ORIGINAL ARTICLE
Iran J Allergy Asthma Immunol
In press.

Correlation between Dynamic Changes of Inflammatory and Immune Biomarkers and Coronary Artery Lesions in Pediatric Patients with Kawasaki Disease

Jiachen Li[1], Nan Dong[2], Mengjin Wang[1], Dong Qi[3], Yuan Wang[1], Yang Liu[1], Jingwei Sun[1], and Quming Zhao[4]

[1] Department of Pediatrics, Bengbu First People's Hospital, Bengbu, China
[2] Department of Ultrasound, Bengbu First People's Hospital, Bengbu, China
[3] Department of Radiology, Bengbu First People's Hospital, Bengbu, China
[4] Pediatric Heart Center, National Children's Medical Center, Children's Hospital, Fudan University, Shanghai, China

Received: 3 June 2026; Received in revised form: 29 July 2026; Accepted: 2 August 2026

ABSTRACT

This study investigated associations between dynamic changes in inflammatory and immune biomarkers and coronary artery lesions (CALs) in children with Kawasaki disease (KD) before and after intravenous immunoglobulin (IVIG) treatment.

We retrospectively analyzed 208 children with KD admitted between January 2020 and September 2025, including 159 with normal coronary arteries and 49 with coronary abnormalities. Inflammatory, hematological, coagulation, and biochemical indicators were assessed before IVIG and at 72 hours, Day 6, and Day 9 after treatment. Between-group differences were evaluated using the Mann–Whitney U test, and associations with CALs were examined using point-biserial Pearson correlation.

Baseline inflammatory and immune indicators did not differ significantly between groups. White blood cell (WBC) counts showed a greater rebound on Days 6 and 9 in children with coronary abnormalities, while platelet counts were higher throughout follow-up. Changes in D-dimer and platelet counts at 72 hours differed significantly between groups and correlated with CALs. WBC counts on Day 9 were also significantly associated with CALs.

Dynamic changes in D-dimer and platelet counts during the early post-treatment period and WBC counts during recovery may provide additional information for assessing coronary involvement in KD. These exploratory associations require prospective validation before routine clinical application.

Keywords: Biomarkers; Blood platelets; Coronary artery disease; Immunoglobulins, Intravenous; Leukocyte count; Mucocutaneous lymph node syndrome

Corresponding Authors: Jingwei Sun, BM;
Department of Pediatrics, Bengbu First People's Hospital, Bengbu, China. Tel/Fax: (+86 153) 95270234, Email: sunjwsun@163.com

*The first, second and third authors contributed equally to this study

Quming Zhao, MD;
Pediatric Heart Center, National Children's Medical Center, Children's Hospital, Fudan University, Shanghai, China. Tel: (+86) 013501878567, Fax: (+86) 013501878567, Email: liuywpwk@163.com

Copyright©, Iran J Allergy Asthma Immunol. All rights reserved.

Published by Tehran University of Medical Sciences (http://ijaai.tums.ac.ir)

IN PRESS

INTRODUCTION

Kawasaki disease (KD) is an acute systemic vasculitis syndrome that mainly occurs in children under 5 years of age, and it is also the most common cause of acquired heart disease in children. In recent years, the incidence of KD has been increasing worldwide and in China.[1-3] The typical clinical features of KD are persistent fever, mucosal hyperemia, pleomorphic rash, acral edema and cervical lymph node enlargement. The most serious complication is a coronary artery lesion (CAL), which can progress to coronary artery dilatation and even coronary artery aneurysm (CAA) and secondary thrombosis.[4,5] In the long term, it can cause myocardial ischemia or infarction, which seriously threatens the life and health of children.[6] Therefore, risk prediction and early biomarker screening for CAL/CAA have become the major difficulties in pediatric clinical diagnosis and treatment.

Current clinical evidence generally suggests that the pathophysiology of KD is closely related to abnormally activated inflammatory cascades and imbalanced immune homeostasis. Studies have shown that an abnormally low baseline platelet count is significantly associated with non-response to intravenous immunoglobulin (IVIG) therapy and with the risk of subsequent CALs.[7] In the acute phase of the disease, extensive inflammatory cell infiltration, the release of acute-phase response proteins, and an activated proinflammatory cytokine network trigger a systemic inflammatory cascade, which causes persistent damage to small and medium-sized vessels throughout the body, particularly the coronary artery endothelium.[8-10] Endothelial injury may further trigger coagulation and platelet activation, thereby exacerbating vascular inflammation and contributing to coronary artery dilation, aneurysm formation, and thrombosis. At present, IVIG combined with acetylsalicylic acid (ASA) is still the standard first-line treatment in the acute phase, with the aim of rapidly controlling the excessive inflammatory response and reducing the risk of coronary artery disease. However, in clinical practice, assessment of disease activity and response to IVIG relies largely on routine indicators such as complete blood count, C-reactive protein and erythrocyte sedimentation rate before treatment. Previous literature is mostly limited to cross-sectional analyses at a single time point, and there is still a lack of longitudinal monitoring data on the dynamic changes of inflammation after IVIG intervention. This gap limits the development of an accurate early warning model for coronary artery disease. Serial assessments at multiple time points are urgently needed to clarify the relationship between dynamic changes in inflammatory indicators and clinical manifestations, thereby providing more reliable evidence to guide the adjustment of individualized treatment plans.

The existing literature has not reached a consensus on the value of baseline inflammatory markers in predicting coronary artery lesions, and several studies have suggested that a single static indicator may have limited ability to distinguish patients who develop CAL from those who do not.[11-13] In contrast, post-IVIG biomarker trajectories may capture early treatment responses and the persistence or rebound of inflammatory, coagulation, and hematological abnormalities that cannot be fully reflected by a single baseline measurement. This potential complementary value provides a rationale for further investigating the temporal changes in these biomarkers and their associations with CAL. In contrast to a single baseline measurement, serial assessments before IVIG treatment and at 72 hours, Day 6, and Day 9 after treatment may better characterize the rates of decline, rebound patterns, and overall temporal trajectories of inflammation-, immune-, and coagulation-related biomarkers. Whether these trajectories are intrinsically related to CAL in KD still requires systematic verification using large clinical longitudinal datasets. Elucidating the dynamic evolution in these indicators and their associations with coronary outcomes is expected to provide more sensitive laboratory evidence for optimizing the risk stratification system of KD. Accordingly, this retrospective study included 208 children with newly diagnosed KD admitted to our hospital between January 2020 and September 2025. The inflammatory cell counts, acute-phase response proteins, cytokine profiles, coagulation function and routine biochemical indicators were systematically monitored and collected at several key time points before IVIG treatment and at 72 hours, Day 6 and Day 9 after IVIG treatment. By comparing the dynamic changes in these indicators between the normal coronary artery group and the abnormal coronary artery group, this study intends to further quantify the statistical relationships between the baseline levels and changes in the indicators and coronary artery lesions.

IN PRESS

The aim of this study is to systematically describe the dynamic evolution trajectories of inflammatory and immune indicators in children with KD after IVIG treatment, and to screen for key dynamic markers closely related to coronary artery damage. The results of this study are expected to provide an objective and quantifiable laboratory reference for early identification of high-risk children, improvement of individualized follow-up strategies and optimization of intervention timing.

MATERIALS AND METHODS

Study Subjects

Study Period and Source of Patients

This retrospective study enrolled 208 children admitted to the Department of Pediatrics at our hospital and clinically diagnosed with KD between January 2020 and September 2025. All children received the standard IVIG combined with aspirin treatment protocol for KD, with doses determined according to body weight and adjusted according to the disease phase and clinical response, and coronary artery imaging results and laboratory data at multiple time points were completely collected. All 208 children included in the final analysis had complete data and follow-up records.

