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Recurrent course of Kawasaki disease with cardiac and joint complications – a case report

Nawrotowy przebieg choroby Kawasakiego z powikłaniami kardiologicznymi i stawowymi – opis przypadku

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Abstract

Kawasaki disease is an acute systemic vasculitis of unknown aetiology. The diagnosis and treatment of the typical course of the disease have already been well described. However, in atypical, recurrent, or complicated Kawasaki disease cases, diagnostic and therapeutic decisions may be challenging. We present the case of a 3.5-year-old boy who presented with typical Kawasaki disease symptoms. Complete Kawasaki disease was diagnosed, and the boy was initially assigned as a low-risk patient, but he developed persisting fever and finally giant coronary aneurysms despite treatment according to the recommendations. Shortly after discharge, the boy was re-admitted due to limping. Two similar relapses were observed over the period of three months and the boy was ultimately diagnosed with recurrent Kawasaki disease complicated by arthritis. The initial typical presentation of Kawasaki disease may not predict its further course, recurrence, or potential complications.

Keywords: paediatrics, arthritis, Kawasaki disease, coronary aneurysm, recurrent Kawasaki disease

Streszczenie

Choroba Kawasakiego to ostre układowe zapalenie naczyń o nieznanej etiologii. Diagnoza i leczenie typowego przebiegu choroby zostały już wielokrotnie opisane. Jednak w nietypowych, nawracających lub powikłanych przebiegach choroby Kawasakiego decyzje diagnostyczne i terapeutyczne mogą być trudne. Przedstawiamy przypadek trzyipółletniego chłopca, u którego zdiagnozowano pełną chorobę Kawasakiego i którego początkowo przydzielono do grupy niskiego ryzyka. Pomimo leczenia zgodnie z zaleceniami gorączka się utrzymywała, a ostatecznie rozwinęły się olbrzymie tętniaki wieńcowe. Krótko po wypisie chłopiec został ponownie przyjęty z powodu dolegliwości bólowych stawu biodrowego. Ostatecznie zaobserwowano dwa podobne nawroty w ciągu trzech miesięcy i ostatecznie zdiagnozowano u chłopca nawracającą chorobę Kawasakiego, powikłaną zapaleniem stawów. Początkowo typowy obraz choroby Kawasakiego może nie wskazywać na dalszy skomplikowany przebieg, nawroty i odległe powikłania.

Słowa kluczowe: pediatria, zapalenie stawów, choroba Kawasakiego, tętniaki tętnic wieńcowych, nawrotowa choroba Kawasakiego

INTRODUCTION

Kawasaki disease (KD) is an acute systemic vasculitis of unknown aetiology[1]. The most serious and, at the same time, the most typical complications of the disease are coronary artery aneurysms. The diagnosis and treatment of the typical course of the disease have already been described in current guidelines and literature[1–3]. However, in atypical, recurrent, or complicated cases of KD, diagnostic and therapeutic decisions may be challenging.

In this paper, we aim to present a rare case of a patient with recurrent KD with cardiac and joint complications and to discuss a possible diagnostic and therapeutic approach based on current literature.

CASE REPORT

A 3.5-year-old boy initially presented with a 6-day history of diarrhoea (up to 10 stools per day, with streaks of blood). On the 7th day, he presented with a fever of up to 39°C requiring regular anti-pyrectics administration, and a small-papular rash (first on the abdomen and spreading onto the extremities). The boy was initially diagnosed with scarlet fever by his general practitioner, and a second-generation cephalosporin was administered. A wet cough developed in the next two days, as well as conjunctivitis without exudate, erythematous mouth with cracked lips, feet and hand joints oedema. The boy was admitted to the paediatrics department with the diagnosis of KD and mycoplasma pneumonia. Laboratory tests on admission showed a white blood count (WBC) of 7.10 × 10^3/μL with 52% neutrophils, anaemia (haemoglobin [Hgb] 10.4 g/dL, red blood cells [RBC] 4.07 × 10^6/μL, haematocrit [HCT] 31.4%), increased platelet count of up to 474 × 10^3/μL, C-reactive protein (CRP) 2.48 mg/dL (normal <0.5 mg/dL), and N-terminal pro-B-type natriuretic peptide (NT-proBNP, normal <125 pg/mL) 259 pg/mL. Na and K ion levels, as well as liver function tests, were normal. Treatment included clarithromycin, intravenous immunoglobulin (IVIG) at a dose of 2 g/kg, and acetylic acid (ASA) in an anti-inflammatory dose (i.e. 50 mg/kg/day in four divided doses). Skin lesions, oedema, and mucosal erythema subsided within hours after IVIG administration, but fever and redness of the conjunctiva persisted. Therefore, steroids (methylprednisolone at an initial dose of 1 mg/kg 2 × daily) and infliximab (a tumour necrosis factor-alpha inhibitor, administered intravenously at a dose of 10 mg/kg) were added to the treatment after which the fever subsided. Echocardiography on admission showed a dilated left coronary artery (LCA) measuring 3.5 mm (*z* score +3.56) and a left anterior descending artery (LAD) measuring 2.6 mm (*z* score +2.68) with an irregular inner surface of the vessels. The right coronary artery (RCA) was normal (all *z* score in the text were based on the Montreal norm and the body surface area was calculated using the Haycock formula)[4]. The systolic function

