Am J Transl Res 2025;17(1):286-293
www.ajtr.org /ISSN:1943-8141/AJTR0160226

Original Article

Expression of Serum Ferritin, Human neutrophil lipocalin, Procalcitonin, and inflammatory factors in children with Kawasaki disease and their relationship to coronary artery lesions

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Received August 31, 2024; Accepted December 6, 2024; Epub January 15, 2025; Published January 30, 2025

Abstract: Objective: To investigate the levels of serum ferritin (SF), human neutrophil lipocalin (HNL), procalcitonin (PCT), and inflammatory cytokines (IL-6, IL-10, IL-17, IL-18, TNF-α, INF-γ, and sCD25) in Kawasaki disease (KD) and their relationship with coronary artery lesions (CAL). Methods: A retrospective analysis was conducted on 76 children with KD treated from May 2022 to May 2024. Participants were classified into a lesion group (n=48) and a no-lesion group (n=28) based on CAL status. Additionally, 76 healthy children were included as controls. Patients with CAL were further categorized into three subgroups based on coronary artery dilation: mild dilation (n=21), moderate dilation (n=18) and coronary artery aneurysm (n=9). The correlation between indicator levels and CAL severity, as well as coronary artery diameter at admission, was analyzed. Partial regression was used to identify inflammatory factors associated with CAL. Results: The lesion group showed significantly higher levels of serum SF, HNL, PCT, and all seven inflammatory cytokines at admission compared to the no-lesion and control groups (all P<0.05). Among patients with CAL, those with coronary artery aneurysms exhibited the highest levels of these indicators compared to the moderate dilation subgroup (P<0.05). Serum levels of SF, HNL, PCT, and inflammatory cytokines were positively correlated with CAL severity and coronary artery diameter. Logistic regression analysis identified these markers as risk factors for CAL in KD. The area under the curve (AUC) for IL-18 was 0.891, with a sensitivity of 0.643 and a specificity of 0.042. Conclusions: Serum SF, HNL, PCT, IL-17, IL-18, and TNF-α are implicated in CAL development in children with KD and may assist in the early clinical diagnosis of CAL.

Keywords: Coronary artery lesion, Kawasaki disease, inflammatory factors, children

Introduction

Kawasaki disease (KD) is a self-limiting, acute systemic vasculitis characterized by fever, cervical lymphadenopathy, rash, oral mucosal changes, and acral changes [1]. The primary complication of KD is coronary artery lesions (CAL), affecting 20%-30% of children with KD. These lesions can lead to thromboembolism, vascular lumen stenosis, or coronary artery aneurysms, with severe cases resulting in aneurysm rupture, myocardial infarction, heart failure, or death [2-4]. KD is now the most common cause of acquired heart disease in children. According to the "Diagnosis, Treatment, and Long-term Management of Kawasaki Disease" guidelines published by the American Heart Association in 2017, the incidence rate for children younger than five years was 243.1 per 100,000 in 2011 and increased to 264.8 per 100,000 in 2012 [5, 6].

Serum ferritin (SF), a positive acute-phase reactant during systemic inflammation, has been reported to be significantly elevated in KD. Aleksandra Stasiak's 2019 study demonstrated notably increased SF levels in children with KD [7]. Similarly, recent research from Fudan Children's Hospital in Shanghai, China, indicated that SF levels in children with KD are significantly higher than those in healthy controls, with even greater elevation in cases involving coronary artery injury [8, 9]. Inflammatory mediators such as interleukin-6 (IL-6), interleukin-10

https://doi.org/10.62347/MIHQ2583

(IL-10), interleukin-17 (IL-17), interleukin-18 (IL-18), tumor necrosis factor-α (TNF-α), interferon-γ (INF-γ), and soluble CD25 (sCD25) play crucial roles in the inflammatory response and may influence SF levels, particularly in children undergoing IVIG therapy for KD.

Given the high risk of CAL in KD, it is essential to explore simple and reliable diagnostic indicators for early intervention [10-12]. SF is considered a sensitive marker for diagnosing pediatric KD, with levels significantly elevated in cases of iron overload [13-16]. IL-17 has also been implicated in coronary atherosclerosis and cardiovascular diseases, highlighting its potential involvement in CAL development. This study aimed to evaluate the expression levels of SF, human neutrophil lipocalin (HNL), procalcitonin (PCT), and inflammatory factors (IL-6, IL-10, IL-17, IL-18, TNF-α, INF-γ, sCD25) in children with KD and to analyze their association with CAL.