Diagnostic criteria: 1) Normal coronary artery: Internal diameter of the left orright coronary artery <2.5 mm in infants <12 months old; <3.0 mm in children aged 1–5 years; <3.5 mm in children >5 years; no intimal thickening or increased echogenicity. 2) Coronary dilatation: Coronary diameter exceeds the above age-based thresholds, without localized saccular orfusiform expansion. 3) Coronary artery aneurysm (CAA): Local coronary artery segment diameter ≥1.5 times that of the adjacent normal vessel, or absolute diameter ≥4 mm in children <5 years old/≥6 mm in children ≥5 years old. 4) Increased coronary intimal echogenicity: Uniformly increased echogenicity of the coronary arterial wall without luminal dilatation, classified as mild coronary vascular injury according to consensus.

Inclusion criteria: 1) patients who fulfilled the clinical diagnostic criteria for KD in the "Expert consensus on the diagnosis and treatment of KD (2020 edition)"; 2) patients who were initially diagnosed with KD and received a single course of IVIG therapy after admission, without receiving other immunosuppressants or glucocorticoids; 3) patients with complete clinical and laboratory data before IVIG treatment and at 72 h, Day 6 and Day 9 after IVIG treatment; 4) children who underwent coronary artery ultrasound and other coronary artery imaging assessments after admission, with sufficient imaging data available to determine coronary artery status.

Exclusion criteria: 1) children with incomplete, refractory, or recurrent KD; 2) patients with congenital heart disease, previous vascular disease, severe infection, autoimmune disease, or hematological disease; 3) those who had received IVIG, hormonal therapy, and other targeted treatments in other hospitals before admission; 4) patients with missing clinical data, unclear coronary imaging results, and missing laboratory indicators at key time points.

This retrospective observational study was conducted in accordance with the ethical principles of the Declaration of Helsinki. The study protocol was reviewed and approved by the Institutional Ethics Committee of Bengbu First People's Hospital (Ethics Approval No.: BBYY2024011). Since this retrospective study only used anonymized clinical, laboratory and imaging data of pediatric patients and did not interfere with routine clinical diagnosis and treatment, the Institutional Ethics Committee waived the requirement forwritten informed consent. All patient-related information was fully de-identified to protect personal privacy and medical confidentiality.

Grouping Criteria

Coronary artery status was assessed by echocardiography after admission. Based on the results of echocardiography, all the children were divided into 2 groups according to the criteria of coronary artery disease:

1) Normal coronary artery group: 159 patients (76.4%) had normal coronary artery diameters without dilatation, aneurysms, or intimal thickening;

2) Abnormal coronary artery group: there were 49 patients with coronary artery dilation, coronary artery aneurysm, or increased echogenicity of the coronary artery intima, accounting for 23.6% of all the patients.

All patients received baseline cardiac ultrasound within 24 hrs after admission before IVIG treatment, followed by repeat ultrasound at 72 hrs after IVIG treatment, and 1–2 weeks after discharge (covering day 6 and day 9 blood-sampling windows). Final coronary grouping was determined by the maximum coronary diameter measured during the entire acute hospitalization period.

IN PRESS

Observation Indicators and Measurement Time Points

Detection Time Point

Four observation time points were set: before IVIG treatment (baseline), 72 hours after IVIG treatment, Day 6 after IVIG treatment, and Day 9 after IVIG treatment. These time-points were selected based on the availability of routine clinical follow-up data and to capture the early response to IVIG at 72 h and the subsequent recovery-phase changes on Day 6 and Day 9. In the figures, "pre-IVIG" denotes the time point before IVIG treatment. Because some clinical follow-up samples at different time points were missing, statistics were calculated using the available samples at each time point.

Detection Indicators

Inflammatory cell indicators: peripheral blood white blood cell (WBC) count, neutrophil count, platelet count; Acute inflammatory markers: C-reactive protein (CRP), serum amyloid A (SAA), erythrocyte sedimentation rate (ESR), procalcitonin (PCT), interleukin-6 (IL-6); Coagulation and myocardial-related indicators: D-dimer, troponin I; Biochemical and other indicators: serum albumin, hemoglobin (HCT), serum sodium, alanine aminotransferase, random blood glucose, serum total bilirubin.

These indicators were selected to reflect complementary aspects of KD-related cardiovascular involvement. Inflammatory markers reflect systemic inflammatory activity; platelet count and D-dimer reflect hematological and coagulation/fibrinolytic responses; biochemical parameters provide information on systemic involvement, while troponin I indicates myocardial injury.

Using the baseline level before IVIG treatment as a reference, the changes in indicators at each time point were calculated as follows:

Change in indicators = values measured at corresponding time points after treatment − baseline values before IVIG treatment

Laboratory Test Methods

All venous blood samples were uniformly tested by the laboratory department of our hospital, and the testing processes, instruments and reagents strictly complied with the standard operating procedures of the hospital: complete blood count and erythrocyte sedimentation rate were measured using an automated blood analyzer; CRP, SAA, PCT and biochemical parameters were measured using automated biochemical and immunological analyzers. Interleukin-6 and troponin I were measured using chemiluminescence immunoassay. D-dimer was measured using immunoturbidimetry. All tests were subjected to daily internal quality control to ensure accurate and reliable results.

Statistical Analysis

Statistical Package for the Social Sciences (SPSS) 26.0 statistical software (IBM, Armonk, NY, USA) was used for data analysis. Descriptive statistics: measurement data were expressed as the mean ± standard deviation ($\bar{x}\pm s$) if they were normally distributed; most of the inflammation and coagulation indicators in this study had right-skewed distributions, so the median (quartiles) (M (Q1, Q3)) was used to describe the central tendency and dispersion of the data. Between-group comparison: The Mann-Whitney U test was used to compare skewed measurement data between the two groups, and the significance level was set at $\alpha=0.05$; $P<0.05$ was considered statistically significant. Correlation analysis: The coronary artery status was assigned to two categories (normal coronary artery=0, abnormal coronary artery=1), and bivariate Pearson correlation was used to assess the associations between coronary artery status and both the baseline levels and changes in the indices. The standardized mean difference and −log 10 (P) were further calculated for the differences in indicators between the groups. These values were visualized to illustrate the magnitude and significance of the differences between the groups.

RESULTS

Basic Information of the Sample

A total of 208 children with KD were included in the analysis. According to the coronary artery status, 159 patients (76.4%) were in the normal coronary artery group and 49 patients (23.6%) were in the abnormal coronary artery group. Overall, 128 patients (61.5%) were male, and the median age at onset (in months) was 24.00 (12.00, 36.00). No statistically significant differences were observed between the two groups in sex distribution, age, height, and weight. The number of patients in the normal coronary artery group was considerably higher than that in the abnormal coronary artery group (159 vs. 49), resulting in an imbalanced sample size between the two groups for subsequent

IN PRESS

comparisons. Therefore, results in the coronary artery abnormality group should be interpreted with caution, given the small sample size and the greater amount of missing data at some follow-up time points (Tables 1-2).

On the basis of the group sizes listed in the table, the sample composition of the 2 groups was further presented graphically, which helped illustrate the relative proportions of the normal coronary artery group and the abnormal coronary artery group (Figure 1). This imbalance in group sizes not only affected the stability of subsequent statistical analyses but also required more caution when interpreting the results for the abnormal coronary artery group.

The proportions between groups showed that the coronary artery abnormality group accounted for about a quarter of all patients. The relatively small size of the abnormal coronary artery group may reduce the stability of statistical estimates and increase their sensitivity to missing data and extreme values. Therefore, both the mean ± standard deviation and the median (interquartile range) were reported to provide a more robust description of the distribution of each indicator.

Descriptive Statistics of Inflammatory and Immune Indicators

The descriptive statistics showed that many inflammatory indicators were elevated before IVIG treatment, suggesting that the included patients were in a relatively active inflammatory response phase. The mean WBC count was 15.05, the median CRP value was 48.42, and the median SAA value was 300.0. These results indicate that WBC count, CRP and SAA can reflect different aspects of acute inflammatory activity. Notably, the relatively large standard deviations of SAA, PCT, and interleukin-6 suggest considerable interindividual variability in the intensity of the inflammatory response (Table 3).

