



Comparison of COVID-19-associated multisystem inflammatory syndrome in children (MIS-C) and Kawasaki disease shock syndrome: case reports and literature review

Songmi Lee, M.D.¹, Danbi Kim, M.D.¹, Beom Joon Kim, M.D., Ph.D.¹, Jung Woo Rhim, M.D., Ph.D.¹, Soo-Young Lee, M.D., Ph.D.^{1,2}, Dae Chul Jeong, M.D., Ph.D.^{1,2}

¹Department of Pediatrics, College of Medicine, The Catholic University of Korea, ²The Vaccine Bio Research Institute, College of Medicine, The Catholic University of Korea, Seoul, Korea

Multisystem inflammatory syndrome in children (MIS-C) is a serious post-infectious complication of COVID-19 characterized by hyperinflammation and multi-organ dysfunction including shock. Shock is also seen in a severe form of Kawasaki disease (KD) called KD shock syndrome (KDSS). Here, we present one MIS-C and one KDSS case and compare similarities and differences between them. Both MIS-C (case 1) and KDSS (case 2) showed hyperinflammation, KD-related features, gastrointestinal problems, hypotension, and coagulopathy. The extent of systemic inflammation and organ dysfunction was more severe in KDSS than in MIS-C. Case 1 was diagnosed as MIS-C because SARS-CoV-2 was confirmed, and case 2 was diagnosed as KDSS because no pathogen was identified in microbiological studies. We believe that the most important difference between MIS-C and KDSS was whether SARS-CoV-2 was identified as an infectious trigger. Organ dysfunction is a hallmark of MIS-C and KDSS, but not KD, so MIS-C shares more clinical phenotypes with KDSS than with KD. Comparison of MIS-C and KDSS will be an interesting and important topic in the field of KD-like hyperinflammatory disease research.

Keywords: COVID-19, Multisystem inflammatory syndrome in children, Kawasaki disease shock syndrome, Kawasaki disease

INTRODUCTION

Children with coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, usually have milder symptoms than adults with this disease [1-3]. However, some develop multisystem inflammatory syndrome in children (MIS-C), a serious complication that can occur 2 to 6 weeks after SARS-CoV-2 infection [2]. MIS-C is characterized by hyperinflammation, multi-organ dysfunction including shock, and infectious trigger (Table 1) [1]. With dermatologic involvement, many patients with MIS-C present with 'Kawasaki disease (KD)-related features' that

constitute the diagnostic criteria for KD [3]. Accordingly, comparing the clinical and laboratory features of MIS-C and KD has become an important research topic during the COVID-19 pandemic [3,4].

KD is a common vasculitis of unknown etiology in children [5,6]. In rare cases, KD can present with shock or hypotension (decrease in blood pressure $\geq 20\%$ from baseline or systolic hypotension for age), which is called KD shock syndrome (KDSS) [7-9]. KDSS is more severe than KD without shock in systemic inflammation, coronary artery abnormalities (CAAs), and intravenous immunoglobulin (IVIG) resistance [10,11]. Although MIS-C and KDSS share important clinical features such as

Received May 31, 2023; Revised June 7, 2023; Accepted June 17, 2023, Published online July 3, 2023

Corresponding author: Soo-Young Lee, <https://orcid.org/0000-0002-5354-3135>

Department of Pediatrics, The Catholic University of Korea, Bucheon St. Mary's Hospital, 327 Sosa-ro, Wonmi-gu, Bucheon 14647, Korea. **E-mail:** sylee@catholic.ac.kr

Copyright © The Korean College of Rheumatology.



