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Original Article

Elevated levels of NT-proBNP, interferon-γ and tumor necrosis factor-α are associated with coronary artery injury in children with severe Kawasaki disease

Dan Li, Wenjing Zhang, Huiqing Feng, Man Li, Jing Zhao, Yanpeng Xu, Yanping Liu

Department of Cardiology, Baoding Hospital of Beijing Children's Hospital Capital Medical University, 3399 Hengxiang North Street, Lianchi District, Baoding 071000, Hebei, China

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Abstract: Objective: To investigate the association between N-terminal pro-brain natriuretic peptide (NT-proBNP), interferon-γ (IFN-γ), tumor necrosis factor-α (TNF-α), and coronary artery injury in children with Kawasaki disease (KD). Methods: This retrospective study included 42 children with KD admitted to Baoding Hospital of Beijing Children's Hospital Capital Medical University from January 2019 to January 2022 (KD group), and 80 healthy children during the same period (control group). Peripheral blood markers including white blood cell count (WBC), red blood cell count (RBC), hemoglobin (Hb), hematocrit (HCT), mean platelet volume (MPV), platelet distribution width (PDW), platelet count (PLT), NT-proBNP, IFN-γ, TNF-α, C-reactive protein (CRP), and circulating endothelial cell count (CEC) were compared between groups. KD patients were further stratified based on the severity of coronary artery involvement for subgroup analysis. Results: Compared with the control group, the KD group showed significantly higher levels of WBC, PDW, PLT, NT-proBNP, IFN-γ, TNF-α, CRP, and CEC, and significantly lower levels of RBC, Hb, and HCT (all $P < 0.05$). MPV levels did not differ significantly between groups ($P > 0.05$). Among the 42 KD patients, 17 had coronary artery involvement. These patients exhibited significantly higher levels of PDW, NT-proBNP, IFN-γ, TNF-α, CRP, and CEC compared to those without coronary artery injury (all $P < 0.05$). There were no significant differences in WBC, RBC, Hb, HCT, MPV, or PLT between the KD subgroups with and without coronary artery lesions (all $P > 0.05$). The diagnostic sensitivity of NT-proBNP, IFN-γ, and TNF-α for coronary artery injury in KD was 96.18%, 83.47%, and 65.20%, respectively, with specificities of 78.71%, 82.16%, and 81.98%, respectively. The area under the ROC curve for NT-proBNP, IFN-γ, and TNF-α was 0.969, 0.875, and 0.704, respectively. Logistic regression analysis identified elevated IFN-γ, TNF-α, CRP, and CEC as independent risk factors for coronary artery injury in children with KD (all $P < 0.05$). Conclusions: NT-proBNP, IFN-γ, and TNF-α levels are significantly elevated in children with severe KD and are closely associated with coronary artery injury. These biomarkers may serve as early predictors of coronary artery complications in KD.

Keywords: N-terminal pro-brain natriuretic peptide, interferon-γ, tumor necrosis factor-α, Kawasaki disease, coronary artery injury

Introduction

Kawasaki disease (KD) is an acute vasculitis syndrome that primarily affects children, characterized by non-specific inflammation of small- and medium-sized arteries as the main pathological feature [1]. Clinical manifestations include fever, rash, and non-suppurative cervical lymphadenopathy, and severe complications may also occur [2]. Among these, coronary artery lesions (CAL) are the most critical complication influencing prognosis and have become a major focus of clinical concern [3]. If left untreated, CAL can progress to coronary artery aneurysm, ischemic myocardial infarction, or even become life-threatening. Therefore, early prediction of coronary artery injury in KD patients is essential [4, 5].

Traditional diagnostic methods mainly depend on clinical symptoms and laboratory parameters. However, due to the wide variability and lack of specificity in KD presentations, diagnosis remains challenging. Biomarkers such as N-terminal pro-brain natriuretic peptide (NT-proBNP), interferon-γ (IFN-γ), and tumor

necrosis factor-α (TNF-α) have emerged as promising indicators, offering early warning signals that may support accurate diagnosis and risk assessment. Their detection is relatively rapid and simple, making them highly valuable for clinical application.