According to the distribution pattern, the mean values of some inflammatory indicators were substantially higher than the medians, indicating that the variables may have right-skewed distributions or extremely high values. For example, the overall level of SAA fluctuates greatly in the acute phase. The mean is more significantly influenced by high values, while the median value better reflects the central tendency of values in most children. Such distribution characteristics suggest that subsequent comparisons between groups should not only rely on differences in mean values but also incorporate the results of nonparametric tests and interquartile ranges for comprehensive assessment.

Between-group Comparison of Inflammatory and Immune Indices in Children with Different Coronary Status

In comparisons between the normal coronary artery group and the abnormal coronary artery group, the differences in most inflammatory and immune indicators before IVIG treatment did not reach the statistical significance level. Among the baseline parameters, the *p* value of SAA (before IVIG) was relatively small (p=0.228), followed by SAA (before IVIG) and platelet count (before IVIG). Taking platelet count as an example, the mean value was 349.6 in the normal coronary artery group, and 395.4 in the abnormal coronary artery group. The platelet level was relatively higher in the abnormal coronary artery group, but the P value for the between-group comparison was 0.256, which did not indicate a statistically significant difference (Table 4).

Because the laboratory indicators varied considerably in measurement scales and value ranges, direct comparison of their raw mean values was not suitable for assessing the direction and relative magnitude of between-group differences across indicators. Therefore, based on the between-group comparison results, standardized mean differences (SMDs) were used to quantify the magnitude and direction of differences between the abnormal and normal coronary artery groups, identifying which indicators showed greater differences in the direction of the abnormal coronary artery group and which showed minimal between-group differences.

CRP and ESR, commonly used markers of inflammation, showed a relatively similar distribution between the two groups. The median CRP value in the normal coronary artery group and the abnormal coronary artery group was 47.52 and 51.36, respectively, and the *p* value of ESR was 0.991. These results suggest that a single inflammatory index measured before IVIG treatment has limited value in distinguishing coronary artery lesions. From a clinical perspective, coronary artery damage may not be fully reflected by the level of inflammation at a single time point, but may instead be associated with the rate of decline in the inflammatory response, changes in hematological parameters, and changes in coagulation-related indicators before and after IVIG treatment (Figure 2).

IN PRESS

Published by Tehran University of Medical Sciences (http://ijaai.tums.ac.ir)

Table 1. Baseline characteristics of children with KD, grouped by coronary artery involvement

Variable	Unit	Total (n=208)	Normal CA (n=159)	Abnormal CA (n=49)	*p*
Sex	n (%)				0.261
Female		80 (38.5)	65 (40.9)	15 (30.6)	
Male		128 (61.5)	94 (59.1)	34 (69.4)	
Pneumonia	n (%)				0.085
No		163 (79.1)	129 (82.2)	34 (69.4)	
Yes		43 (20.9)	28 (17.8)	15 (30.6)	
Age	months	24.00 (12.00, 36.00)	24.00 (12.00, 36.00)	12.00 (12.00, 48.00)	0.890
Height	cm	90.00 (76.00, 100.00)	90.00 (76.00, 100.00)	85.00 (76.00, 106.00)	0.720
Weight	kg	12.50 (10.00, 16.00)	12.50 (10.00, 16.00)	12.00 (10.00, 16.25)	0.934

Continuous variables were tested for normality; normally distributed variables are presented as mean ± SD and compared using the independent-samples t test, while non-normally distributed variables are presented as median (P25, P75) and compared using the Mann-Whitney U test. Categorical variables are presented as n (%) and compared using the chi-square test or Fisher's exact test. The two-sided significance level was set at $\alpha = 0.05$.

Table 2. Sample Assignment

Coronary Grouping	Number of Patients	Constituent ratio (%)
Normal coronary artery	159	76.4
Coronary artery abnormalities	49	23.6

The coronary artery group was determined according to the classification of patients as having normal or abnormal coronary arteries in the data table.

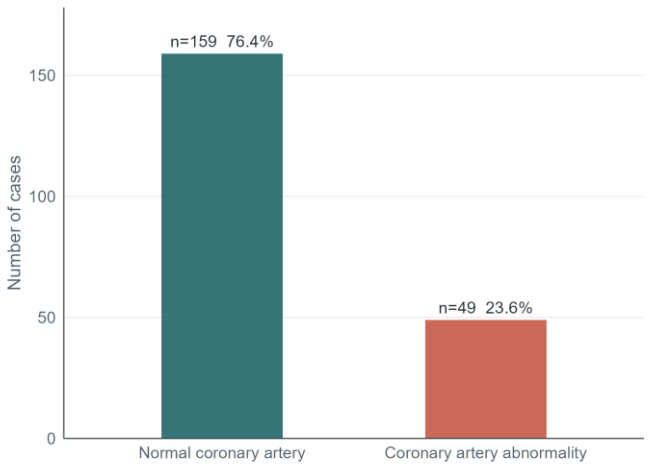

Figure 1. Composition of the sample groups

IN PRESS

Table 3. Descriptive statistics of the main inflammatory and immune indicators before IVIG treatment

Variables	n	Mean ± standard deviation	Median (Q1, Q3)
Age (months)	208	28.65±24.19	24.00 (12.00, 36.00)
White blood cells (before IVIG)	208	15.05±5.82	13.88 (11.06, 18.23)
Neutrophils (before IVIG)	206	9.86±4.71	8.96 (6.88, 12.09)
Platelets (before IVIG)	208	360.4±155.4	345.0 (263.5, 437.2)
CRP (before IVIG)	205	68.07±87.92	48.42 (30.52, 80.79)
SAA (before IVIG)	185	293.6±545.0	300.0 (112.1, 320.0)
Blood sedimentation (before IVIG)	205	54.11±31.60	54.00 (30.00, 78.00)
PCT (before IVIG)	203	0.91±1.44	0.42 (0.24, 0.94)
Interleukin-6 (before IVIG)	190	96.18±175.8	46.58 (18.84, 113.9)

Age in months; continuous variables are reported both as means ± standard deviations and medians (quartiles).

Table 4. Comparison of main indicators before IVIG treatment among children with different coronary artery statuses

Variables	Mean±SD in the normal coronary artery group (n)	Mean±SD in the abnormal coronary artery group (n)	Median in the normal coronary artery group (Q1, Q3)	Median in the abnormal coronary artery group (Q1, Q3)	*p*
White blood cells (before IVIG)	15.12±5.87	14.82±5.70	14.12 (11.05, 18.17)	13.62 (11.25, 18.28)	0.851
Neutrophils (before IVIG)	10.02±4.77	9.36±4.51	9.38 (6.95, 12.10)	8.04 (6.71, 11.10)	0.339
Platelets (before IVIG)	349.6±145.1	395.4±182.3	335.0 (261.5, 431.0)	362.0 (273.0, 484.0)	0.256
CRP (before IVIG)	71.05±98.08	58.35±38.65	47.52 (30.38, 81.93)	51.36 (31.71, 67.76)	0.977
SAA (before IVIG)	251.3±170.1	442.4±1110.8	288.3 (98.74, 320.0)	300.0 (236.6, 320.0)	0.228
Blood sedimentation (before IVIG)	54.06±31.11	54.28±33.56	54.50 (30.00, 78.00)	52.00 (27.50, 75.50)	0.991
Albumin (before IVIG)	40.48±4.66	40.23±6.54	40.72 (37.95, 42.99)	39.85 (36.53, 42.31)	0.247
D-dimer (before IVIG)	1.92±2.91	1.80±4.76	0.98 (0.65, 1.83)	0.93 (0.70, 1.40)	0.537
PCT (before IVIG)	0.90±1.42	0.96±1.50	0.40 (0.24, 0.97)	0.47 (0.28, 0.85)	0.524
Interleukin-6 (before IVIG)	94.45±176.9	102.1±173.9	44.65 (19.25, 111.5)	52.14 (15.29, 127.0)	0.836

p values were calculated using the normal approximation of the Mann-Whitney U test; a P value of less than 0.05 was considered statistically significant; available-case analysis was used for each variable.