This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

Table 1. Case definition of MIS-C and clinical and laboratory features of case 1 and case 2

MIS-C (CDC and RCPCH definition)	Case 1 (MIS-C)	Case 2 (KDSS)
Individual aged <21 years presenting with fever (>38.0°C)	9-year-old boy	5-year-old boy
1. Multi-organ involvement, ≥2/8 organs	4 organs	6 organs
Cardiovascular: hypotension or shock, inotropic support, pulmonary edema, pericarditis or PE, dysrhythmia, NT-proBNP >400 pg/mL, elevated troponin, EF <55%, CAAs	+++ (Hypotension, PE, NT-proBNP)	+++ (Hypotension, inotropics, PE, CAAs)
Respiratory: ventilator support or O ₂ therapy due to ARDS, X-ray infiltration, pleural effusion, pulmonary hemorrhage	-	-
Hematologic: fibrinogen >400 mg/dL, INR >1.1, DIC, Hb <9 g/dL, WBC <4,000/μL, PLT <150,000/μL, DVT	+++ (Fibrinogen, INR, PLT)	+++ (INR, DIC, PLT)
Gastrointestinal: vomiting, diarrhea, abdominal pain, appendicitis, pancreatitis, hepatitis or hepatomegaly, gallbladder hydrops or edema	+++ (Vomiting, diarrhea, abdominal pain, hepatitis)	+++ (Vomiting, diarrhea, abdominal pain, hepatitis)
Neurological: seizures, coma, encephalitis, meningitis, uveitis	-	-
Renal: AKI (creatinine >1.05 mg/dL, 1~10 years), dialysis	-	+
Dermatologic: ≥4/5 KD-related features*	+++	+++
Musculoskeletal: arthritis, arthralgia, myositis, myalgia	-	+
2. Hyperinflammation	+++	+++
Elevated markers: CRP, ESR, procalcitonin, or IL-6, Ferritin >500 ng/mL, albumin ≤3.0 g/dL, Elevated AST, ALT, LDH, or TG	(CRP, ESR, ferritin, albumin, AST, ALT, LDH)	(CRP, ESR, albumin, AST, ALT, LDH)
3. Infectious trigger	+	-
No other cause but evidence of current or recent SARS-CoV-2 infection by RT-PCR or exposure within 4 weeks	(Positive RT-PCR: 3 weeks earlier and at admission)	(No pathogens isolated from microbiological studies)

+: present, -: absent, +++: 3 or more parameters, MIS-C: multisystem inflammatory syndrome in children, KDSS: Kawasaki disease shock syndrome, CDC: Centers for Disease Control and Prevention, RCPCH: Royal College of Paediatrics and Child Health, PE: pericardial effusion, NT-proBNP: N-terminal prohormone of brain natriuretic peptide, EF: ejection fraction, CAAs: coronary artery abnormalities, ARDS: acute respiratory distress syndrome, INR: international normalized ratio, DIC: disseminated intravascular coagulopathy, Hb: hemoglobin, WBC: white blood cells count, PLT: platelet count, DVT: deep vein thrombosis, AKI: acute kidney injury, KD: Kawasaki disease, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, IL: interleukin, AST: aspartate transaminase, ALT: alanine transaminase, LDH: lactate dehydrogenase, TG: triglycerides, SARS-CoV-2: severe acute respiratory syndrome coronavirus 2, RT-PCR: real-time reverse transcription-polymerase chain reaction. *KD-related features include rash, conjunctival injection, red lips and strawberry tongue, cervical lymphadenopathy, and extremity changes.

shock, few studies have compared the two [8]. Here, we report our experience with MIS-C and KDSS and review the literature to discuss the similarities and differences between the two diseases.

CASE REPORT

Case 1 (MIS-C)

