets (10^9/L)	189.32 ± 65.88	193.25 ± 66.55	185.85 ± 65.14	0.081
Preoperative mean platelet volume (fL)	11.1 ± 2.15	11.3 ± 2.32	10.9 ± 1.94	0.382
Albumin (g/L)	39.22 ± 3.72	39.70 ± 3.44	38.83 ± 3.90	< 0.001
BUN (mmol/L)	9.65 ± 4.40	9.38 ± 4.58	9.88 ± 4.22	0.087
Serum creatinine (μmol/L)	127.78 ± 37.21	122.82 ± 33.90	132.16 ± 39.42	< 0.001
eGFR (ml/min/1.73m^2)	49.10 ± 9.58	50.34 ± 9.15	48.00 ± 9.83	< 0.001
Uric acid (μmol/L)	481.99 ± 187.11	463.91 ± 149.50	498.00 ± 213.86	0.005
NT-proBNP (pg/ml)	1085.00 (458.30–2482.00)	1024.00 (457.72–2321.00)	1172.00 (471.65–2636.00)	0.351
PT (s)	15.00 (13.00, 16.00)	15.00 (13.00, 16.00)	15.00 (13.00, 16.00)	0.460
APTT (s)	44.00 (35.25, 53.00)	44.00 (35.00, 53.00)	45.00 (36.00, 53.00)	0.300
PDW Parameters				
Preoperative PDW (%)	13.83 ± 2.61	13.94 ± 2.78	13.74 ± 2.45	0.250
Postoperative PDW (%)	14.62 ± 3.04	14.46 ± 2.93	14.76 ± 3.13	0.121
Postoperative PDW higher than normal range (%)	26 (2.69)	10 (2.21)	16 (3.12)	0.382
dPDW[a] (%)	0.90 (-0.20, 2.00)	0.60 (-0.40, 1.70)	1.20 (0.10, 2.20)	< 0.001
Surgery				
Sole Valve (%)	507 (52.48)	254 (56.07)	253 (49.32)	0.036
Sole CABG (%)	373 (38.61)	175 (38.63)	198 (38.60)	0.992
Valve & CABG (%)	86 (8.90)	24 (5.30)	62 (12.09)	< 0.001
CPB duration (mins)	101.81 ± 35.75	93.60 ± 28.86	108.93 ± 39.48	< 0.001
Major bleeding[b] (%)	78 (8.07)	38 (8.39)	40 (7.80)	0.736
Transfusion[c]				
Erythrocyte (U)	2.00 (1.00–3.00)	2.00 (1.00–2.00)	2.00 (1.00–4.00)	< 0.001
Platelet (U)	0.00 (0.00–0.00)	0.00 (0.00–0.00)	0.00 (0.00–1.00)	< 0.001
Vasopressors on the surgery day				
Epinephrine > 0.5 μg/kg/min	73 (7.55)	31 (6.04)	42 (9.27)	0.505
Norepinephrine > 1 μg/kg/min	108 (11.18)	43(9.49)	65(12.67)	0.144
Dopamine > 10 μg/kg/min	23 (2.38)	10 (1.94)	13 (2.86)	0.904
Dobutamine > 10 μg/kg/min	16 (1.65)	7 (1.36)	9(1.98)	0.615
Prognosis				
In-hospital mortality (%)	28 (2.90)	3 (0.66)	25 (4.87)	< 0.001
Length of ICU stay (hours)	46.00 (22.00–92.00)	39.00 (21.00–69.00)	66.00 (24.00–115.00)	< 0.001

Table 1 (continued)

Characteristics	Total (*n* = 966)	No-AKI (*n* = 453)	AKI (*n* = 513)	*P* value
Length of hospital stay (days)	14.00 (11.00–19.00)	14.00 (11.00–18.00)	15.00 (12.00–21.00)	< 0.001
Hospitalization cost (CNY)	99147.35 (71926.27–131647.75)	94489.48 (67545.60–123800.00)	102961.00 (76842.50–141899.60)	< 0.001