Materials and methods

Participants

A retrospective analysis was conducted on 44 children with KD who visited the Department of Cardiology at Baoding Hospital of Beijing Children's Hospital from May 2022 to May 2024. The participants were divided into a lesion group (28 cases) and a no-lesion group (16 cases) based on the presence of CAL. Additionally, 44 healthy children during the same period were included as the control group. The study was approved by the Ethics Committee of Baoding Hospital of Beijing Children's Hospital.

Inclusion criteria: (1) Diagnosis of KD met established diagnostic criteria [1]; (2) The control group consisted of healthy children.

Exclusion criteria: (1) Severe infections; (2) Congenital heart disease; (3) Allergies; (4) Congenital malformations; (5) Vital organ insufficiency; (6) Immune dysfunction; (7) Viral myocarditis; (8) Scarlet fever; (9) Measles.

Detection method

Blood samples (fingerstick or venous) were collected immediately upon hospital admission. SF, HNL, and PCT levels were measured using a medical centrifuge (Jintan Hengfeng Instrument Manufacturing Co., Ltd.) with settings of 10 minutes at 3,000 rpm (centrifugation radius: 16.5 cm). An enzyme-linked immunoassay kit (Shanghai Xitang Biological Company) was used for detection. For inflammatory factor analysis, anticoagulant blood samples were centrifuged at 1,690 × g for 10 minutes to obtain the supernatant. Levels of IL-6, IL-10, IL-17, IL-18, TNF-α, INF-γ, and sCD25 were measured using a Luminex 200 flow cytometer (United States) and Invitrogen Human Custom Procartaplex 13-plex kit.

Observation indicators

Serum levels of SF, HNL, PCT, and inflammatory factors (IL-6, IL-10, IL-17, IL-18, TNF-α, INF-γ, and sCD25) were compared among the three groups at admission. Children with CAL were categorized into three subgroups based on coronary artery internal diameter: Mild dilation: Coronary artery/aortic root diameter ratio of 0.16-0.30; coronary inner diameter of 3.00-8.00 mm (12 cases). Moderate dilation: Coronary artery/aortic root diameter ratio of 0.30-0.60; coronary inner diameter of 4.00-8.00 mm (9 cases). Coronary artery aneurysm (7 cases). Correlation analysis of serum SF, HNL, PCT, and inflammatory factors with CAL and coronary artery diameter at admission. Identification of factors influencing CAL in children with KD. Diagnostic evaluation of SF, HNL, PCT, and inflammatory factors for CAL at admission using receiver operating characteristic (ROC) curves.

Statistical analysis

Statistical analysis was performed using SPSS version 20.0 (SPSS Inc., Chicago, IL, USA). Non-normally distributed variables were analyzed using the Kruskal-Wallis test and the Mann-Whitney U-test. Correlation analysis was conducted with Spearman's rank correlation. Logistic regression was used to identify factors influencing CAL. ROC curves and area under the curve (AUC) analyses were used to assess predictive value. A *P*-value of <0.05 was considered statistically significant. Results were presented as mean ± standard deviation unless otherwise stated.

Table 1. Comparison of serum levels of SF, HNL, PCT and inflammatory factors (IL-6, IL-10, IL-17, IL-18, TNF-α, INF-γ, sCD25) between the three groups ($\bar{x}\pm s$)

Groups	Control Group	Lesion Group	No-Lesion Group	t	P
	n=76	n=48	n=28		
SF	350.53±44.47	1214.11±99.92*	1897.76±105.85	32.231	0.001
HNL	4.96±1.12	11.02±2.21*	20.33±3.5	18.642	0.002
PCT	10.46±2.11	12.81±0.15*	20.65±4.22	6.311	0.001
IL-6	61.95±8.61	113.84±83.87	151.79±126.56	1.687	0.054
IL-10	0.02±0.01	0.37±0.04*	0.45±0.07	49.321	0.056
IL-17	2.79±0.82	8.19±2.08*	17.73±3.76	17.442	0.001
IL-18	23.27±0.08	20.21±2.23*	25.61±3.01	23.171	0.001
TNF-α	6.69±4.52	2.39±0.99	2.58±1.13	0.356	0.001
INF-γ	0.91±0.63	1.17±0.96	1.28±0.83	0.886	0.169
sCD25	41847.97±21517.21	57926.1±36347.75	58163.05±36072.95	1.296	0.099