A 9-year-old boy was hospitalized with fever and abdominal pain for 5 days. He had no known medical illness or family history except SARS-CoV-2 infection confirmed by real-time reverse transcription-polymerase chain reaction (RT-PCR) three weeks prior to hospitalization. His height was 135 cm (50-75th percentile) and his weight was 38.3 kg (75-90th percentile). Vital signs were as follows: blood pressure, 74/40 mmHg; respiratory

rate, 25 breaths/min; heart rate, 150 beats/min; and body temperature, 37.3°C. Physical examination revealed four KD-related features of maculopapular rash, conjunctival injection, red lips and strawberry tongue, and extremity changes. Important laboratory features included hemoglobin, 10.5 g/dL; white blood cell (WBC) count, 18,060/μL (neutrophil 80%); platelet count, 96,000/μL; erythrocyte sedimentation rate (ESR), 36 mm/h; C-reactive protein (CRP), 82 mg/L (normal <5 mg/L); aspartate transaminase (AST), 51 U/L; alanine transaminase (ALT), 92 U/L; albumin, 2.9 g/dL; international normalized ratio (INR), 1.39 (normal <1.1); fibrinogen, 431 mg/dL; N-terminal prohormone of brain natriuretic peptide (NT-proBNP), 749 pg/mL; and ferritin, 690 ng/mL. Nasopharyngeal swab RT-PCR for SARS-CoV-2 was positive (*E/RdRp* gene *Ct* value, 32.09/34.24). Chest X-ray and urinalysis were unremarkable. Echocardiography

showed pericardial effusion but no CAAs.

The patient was diagnosed with MIS-C based on multisystem organ involvement (cardiovascular, hematologic, gastrointestinal, and dermatologic), laboratory evidence of inflammation, and evidence of SARS-CoV-2 infection. Fluid and IVIG (2 g/kg/dose) were administered, and his blood pressure (110/70 mmHg) was stable the next day. However, his fever persisted, and he developed oliguria (<0.5 mL/kg/h; blood urea nitrogen [BUN], 25.3 mg/dL; creatinine, 0.6 mg/dL) and generalized edema. Intravenous methylprednisolone (IVMP, 30 mg/kg/day for 5 days), albumin, and diuretics were added to the treatment. His symptoms, including oliguria and edema, improved after 3 days of IVMP treatment, and no subsequent fever was observed. He was discharged on the 9th day of hospitalization. Oral steroids (prednisolone 1 mg/kg) were tapered over 2 weeks and discontinued. During the 6-month follow-up, no further CAAs or recurrence of MIS-C or KD was observed.

Case 2 (KDSS)

A 5-year-old boy was referred to our department due to fever and abdominal pain. The patient had fever and cervical lymphadenopathy for 5 days and was treated with antibiotics for 3 days at the Department of Otorhinolaryngology. However, his symptoms worsened and he developed vomiting, diarrhea, and abdominal pain. He was previously healthy with no specific medical or family history. His height was 118 cm (75-90th percentile) and his weight was 24.7 kg (75-90th percentile). Vital signs were as follows: blood pressure, 90/60 mmHg; respiratory rate, 28 breaths/min; heart rate, 100 beats/min; and body temperature, 38.7°C. Physical examination revealed five KD-related features of urticarial rash, conjunctival injection, red lips and

strawberry tongue, cervical lymphadenopathy, and extremity changes. Important laboratory features included hemoglobin, 11.2 g/dL; WBC count, 37,420/ μ L (neutrophil 93%); platelet count, 103,000/ μ L; ESR, 59 mm/h; CRP, 205 mg/L; AST, 299 U/L; ALT 181 U/L; albumin, 2.5 g/dL; and INR, 1.51. Chest X-ray and urinalysis were unremarkable. Initial echocardiography showed pericardial effusion and mitral regurgitation without CAAs.