Recent studies have found that elevated levels of inflammatory markers including NT-proBNP, IFN-γ, and TNF-α are associated with an increased risk of CAL in KD patients [6]. NT-proBNP is a well-established biomarker for heart failure [7]; IFN-γ is a potent antiviral cytokine secreted primarily by T cells and NK cells [8]; and TNF-α is a pro-inflammatory cytokine mainly produced by activated monocytes and macrophages [9].

To identify potential predictive markers, this study was conducted to evaluate the diagnostic value of NT-proBNP, IFN-γ, and TNF-α for coronary artery injury in children with severe KD. This study provides a comprehensive analysis of the roles of NT-proBNP, IFN-γ, and TNF-α in KD and its related coronary artery injury. These biomarkers not only reflect the systemic inflammatory response but also correlate with the extent of myocardial damage, offering a novel perspective for the clinical diagnosis and prognostic evaluation of KD. Furthermore, through stratified analysis based on the severity of CAL, this study highlights the specific biomarker changes associated with coronary injury, laying a scientific foundation for early identification and intervention in high-risk KD children. Overall, the findings may contribute to a deeper understanding of KD pathogenesis and support evidence-based strategies for its early diagnosis and treatment.

Materials and methods

Case selection

This retrospective study included 42 children diagnosed with KD who were admitted to Baoding Hospital of Beijing Children's Hospital Capital Medical University from January 2019 to January 2022 (KD group), and 80 healthy children during the same period as the control group.

Inclusion criteria: (1) Diagnosis met the criteria established by the Japanese Kawasaki Disease Research Committee (5th edition, 2002) [10], which include: bilateral bulbar conjunctival injection without purulent discharge, fissured lips, strawberry tongue, non-suppurative cervical lymphadenopathy, and erythema/edema of the hands and feet. (2) Children aged 1-9 years in the acute phase of KD. (3) All children in the KD group underwent echocardiography to assess for coronary artery dilation or aneurysm. Patients were further categorized into coronary artery lesion (CAL) and without-CAL subgroups based on findings. (4) The control group consisted of age-matched healthy children during the same period. (5) The study protocol complied with ethical standards.

Exclusion criteria: (1) Children who had received vitamin D, gamma globulin, or related drug treatments prior to admission [11]. (2) Children with hematologic disorders. (3) Children with other immune-related diseases. (4) Children diagnosed with scarlet fever. (5) Children with sepsis or other infectious diseases.

Data collection

The following peripheral blood parameters were measured and compared between the KD and control groups: white blood cell count (WBC), red blood cell count (RBC), hemoglobin (Hb), hematocrit (HCT), mean platelet volume (MPV), platelet distribution width (PDW), platelet count (PLT), NT-proBNP, IFN-γ, TNF-α, C-reactive protein (CRP), and circulating endothelial cell count (CEC).

For the KD group, 5 mL of fasting venous blood was collected the day after admission. Samples were centrifuged at room temperature using a desktop low-speed centrifuge (TD5A) at 5500 rpm for 10 minutes to separate serum. For the control group, fasting venous blood was collected and processed in the same manner. Serum levels of NT-proBNP, IFN-γ, and TNF-α were measured using enzyme-linked immunosorbent assay kits (Wuhan Aidi Anti-Biotechnology Co., Ltd.).

Echocardiography was performed on children with KD using an ultrasound diagnostic system (Tianjin Shenghong Medical Equipment Co., Ltd.). The coronary arteries were examined via transthoracic ultrasound to assess vascular structure and detect coronary lesions.