* represents an indicator that showed a statistically significant difference in results compared to the control group (P<0.05). SF = serum ferritin; HNL = Human neutrophil lipocalin; PCT = procalcitonin; IL-6 = interleukin-6; IL-10 = interleukin-10; IL-17 = interleukin-17; IL-18 = interleukin-18; TNF-α = tumor necrosis factor-α; INF-γ = Interferon-γ; sCD25 = soluble CD25.

Results

Comparison of baseline characteristics between groups

The lesion group included 26 males and 22 females, with an age range of 7-67 months (42.59±12.19 months). The lesion-free group consisted of 12 males and 16 females, aged 8-66 months (40.71±12.63 months). The control group comprised 35 males and 41 females, aged 6-68 months (43.42±12.28 months). The general characteristics of the three groups were balanced and comparable (all P>0.05). Ultrasound findings indicated that in the control group, the coronary artery walls were smooth, with a coronary artery-to-aortic root inner diameter ratio <0.16 and a coronary inner diameter of 0-3 mm. Coronary artery dilation or aneurysm was classified as CAL.

Comparison of serum levels of SF, HNL, PCT, and inflammatory factors

The serum levels of SF, HNL, PCT and inflammatory factors (IL-6, IL-10, IL-17, IL-18, TNF-α, INF-γ, sCD25) were compared among the three groups at admission. The lesion group had significantly higher levels than the no-lesion group, which in turn had higher levels than the control group (P<0.05). Specifically, levels of SF, HNL, PCT, IL-17, IL-18, and TNF-α in the lesion group were significantly higher compared to the no-lesion group (all P<0.05). See **Table 1**.

Correlation analysis

The results of correlation analysis showed that serum levels of SF, HNL, PCT, IL-6, IL-10, IL-17A, IL-18, TNF-α, INF-γ, and sCD25 were positively correlated with CAL (r=0.726, 0.637, 0.542, P<0.001), and coronary artery diameter (r= 0.629, 0.701, 0.588, P<0.001). See **Figure 1**.

Partial regression analysis

Using CAL as the dependent variable and serum levels of SF, HNL, PCT, IL-6, IL-10, IL-17A, IL-18, TNF-α, INF-γ, and sCD25 as independent variables, logistic regression analysis revealed that these factors were significant risk factors for CAL in children with KD (all P<0.05). See **Table 2** and **Figure 2**.

ROC analysis

The ROC curve showed that IL-18 had the largest AUC for diagnosing CAL in children with KD (AUC=0.891), with a sensitivity of 0.643 and a specificity of 0.042. When comparing KD patients without CAL (negative samples) and those with CAL (positive samples), the AUCs of SF, HNL, PCT, and inflammatory factors (IL-17, IL-18, TNF-α) were plotted. The results indicate significant diagnostic value at admission. See **Table 3** and **Figure 3**.

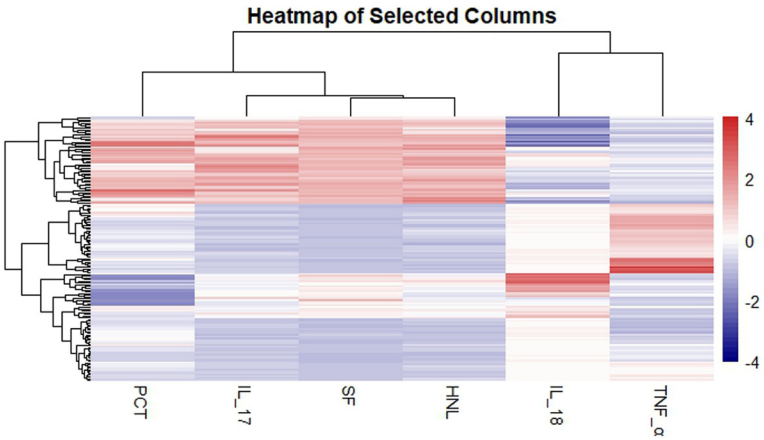

Figure 1. Indicator correlation heatmap.