He was diagnosed with typical KD ($\geq 4/5$ KD-related features) and administered IVIG (2 g/kg/dose) for treatment. However, fever was uncontrolled with oliguria (<0.5 mL/kg/h) and decreased renal function (BUN, 44.7 mg/dL; creatinine, 1.1 mg/dL). In addition, he developed hypotension (68/30 mmHg) that was unresponsive to fluid therapy. Follow-up echocardiography showed a significant increase in pericardial effusion without CAAs. Considering the possibility of septic shock with acute kidney injury, he was transferred to the intensive care unit (ICU) on the 3rd day of hospitalization. Inotropics (dopamine and norepinephrine), empirical antibiotics (vancomycin and meropenem), IVMP (30 mg/kg/day for 5 days), antithrombin III, albumin, and diuretics were added for treatment. After 6 days of treatment in the ICU, body temperature and blood pressure normalized, and the patient was transferred to the general ward. He was hospitalized for another week to manage scrotal edema and joint pain and was discharged on the 16th day of hospitalization. No pathogens were identified in microbiological studies, including bacterial (blood, urine, and stool cultures) and viral (Epstein-Barr virus [EBV] and multiplex respiratory virus PCR) tests. One week later, outpatient echocardiography revealed a 4.1 mm aneurysm in the left anterior descending (LAD) coronary artery (Figure 1). Oral steroids (prednisolone 1

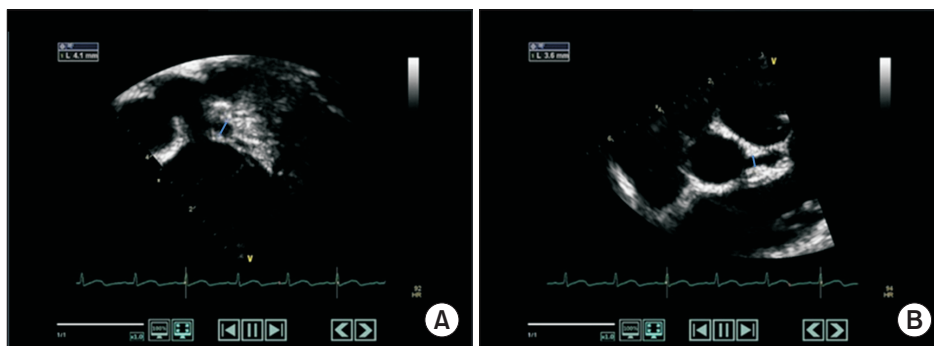


Figure 1. Echocardiography of a patient with Kawasaki disease shock syndrome (case 2) at 1 week after discharge. The apical four-chamber view (A) shows an aneurysm of 4.1 mm in the left anterior descending (LAD) and the parasternal short-axis view (B) shows an aneurysmal dilatation of 3.6 mm in the proximal LAD. Each ventricular function is normal, with no additional pericardiac effusion.

mg/kg) were tapered over 3 weeks and discontinued. During the 8-year follow-up, no exacerbation of CAAs and recurrence of KDSS or KD was observed.

DISCUSSION

MIS-C was first reported as hyperinflammatory shock in 8 children in the UK in April 2020 during the COVID-19 pandemic [12]. Most patients with MIS-C show KD-related features, gastrointestinal problems, and hypotension, as did our two patients in this study. The presence of KD-related features in patients with MIS-C has received clinical attention, but the extent of organ dysfunction in patients with MIS-C is of greater importance as it directly affects the disease course and outcome [4,13]. In children and in adults, MIS can occur as a serious post-infectious complication of COVID-19. Chung et al. [14] have recently reported the first Korean case of MIS in adults (MIS-A). Patients with MIS-C or MIS-A may be misdiagnosed with septic shock due to gastrointestinal infection, as severe abdominal pain and shock are common initial symptoms [8,9]. Therefore, MIS-C and MIS-A should be considered as a differential diagnosis in patients with unexplained shock, heart failure, and gastrointestinal symptoms [14,15].

Like MIS-C, KDSS has a relatively short history. The term 'KD shock syndrome' (KDSS) was first introduced in 2009 to distinguish this severe form of KD from KD without shock [9]. In actual practice, KDSS shows distinct characteristics in various aspects as KDSS patients have frequent organ dysfunction (gastrointestinal, cardiovascular, hematologic, neurologic, and renal) and more severe systemic inflammation (CRP elevation, hyperferritinemia, anemia, thrombocytopenia, and hypoalbu-

minemia) [9-11]. Epidemiologically, KD and KDSS show different prevalence according to age, region, and race [6]. Therefore, some experts believe that KDSS should be regarded as a distinct disease entity [10]. Like MIS-C, KDSS is sometimes confused with septic shock or toxic shock syndrome [7]. Clinical suspicion of MIS-C or KDSS is a prerequisite for early diagnosis and prompt treatment of the disease [14,15].

