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Original Article

Do hematologic parameters predict coronary artery involvement in children with Kawasaki disease? A retrospective study

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ABSTRACT

Objective: Kawasaki disease (KD) is an acute systemic vasculitis of childhood that may lead to coronary artery involvement (CAI) if not promptly treated. Early identification of laboratory predictors for coronary complications is essential. This study aimed to identify early laboratory predictors of coronary complications in KD, to help clinicians assess risk during the acute phase.

Material and Methods: We retrospectively analyzed 38 pediatric KD patients. Clinical and laboratory data—including hemoglobin (Hb), hematocrit (Htc), white blood cell count (WBC), platelet counts (PLT), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), liver enzymes, and Harada scores—were collected and compared between patients with and without CAI. Receiver operating characteristic (ROC) and logistic regression analyses were performed.

Results: The mean age was 35.0±32.6 months, with 65.8% male. CAI was observed in 16 of 38 patients (42.1%). No statistically significant differences were found in the laboratory values at diagnosis between patients with and without CAI.

Conclusion: Routine hematological and inflammatory markers at admission were not predictive of CAI in KD. These findings highlight the need for high clinical suspicion in incomplete cases and suggest that incorporating clinical features and risk scores may improve early risk stratification.

Keywords: Blood cell count, coronary aneurysm, C-reactive protein, Kawasaki disease

INTRODUCTION

Kawasaki disease (KD) is an acute, self-limited vasculitis predominantly affecting children under five years of age (1, 2). It is now the leading cause of acquired heart disease in children in developed countries (3, 4). KD is characterized by fever plus a constellation of clinical signs and has a marked predilection for the coronary arteries (5). Although the exact cause remains unknown, an exaggerated immune response to an infectious trigger in genetically susceptible hosts is thought to underlie its pathogenesis (6). Timely administration of intravenous immunoglobulin (IVIG) within the acute phase dramatically reduces the risk of coronary artery aneurysms (CAA) from an estimated 20–25% in untreated cases to around 2–4% in those treated with IVIG (7, 8).

Despite appropriate therapy, a subset of children still develop coronary artery involvement (CAI). Incomplete presentations of KD, characterized by fewer diagnostic criteria, are prone to diagnostic delays and have been associated with a higher risk of CAI (9). Laboratory features of acute KD such as elevated CRP and ESR, leukocytosis, mild anemia and hypoalbuminemia reflect intense systemic inflammation but their predictive value for coronary outcomes remains unclear (10-12).

Given this uncertainty, we conducted a retrospective study of children with KD to evaluate whether baseline hematological parameters are associated with CAI. We also examined the impact of incomplete clinical presentation on coronary risk. Our aim was to identify any early laboratory predictors of coronary complications in KD, which could help guide clinicians in risk stratification during the acute phase.

MATERIALS and METHODS

This study was a retrospective observational analysis conducted at a Baskent University Ankara Hospital. We reviewed the medical records of children diagnosed with KD between 2003 and 2017. The diagnosis of complete KD was established based on the American Heart Association (AHA) criteria, which require fever for at least five days together with at least four of the five principal clinical features of KD. In cases of

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suspected incomplete KD, patients presented with prolonged fever and fewer than four of the principal clinical features, and the diagnosis was supported by compatible laboratory findings or echocardiographic evidence of KD in the absence of an alternative explanation (13, 14). Patients were categorized as having complete KD or incomplete KD according to these definitions. We excluded patients with known congenital heart disease or other systemic inflammatory conditions that could confound the assessment.

For each patient, we recorded demographic information and clinical presentation (complete vs. incomplete KD). Laboratory data were collected from the initial evaluation at the time of diagnosis. The parameters of interest included hemoglobin (Hb, g/dL), hematocrit (Hct, %), white blood cell count (WBC, per mm^3), and platelet count (PLT, per mm^3) at baseline, as well as in the 1st, 2nd, and 3rd weeks, and at the 1st and 2nd months of follow-up. Inflammatory markers included CRP, time to CRP normalization (days), ESR, and time to ESR normalization (days). In addition, biochemical markers such as aspartate aminotransferase (AST, U/L), alanine aminotransferase (ALT, U/L), gamma-glutamyl transferase (GGT, U/L), and serum albumin (Alb, g/dL) were assessed. Anemia was defined according to age-specific reference ranges of Hb.