Statistical methods

All data were analyzed using SPSS version 21.0. Continuous variables such as WBC, PDW, PLT, NT-proBNP, IFN-γ, TNF-α, CRP, and CEC

Table 1. Comparison of general data between KD and control groups

Group	KD group (n = 42)	Control group (n = 80)	t/χ^2	P
Age (years)	5.72 ± 1.80	5.40 ± 1.94	0.887	0.377
Weight (kg)	20.61 ± 3.10	19.85 ± 2.96	1.326	0.187
Height (cm)	114.96 ± 5.21	113.53 ± 5.71	1.354	0.178
Gender (%)			1.243	0.265
Male	29 (69.05)	47 (58.75)		
Female	13 (30.95)	33 (41.25)		
HR (times/min)	110.4 ± 8.9	108.7 ± 8.1	1.064	0.289
SBP (mmHg)	112.1 ± 7.5	110.6 ± 7.2	1.078	0.283
DBP (mmHg)	69.2 ± 6.0	68.5 ± 6.5	0.580	0.563

HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 2. Comparison of peripheral blood indexes between KD and control groups ($\bar{x} \pm s$)

Group	KD group (n = 42)	Control group (n = 80)	T	P
WBC ($\times 10^9$/L)	13.82 ± 2.77	7.61 ± 1.43	16.361	0.000
RBC ($\times 10^{12}$/L)	4.09 ± 0.60	4.72 ± 0.74	-4.755	0.000
Hb (g/L)	113.0 ± 8.3	118.5 ± 7.6	-3.679	0.000
HCT (%)	33.86 ± 4.40	37.92 ± 4.81	-4.559	0.000
MPV	10.29 ± 1.67	9.84 ± 1.33	1.623	0.107
PDW (fL)	13.41 ± 2.86	10.81 ± 2.30	5.446	0.000
PLT ($\times 10^9$/L)	411.75 ± 59.81	208.24 ± 32.55	24.376	0.000

KD, Kawasaki disease; WBC, white blood cell; RBC, red blood cell; Hb, hemoglobin; HCT, hematocrit; MPV, mean platelet volume; PDW, platelet distribution width; PLT, platelet count.

Table 3. Comparison of inflammatory indicators and NT proBNP measurement values between KD and control groups ($\bar{x} \pm s$)

Group	KD group (n = 42)	Control group (n = 80)	T	P
NT-proBNP (ng/L)	1342.7 ± 498.1	114.8 ± 34.8	22.029	0.000
IFN-γ (ng/L)	30.88 ± 5.83	21.75 ± 4.20	9.942	0.000
TNF-α (ng/L)	67.30 ± 16.41	32.81 ± 8.54	15.296	0.000
CRP (mg/L)	96.43 ± 18.03	4.28 ± 1.40	45.623	0.000
CEC (n/μL)	4.28 ± 1.40	1.81 ± 0.66	13.254	0.000

KD, Kawasaki disease; NT-proBNP, N-terminal pro-brain natriuretic peptide; IFN-γ, interferon-γ; TNF-α, tumor necrosis factor-α; CRP, C-reactive protein; CEC, count of endothelial cell.

were expressed as mean ± standard deviation ($\bar{x} \pm s$) when normally distributed. Group comparisons were made using independent sample t-tests. Categorical variables (e.g., gender) were described as frequencies and percentages, and comparisons were made using the chi-square (χ^2) test.

Receiver operating characteristic (ROC) curve analysis was used to evaluate the diagnostic value of NT-proBNP, IFN-γ, and TNF-α for coronary artery injury in KD. Logistic regression analysis was employed to identify risk factors associated with coronary artery lesions in KD. A *p*-value < 0.05 was considered statistically significant.

Results

Comparison of basic data between the KD and control groups

There were no statistically significant differences in baseline characteristics between the KD group and the control group (all P > 0.05). See **Table 1**.

Comparison of peripheral blood indexes between the KD group and control group

The levels of WBC, PDW, and PLT were significantly higher in the KD group compared to the control group, while RBC, Hb, and HCT levels were significantly lower (all P < 0.05). No significant difference in MPV was observed between the two groups (P > 0.05). See **Table 2**.

Comparison of inflammatory indexes between the KD group and control group

The levels of NT-proBNP, IFN-γ, TNF-α, CRP, and CEC were significantly elevated in the KD group compared to the control group (all P < 0.05). See **Table 3**.