Here, we describe two cases with KD-related features, systemic inflammation, and organ dysfunction (Table 1). The overall disease course of these two cases was severe but typical, so the diagnosis itself was not difficult. MIS-C is diagnosed when SARS-CoV-2 is identified in patients with KD-related features and organ dysfunction [1]. KDSS is diagnosed when shock or hypotension is observed in patients with KD [9]. The presence or absence of pathogens is not essential for KDSS diagnosis, but various pathogens can be found in KDSS patients (e.g., EBV-confirmed KDSS, mycoplasma-confirmed KDSS, or SARS-CoV-2-confirmed KDSS) [12,13]. Case 1 was diagnosed as MIS-C because SARS-CoV-2 was confirmed, and case 2 was diagnosed as KDSS because no pathogen was identified in microbiological studies. MIS-C (case 1) and KDSS (case 2) showed similar clinical manifestations, but the extent of systemic inflammation and organ dysfunction was more severe in KDSS than in MIS-C. Suzuki et al. [8] reported that MIS-C and KDSS present similar clinical signs and symptoms but differ in severity and are unlikely to be the same disease entity. On the contrary, it has been proposed that MIS-C and KDSS have a common host immune response and are of a single disease spectrum with varying severity [13]. Regardless of whether MIS-C and KDSS are the same disease entity, at least phenotypically, MIS-C and KDSS share many overlapping features [3,4]. We found that the

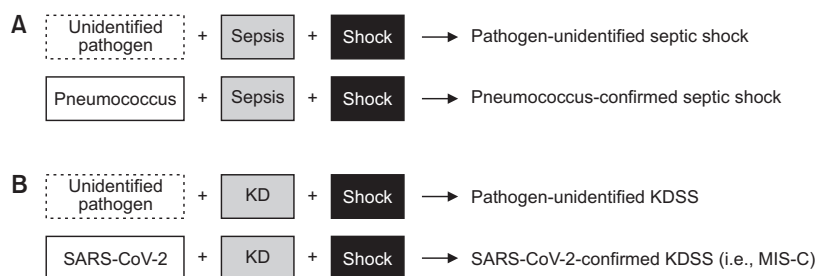


Figure 2. The relationship between KDSS and MIS-C similar to that between pathogen-unidentified septic shock and pneumococcal septic shock. (A) When pneumococcus is identified in a patient with septic shock, the patient is diagnosed with 'pneumococcus-confirmed septic shock.' (B) When SARS-CoV-2 is identified in a patient with KDSS, the patient should be diagnosed with 'SARS-CoV-2-confirmed KDSS (i.e., MIS-C).' KD: Kawasaki disease, KDSS: Kawasaki disease shock syndrome, MIS-C: multisystem inflammatory syndrome in children, SARS-CoV-2: severe acute respiratory syndrome coronavirus 2.

most important factor that differentiates MIS-C from KDSS is whether SARS-CoV-2 has been identified as an infectious trigger. In other words, case 1 (MIS-C) would have been diagnosed as KDSS if there was no evidence of SARS-CoV-2 infection. Similarly, case 2 (KDSS) could have been diagnosed with MIS-C if SARS-CoV-2 was confirmed. The relationship between KDSS and MIS-C is similar to that between pathogen-unidentified septic shock and pneumococcal septic shock (Figure 2).