Transthoracic echocardiography was performed for all patients at diagnosis (baseline) and repeated during follow-up at approximately 1 month, 2 months, and 6 months after disease onset. The coronary arteries (including the left main, left anterior descending, left circumflex, and right coronary arteries) were imaged, and their internal diameters were measured. Coronary artery Z-scores were calculated using body surface area–adjusted models. CAI was defined and classified based on Z-score criteria: normal if Z-score < 2.0, dilatation (ectasia) if Z-score ≥ 2.0 and < 2.5, small aneurysm if Z-score 2.5–5.0, medium aneurysm if Z-score 5.0–10 (and absolute diameter < 8 mm), and giant aneurysm if Z-score ≥ 10 (or absolute diameter ≥ 8 mm) (15). In our analysis, we grouped coronary outcomes into a binary variable indicating the presence versus absence of any CAI.

We calculated the Harada score for patients who presented within the first 9 days of illness. The Harada scoring system is a clinical tool developed to identify KD patients at higher risk for coronary artery complications, originally described in a Japanese cohort (16). It assigns one point for each of the following risk factors present early in the disease course: (1) age < 12 months, (2) male sex, (3) fever duration ≥ 8 days, (4) PLT count ≥ 350.000/mm^3 at presentation, (5) CRP ≥ 3 mg/dL, (6) ESR ≥ 40 mm/h, (7) WBC count ≥ 12,000/mm^3, and (8) Htc ≤ 35%. A total score ≥ 4 has been used as a threshold to predict increased risk of CAI. In our study, we applied the Harada score only to those patients who were diagnosed and treated within day 9 of illness, in line with its intended use for early risk stratification.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro-Wilk test. Data were presented as mean and standard deviation (SD) or as median with interquartile range (IQR), as appropriate. Categorical variables were summarized as counts and percentages. Group comparisons between patients with and without CAI were conducted using the independent samples t-test. To evaluate potential predictors of CAI, univariate logistic regression analyses were performed for key laboratory variables, including Hb, Htc, WBC count, PLT counts across different timepoints, CRP, ESR, liver function tests (AST, ALT, GGT, Alb) levels. Odds ratios (ORs) with 95% confidence intervals (CIs) and corresponding p-values were reported. ROC curve analyses were conducted to assess the diagnostic performance of selected variables in predicting CAI. The area under the curve (AUC) was calculated for each parameter. A p-value < 0.050 was considered statistically significant.

RESULTS

The demographic and clinical characteristics of patients with KD are presented in Table I. In 2 cases, classification could not be determined due to missing data.

Table I: Demographic and clinical characteristics

Variable	n	Values
Gender (Male)*	38	25 (65.8)
Harada Score ≥4*	38	20 (52.6)
Age (months)†	38	25.0 [11.2–55.0]
Fever (°C)‡	36	39.0 ± 0.6
Duration of fever (days) ‡	36	8.2 ± 3.4
Heart rate (beats/min) ‡	32	124.3 ± 19.7
Systolic BP (mmHg) ‡	30	94.3 ± 8.2
Diastolic BP (mmHg) ‡	30	58.8 ± 6.1
Season at diagnosis*	38	
Spring		10 (26.3)
Summer		14 (36.8)
Autumn		4 (10.5)
Winter		10 (26.3)
Diagnostic Findings*	38	
Lip and oral mucosa changes		33 (86.8)
Conjunctivitis		32 (84.2)
Rash		30 (78.9)
Extremity changes		27 (71.1)
Lymphadenopathy		26 (68.4)
Fever duration (days) ‡	36	8.2 ± 3.4
Time to diagnosis (days) ‡	37	6.4 ± 4.8
Length of hospital stay (days) ‡	35	10.2 ± 7.1
Complet KD*	38	28 (73.7)
Coronary artery involvement*	38	16 (42.1)
Harada score ‡	37	4.5 ± 1.2

:** *n(%),* **†:** *median (IQR),* **‡:** *mean±SD,* ***IQR: *Interquartile Range,* ***BP:*** *Blood pressure,* ***KD:*** *Kawasaki Disease,* ***CAI:*** *Coronary artery involvement,* ***Harada score:*** *a clinical risk score used to predict coronary artery involvement. Calculated only for patients who presented within the first 9 days of illness.*