AKI: Acute Kidney Injury; APTT: Activated Partial Thromboplastin Time; BMI: Body Mass Index; BUN: Blood Urea Nitrogen; CABG: Coronary Artery Bypass Grafting; COPD: Chronic Obstructive Pulmonary Disease; CPB: Cardiopulmonary Bypass; DM: Diabetes Mellitus; eGFR: Estimated Glomerular Filtration Rate; ICU: Intensive Care Unit; LVEF: Left Ventricular Ejection Fraction; NT-proBNP: N-terminal pro-B-type Natriuretic Peptide; PDW: Platelet Distribution Width; PT: Prothrombin Time

The values are expressed as the median (IQR) and mean ± SD or number (%)

P-values are the results of unpaired t-test or Mann–Whitney U test for continuous variables, and χ2 test or Fisher's exact test for categorical variables

a: The change in PDW (dPDW) defined as the difference between PDW values preoperatively and upon admission to the ICU immediately after surgery

b: "Major bleeding" refers to bleeding events classified as grade 3 to 4 according to the BARC (Bleeding Academic Research Consortium) criteria

c: "Transfusion" refers to the volume of blood transfused during surgery and on the day of the surgery

Table 2 Multivariate logistic regression models of dPDW with AKI

dPDW	Model1		Model2		Model3	
	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
Continuous	1.07 (1.02 ~ 1.13)	0.005	1.08 (1.02 ~ 1.13)	0.005	1.09 (1.02 ~ 1.16)	0.012
dPDW Quantile						
1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
2	1.41 (0.98 ~ 2.02)	0.061	1.43 (1.00 ~ 2.05)	0.053	2.16 (1.29 ~ 3.60)	0.003
3	1.80 (1.25 ~ 2.61)	0.002	1.83 (1.26 ~ 2.65)	0.001	2.32 (1.38 ~ 3.89)	0.001
4	2.36 (1.64 ~ 3.40)	< 0.001	2.45 (1.70 ~ 3.54)	< 0.001	2.95 (1.78 ~ 4.90)	< 0.001
P for trend		< 0.001		< 0.001		< 0.001

CI: Confidence Interval; OR: Odds Ratio; PDW: Platelet Distribution Width

Model 1: Crude

Model 2: Adjust: gender, age

Model 3: Adjust: gender, age, surgical type, pre-op eGFR, CPB duration, BMI, hypertension, diabetes, pre-op hemoglobin, pre-op platelet, pre-op albumin, platelet transfusion, pre-op antiplatelet medicine and post-op major bleeding

Discussion

Our study provided novel insights into the predictive role of PDW changes in post-cardiac AKI in patients with preoperative renal insufficiency. The findings highlighted PDW's potential as a biomarker for AKI risk assessment among this specific patient population.

PDW, indicating platelet size variability, links to platelet activation and inflammation [15–18]. In cardiac surgery, perioperative factors such as CPB use, hemodynamic alterations, and antiplatelet medication can directly or indirectly influence PDW [12, 13, 19]. Our study has shown that perioperative changes in PDW (dPDW) are significantly associated with the occurrence of postoperative AKI, particularly in patients with pre-existing renal dysfunction.

The study specifically targeted patients with preoperative renal dysfunction due to their heightened risk and altered baseline renal physiology, which makes them a critical subgroup for studying AKI post-cardiac surgery. This focus allows for a clearer assessment of PDW's predictive value in a population already at a higher baseline risk, thus providing valuable insights into managing and stratifying risk in this vulnerable group.

