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Contents

Thrice Monthly Volume 10 Number 35 December 16, 2022

EVIDENCE REVIEW

REVIEW

MINIREVIEWS

ORIGINAL ARTICLE

Retrospective Study

Observational Study

World Journal of Clinical Cases

Thrice Monthly Volume 10 Number 35 December 16, 2022

Contents

World Journal of Clinical Cases

Thrice Monthly Volume 10 Number 35 December 16, 2022

Contents

LETTER TO THE EDITOR

World Journal of Clinical Cases

Thrice Monthly Volume 10 Number 35 December 16, 2022

Contents

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CASE REPORT

Kawasaki disease without changes in inflammatory biomarkers: A case report

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Abstract

BACKGROUND

Kawasaki disease (KD) is diagnosed based on clinical features. Blood tests and other tests are auxiliary diagnostic tools. Since KD is a disease caused by arterial inflammation, many patients with KD have elevated levels of inflammatory biomarkers, such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and serum amyloid A protein (SAA) in blood tests. We report our experience of a patient with KD who did not have elevated levels of inflammatory biomarkers.

CASE SUMMARY

A 1-year-old boy presented with a 3-day history of fever. Five of the six symptoms of KD were observed, except for changes in the lips and oral cavity. Blood tests revealed no elevation in CRP, ESR, or SAA levels. Although the blood test results were atypical, the patient was diagnosed with KD based on clinical symptoms and was admitted to the hospital for treatment. The patient was administered intravenous immunoglobulin (IVIG) and aspirin. Despite commencing treatment, the fever persisted; therefore, additional IVIG was administered, the dosage of aspirin was increased, and ulinastatin was added. Three doses of IVIG were administered and the fever resolved on day 11 of KD symptoms started. Blood tests performed during hospitalization showed normal levels of inflammatory biomarkers. We examined leucine-rich alpha-2-glycoprotein 1 - a protein that is elevated during the acute phase of KD. The protein levels did not increase during hospitalization.

CONCLUSION

This case suggests the need to identify criteria and biomarkers for detecting KD conditions that do not require KD treatment.

Key Words: Blood test; C-reactive protein; Erythrocyte sedimentation rate; Intravenous immunoglobulin; Kawasaki disease; Case report

Core Tip: Cases of Kawasaki disease in children with not elevated levels of inflammatory biomarkers, such as C-reactive protein, erythrocyte sedimentation rate, and serum amyloid A protein, are reported. This study clarified the need to identify criteria and biomarkers to detect Kawasaki disease conditions that do not require treatment.

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INTRODUCTION

Kawasaki disease (KD) is a systemic vasculitis that primarily affects children[1]. The cause of the KD remains unknown. A serious complication of KD is the development of coronary artery lesions (CAL), that affect 15%-25% of untreated patients[2]. Hence, early detection and treatment of KD are necessary. The diagnosis of KD is essentially based on the presence of clinical symptoms and not laboratory data [3]. Due to arterial inflammation, most patients with KD have elevated levels of inflammatory biomarkers, such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and serum amyloid A protein (SAA)[4]. These biomarkers support the diagnosis of KD. Herein, we report a case of complete KD in a child with normal levels of inflammatory biomarkers.

CASE PRESENTATION

Chief complaints

A 3 d history of fever and swelling of the hands and feet.

History of present illness

A 1-year-old healthy boy was referred to our hospital with a 3 d history of fever and swelling of the hands and feet. Excluding changes in the lips and oral cavity, the patient had five of the six symptoms of KD such as fever, bilateral bulbar conjunctival injection, rash, changes in the peripheral extremities, and nonsuppurative cervical lymphadenopathy (Figure 1).

History of past illness

The patient's medical history was unremarkable. Physical and neurological findings were also normal.

Personal and family history

The patient's family and genetic history of was unremarkable.