Comparison of peripheral blood indexes in KD children with or without CAL

Among the 42 children in the KD group, 17 were diagnosed with CAL based on echocar-

Table 4. Comparison of peripheral blood indexes of KD children with or without coronary artery lesions ($\bar{x} \pm s$)

Index	Coronary artery lesions (n = 17)	Without coronary artery lesions (n = 25)	T	P
WBC (× 10^9/L)	14.10 ± 2.58	13.63 ± 2.61	0.575	0.568
RBC (× 10^{12}/L)	4.01 ± 0.55	4.14 ± 0.58	-0.728	0.471
Hb (g/L)	112.1 ± 7.9	113.6 ± 8.1	-0.595	0.555
HCT (%)	32.76 ± 4.26	34.61 ± 4.31	-1.372	0.178
MPV	10.62 ± 1.61	10.07 ± 1.45	1.154	0.255
PDW (fL)	14.46 ± 2.67	12.70 ± 2.70	2.083	0.044
PLT (× 10^9/L)	418.62 ± 56.60	407.08 ± 52.74	0.676	0.503

KD, Kawasaki disease; WBC, white blood cell; RBC, red blood cell; Hb, hemoglobin; HCT, hematocrit; MPV, mean platelet volume; PDW, platelet distribution width; PLT, platelet count.

Table 5. Comparison of inflammatory markers and NT proBNP measurement values in KD children with or without coronary artery lesions ($\bar{x} \pm s$)

Index	Coronary artery lesions (n = 17)	Without coronary artery lesions (n = 25)	T	P
NT-proBNP (ng/L)	1981.6 ± 441.4	908.2 ± 289.5	9.536	0.000
INF-γ (ng/L)	35.41 ± 5.76	27.80 ± 4.30	4.904	0.000
TFN-α (ng/L)	74.52 ± 15.28	62.39 ± 12.67	2.801	0.008
CRP (mg/L)	113.20 ± 15.20	85.03 ± 14.94	5.956	0.000
CEC (n/μL)	5.91 ± 1.28	3.17 ± 1.14	7.276	0.000

KD, Kawasaki disease; NT-proBNP, N-terminal pro-brain natriuretic peptide; IFN-γ, interferon-γ; TNF-α, tumor necrosis factor-α; CRP, C-reactive protein; CEC, count of endothelial cell.

diography. PDW levels were significantly higher in KD children with CAL than in those without ($P < 0.05$). There were no significant differences in WBC, RBC, Hb, HCT, MPV, or PLT between the two subgroups (all $P > 0.05$). See **Table 4**.

Comparison of inflammatory indexes in KD children with or without CAL

The levels of NT-proBNP, IFN-γ, TNF-α, CRP, and CEC were significantly higher in KD children with CAL than in those without CAL (all $P < 0.05$). See **Table 5**.

Diagnostic value of NT-proBNP, IFN-γ, and TNF-α for CAL in KD

ROC curve analysis was conducted to evaluate the diagnostic value of NT-proBNP, IFN-γ, and TNF-α for CAL in KD. The sensitivity of NT-proBNP, IFN-γ, and TNF-α was 96.18%, 83.47%, and 65.20%, respectively; the specificity was 78.71%, 82.16%, and 81.98%, respectively. The area under the ROC curve (AUC) was 0.969, 0.875, and 0.704, respectively. See **Figure 1** and **Table 6**.

Risk factor analysis for CAL in KD

A logistic regression model was constructed using PDW, NT-proBNP, IFN-γ, TNF-α, CRP, and CEC as independent variables, with CAL as the dependent variable. The results indicated that elevated levels of IFN-γ, TNF-α, CRP, and CEC were independent risk factors for CAL in children with KD (all $P < 0.05$). See **Table 7**.

Discussion

KD is an acute febrile illness of childhood characterized by systemic vasculitis. High-grade fever (often > 39°C) is usually the initial clinical manifestation. Coronary artery involvement occurs in approximately 5-9% of KD cases and may result in serious complications such as thrombosis, myocardial infarction, myocarditis, or even death [12-14]. Increasing evidence suggests that inflammation plays a central role in the pathogenesis of CAL in KD [15].