IN PRESS

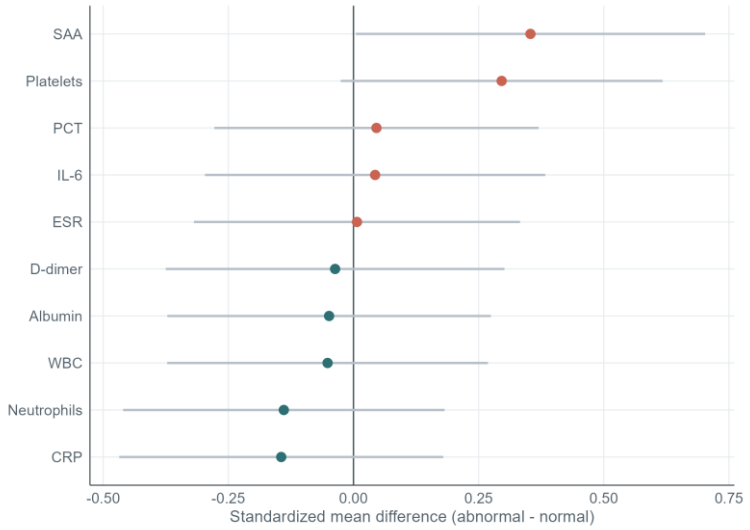

Figure 2. The standardized mean difference of the main indicators before IVIG treatment

Dynamic Changes of Inflammatory and Immune Indicators

Further analysis of changes in the indices at baseline and at 72 hours, day 6, and day 9 after IVIG treatment showed that the dynamic trajectories were not consistent across indices. White blood cells in the normal coronary artery group decreased from 15.12×10⁹/L before IVIG treatment to 13.09×10⁹/L at 72 hours, and then increased to 14.83×10⁹/L on day 6 after IVIG treatment. In the abnormal coronary artery group, the mean values of white blood cells on day 6 and day 9 were 19.36×10⁹/L and 17.93×10⁹/L, respectively, both of which were higher than those in the normal coronary artery group. This change suggests that compared with the cross-sectional difference before treatment, the recovery trajectory of white blood cells in the middle and late stages after treatment may better reflect the difference in inflammatory response between the 2 groups.

Table 5 shows the means and standard errors of the main measures at different time points, but it does not adequately represent the continuous trends of change over the course of treatment. Therefore, dynamic curves were plotted by indicator category. First, the inflammatory cell response reflected by white blood cells and neutrophils was observed, and then the changes in platelets and acute-phase inflammatory markers were analyzed.

As shown in Figure 3, the white blood cell dynamic curve showed a decrease in both groups at 72 hours after IVIG treatment, but the increase was more pronounced in the coronary artery abnormality group on Day 6 and Day 9 after treatment. This trend suggests that observing WBC levels before treatment alone is insufficient to reflect the between-group differences in the inflammatory recovery process after treatment. Therefore, it is necessary to further examine neutrophil counts, which are a more direct indicator of the granulocyte inflammatory response.

As shown in Figure 4, the changes in neutrophils were consistent with the trend of white blood cells, with a decrease in the early post- treatment phase and an increase on day 6. However, the difference in neutrophils between the two groups at the subsequent time points was relatively modest, indicating that the change in the total WBC count may be affected by other cellular components. In addition to inflammatory cells, platelets—a common indicator of recovery in KD—also warrants separate observation.

As shown in Figure 5, platelets showed a different dynamic pattern from white blood cells and neutrophils, gradually increasing after treatment in both groups and reaching higher levels around day 6. In the abnormal coronary artery group, platelet levels remained relatively high both before and after treatment, suggesting that the platelet response may be associated with the severity of coronary artery lesions. In contrast, CRP and SAA more directly reflect the burden of acute inflammation, and their trajectories may further help assess the resolution of inflammation after treatment.

IN PRESS

Table 5. Dynamic changes in the main inflammatory and immune indices

Indicators	Time point	Normal coronary artery group mean±E (n)	Abnormal coronary artery group mean±SE (n)
Leukocytes	Before IVIG	15.12±0.47 (n=159)	14.82±0.81 (n=49)
Leukocytes	IVIG72h	13.09±0.52 (n=157)	11.89±0.82 (n=49)
Leukocytes	IVIG6d	14.83±0.94 (n=56)	19.36±2.48 (n=18)
White cells	IVIG9d	12.42±1.12 (n=31)	17.93±1.99 (n=9)
Neutrophils	Before IVIG	10.02±0.38 (n=157)	9.36±0.64 (n=49)
Neutrophils	IVIG72h	5.69±0.35 (n=157)	5.16±0.56 (n=49)
Neutrophils	IVIG6d	7.67±0.74 (n=54)	8.51±1.52 (n=18)
Neutrophils	IVIG9d	5.59±0.79 (n=28)	6.08±1.33 (n=9)
Platelets	Before IVIG	349.6±11.51 (n=159)	395.4±26.05 (n=49)
Platelets	IVIG72h	537.5±14.18 (n=157)	521.2±22.24 (n=49)
Platelets	IVIG6d	601.5±22.61 (n=56)	654.0±37.14 (n=18)
Blood platelets	IVIG9d	500.7±26.42 (n=31)	581.6±63.97 (n=9)
CRP	Before IVIG	71.05±7.83 (n=157)	58.35±5.58 (n=48)
CRP	IVIG72h	11.94±1.38 (n=155)	10.30±1.83 (n=48)
CRP	IVIG6d	9.16±2.18 (n=54)	4.61±0.76 (n=15)
CRP	IVIG9d	6.16±1.02 (n=23)	3.12±0.96 (n=5)
SAA	Before IVIG	251.3±14.17 (n=144)	442.4±173.5 (n=41)
SAA	IVIG72h	36.66±5.94 (n=134)	45.79±10.67 (n=40)
SAA	IVIG6d	43.02±11.44 (n=31)	43.91±26.02 (n=12)

SE is the standard error; sample sizes varied at different time points due to missing test records.

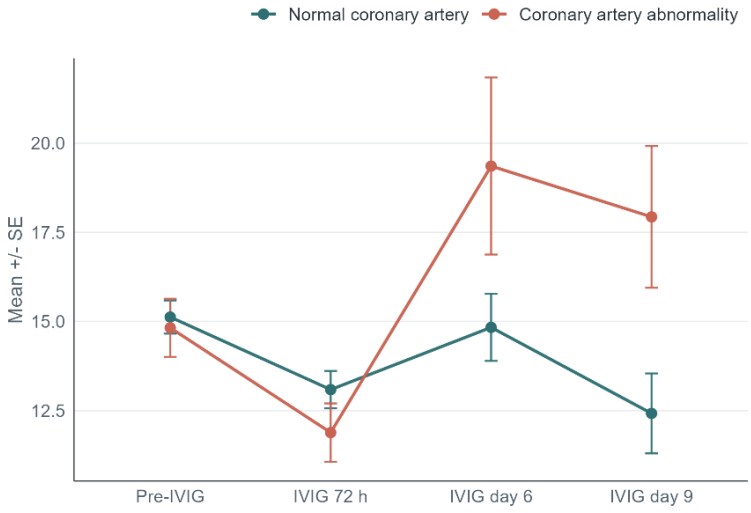

Figure 3. Dynamic changes of white blood cells

Published by Tehran University of Medical Sciences (http://ijaai.tums.ac.ir)