Physical examination

The patient's height and weight were 77 cm and 8.6 kg, respectively. His vital signs at admission were as follows: body temperature, 38.2 °C; blood pressure, 98/56 mmHg; heart rate, 142 beats/min; respiratory rate, 34 breaths/min; and oxygen saturation, 98% in room air. At the time of admission, he had bilateral bulbar conjunctival injection and rash over the torso and extremities. He had changes in the peripheral extremities, with edema of the hands and feet. Redness at the site of Bacille Calmette-Gué rin inoculation was observed. Bilateral cervical lymph nodes were nonsuppurative and enlarged to 15-16 mm.

Laboratory examinations

The hematological values are shown in Table 1. On hospitalization, serum laboratory results showed

Table 1 Patient's laboratory findings on hospitalization

Day	3	5	7	10	13	21
WBC (/μL)	8080	7120	4720	5890	5030	6810
Neut (%)	34	31	31	31	31	31
Platelet (10^4/μL)	27.5	23.3	20.3	26.9	26.5	28.3
ESR (mm/h)	-	3	-	-	-	-
SAA (μg/mL)	-	3.7	-	-	-	-
PCT (ng/mL)	-	0.1	-	-	-	-
D dimer (μg/dL)	< 0.5	0.6	0.6	0.6	0.9	1.0
Total protein (g/dL)	6.3	5.9	7.3	8.2	9.1	8.6
Albumin (g/dL)	4.3	4.1	3.5	3.3	3.3	3.7
T-Bil (mg/dL)	0.3	0.3	0.3	0.2	0.2	-
AST (U/L)	39	33	34	39	36	48
ALT (U/L)	20	17	16	14	13	14
LDH (U/L)	354	368	316	310	280	308
BUN (mg/dL)	9.4	12.6	13.8	22.2	18.9	21.5
Cr (mg/dL)	0.21	0.25	0.34	0.30	0.30	0.29
Uric acid (mg/dL)	3.1	3.7	4.5	5.6	-	-
Total cholesterol (mg/dL)	139	134	114	120	-	-
HDL-C (mg/dL)	37	31	30	31	33	45
Na (mEq/L)	132	134	142	138	137	138
K (mEq/L)	4.3	4.2	3.7	4.2	3.8	3.8
Cl (mEq/L)	103	105	112	12	107	106
CRP (mg/dL)	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05	< 0.05
IgG (mg/dL)	512	466	2620	3527	3842	2936
BNP (pg/mL)	< 3.9	< 3.9	-	-	-	< 3.9
LRG1 (μg/mL)	-	32.6	30.6	27.0	-	23.4

LRG1 reference range: < 93.0 μg/mL. WBC: White blood cell count; Neut: Neutrophils; ESR: Erythrocyte sedimentation rate; SAA: Serum amyloid A protein; PCT: Procalcitonin; T-Bil: Total bilirubin; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; LDH: Lactate dehydrogenase; BUN: Blood urea nitrogen; Cr: Creatinine; HDL-C: High-density lipoprotein cholesterol; Na: Sodium; K: Potassium; Cl: Chlorine; CRP: C-reactive protein; IgG: Immunoglobulin G; BNP: Brain natriuretic peptide; LRG1: Leucine-rich alpha-2-glycoprotein

normal levels of inflammatory biomarkers, such as C-reactive protein, erythrocyte sedimentation rate, and serum amyloid A protein, which were as follows: white blood cell count, 7.1 × 10^3/mm^3; platelet count, 23.3 × 104/mm^3; ESR, 3 mm/h; CRP, < 0.05 mg/dL; serum amyloid A protein, 3.7 μg/mL; procalcitonin, 0.1 (ng/mL): aspartate aminotransferase, 33 U/L; and alanine aminotransferase, 17 U/L. The Kobayashi risk score for predicting intravenous immunoglobulin (IVIG) resistance was 1 point, indicating a low risk[5]. Rapid *Streptococcus pyogenes* and adenovirus antigen test results were negative. The antibody titers of Epstein-Barr virus, cytomegalovirus, *Mycoplasma pneumoniae*, human parvovirus B19, measles, and mumps were not elevated. Polymerase chain reaction was negative for severe acute respiratory syndrome coronavirus 2. Bacteriological cultures of blood, urine, and stool samples were negative. A urinalysis revealed no pyuria.