NT-proBNP is a cardiac neurohormone synthesized and released by ventricular myocytes, commonly used as a biomarker for heart failure and cardiovascular injury [16]. IFN-γ is a cytokine with broad immunomodulatory effects, and TNF-α is a pro-inflammatory cytokine primarily secreted by monocytes and macrophages, both involved in immune and inflammatory responses [17, 18]. This study aimed to explore the diagnostic value of NT-proBNP, IFN-γ, and TNF-α in detecting coronary artery injury in children with severe KD.

The findings of this study showed that WBC, PDW, and PLT levels were significantly elevated in KD patients compared to controls. Notably,

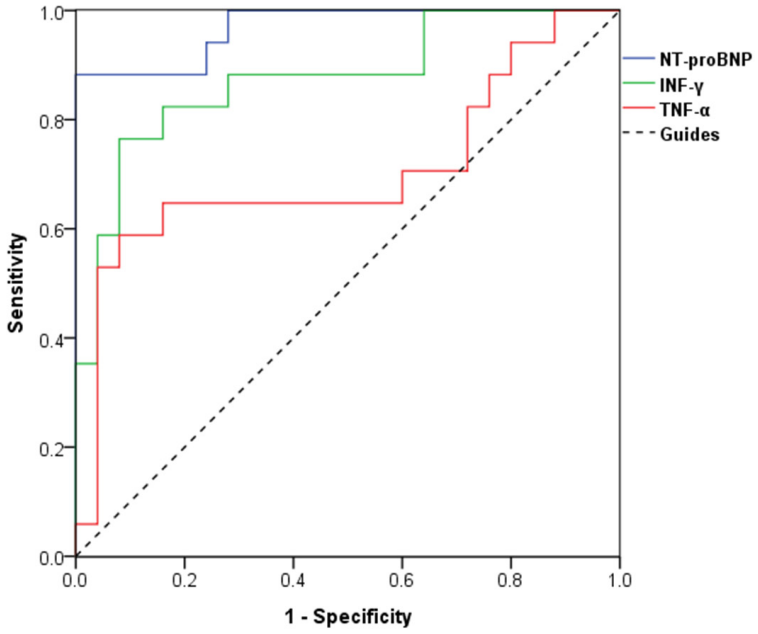

Figure 1. ROC curve of NT-proBNP, IFN-γ and TNF-α in diagnosis of coronary artery lesions in children with KD. KD, Kawasaki disease; NT-proBNP, N-terminal pro-brain natriuretic peptide; IFN-γ, interferon-γ; TNF-α, tumor necrosis factor-α.

Table 6. Diagnostic Value of NT-proBNP, IFN-γ, and TNF-α for coronary artery lesions in KD

	Sensitivity (%)	Specificity (%)	Missed diagnosis (%)	Misdiagnosis (%)	AUC
NT-proBNP	96.18	78.71	3.82	21.29	0.969
INF-γ	83.47	82.16	16.53	17.84	0.875
TFN-α	65.2	81.98	34.8	18.02	0.704

KD, Kawasaki disease; NT-proBNP, N-terminal pro-brain natriuretic peptide; IFN-γ, interferon-γ; TNF-α, tumor necrosis factor-α.

Table 7. Risk factor analysis for coronary artery lesions in children with KD

Index	β	SE	Walds	P	OR	95% *CI*	
PDW	0.621	0.391	2.522	0.143	1.861	0.865	4.004
NT-proBNP	0.448	0.403	1.236	0.402	1.565	0.710	3.448
IFN-γ	0.571	0.266	4.608	0.045	1.770	1.051	2.981
TNF-α	0.801	0.301	7.082	0.000	2.228	1.235	4.019
CRP	0.399	0.183	4.754	0.041	1.490	1.041	2.133
CEC	0.628	0.201	9.762	0.000	1.874	1.264	2.779
Constant term	1.404	0.778	3.257	0.094	4.071	0.886	18.707

KD, Kawasaki disease; PDW, platelet distribution width; NT-proBNP, N-terminal pro-brain natriuretic peptide; INF-γ, interferon-γ; TFN-α, tumor necrosis factor-α; CRP, C-reactive protein; CEC, count of endothelial cell.