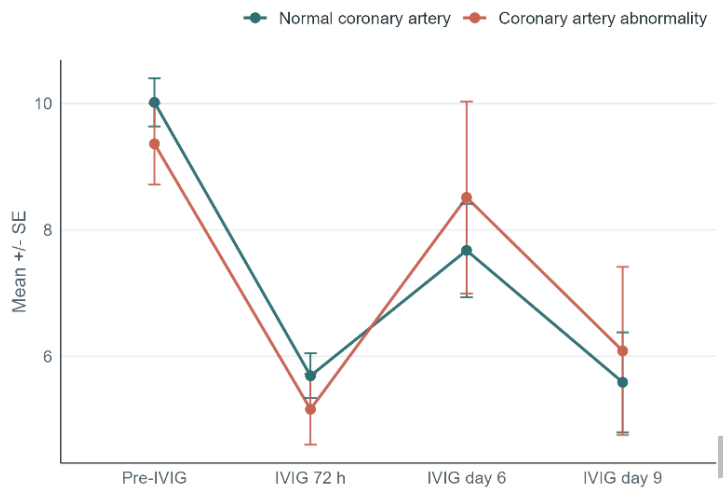

Figure 4. Dynamic changes of neutrophils

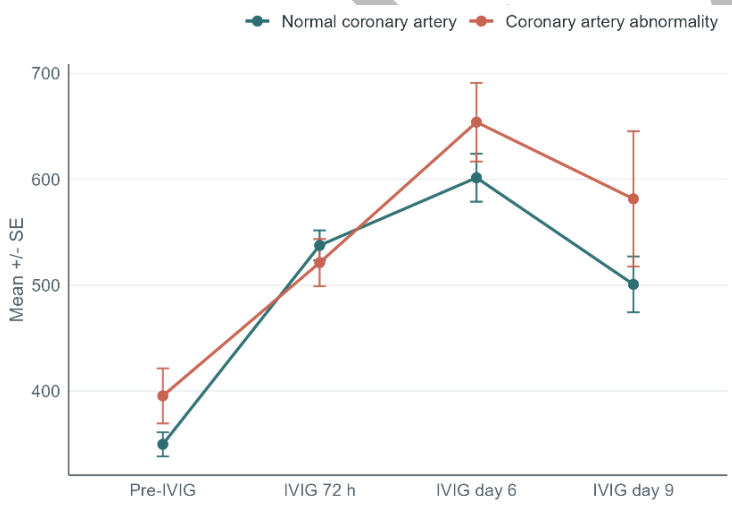

Figure 5. Dynamic changes of platelets

As shown in Figure 6, CRP showed a rapid post-treatment decline in both groups, indicating an overall attenuation of the acute inflammatory response after IVIG treatment. In the normal coronary artery group before treatment, the mean CRP level was slightly higher, but CRP levels decreased to lower levels after treatment in both groups, indicating that CRP dynamically reflects the overall process of inflammation resolution, but its ability to distinguish between normal and abnormal coronary artery status alone is relatively limited. In order to further verify the consistency of changes in acute-phase inflammatory markers, the dynamic trajectory of SAA was examined.

As shown in Figure 7, neutrophils decreased from 10.02×10^9/L to 5.69×10^9/L in the normal coronary artery group and from 9.36×10^9/L to 5.16×10^9/L in the abnormal coronary artery group at 72 hours after treatment. Platelets showed an increasing trend after treatment. The mean platelet count was 601.5×10^9/L in the normal coronary artery group and 654.0×10^9/L in the abnormal coronary group on day 6 after treatment. CRP and SAA generally decreased after treatment, reflecting declining inflammatory burden as treatment progressed. However, given the considerable amount of missing data on day 6 and day 9, these trends should be interpreted as general patterns rather than definitive conclusions.

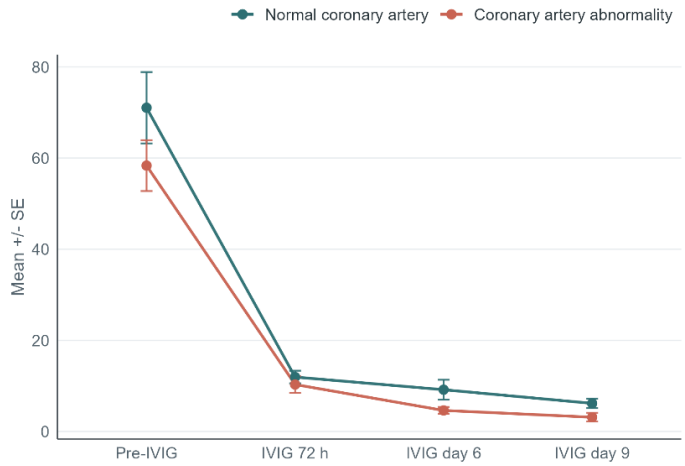

Figure 6. Dynamic changes of CRP

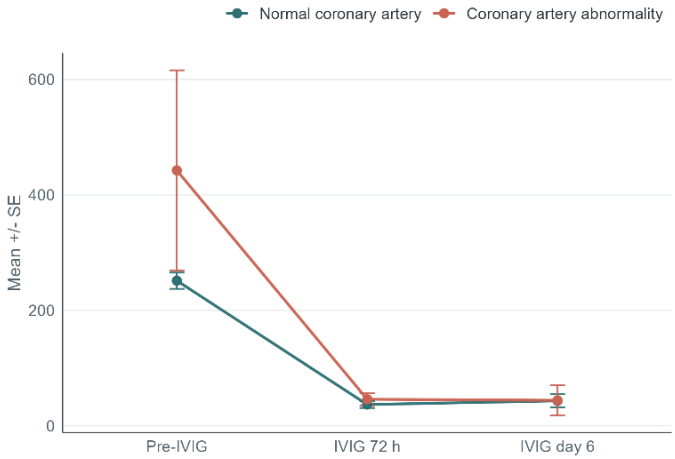

Figure 7. Dynamic changes of SAA

Relationship between Changes in Inflammatory and Immune Markers and Coronary Artery Lesions

Based on the dynamic changes, changes at each time-point after IVIG treatment compared with those before IVIG treatment were further calculated to more directly reflect the extent of improvement or rebound in each indicator. The difference in the change in D-dimer at 72 hours between the 2 groups was the most prominent ($p \leq 0.001$). In the normal coronary artery group, D-dimer decreased by an average of 1.68 ng/mL at 72 hours, compared with a decrease of only 0.25 ng/mL in the abnormal coronary artery group. This suggests that the overall decline in D-dimer after treatment was more pronounced in the normal coronary artery group, whereas the decrease was smaller—or nearly negligible—in the abnormal coronary artery group.

Table 6 shows the specific numerical differences for each change between the two groups. In order to more clearly compare the statistical signal strength of multiple changes, the p value for each change is transformed into $-\log_{10}(P)$. The transformation does not alter the test results per se, but serves to highlight which changes are more notable in between-group comparisons.

As shown in Figure 8, in addition to D-dimer, the change in platelet count at 72 hours also showed a significant difference ($p=0.011$) between groups. The mean increase in platelet count within 72 hours was

Published by Tehran University of Medical Sciences (http://ijaai.tums.ac.ir)

188.4×10^9/L in the normal coronary artery group and 125.9×10^9/L in the abnormal coronary artery group, suggesting a greater early increase in platelet count in the normal coronary artery group after treatment. The P values for the 72-hour changes in sodium and troponin I were 0.026 and 0.044, respectively, indicating that in addition to the classical inflammatory markers, short-term changes in coagulation, electrolyte and myocardial injury-related markers may also be associated with the coronary artery status. However, these findings are based on univariate comparisons and do not establish independent predictive value.

Correlation Analysis between Inflammatory and Immune Indicators and Coronary Artery Lesions

In the correlation analysis, abnormal coronary artery status was assigned a value of 1, and normal coronary artery status was assigned a value of 0 to investigate the directional associations between the levels and changes in each index and the status of coronary artery disease. The results showed that the change in D-dimer within 72 hours was positively correlated with coronary artery abnormality (r=0.26, *p*=0.005, n=112), indicating that children with an insufficient decrease—or even an increase—in D-dimer were more likely to be in the abnormal coronary artery group. There was a negative correlation between the change in platelet count at 72 hours and coronary artery abnormalities (r=-0.19, *p*=0.006, n=206), which was consistent with the finding that platelet increases were more pronounced in the normal coronary artery group.