Table 2 compares the characteristics of MIS-C cases in the US (n=186), KDSS cases in the literature review (n=103), and KD cases from a nationwide survey in Korea (n=14,916) [2,10,11]. Although KDSS is a subtype of KD, KDSS is closer to MIS-C than to KD in terms of demographics, clinical and laboratory

features, organ involvement, and treatment and outcomes. Most patients with KD are younger than 5 years, but two-third of patients with MIS-C and nearly half of patients with KDSS are older than 5 years [5,10]. KD is most frequent in East Asia, including Korea, Japan, and Taiwan. However, MIS-C and KDSS are relatively frequent in Europe and North America (i.e., Hispanic and black populations) and rare in East Asia (i.e., Asian populations) [8,9]. Systemic inflammation is more severe in MIS-C and KDSS than in KD. Organ involvement, such as gastrointestinal problems or hypotension, rarely occurs in KD but is a principal feature of MIS-C and KDSS. Despite many similarities and the close relationship between MIS-C and KDSS, few studies have addressed the relationship between the two diseases. As this

Table 2. Characteristics of MIS-C, KDSS, and KD

	MIS-C, US [2] (n=186)	KDSS, literature review [10] (n=103)	KD, Korea [11] (n=14,916)
Demographic and clinical			
Median age (yr)	8.3	5.2	2.4
Sex, male (%)	61.8	55.3	58.4
Race, dominant	Hispanic, black	Hispanic	Asian
Median fever duration (day)	6.0	7.2	5.7
≥4/5 KD-related features (%)*	39.8	69.9	67.2
Laboratory abnormalities (%)			
Anemia	47.6	58.8	-
Neutrophilia	68.5	64.1	-
Thrombocytopenia	54.6	79.5	-
Elevated CRP	90.6	91.9	-
Elevated AST or ALT	63.7	61.4	-
Hypoalbuminemia	80.3	93.4	-
Organ involvement (%)			
Cardiovascular	80.1	100.0	10.8
Respiratory	70.4	50.0	-
Hematologic	76.9	54.8	-
Gastrointestinal	91.9	74.6	-
Neurologic or renal	14.0	18.4	-
Dermatologic (e.g., rash)	59.1	76.9	83.1
Treatment and outcome (%)			
Initial therapy with IVIG	77.4	95.1	95.4
Additional therapies	69.3	52.4	11.9
Systemic steroids	48.9	35.9	3.0
Biologics or others [†]	20.4	16.5	0.005
Mortality	2.1 (4 of 186)	6.8 (7 of 103)	0.0001 (2 of 14,916)

MIS-C: multisystem inflammatory syndrome in children, KDSS: Kawasaki disease shock syndrome, KD: Kawasaki disease, CRP: C-reactive protein, AST: aspartate transaminase, ALT: alanine transaminase, IVIG: intravenous immunoglobulin. *KD-related features include rash, conjunctival injection, red lips and strawberry tongue, cervical lymphadenopathy, and extremity changes. [†]Biologics or others include anakinra, tocilizumab, infliximab, cyclosporin, and methotrexate.

study is a simple case report, large-scale clinical and experimental studies are needed to support our findings. Understanding the similarities and differences between MIS-C and KDSS will be helpful for related research and proper treatment.

SUMMARY

In this study, we present one case each of MIS-C and KDSS, which share many overlapping features in terms of systemic inflammation and organ dysfunction. The clinical manifestations and disease course are more severe in KDSS than in MIS-C. We found that the fundamental difference between MIS-C and KDSS was SARS-CoV-2 identified as an infectious trigger.

FUNDING

This study was supported by the Institute of Clinical Medicine Research of Bucheon St. Mary's Hospital, Research Fund, 2023.

ACKNOWLEDGMENTS

None.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

AUTHOR CONTRIBUTIONS

Conceptualization: S.L. and S.Y.L. Data curation: D.K. and B.J.K. Formal analysis: B.J.K., J.W.R., and D.C.J. Writing-original draft: S.L. Writing-review & editing: J.W.R., S.Y.L., and D.C.J. All authors approved the final manuscript.