Table II: Laboratory parameters by coronary artery involvement status

	n	Total*	CAI (+)*	CAI (-)*	p†
Hb (g/dl)	38	11.0±1.05	10.8±1.20	11.1±1.01	0.534
Hct (%)	38	32.5±2.87	31.9±2.82	32.9±2.93	0.315
WBC (x10³/mm³)	38	16.0±6.60	18.0±7.94	14.9±5.73	0.204
PLT (x10³/mm³)	38	467.11±179	471.05±173.00	321.00±169.00	0.362
1st week		682.05±271	747.60±325.00	630.32±206.00	0.405
2nd week		695.69±297	724.17±338.00	664.64±264.00	0.975
3rd week		518.62±290	565.29±267.00	482.33±368.00	0.470
1st month		431.50±123	438.29±132.00	424.71±123.00	0.902
2nd month		404.92±70	414.00±63.00	400.50±90.00	0.886
CRP (mg/L)	37	121.1±83.23	105.0±65.72	139.4±96.20	0.286
Time to CRP decrease (days)	38	13.8±11.39	12.7±8.46	14.6±14.90	0.692
ESR (mm/h)	36	75.5±25.44	73.8±25.92	77.4±25.93	0.895
Time to ESR decrease (days)	38	30.0±20.34	29.0±21.57	30.7±19.39	0.875
AST (U/L)	37	66.6±85.19	77.1±110.88	58.5±58.02	0.988
ALT (U/L)	37	88.4±96.69	110.4±130.50	71.6±47.27	0.951
Alb (mg/dl)	29	3.5±0.51	3.4±0.50	3.5±0.52	0.643
GGT (U/L)	22	94.5±88.55	115.4±87.38	73.5±100.73	0.149

***:** *mean±SD,* **†:** *Independent samplet test*, **CAI:** *Coronary artery involvement,* **Hb:** *Hemoglobin,* **Hct:** *Hematocrit,* **WBC:** *White Blood Cell,* **PLT:** *Platelet,* **CRP:** *C-reactive protein,* **ESR:** *Erythrocyte Sedimentation Rate,* **AST:** *Aspartate Aminotransferase,* **ALT:** *Alanine Aminotransferase,* **Alb:** *Albumin,* **GGT:** *Gamma-Glutamyl Transferase*

Table III: Univariate logistic regression of laboratory parameters for predicting coronary artery involvement

	n	OR	95% CI Lower	95% CI Upper	p
Hb (g/dl)	38	0.759	0.397	1.453	0.405
Hct (%)	38	0.876	0.679	1.132	0.312
WBC (x10³/mm³)	38	1.000	0.999	1.000	0.216
PLT (x10³/mm³)	38	1.000	1.000	1.000	0.651
1st week		1.000	1.000	1.000	0.621
2nd week		1.000	1.000	1.000	0.862
3rd week		1.000	1.000	1.000	0.995
1st month		1.000	1.000	1.000	0.925
2nd month		1.000	1.000	1.000	0.972
CRP (mg/L)	37	0.995	0.987	1.004	0.313
Time to CRP decrease (days)	38	0.986	0.916	1.060	0.696
ESR (mm/h)	36	0.007	–0.043	0.057	0.795
Time to ESR decrease (days)	38	1.001	0.957	1.048	0.950
AST (U/L)	37	1.004	0.994	1.013	0.453
ALT (U/L)	37	1.005	0.996	1.014	0.251
Alb (mg/dl)	29	0.417	0.083	2.089	0.287
GGT (U/L)	11	0.003	–0.007	0.014	0.535

***OR:** Odds ratios,* **CI:** *confidence intervals,* **Hb:** *Hemoglobin,* **Hct:** *Hematocrit,* **WBC:** *White Blood Cell,* **PLT:** *Platelet,* **CRP:** *C-reactive protein,* **ESR:** *Erythrocyte Sedimentation Rate,* **AST:** *Aspartate Aminotransferase,* **ALT:** *Alanine Aminotransferase,* **Alb:** *Albumin,* **GGT:** *Gamma-Glutamyl Transferase*

All patients were treated with IVIG (2 g/kg single infusion) and high-dose aspirin in the acute phase, followed by low-dose aspirin in the subacute phase as per standard protocol.

The laboratory parameters are presented in Table II. Inflammatory markers were elevated in nearly all patients.