After comparing the changes, correlation analysis was used to further examine the relationship between each index and coronary artery abnormality in terms of both direction and strength. Table 7 shows the top-ranked correlation results, and Figure 9 visualizes the direction of the correlation coefficients (positive vs. negative) to distinguish indicators that are positively associated with coronary artery abnormality from those that are inversely associated with it.

Among the post-treatment indicators, the correlation coefficient between white blood cells on day 9 after IVIG and coronary artery abnormalities was 0.36, and the P value was 0.023, suggesting that the elevated white blood cells at later time points after treatment may be associated with coronary artery abnormalities. Consistent with the dynamic changes, the mean count of white blood cells in the abnormal coronary artery group was higher on day 6 and day 9, which may reflect the slow resolution of the inflammatory response in some children after treatment. In general, the biomarkers highly associated with CALs included not only changes in coagulation-related indicators, but also post-treatment changes in platelet counts, white blood cell levels, and inflammatory indicators such as SAA. These findings suggest that coronary artery involvement in KD may reflect a multidimensional clinical phenotype characterized by concurrent alterations in inflammatory, hematologic, and coagulation-related biomarkers.

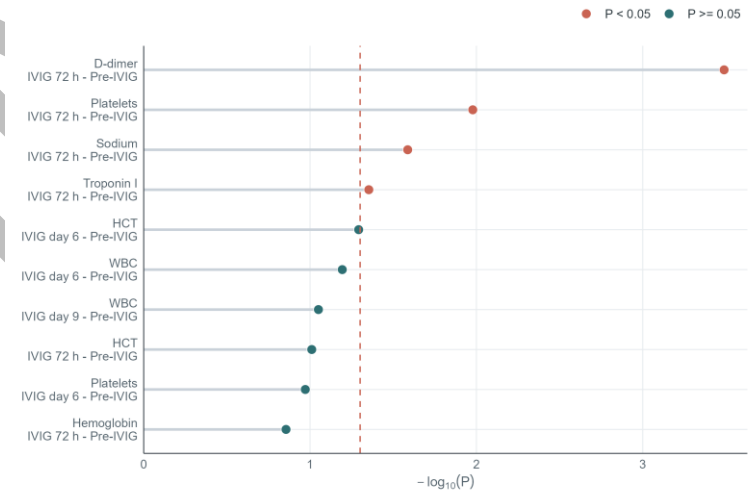

Figure 8. Signal of between-group differences in changes in the main indicators

Published by Tehran University of Medical Sciences (http://ijaai.tums.ac.ir)

Table 6. Between-group comparison of changes in the main indicators

Amount of Change	Mean change in the normal coronary artery group	Mean change in the abnormal coronary artery group	Median in the normal coronary artery group (Q1, Q3)	Median in the abnormal coronary artery group (Q1, Q3)	*p*
D-dimer _IVIG72 h-before IVIG	-1.68	0.25	-0.77 (-1.86, -0.17)	-0.13 (-0.41, 0.14)	<0.001
Platelet _IVIG72 h-before IVIG	188.4	125.9	176.0 (93.00, 271.0)	142.0 (25.00, 206.0)	0.011
Sodium _IVIG72 h-before IVIG	2.79	4.98	2.65 (0.38, 5.17)	5.80 (1.50, 9.03)	0.026
Troponin I_IVIG72 h-before IVIG	-0.51	-1.19	-0.22 (-0.47, -0.12)	-0.38 (-1.20, -0.21)	0.044
Hct_ivig6d-before IVIG	-0.69	-3.12	-0.15 (-2.42, 2.70)	-2.20 (-2.80, 0.40)	0.051
White blood cells _IVIG6d-before IVIG	-0.53	5.23	-1.60 (-6.13, 6.15)	3.81 (-3.51, 13.76)	0.064
Leukocytes _IVIG9d-before IVIG	-1.10	4.35	-2.45 (-7.08, 4.79)	2.78 (-0.87, 6.75)	0.089
Hct_ivig72 h-before IVIG	-1.36	-2.05	-0.60 (-3.30, 1.35)	-2.15 (-4.83, 0.48)	0.098

Amount of change=index value at the time point after treatment − index value before IVIG treatment; P values were calculated using the normal approximation of the Mann-Whitney U test.

Table 7. Ranking of correlations between inflammatory and immune indicators and coronary artery abnormalities

Variables	Type	n	Correlation coefficient r	*p*
D-dimer _IVIG72 h-before IVIG	Amount of change	112	0.26	0.005
Platelet _IVIG72 h-before IVIG	Amount of change	206	-0.19	0.006
IVIG9d white blood cells	Raw metrics	40	0.36	0.023
White blood cells _IVIG6D-before IVIG	Amount of change	74	0.25	0.031
IVIG6d white blood cells	Original index	74	0.24	0.040
IVIG72h alanine	Original metrics	106	0.20	0.040
Random blood glucose before IVIG	Raw metrics	194	0.14	0.046
SAA before IVIG	Raw metrics	185	0.15	0.047
Total serum bilirubin before IVIG	Raw indicators	205	0.14	0.052
Sodium _IVIG72 h-before IVIG	Amount of change	76	0.22	0.056

Abnormal coronary artery status=1, normal coronary artery status=0; correlation coefficients are approximate point-biserial Pearson correlation coefficients.

IN PRESS

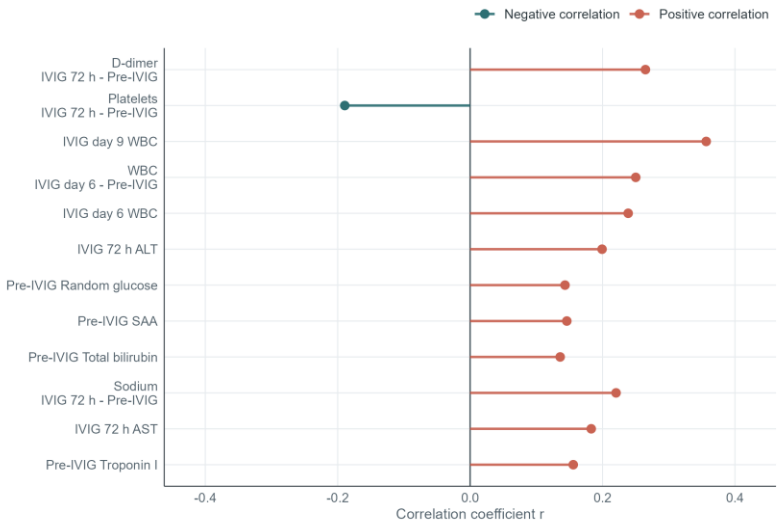

Figure 9. Ranking of correlations between inflammatory and immune indicators and coronary artery abnormalities

DISCUSSION

This retrospective study enrolled a total of 208 children diagnosed with KD between January 2020 and September 2025, including 159 with normal coronary arteries and 49 patients complicated by CALs. We systematically analyzed the baseline levels, dynamic changes, and variation magnitudes of multiple inflammatory, immune, coagulation and biochemical indicators before and after IVIG treatment, and further explored their correlations with CALs. Overall results demonstrated that static baseline indicators alone before IVIG treatment had limited value in distinguishing CALs, whereas the dynamic trends and early change values of laboratory markers after IVIG intervention were closely associated with the occurrence of coronary artery damage in children with KD.