PDW was significantly higher in KD children with CAL than in those without CAL. Coronary artery injury may lead to vascular endothelial damage, resulting in exposure of subendothelial structures, activation of platelets and leukocytes, and impaired immune function [19, 20]. The elevated PDW suggests increased platelet activation. Prior research has identified PDW as a potential biomarker in immune-mediated diseases and cardiovascular disorders, including atherosclerosis and infection-related thrombosis [21-23]. Therefore, elevated PDW in KD patients, particularly those with CAL, warrants clinical attention.

Inflammatory and cardiovascular injury markers (NT-proBNP, IFN-γ, TNF-α, CRP, and CEC) were also significantly elevated in KD patients, especially in those with CAL, while RBC, Hb, and HCT were lower. NT-proBNP is biologically inactive but stable in plasma and is rapidly secreted in response to myocardial ischemia or necrosis [24, 25]. IFN-γ enhances antigen presentation, facilitates immune complex clearance, and modulates lymphocyte activation and extracellular matrix secretion. Its upregulation during coronary injury reflects a stress-induced immune response [26, 27]. TNF-α binds to receptors on endothelial cells, induces the expression of new endothelial antigens, promotes antibody-mediated cytotoxicity, and accelerates vascular injury [28, 29]. CRP levels increase rapidly in response to inflammation, infection, or myocardial damage. CECs play roles in vascular repair and neovascularization, and their elevated levels may reflect endothelial injury and regenerative activity [30, 31].

ROC curve analysis further confirmed the diagnostic value of NT-proBNP, IFN-γ, and TNF-α in

identifying CAL in KD, with good sensitivity and specificity, supporting their utility as early diagnostic markers. The logistic regression analysis revealed that elevated IFN-γ, TNF-α, CRP, and CEC were independent risk factors for CAL in KD. IFN-γ is implicated in increased vascular permeability and inflammatory cell infiltration, while TNF-α contributes to endothelial dysfunction, thrombosis, hemorrhage, and tissue hypoxia [32, 33]. CRP, though non-specific, is an established predictor of cardiovascular disease and actively participates in atherosclerotic processes [34]. CECs are also linked to tumor angiogenesis and may serve as markers of endothelial injury and treatment response [35]. Thus, the elevation of these biomarkers may serve as independent predictors of coronary complications in KD.

This studies limitations include the following, the sample size was relatively small (42 KD cases), potentially affecting the generalizability and statistical power of the results. Additionally, the study was conducted at a single center, and the patient population may not represent broader demographics. Therefore, large-scale, multicenter studies are needed to validate these findings and enhance their clinical applicability.

Our findings also suggest that combining multiple biomarkers can enhance diagnostic accuracy and risk stratification. Future studies should investigate the clinical utility of biomarker panels for early diagnosis and individualized management of KD. Further mechanistic research on NT-proBNP, IFN-γ, and TNF-α may reveal deeper insights into the pathogenesis of KD and guide the development of novel therapeutic targets. Translating these findings into clinical tools such as diagnostic kits or risk prediction models may help clinicians implement early interventions and personalized treatment strategies, ultimately improving the long-term outcomes of children with KD. In conclusion, NT-proBNP, IFN-γ, and TNF-α levels were significantly elevated in children with severe KD and were associated with coronary artery injury. These markers may serve as early predictors of coronary complications in KD.

Disclosure of conflict of interest

None.

Address correspondence to: Yanping Liu, Department of Cardiology, Baoding Hospital of Beijing Children's Hospital Capital Medical University, 3399 Hengxiang North Street, Lianchi District, Baoding 071000, Hebei, China. E-mail: nsklyp@163.com

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