Imaging examinations

On admission, echocardiogram and electrocardiogram findings were normal. Chest radiography showed no abnormalities, such as cardiac enlargement or infiltrates.

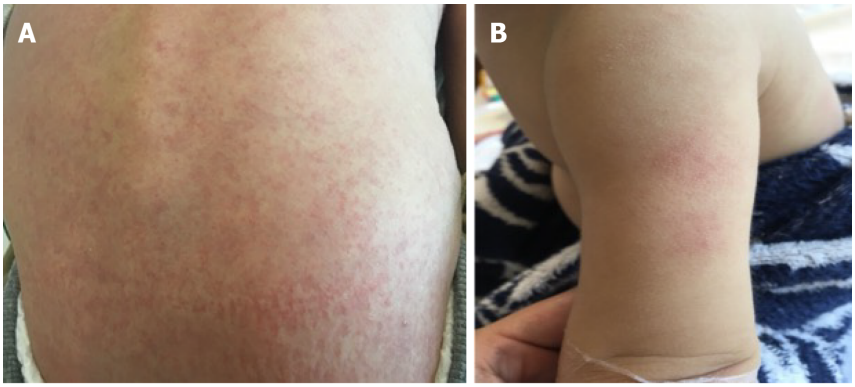

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Figure 1 Clinical features on admission. A: Rash on the back; B: Redness at the site of Bacille Calmette-Guérin inoculation.

FINAL DIAGNOSIS

Kawasaki disease.

TREATMENT

The treatment course and changes in the patient's body temperature are shown in Figure 2. Treatment with IVIG and aspirin failed to relieve the fever despite low point on the Kobayashi risk score. The patient appeared to be resistant to IVIG treatment; therefore, the dosage of aspirin was increased and ulinastatin was added. After the 2nd line treatment, clinical symptoms of KD tended to improve, but fever persisted. Based on Kawasaki disease guidelines, the patient required 3rd line treatment of IVIG. To avoid inducing hyperviscosity syndrome, the dosage of IVIG was reduced to 1 g/kg[6]. The treatment was intensified, and the fever broke on day 11 of KD symptoms started. Membrane-like desquamation was observed on Day 13. Ultrasonography performed during hospitalization showed no coronary artery dilation. Levels of inflammatory biomarkers and platelets did not increase during hospitalization. There was a discrepancy between the clinical course of the disease and the results of blood tests and ultrasonographic findings. Considering the possibility that it was not KD, we examined leucine-rich alpha-2-glycoprotein 1 (LRG1), a protein that is elevated in the acute phase of KD[7]. The protein level did not increase during hospitalization (Table 1).

OUTCOME AND FOLLOW-UP

After the patient was discharged from the hospital, no coronary artery dilation and aneurysm were observed. Blood tests showed no increase in the level of CRP < 0.05 and platelet count.

DISCUSSION

Our patient's symptoms were consistent with those of typical KD and did not deviate from the diagnostic criteria. Nevertheless, his laboratory data were negative for inflammatory biomarkers, such as CRP, ESR, and SAA. Other diseases with KD symptoms have not been identified. Additionally, the drug-induced lymphocyte stimulation test for immunoglobulin was negative; it was performed considering the transient fever caused by IVIG treatment. Furthermore, the levels of LRG1 (reference range: < 93.0 ng/mL), a novel KD biomarker, were not elevated in our patient[7].