The mechanisms underlying this association are hypothesized to involve the interplay between inflammatory response [20] and endothelial dysfunction [21, 22] triggered by cardiac surgery. Persistent, low-grade inflammation is recognized as a hallmark feature of CKD, contributing to the development of all-cause mortality among these patients [23]. In patients with pre-existing renal insufficiency, renal vulnerability to inflammatory insults becomes heightened. Systemic inflammatory responses induced by CPB [24] and other surgical stresses can further exacerbate this vulnerability, potentially leading to AKI [25]. An acute inflammatory response enhances endothelial cell-leukocyte adhesion, impairing blood flow within the renal microvasculature. This adhesion is intensified by inflammatory mediators such as cytokines, ROS, RNS, and eicosanoids, which also activate leukocytes. Concurrently, renal tubular epithelial cells release mediators amplifying the inflammation cascade, further impairing microvascular flow and kidney function [26]. Elevated PDW levels, indicative of increased platelet activation and inflammation, may signal a heightened inflammatory state and endothelial dysfunction, serving as precursors to renal injury post-surgery.

Our multivariate analysis adjusted for potential confounders such as CPB duration, surgery duration, platelet transfusion, and anti-platelet medication, which might

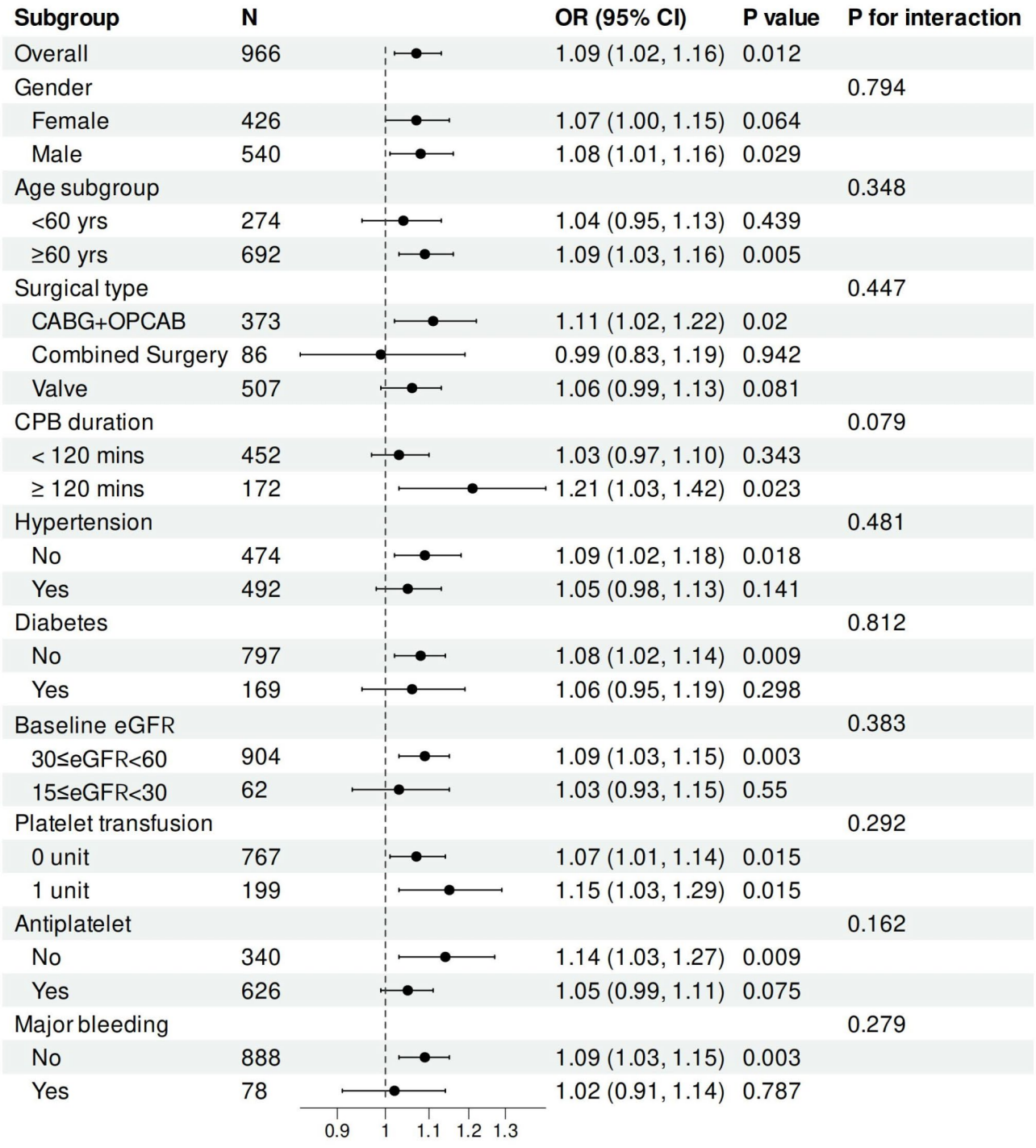

Fig. 2 The subgroup analysis between dPDW and AKI in various subsets. Fig. 2 illustrated the odds ratios (OR) and 95% confidence intervals (CI) for the association between perioperative changes in platelet distribution width (dPDW) and postoperative acute kidney injury (AKI) across various subgroups. Subgroups include gender, age, surgical type, CPB duration, hypertension, diabetes status, baseline eGFR, platelet transfusion, use of antiplatelet medication, and major bleeding. The analysis demonstrates a consistent association between dPDW and AKI in most subgroups, with P for interaction > 0.05 in all cases, indicating no significant heterogeneity in the effect of dPDW across these subgroups