ORCID

Songmi Lee, <https://orcid.org/0000-0003-1639-5310>

Danbi Kim, <https://orcid.org/0000-0002-5145-7620>

Beom Joon Kim, <https://orcid.org/0000-0001-5033-4568>

Jung Woo Rhim, <https://orcid.org/0000-0002-0227-3809>

Soo-Young Lee, <https://orcid.org/0000-0002-5354-3135>

Dae Chul Jeong, <https://orcid.org/0000-0003-0934-817X>

REFERENCES

1. Kwak JH, Lee SY, Choi JW, the Korean Society of Kawasaki Disease. Clinical features, diagnosis, and outcomes of multisystem inflammatory syndrome in children associated with coronavirus disease 2019. *Clin Exp Pediatr* 2021;64:68-75.
2. Feldstein LR, Rose EB, Horwitz SM, Collins JP, Newhams MM, Son MBF, et al. Multisystem inflammatory syndrome in U.S. children and adolescents. *N Engl J Med* 2020;383:334-46.
3. Sharma C, Ganigara M, Galeotti C, Burns J, Berganza FM, Hayes DA, et al. Multisystem inflammatory syndrome in children and Kawasaki disease: a critical comparison. *Nat Rev Rheumatol* 2021;17:731-48.
4. Hufnagel M, Armann J, Jakob A, Doenhardt M, Diffloth N, Hospach A, et al. A comparison of pediatric inflammatory multisystem syndrome temporarily-associated with SARS-CoV-2 and Kawasaki disease. *Sci Rep* 2023;13:1173.
5. McCrindle BW, Rowley AH, Newburger JW, Burns JC, Bolger AF, Gewitz M, et al. Diagnosis, treatment, and long-term management of Kawasaki disease: a scientific statement for health professionals from the American Heart Association. *Circulation* 2017;135:e927-99.
6. Park WY, Lee SY, Kim GB, Song MK, Kwon HW, Bae EJ, et al. Clinical aspects for differential diagnosis of Kawasaki disease shock syndrome: a case control study. *BMC Pediatr* 2021;21:25.
7. Lee KY, Park JE, Park WS. Atypical Kawasaki disease presented with toxic shock syndrome. *J Korean Pediatr Soc* 2002;45:1048-51.
8. Suzuki J, Abe K, Matsui T, Honda T, Yasukawa K, Takanashi JI, et al. Kawasaki disease shock syndrome in Japan and comparison with multisystem inflammatory syndrome in children in European countries. *Front Pediatr* 2021;9:625456.
9. Gámez-González LB, Ramírez-López MJ, Colmenero-Rascón M, Yamazaki-Nakashimada MA. Kawasaki disease shock syndrome in the COVID-19 pandemic. *Cardiol Young* 2022;32:506-7.
10. Gamez-Gonzalez LB, Moribe-Quintero I, Cisneros-Castolo M, Varela-Ortiz J, Muñoz-Ramírez M, Garrido-García M, et al. Kawasaki disease shock syndrome: unique and severe subtype of Kawasaki disease. *Pediatr Int* 2018;60:781-90.
11. Kim GB, Park S, Eun LY, Han JW, Lee SY, Yoon KL, et al. Epidemiology and clinical features of Kawasaki disease in South Korea, 2012-2014. *Pediatr Infect Dis J* 2017;36:482-5.
12. Riphagen S, Gomez X, Gonzalez-Martinez C, Wilkinson N, Theocharis P. Hyperinflammatory shock in children during COVID-19 pandemic. *Lancet* 2020;395:1607-8.
13. Tsoukas P, Yeung RSM. Kawasaki disease and MIS-C share a host immune response. *Nat Rev Rheumatol* 2022;18:555-6.
14. Chung H, Seo H, Park S, Kim H, Jung J, Chong YP, et al. The first case of multisystem inflammatory syndrome in adult after COVID-19 in Korea. *J Korean Med Sci* 2021;36:e181.
15. Rhee S, Kim D, Cho K, Rhim JW, Lee SY, Jeong DC. Under-recognized macrophage activation syndrome in refractory Kawasaki disease: a wolf in sheep's clothing. *Children (Basel)* 2022;9:1588.