CAI were identified in 16 out of 38 patients (42.1%). Most coronary lesions were detected either at diagnosis or during follow-up echocardiography within the first 2–6 weeks of illness. Among the affected patients, 12 had CAA only, two had coronary dilatation (ectasia) without meeting aneurysm criteria, and two patients exhibited both aneurysms and ectasia. For instance, one patient presented with a small aneurysm in the right coronary artery and diffuse ectatic changes in the left coronary system.

Table IV: Association between clinical type and coronary artery involvement

	CAI (+)*	p†
Complet KD (n:28)	11 (39.3)	
Inkomplet KD (n:8)	5 (62.5)	
Total (n:36)	16	0.422

: n(%), †: Fisher's exact test, **CAI: Coronary artery involvement, **KD:** Kawasaki Disease. Among the cases with a known classification of KD (complete or incomplete), CAI data were missing for two patients.*

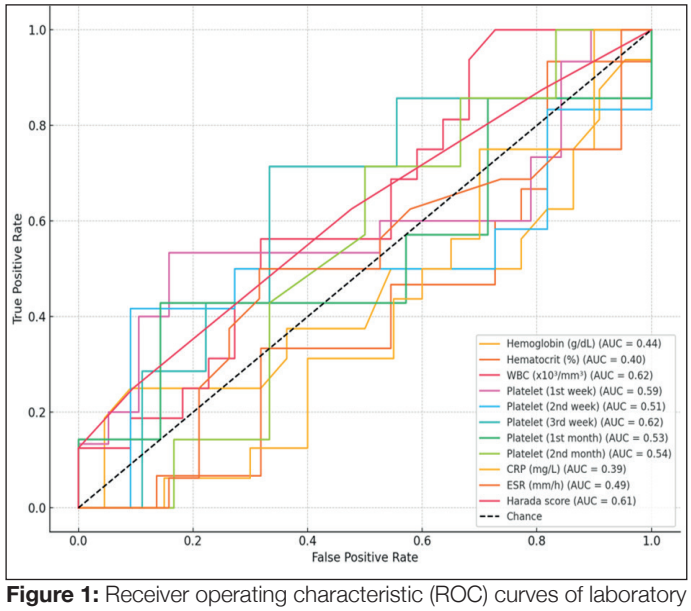

Figure 1: Receiver operating characteristic (ROC) curves of laboratory parameters for predicting coronary artery involvement in Kawasaki Disease. AUC values are shown in parentheses. WBC and platelet values in the third week showed the highest predictive performance, though no parameter exceeded an AUC of 0.65.

Based on Z-score classification, seven aneurysms were categorized as small (Z-score 2.5–5), seven as medium (Z-score 5–10 or absolute diameter 5–8 mm), and two as giant (Z-score ≥10). All patients with CAI received appropriate adjunctive therapy and were closely monitored by pediatric cardiology according to the severity of coronary findings, including continuation of aspirin therapy.

The distributions of key laboratory parameters—including Hb, Htc, WBC, PLT count at various time points, CRP, ESR, and Harada score—were compared between patients with and without CAI (Table II). No statistically significant differences were found in any of these baseline parameters.

ROC analysis was conducted to assess the predictive value of laboratory parameters for CAI (Figure 1). The highest AUC was observed for WBC count (AUC = 0.62), followed by PLT count in the third week (AUC = 0.62) and the Harada score (AUC = 0.61). However, none of the parameters demonstrated strong predictive performance (AUC > 0.70).

In univariate logistic regression analyses, none of the evaluated laboratory parameters—including Hb, Htc, WBC count, PLT count across various time points, CRP, ESR, liver enzymes (AST, ALT, Alb, GGT)—were found to be statistically significant predictors of CAI (Table III).

Among 36 patients with available data, 28 were diagnosed with complete KD and 8 with incomplete KD (Table IV). CAI was observed in 11 of the 28 complete KD cases (40.7%) and in 5 of the 8 incomplete KD cases (83.3%). Although the rate of CAI appeared higher in the incomplete group, the difference did not reach statistical significance (Fisher's exact test, p = 0.422).