Table 2. Logistic regression analysis

Influencing factors	β	SE	Wald χ^2	P	95% CI
SF	0.001	0.001	14.223	0.001	0.001-0.002
HNL	0.005	0.004	1.145	0.254	-0.004-0.014
PCT	-0.017	0.002	-6.782	0.001	-0.021--0.012
IL-17	0.004	0.004	0.897	0.371	-0.004-0.012
IL-18	0.02	0.005	3.886	0.001	0.010-0.030
TNF-α	-0.007	0.003	-2.323	0.022	-0.013--0.001

SF = serum ferritin; HNL = Human neutrophil lipocalin; PCT = procalcitonin; IL-6 = interleukin-6; IL-10 = interleukin-10; IL-17 = interleukin-17; IL-18 = interleukin-18; TNF-α = tumor necrosis factor-α; INF-γ = Interferon-γ; sCD25 = soluble CD25.

Discussion

Currently, the pathogenesis of KD remains unclear, but it is widely accepted to involve abnormal inflammatory responses [17-19]. KD primarily affects small- and medium-sized arteries, particularly the coronary arteries, leading to CAL. CAL is a key risk factor for acquired heart disease in children and significantly impacts the prognosis of KD [20]. Therefore, identifying effective biomarkers for the early diagnosis of CAL in children with KD is of critical importance [21].

IL-17, predominantly secreted by helper T cells, is a crucial mediator of inflammatory responses. It promotes the release of inflammatory factors such as TNF-α, exacerbates vascular inflammation, increases plaque instability and rupture risk, and induces thrombosis [20, 21]. Yang et al. [13] demonstrated that IL-17 aggravates inflammation by stimulating fibroblasts and epithelial cells to release inflammatory cytokines, mediating tissue and endothelial cell damage, and contributing to the pathogenesis and progression of KD [15, 22].

A study comparing 13 inflammatory factors in children with acute KD and children with infection-related fever (but without KD) detected significantly higher levels of cytokines in the KD group. These included IL-8, IL-1β, IL-2, IL-6, IL-10, IL-4, IL-5, IL-12P70, IL-17A, IL-18, TNF-α, INF-γ, and sCD25. The findings suggest that the inflammatory response in children with KD is more pronounced during the early stages of the disease compared to those with infection-related fever [3, 11, 14-16].

This study further confirmed elevated serum levels of IL-17A, IL-18, and sCD25 in children with acute KD, particularly in those with CAL, consistent with prior research. Weng et al. [23] highlighted that IL-18 could serve as an independent prognostic factor for KD, unaffected by traditional inflammatory markers like C-reactive protein. This might occur through IL-18's role in modulating the production of other inflammatory factors.

Studies have reported significant decreases in serum IL-8, IL-1β, IL-2, IL-6, IL-18, TNF-α, sCD25, and IL-10 levels in children with KD after intravenous immunoglobulin treatment. Although the differential changes in IL-1β and IL-2 levels were observed in only a small subset of patients and were deemed insufficiently representative for inclusion in figures, these findings suggest that IVIG treatment effectively reduces systemic inflammation. This supports the hypothesis that inflammatory factors play a critical role in the onset and progression of KD.

This study demonstrated that serum levels of SF, HNL, PCT, and inflammatory factors (IL-17, IL-18, TNF-α) were significantly higher in children with KD compared to the control group, with the lesion group > no-lesion group > con-