influence PDW. The significant association of dPDW with AKI across different models and the robustness of these findings in subgroup analyses underscore dPDW's potential as an independent predictor of AKI, not merely an indirect marker influenced by these variables. While platelet transfusion can affect PDW values, our analysis included this variable as a confounder. Moreover, the subgroup analysis further strengthened the predictive

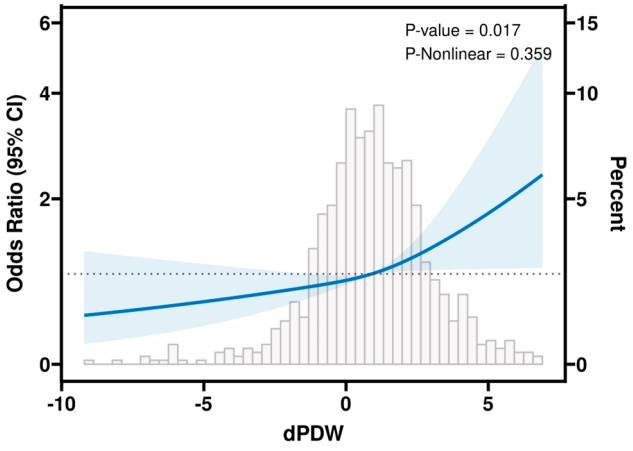

Fig. 3 Association between dPDW and AKI with the RCS anaylsis. Fig. 3 depicted the association between perioperative changes in platelet distribution width (dPDW) and the risk of postoperative acute kidney injury (AKI) using restricted cubic spline (RCS) regression analysis. The analysis suggested a linear association with no significant non-linear relationship (P for non-linearity = 0.359)

value of dPDW for AKI, suggesting that the observed changes in PDW are not solely artifacts of transfusion but reflect a true physiological response to surgical stress and its impact on renal outcomes.

Given the limited literature on PDW as a predictor for AKI, our study addresses a significant gap, particularly regarding patients with preoperative renal insufficiency. This necessitates a re-evaluation of perioperative care, underscoring the importance of closely monitoring PDW changes in high-risk groups. Future research should aim to elucidate the specific pathophysiological mechanisms linking PDW changes to renal outcomes post-cardiac surgery, potentially paving the way for targeted interventions to reduce AKI risk.