DISCUSSION

In this retrospective study of 38 children with KD, we investigated whether initial hematological and inflammatory parameters are associated with the development of CAI. Our findings indicate that traditional laboratory markers measured at presentation – including Hb, Htc, WBC count, PLT count, CRP and ESR, AST, ALT, Alb and GGT – did not show a statistically significant difference between patients who developed coronary artery changes and those who did not. In other words, no routine blood parameter at diagnosis emerged as a clear predictor of coronary complications in our cohort. This is consistent with much of the existing literature, which has shown that while KD is invariably accompanied by systemic inflammation and characteristic laboratory abnormalities, these metrics alone often lack the specificity needed to predict CAI formation (12). The acute phase reactants (CRP, ESR) and blood counts reflect the intensity of inflammation but do not necessarily distinguish which children will have CAI. Our results reinforce the notion that clinicians cannot rely solely on common lab tests at presentation to assess the risk of coronary sequelae in KD.

It is worth noting that some previous studies have reported potential links between certain laboratory extremes and coronary risk, although findings have been variable. For example, extremely elevated CRP levels have been associated with increased likelihood of coronary artery lesions in some analyses (17, 18). A meta-analysis reported that for each 1 mg/L increase in CRP, the odds of coronary artery abnormality increased slightly (OR ~1.02), underscoring CRP as a continuous contributor to risk (19). Similarly, anemia and hypoalbuminemia have been observed more frequently in KD patients who develop CAA in some studies (12). Increased neutrophil counts and higher PLT counts in the subacute phase have also been proposed as risk factors in prior reports (12). However, these associations are not consistently reproducible across all populations. For instance, Rahbari-Manesh et al. (20) found no significant correlation between PLT count or ESR/CRP levels and CAI. Our findings are consistent with previous studies that question the predictive value of individual hematologic markers. The absence of significant differences in our analysis supports the notion that the pathogenesis of coronary lesions in KD is multifactorial and cannot be adequately reflected by a single laboratory parameter measured at a single time point.

The ROC analysis revealed that the WBC count had an AUC value of 0.62. Similarly, the PLT count in the third week and the Harada score showed AUC values in the range of 0.61–0.62. However, none of these parameters demonstrated adequate predictive performance (AUC > 0.70). This suggests that relying solely on laboratory values may be insufficient to predict CAI in KD.

Limitations

This study has several limitations. First, its retrospective and single-center design may limit the generalizability of the findings to broader populations. Most importantly, the sample size (38 patients) was small, which is the main limitation of our study. A small sample size reduces the statistical power to detect potentially meaningful differences, increasing the risk of type II error. In our analysis, this means subtle predictive effects of laboratory variables could have been missed. Additionally, among the cases with a known KD classification (complete or incomplete), CAI data were missing for two patients, which may have affected the analysis of the association between KD type and CAI. Although a wide range of laboratory parameters was evaluated, it should be noted that the dynamic nature of inflammation in KD may not be fully captured by measurements at a single time point. Serial measurements and trends over time might offer more meaningful predictive insights, but this aspect could not be assessed within the scope of this study.

CONCLUSION

In this retrospective study, no significant association was found between initial hematological parameters, inflammatory markers, or liver enzymes and the development of CAI in children diagnosed with KD. These results suggest that the commonly measured laboratory indices at presentation, while reflective of the systemic inflammation in KD, are not reliable predictors of which children will develop CAI. The Harada risk score and other risk factors were examined, but their predictive value in our population was limited, indicating that current risk stratification tools may need to be tailored for different populations or supplemented with additional biomarkers.

In summary, routine blood test abnormalities in KD should alert physicians to the diagnosis but do not on their own predict coronary outcomes. Clinicians should be particularly vigilant in cases of incomplete KD, as delayed diagnosis in these cases can lead to higher rates of CAI. Future prospective studies and larger multicenter analyses are needed to identify early predictors of coronary artery lesions in KD. Such research may include advanced inflammatory markers, genetic factors, or refined clinical scoring systems. Improved risk stratification would allow for more targeted therapy with the ultimate goal of further reducing the incidence of CAI in KD. With ongoing investigations into the pathogenesis and biomarkers of KD, we hope that early identification of high-risk patients will become more feasible, thereby improving outcomes for this serious pediatric condition.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study protocol was reviewed and approved by the Baskent University Institutional Review Board (date:30.07.2025, number: KA25/291)

Contribution of the authors

Study conception and design: ***MKK, İE;*** data collection: MKK; analysis and interpretation of results: ***MKK, İE, EB;*** draft manuscript preparation: ***MKK, İE.*** All authors reviewed the results and approved the final version of the article.

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Conflict of interest

The authors declare that there is no conflict of interest.

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