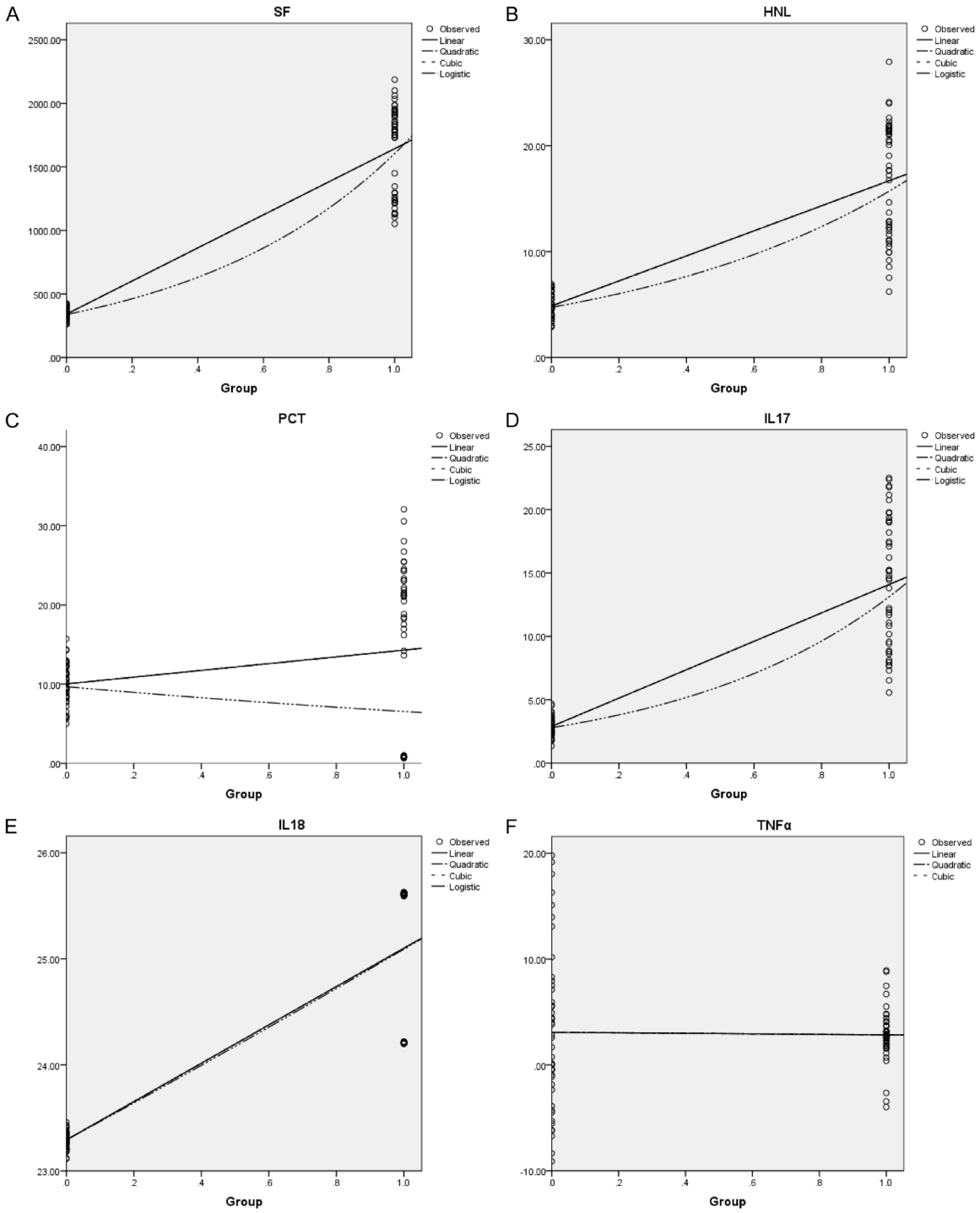

Figure 2. Linear analysis of SF, HNL, PCT and inflammatory cytokines (IL-6, IL-10, IL-17, IL-18, TNF-α, INF-γ, sCD25). A. Linear analysis result of SF for KD; B. Linear analysis result of HNL for KD; C. Linear analysis result of PCT for KD; D. Linear analysis result of IL-17 for KD; E. Linear analysis result of IL-18 for KD; F. Linear analysis result of TNF-α for KD.

trol group. Among patients with CAL, those with coronary artery aneurysms exhibited the highest levels of these markers, followed by those with moderate and mild coronary artery dilation. These findings suggest that SF, HNL, PCT, IL-17, IL-18, and TNF-α are involved in the development of CAL in children with KD. The potential mechanisms underlying these obser-

Table 3. ROC analysis

Indicators	AUC	95% CI	Cut-Off Value	Sensitivity/%	1-Specificity/%	P
IL-18	0.891	0.992-1.000	6.0492	0.643	0.042	<0.05

SF = serum ferritin; HNL = Human neutrophil lipocalin; PCT = procalcitonin; IL-6 = interleukin-6; IL-10 = interleukin-10; IL-17 = interleukin-17; IL-18 = interleukin-18; TNF-α = tumor necrosis factor-α; INF-γ = ilullor necrosis factor-γ; sCD25 = soluble CD25.