The AUC analysis revealed a modest increase in predictive accuracy when dPDW was included in the model (AUC: 0.69 vs. 0.67 without dPDW). While this improvement suggested that dPDW contributes to the overall predictive value, it also highlights the need for additional biomarkers or parameters to enhance the accuracy of AKI prediction models in this patient population. Likewise, while the identified optimal cut-off value of dPDW = 1.1 with a sensitivity of 52.6% and specificity of 63.7% may suggest moderate discriminative power in isolation, it is important to consider the utility of dPDW in conjunction with other clinical parameters and biomarkers. The predictive value of biomarkers can often be enhanced when used as part of a composite index that includes multiple indicators of patient status and surgical impact. Our study positioned dPDW as a significant contributor to a multimodal predictive model, where its integration with other risk factors could substantially refine risk stratification and early diagnostic processes for AKI in post-cardiac surgery settings.

Moreover, the moderate sensitivity and specificity underscore the need for further investigation into the threshold levels and possibly dynamic monitoring of dPDW changes rather than reliance on a single cut-off point. This approach is aligned with the evolving understanding of AKI as a complex syndrome where no single biomarker may suffice for effective prediction on its own. Future studies could focus on longitudinal measurements of dPDW and their correlation with AKI progression, potentially improving the predictive accuracy of this biomarker.

Our study is subject to certain limitations. The retrospective nature and single-center design may restrict the generalizability of our findings. Another limitation is the study's observational design, preventing the preoperative assessment of inflammatory markers and cytokines in patients undergoing cardiac surgery. Due to the retrospective nature of our study, we were unable to establish a definitive causal relationship between changes in PDW and the development of postoperative AKI. As a result, the precise relationship between PDW and inflammation remains to be clarified. Future research should incorporate the evaluation of inflammatory factors to clearly elucidate the relationship between preoperative renal insufficiency, inflammation, and PDW. Additionally, our study did not account for the inherent intra-individual variability in PDW measurements, which could influence the interpretation of small changes in dPDW values. Furthermore, the multifactorial nature of AKI following cardiac surgery necessitates cautious interpretation of PDW as an isolated predictive marker. Despite these limitations, the study serves as a crucial stepping stone for further investigation in this field.

Conclusion

In conclusion, our study underscores the potential of perioperative PDW changes as a predictive biomarker for postoperative AKI in patients with pre-existing renal insufficiency undergoing cardiac surgery. These findings could significantly impact preoperative risk stratification and perioperative management strategies in this vulnerable patient cohort.

Abbreviations

AKI	Acute Kidney Injury
BMI	Body Mass Index
BUN	Blood Urea Nitrogen
CABG	Coronary Artery Bypass Grafting
CPB	Cardiopulmonary Bypass
DM	Diabetes Mellitus
eGFR	Estimated Glomerular Filtration Rate
ICU	Intensive Care Unit
OPCAB	Off-Pump Coronary Artery Bypass
PDW	Platelet Distribution Width
SCr	Serum Creatinine
KDIGO	Kidney Disease: Improving Global Outcomes
LVEF	Left Ventricular Ejection Fraction
NT-proBNP	N-terminal pro-B-type natriuretic peptide
OR	Odds Ratio

CI	Confidence Interval
ROC	Receiver Operating Characteristic
RCS	Restricted Cubic Spline

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-024-03802-9.

Supplementary Material 1

Supplementary Material 2

Acknowledgements
We are grateful for the contribution of the study personnel from the department of nephrology, cardiac surgery and critical care for persistent contribution to the maintenance of the cardiac surgery database.

Author contributions
WJ, XD and JT designed and directed the study, YS, SG, WJ and XX participated in data collection and maintenance, WJ, SG, JX and YS analyzed the data, WJ, YS and YF interpreted the results and writing. XX and ZL participated in reviewing the manuscript, the maintenance of dataset and facilitating the acquisition of data. All authors read and approved the final manuscript.