To prevent CAL, risk scores, such as the Kobayashi risk score, have been developed to identify patients with a high risk of IVIG resistance[5]. However, there are few reports on detecting risk scores for IVIG-unnecessary KD patients. Considering the present case, this case may be bound by the KD diagnostic criteria. In other words, this case raises inquiries whether it was a "KD" patient, one who required IVIG treatment, or not. The number of patients with Kawasaki disease increase, while the number of patients with sequelae such as CAL is on the decline[3]. These findings imply the possibility that patients who do not require IVIG treatment are mixed in. KD treatment leads to a greater hospital cost, rescheduling vaccination, and longer follow-up of coronary arteries using ultrasonographic studies.

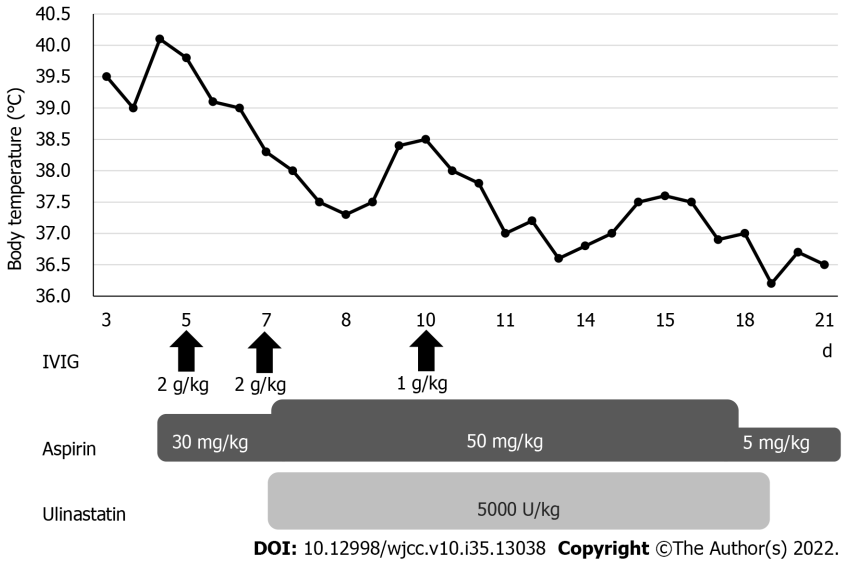

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Figure 2 Clinical course. IVIG: Intravenous immunoglobulin.

An accurate diagnosis of KD is important. A previous study reported that significantly higher serum CRP levels are associated with CAL and IVIG resistance[8]. Inflammatory biomarkers may be helpful in the diagnosing of KD[4]. Procalcitonin and D dimer are biomarkers to predict the treatment-resistant group[9,10]. In addition, BNP and NT-proBNP have been reported as useful biomarkers for diagnosis [11]. In our patient, those values were not elevated, interestingly. However, even with low levels of inflammatory biomarkers, KD could not be ruled out and CALs could dilate[9]. Therefore, to avoid over diagnosis and unnecessary treatment, novel biomarkers should be established to detect KD cases that require no treatment.

The limitations of this study are that we did not identify other diseases that were not KD, first. Therefore, this case was still be treated as KD. Second, we did not measure IL-1, IL-6, and TNF-α themselves in this patients, and SAA and ESR were measured only once. Here, we reported that one year old KD boy with unelevated level of inflammatory biomarkers, such as CRP, ESR, and SAA protein. In the future, new biomarkers should be established to detect KD cases that do not require treatment.

CONCLUSION

Currently, it is important to identify the clinical symptoms of KD. The present case suggests the need to identify criteria and biomarkers for detecting KD conditions that do not require KD treatment.

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FOOTNOTES

Author contributions: Yamashita K was the first author of this manuscript; Kanazawa T advised and helped revise the manuscript; Abe Y analyzed the data and drafted the manuscript; Naruto T was measured as leucine-rich alpha-2-glycoprotein; Mori M measured leucine-rich alpha-2-glycoprotein and provided critical comments on the manuscript; all authors have read and approved the final manuscript.

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