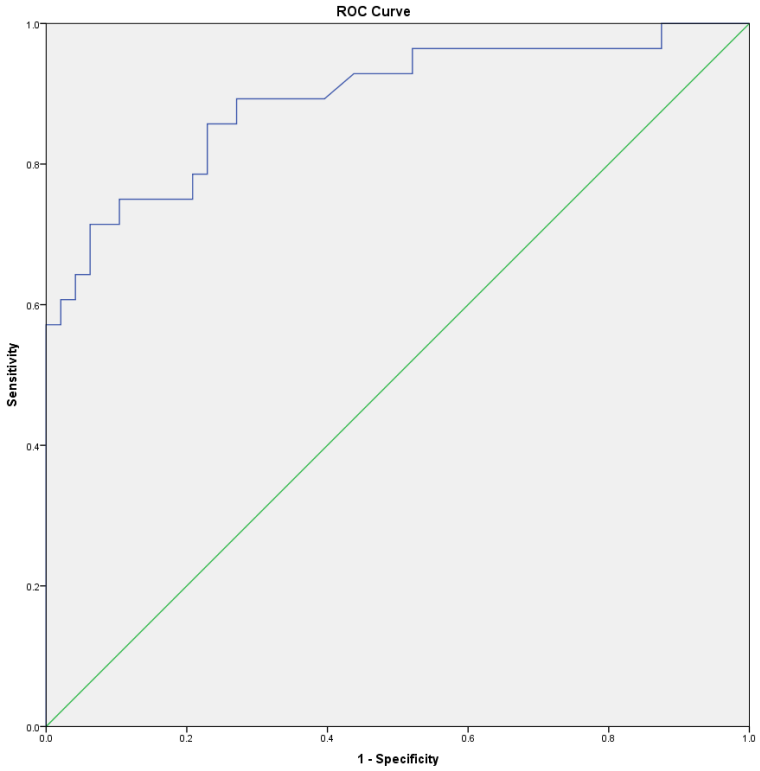

Figure 3. ROC curve.

vations are as follows: Ferritin, a large iron-containing protein, serves as an indicator of iron overload or deficiency [22, 24]. It is stored in the heart, liver, spleen, and bone marrow and functions as a sensitive marker for evaluating iron storage [25-27]. Elevated SF levels are observed in various conditions such as autoimmune diseases (e.g., systemic lupus erythematosus, juvenile idiopathic arthritis), tumors, acute pancreatitis, liver disease, and inflammatory disorders [11, 29-31]. These elevations may result from increased ferritin synthesis, release from macrophages and hepatocytes, decreased glycosylation capacity, or downregulated ferritin receptors [28, 29].

In KD, abnormal activation of monocyte-macrophages triggers cytokine and chemokine release, promoting immune cell homing and vascular endothelial cell activation. This process increases vascular permeability and inflammation, leading to coronary artery damage. Cytokines like TNF-α further stimulate serum ferritin synthesis [6, 20]. Elevated SF levels in KD may contribute to vascular remodeling and CAL development, although this mechanism requires further laboratory confirmation [28-31].

Further correlation analysis revealed that SF, HNL, and PCT levels at admission were positively correlated with CAL and coronary artery diameter. Logistic regression analysis identified these markers as significant risk factors for CAL in children with KD. ROC curve analysis showed that the AUC for combined detection of SF, HNL, and PCT in diagnosing CAL was 0.818, higher than that of individual markers, indicating superior diagnostic efficacy.

This study has several limitations. The relatively small sample size limits the robustness of the evidence. Future studies will involve a larger cohort to strengthen the findings. Current diagnostic criteria for CAL rely heavily on ultrasound measurements, which may introduce bias. Future studies will involve designated sonographers to minimize variability and improve diagnostic accuracy.

In conclusion, serum levels of SF, HNL, PCT, IL-17, IL-18, and TNF-α are closely associated with CAL development in children with KD. Among these, IL-18 shows particularly high clinical value for predicting CAL in KD, emphasizing its potential as a biomarker for early diagnosis.

Active monitoring of SF, HNL, and PCT levels in children with KD could aid in the early diagnosis of CAL, allowing for timely optimization of treatment plans and improved prognosis.

Disclosure of conflict of interest

None.

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