Funding
This work was supported by Shanghai Clinical Research Center for Kidney Disease, No. 22MC1940100; Shanghai Key laboratory of Kidney and Blood Purification (14DZ226022, 20DZ2271600); National Nature Science Foundation of China, No. 82102289; Natural Science Foundation of Xiamen, 3502Z20227114; Shanghai Federation of Nephrology Project supported by Shanghai ShenKang Hospital Development Center, No. SHDC2202230; Shanghai "science and technology innovation plan" Yangtze River Delta scientific and technological Innovation Community project, No. 21002411500; Nature Science Foundation of Xiamen, No. 2022J011419; Hospital Management Research Fund of Shanghai Hospital Association, No. X2022185.

Data availability
The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study was conducted as a observational retrospective analysis of existing patient records and data. As such, it did not require prospective registration in a clinical trials registry. This study was approved by the ethical board from Zhongshan Hospital, Fudan University (Approval Number B2021–873R). The written informed consent was obtained from participants to participate in the study. The study was conducted in accordance with the Helsinki Declaration (WMA Declaration of Helsinki, 2013).

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Nephrology, Zhongshan Hospital, Fudan University, No 180 Fenglin Rd, Shanghai, China
[2]Department of Nephrology, Zhongshan Hospital (Xiamen), Fudan University, Fujian, China
[3]Xiamen Nephrology Clinical Quality Control Center, Shanghai, China
[4] Shanghai Institute of Kidney and Dialysis, Shanghai, China
[5]Department of Cardiac Surgery Intensive Care Unit, Zhongshan Hospital, Fudan University, Shanghai, China

Received: 31 July 2024 / Accepted: 7 October 2024
Published online: 01 November 2024