First of all, all enrolled children presented with pronounced acute inflammatory responses at baseline before IVIG administration, manifested as elevated WBC count, platelet count, CRP, SAA, PCT and IL-6. A considerable number of indicators showed a right-skewed distribution with pronounced outliers, especially SAA, PCT and IL-6, which exhibited large standard deviations and reflected heterogeneity in inflammatory intensity among individual patients.[14-16] For this reason, we used both mean ± standard deviation and median (interquartile range) for descriptive statistics, and applied the non-parametric Mann-Whitney U test for intergroup comparison to ensure the robustness of statistical analysis. Consistent with most previous clinical studies, our data revealed that no statistically significant intergroup differences were observed in any single inflammatory or immune indicator between the normal coronary artery group and the CAL group at baseline (all $P > 0.05$), including SAA and platelet count, which had relatively low P values but still did not reach statistical significance.17,18 This finding supports the notion that acute inflammatory activation is a universal pathological feature of KD patients in the acute phase.3,19 Since all patients experience a pronounced inflammatory response in the early stage of the disease, relying solely on pre-treatment single-time-point laboratory results is unlikely to effectively identify children at high risk of CALs.

Regarding the dynamic trajectories of indicators after IVIG treatment, distinct variation patterns were observed among different biomarkers.[20] For WBC and neutrophils, both groups experienced a marked decline at 72 hours after IVIG, indicating that standard IVIG therapy can rapidly suppress acute cellular inflammation.[21] However, WBC levels in the CAL group rebounded markedly on day 6 and day 9 after treatment and remained persistently higher than those in the normal coronary group. This inflammatory rebound suggests that the inflammatory response in KD children with CALs resolves more slowly, and immune dysregulation cannot be fully corrected by conventional IVIG therapy in a timely manner. Unlike inflammatory cells, platelets showed a continuous upward trend after IVIG treatment in both groups, which is a typical hematological feature during the recovery phase of KD.

IN PRESS

Notably, platelet levels in the CAL group were consistently higher throughout the observation period, implying that excessive platelet activation and increased platelet production may contribute to the progression of coronary vascular injury. Regarding the acute-phase inflammatory proteins CRP and SAA, both decreased sharply after IVIG intervention in all patients, reflecting the general remission of systemic inflammation. However, their similar declining trends between the two groups meant that these two classic inflammatory markers have limited capability to differentiate CAL status.

Calculating the change values of indicators (post-treatment value minus baseline value) is a more intuitive method to evaluate the responses of various markers to IVIG treatment. In the present study, the change in D-dimer at 72 hours post-IVIG showed the most prominent intergroup difference ($P < 0.001$).[22,23] The D-dimer level decreased markedly in children with normal coronary arteries, while the reduction was minimal in the CAL group. D-dimer is a core marker reflecting in vivo thrombosis and fibrinolytic activity. Coronary endothelial injury caused by persistent inflammation can activate the coagulation system and induce micro thrombosis in coronary vessels. Our results indicate that incomplete recovery of fibrinolytic function and an insufficient decline in D-dimer within 72 hours after IVIG are strongly correlated with CALs. In addition, the change values of platelet count, serum sodium and cardiac troponin I at 72 hours also differed significantly between groups ($p<0.05$). These findings reveal that not only conventional inflammatory indicators, but also coagulation function, electrolyte balance and myocardial injury markers are involved in the pathological process of KD complicated by CALs, forming a multi-system abnormal profile.[24,25]

Point-biserial Pearson correlation analysis was further performed to determine the relationship between each indicator and the occurrence of CALs. The change in D-dimer at 72 hours was positively correlated with CALs ($r=0.26$, $p=0.005$), meaning that children with a smaller decline in D-dimer were more likely to develop coronary lesions. The change in platelet count at 72 hours was negatively correlated with CALs ($r=-0.19$, $p=0.006$), which was consistent with the results of intergroup comparison of change values. Moreover, the WBC level on day 9 after IVIG had a moderate positive correlation with CALs ($r=0.36$, $p=0.023$), further supporting the notion that delayed resolution of inflammation is an important feature of KD patients with coronary damage.[26,27] Persistent inflammation may induce coronary endothelial injury and coagulation activation, while platelet activation may further amplify vascular inflammation. Collectively, CALs in KD are not caused by abnormality in a single marker, but result from the combined changes of multiple inflammatory, immune, coagulation and myocardial-related indicators. From a clinical perspective, dynamic changes in D-dimer, platelet count, and WBC may serve as adjunctive markers for CAL risk assessment and help identify children who would benefit from closer clinical and echocardiographic follow-up.

This study has several strengths. Firstly, we enrolled 208 consecutive KD patients over a period of nearly 5 years, which reflected the real clinical characteristics of KD in recent years. Secondly, four sequential time points (pre-IVIG, 72 h, day 6, day 9 post-IVIG) were set to dynamically monitor indicators, which completely depicted the evolution of inflammation after IVIG treatment. Thirdly, by integrating baseline levels, dynamic trends and magnitude of change, and by expanding the analysis to coagulation, myocardial and biochemical indicators, we established a more comprehensive framework for CAL risk assessment.

However, several limitations of this study must be acknowledged. First, this was a single-center retrospective, observational study, which may be subject to selection bias, and the generalizability of our findings needs to be validated by multicenter research with large samples. In addition, local patient demographics, referral patterns, laboratory practices, and treatment protocols may have influenced the observed biomarker trends and potentially limit the generalizability of our findings to other clinical settings. Only hospitalized patients were enrolled, whereas patients with mild KD managed in outpatient settings were excluded, so our conclusions have limited external generalizability to populations with mild KD. Second, the group sizes were unequal (159 vs. 49), and the number of available observations decreased gradually at the later follow-up time points due to missing laboratory data, which may affect the stability and robustness of statistical results. Variations in the sampling intervals around the predefined time points may also have affected the interpretation of dynamic biomarker changes. Therefore, we attempted to use linear mixed-effects models for repeated-measures analysis; however, substantial missing data resulted in convergence

IN PRESS

difficulties and limited the reliability of longitudinal trajectory comparisons. Third, this study only used univariate comparison and correlation analysis, and did not establish multivariate regression models to screen for independent risk factors for CALs. Key confounders including the fever-to-IVIG interval and IVIG resistance were unavailable in archived records and could not be adjusted for; multiple univariate tests may have increased the risk of type I error, and stable ROC diagnostic thresholds could not be calculated due to limited subgroup samples. Fourth, we did not detect immune cell subsets or inflammatory signaling pathways, so the specific molecular mechanisms underlying indicator changes remain to be further explored. The observed correlations do not establish causality, and hypothesized inflammatory-coagulation pathways require further validation. Fifth, coronary classification was based on the age-specific cutoff values recommended in the 2020 consensus rather than body surface area–adjusted Z scores, and incomplete long-term echocardiographic follow-up after discharge precluded assessment of long-term coronary outcomes. In clinical practice, clinicians should not rely merely on baseline inflammatory indicators to assess the risk of CALs in KD children. Dynamic monitoring of multiple markers within 1 week after IVIG treatment, especially the change in D-dimer and platelet count at 72 hours, and WBC levels at later follow-up, may provide additional information for the early identification of patients at increased risk of coronary artery lesions. Further prospective studies with larger sample sizes[1,28,29] and multivariable analysis are required to optimize the predictive model for CALs and inform individualized intervention and follow-up strategies for KD.

Through analysis of data from 208 children with KD, this study confirmed that single baseline inflammatory and immune indicators before IVIG treatment could not effectively distinguish patients with coronary artery lesions. The changes in D-dimer and platelet count at 72 hours after treatment, as well as white blood cell levels during the late phase, were significantly correlated with coronary damage. These indicators are only exploratory candidates and need external multi-center prospective validation for routine clinical application. Overall, dynamic changes in laboratory markers after IVIG intervention may provide greater clinical value than static baseline measurements for assessing coronary lesion risk and can provide reliable evidence for clinical monitoring and risk stratification.