References

1. Xu J, Jiang W, Shen B, Fang Y, Teng J, Wang Y, Ding X. Acute kidney Injury in Cardiac surgery. Contrib Nephrol. 2018;193:127–36.
2. Hoste EAJ, Kellum JA, Selby NM, Zarbock A, Palevsky PM, Bagshaw SM, Goldstein SL, Cerda J, Chawla LS. Global epidemiology and outcomes of acute kidney injury. Nat Rev Nephrol. 2018;14(10):607–25.
3. Massoth C, Zarbock A, Meersch M. Acute kidney Injury in Cardiac surgery. Crit Care Clin. 2021;37(2):267–78.
4. Wang Y, Bellomo R. Cardiac surgery-associated acute kidney injury: risk factors, pathophysiology and treatment. Nat Rev Nephrol. 2017;13(11):697–711.
5. Pannu N, James M, Hemmelgarn BR, Dong J, Tonelli M, Klarenbach S, Alberta Kidney Disease N. Modification of outcomes after acute kidney injury by the presence of CKD. Am J Kidney Dis. 2011;58(2):206–13.
6. Akturk S, Buyukavci R. Evaluation of blood neutrophil-lymphocyte ratio and platelet distribution width as inflammatory markers in patients with fibromyalgia. Clin Rheumatol. 2017;36(8):1885–9.
7. Su N, Tang X, Zhan X, Wang X, Peng F, Wen Y, Feng X, Zhou Q, Wang Q, Chen X, et al. The relationship between platelet distribution width and new-onset cardiovascular disease events in patients with peritoneal dialysis. Ren Fail. 2022;44(1):1640–8.
8. Wei Y, Feng J, Ma J, Chen D, Xu H, Yin L, Chen J. Characteristics of platelet-associated parameters and their predictive values in Chinese patients with affective disorders. BMC Psychiatry. 2022;22(1):150.
9. Yu Z, Xiong J, Yang K, Huang Y, He T, Yu Y, Zhao J. The association between platelet indices and cardiovascular events in chronic kidney disease patients without dialysis. Int Urol Nephrol. 2021;53(5):961–71.
10. Zhu HM, Xiong YY, Chen YB, Xiao J. Serum platelet distribution width predicts cardiovascular and all-cause mortality in patients undergoing peritoneal dialysis. Postgrad Med. 2023;135(4):394–401.
11. Ruiyan W, Bin X, Jianhua D, Lei Z, Dehua G, Tang Z. Platelet Distribution Width and Mortality in Hemodialysis Patients. *Evid Based Complement Alternat Med* 2021, 2021:6633845.
12. Paparella D, Yau TM, Young E. Cardiopulmonary bypass induced inflammation: pathophysiology and treatment. An update. Eur J Cardiothorac Surg. 2002;21(2):232–44.
13. Tao YQ, Wang Q, Li YW, Yu-Lan JZ, Tang ZS. [Effects of Apheresis platelet transfusion on PLT, MPV, PDW and PCT]. Zhongguo Shi Yan Xue Ye Xue Za Zhi. 2023;31(6):1820–4.
14. Kellum JA, Lameire N, Group KAGW. Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (part 1). Crit Care. 2013;17(1):204.
15. Izzi B, Gialluisi A, Gianfagna F, Orlandi S, De Curtis A, Magnacca S, Costanzo S, Di Castelnuovo A, Donati MB, de Gaetano G et al. Platelet distribution width is Associated with P-Selectin dependent platelet function: results from the Moli-Family Cohort Study. Cells 2021, 10(10).
16. Ligi D, Della Franca C, Notarte KI, Goldrich N, Kavteladze D, Henry BM, Mannello F. Platelet distribution width (PDW) as a significant correlate of COVID-19 infection severity and mortality. Clin Chem Lab Med. 2024;62(3):385–95.
17. Park J, Kym D, Kim M, Cho YS, Hur J, Chun W, Yoon D, Yoon J. Pioneering predictions of AKI and AKIN severity in burn patients: a comprehensive CBC approach. Sci Rep. 2024;14(1):675.
18. Vagdatli E, Gounari E, Lazaridou E, Katsibourlia E, Tsikopoulou F, Labrianou I. Platelet distribution width: a simple, practical and specific marker of activation of coagulation. Hippokratia. 2010;14(1):28–32.
19. Verma R, Mahapatro S, Kumar A, Rizvi I, Garg RK, Malhotra HS, Sharma PK, Uniyal R. Platelet dysfunction and coagulation assessment in patients of tuberculous meningitis. Neurol Sci. 2020;41(8):2103–10.
20. McGuinness J, Bouchier-Hayes D, Redmond JM. Understanding the inflammatory response to cardiac surgery. Surgeon. 2008;6(3):162–71.
21. Brettner F, Chappell D, Schwartz L, Lukasz A, Kumpers P, Becker BF, Reichart B, Rehm M, Bruegger D. Vascular endothelial dysfunction during cardiac surgery: On-Pump versus off-pump coronary surgery. Eur Surg Res. 2017;58(5–6):354–68.
22. Knezevic D, Curko-Cofek B, Batinac T, Laskarin G, Rakic M, Sostaric M, Zdravkovic M, Sustic A, Sotosek V, Baticic L. Endothelial dysfunction in patients undergoing cardiac surgery: a narrative review and clinical implications. J Cardiovasc Dev Dis 2023, 10(5).
23. Mihai S, Codrici E, Popescu ID, Enciu AM, Albulescu L, Necula LG, Mambet C, Anton G, Tanase C. Inflammation-Related Mechanisms in Chronic Kidney Disease Prediction, Progression, and Outcome. *J Immunol Res* 2018, 2018:2180373.

24. Bronicki RA, Hall M. Cardiopulmonary Bypass-Induced Inflammatory response: pathophysiology and treatment. Pediatr Crit Care Med. 2016;17(8 Suppl 1):S272–278.
25. Giacinto O, Satriano U, Nenna A, Spadaccio C, Lusini M, Mastroianni C, Nappi F, Chello M. Inflammatory response and endothelial dysfunction following cardiopulmonary bypass: pathophysiology and pharmacological targets. Recent Pat Inflamm Allergy Drug Discov. 2019;13(2):158–73.
26. Cantaluppi V, Quercia AD, Dellepiane S, Ferrario S, Camussi G, Biancone L. Interaction between systemic inflammation and renal tubular epithelial cells. Nephrol Dial Transpl. 2014;29(11):2004–11.

Publisher's note