STATEMENT OF ETHICS

This retrospective observational study was approved by the Institutional Ethics Committee of Bengbu First People's Hospital (Ethics Approval No.: BBYY2024011) and was conducted in accordance with the Declaration of Helsinki. The committee waived the requirement for written informed consent because the study used only anonymized clinical, laboratory, and imaging data and did not interfere with routine patient care. All patient information was de-identified to protect privacy and confidentiality.

FUNDING

This study was supported by the Key Research Project of the Anhui Provincial Health Commission (Grant No. AHWJ2022a040), the Key Scientific Research Projects of Bengbu Municipal Health Commission (Grant Nos. BBWK2024A102, BBWK2025A07), and the Guiding Science and Technology Innovation Project of Bengbu City (Grant No. 20230108).

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

ACKNOWLEDGMENTS

This study was supported by the Key Research Project of the Anhui Provincial Health Commission (Grant No. AHWJ2022a040), the Bengbu Municipal Key Health Research Projects (Grant Nos. BBWK2024A102 and BBWK2025A07), and the Bengbu Municipal Science and Technology Innovation Guidance Project (Grant No. 20230108).

DATA AVAILABILITY

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

AI ASSISTANCE DISCLOSURE

Not applicable.

IN PRESS

REFERENCES

1. Zhang X, He Y, Shao Y, Hang B, Xu Z, Chu M. Factors affecting the duration of coronary artery lesions in patients with the Kawasaki disease: a retrospective cohort study. Pediatr Rheumatol. 2021;19(1):96.
2. Seki M, Minami T. Kawasaki Disease: Pathology, Risks, and Management. Vasc Health Risk Man. 2022;18:407-16.
3. Navarro FB, Mulinari L. Kawasaki disease: Surgical treatment. J Cardiac Surg. 2022;37(12):4340-1.
4. Rahiman HH, Kar SS, Mohsin MM, Dube R, Alboot NMJ. Atypical Kawasaki Disease in a 5-month-old Infant. Ann Afr Med. 2025;24(4):944-8.
5. Onoki T, Metoki T, Iwasawa S, Kawano K, Kimura M, Kure S, et al. Two Distinct Cases of Adult-onset Kawasaki Disease. Intern Med. 2022;61(23):3525-9.
6. Huang T, Peng Q, Zhang Y, Zhu Z, Fan X. The Systemic Immune-Inflammation Index (SII) and coronary artery lesions in Kawasaki disease. Clin Exp Med. 2024;24(1):4.
7. Chantasiriwan N, Silvilairat S, Makonkawkeyoon K, Pongprot Y, Sittiwangkul R. Predictors of intravenous immunoglobulin resistance and coronary artery aneurysm in patients with Kawasaki disease. Paediatr Int Child H. 2018;38(3):209-12.
8. Chen Y, Yang M, Zhang M, Wang H, Zheng Y, Sun R, et al. Single-Cell Transcriptome Reveals Potential Mechanisms for Coronary Artery Lesions in Kawasaki Disease. Arterioscler Thromb Vasc Biol. 2024;44(4):866-82.
9. Tsoukas P, Yeung RSM. Kawasaki Disease-Associated Cytokine Storm Syndrome. Adv Exp Med Biol. 2024;1448:365-83.
10. Bordea MA, Costache C, Grama A, Florian AI, Lupan I, Samasca G, et al. Cytokine cascade in Kawasaki disease versus Kawasaki-like syndrome. Physiol Res. 2022;71(1):17-27.
11. Ferencik M, Mayrhofer T, Lu MT, Bittner DO, Emami H, Puchner SB, et al. Coronary Atherosclerosis, Cardiac Troponin, and Interleukin-6 in Patients With Chest Pain: The PROMISE Trial Results. JACC. Cardiovascular Imaging. 2022;15(8):1427-38.
12. Yan ZS, Zhang NC, Li K, Sun HX, Dai XM, Liu GL. Upregulation of long non-coding RNA myocardial infarction-associated transcription is correlated with coronary artery stenosis and elevated inflammation in patients with coronary atherosclerotic heart disease. Kaohsiung J Med Sci. 2021;37(12):1038-47.
13. Liu X, Dong M, Li T, Wang J. Correlation of circulating fibroblast growth factor 21 levels with inflammatory factors and the degree of coronary artery stenosis in patients with acute myocardial infarction. Cytokine. 2024;178:156591.
14. Huang XB, Zhao S, Liu ZY, Xu YY, Deng F. Serum amyloid A as a biomarker for immunoglobulin resistance in Kawasaki disease. Ann Med. 2023;55(2):2264315.
15. Feng H, Chai H, Li D, Shi C, Xu Y, Liu Y. Expression of PCT, BNP and Inflammatory Factors in Children with Kawasaki Disease and Their Correlation with Coronary Artery Lesions. Altern Ther Health M. 2024;30(1):302-6.
16. Wang QQ, Zheng LY, Zhao S. The effects of glucocorticoid plus intravenous immunoglobulin (IVIG) vs IVIG alone on platelet activation in children with Kawasaki disease. Turk J Pediatr. 2023;65(4):640-9.
17. Zhang X, Ya-Wang S, Ya-Lan Z, Yi L. Serum Lp-PLA2 Level Predicting Coronary Artery Lesions in Children with Kawasaki Disease. Heart Surg Forum. 2021;24(4):E611-8.
18. Liang J, Ma Y, Han N, Ai K, Zhang H, Zhang J. MRI Diagnosis of Coronary Artery Lesions in Children With Kawasaki Disease and Their Correlation With Inflammatory Factors. J Magn Reson Imaging. 2024;60(6):2369-77.
19. Burns JC. The etiologies of Kawasaki disease. J Clin Invest. 2024;134(5):e176938.
20. Lv D, Zhao X, Li X, Meng T, Zhang W. Evaluation and Dynamic Change Characteristics of Cytokines in Severe Fever With Thrombocytopenia Syndrome Infected Patients. J Med Virol. 2026;98(2):e70826.
21. Wagner Y, Adam D, Pomeranz Engelberg G, Pomeranz A, Messinger YH. HLH Syndrome in a Community Hospital: The Challenge of an Early Diagnosis. Pediatr Health Med T. 2024;15:111-20.
22. Cavalera MA, Gernone F, Uva A, D'Ippolito P, Roura X, Paltrinieri S, et al. Effect of domperidone (leisguard®) on antibody titers, inflammatory markers and creatinine in dogs with leishmaniosis and chronic kidney disease. Parasite Vector. 2021;14(1):525.
23. Li Y, Chen B. Gamma globulin combined with acyclovir for children with infectious mononucleosis and their effect on immune function. Am J Transl Res. 2023;15(6):4399-407.
24. Ajmi H, Ghorbel S, Ezzi O, Mabrouk S, Mansour K, Kahloul N, et al. Coronary artery aneurysm in Kawasaki disease and its risk factors: a retrospective study about 65 Tunisian children. Ann Cardiol Angeiol (Paris). 2022;71(2):86-9.
25. Hwang B. Echocardiographic Evaluation in Kawasaki Disease. J Med Ultrasound. 2025;33(3):206-15.

IN PRESS

26. Jone PN, Romanowicz J, Browne L, Malone LJ. Imaging Evaluation of Kawasaki Disease. Curr Cardiol Rep. 2022;24(10):1487-94.
27. Bajolle F. Diagnosis and management of Kawasaki disease. La Revue Du Praticien. 2023;73(2):179-86.
28. Zhao J, Ma Y, Xin L, Wang L, Gao H. Impact of intravenous immunoglobulin treatment on peripheral blood cells in children with Kawasaki disease complicated with coronary artery lesion. Ital J Pediatr. 2025;51(1):44.
29. Cai JH, Tang M, Zhang HX, Dan Luo E, Zhang R, Shuai SP, et al. Therapeutic Window of Intravenous Immunoglobulin (IVIG) and its correlation with IVIG-resistant in Kawasaki Disease: a retrospective study. J Pediat-Brazil. 2023;99(2):161-7.

IN PRESS