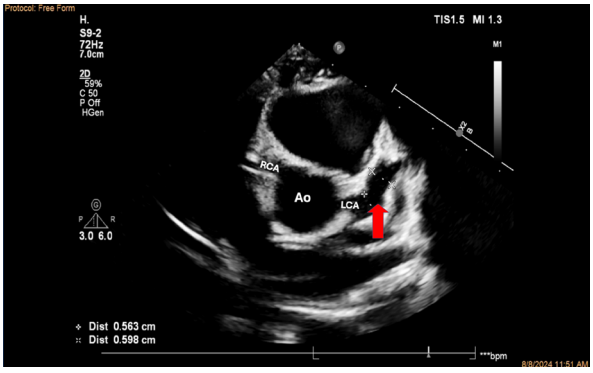

Fig. 1. Echocardiography, parasternal short-axis view, demonstrating an LCA aneurysm (red arrow)

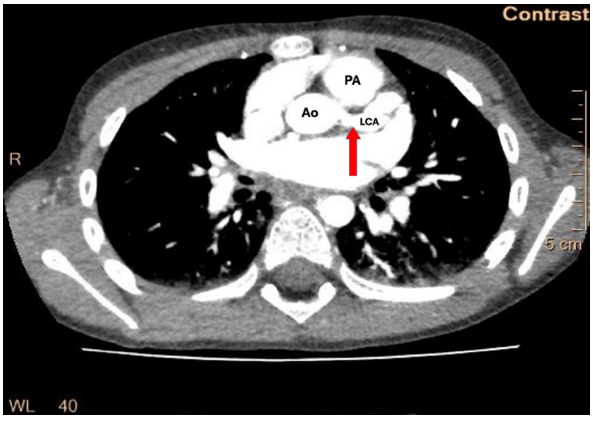

Fig. 2. Computed tomography angiogram of the heart demonstrating a giant coronary aneurysm of the LCA extending into the LAD (red arrow)

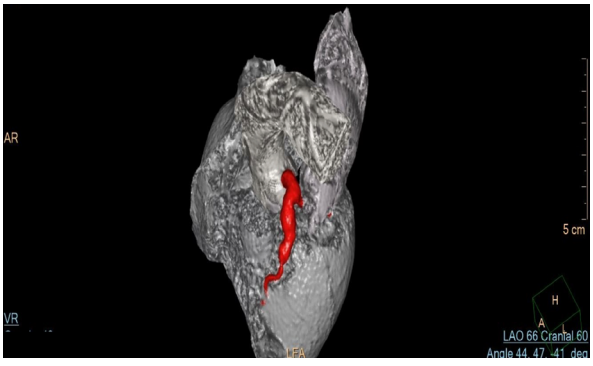

Fig. 3. 3D reconstruction of a computed tomography angiogram reconstruction demonstrating coronary aneurysms of the LCA extending into the LAD

in echocardiography as well as electrocardiography were normal.

The boy was then referred to the cardiology department. Forty-eight hours after the fever subsided, ASA was reduced to an anti-platelet dose (i.e. 3–5 mg/kg/day). After five days, further dilation of the coronaries: LCA to 4.5 mm (*z* score +6.18) and LAD to 2.7 mm (*z* score +2.97) were observed, and clopidogrel was added to the anti-platelet therapy (dosage of 0.2 mg/kg). After the next

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seven days, the boy presented with aneurysms: LCA up to 6 mm (*z* score +10.12) (Fig. 1) and LAD up to 4 mm (*z* score +6.65). Warfarin with initial heparin bridging was administered. Computed tomography angiography (CTA) confirmed giant coronary aneurysms in the LCA and a medium-sized aneurysm in the LAD (Figs. 2 and 3) as well as a giant aneurysm of the RCA measuring up to 8 mm, *z* score +15.63 (previously not described in echocardiograms), with undisturbed blood flow. The patient was discharged home on anti-coagulant (warfarin) and anti-platelet (ASA) therapy and steroids with gradually decreasing doses, in a good general condition.

The day after the discharge, the patient began to limp, complaining of pain in his left lower limb. After a few days, ultrasound examination of the knee and hip joints was performed in the emergency room (ER) and an increased amount of fluid in the left hip joint was found. Transient synovitis (*coxitis fugax*) was diagnosed, and the boy was discharged home with a recommendation for a conservative lifestyle.

The next day (after another reduction in steroid doses), the boy presented with a fever of up to 38.5°C, petechiae, desquamation of skin on the feet and redness of the conjunctiva, so he was re-admitted to the paediatrics department. Recurrent KD was diagnosed. The steroid doses were increased (prednisone 2 mg/kg/day), and cyclosporine (5–8 mg/kg/day) was initiated. The fever and limping subsided. The boy was then again transferred to the paediatric cardiology department. His condition gradually improved; the skin and mucosal symptoms resolved, and the fever did not return. Initially a possible slight dilatation of the LCA in relation to the previous hospitalisation was observed. However, echocardiography before discharge showed findings comparable to the previous examinations and CTA.

The patient was re-admitted to the cardiology department again three weeks later due to fever and joint pain in hips and knees, and reduced appetite. The recent history included COVID-19 infection confirmed by antigen test a few days before admission.

The patient's physical examination was normal, but persistent fever as well as general malaise were observed. The boy was constantly complaining of leg pain and gradually refused to walk.

The differential diagnoses included macrophage activation syndrome (MAS), vasculitis, Still's disease, antiphospholipid syndrome (APS), thromboembolic complications, bacterial infections (tuberculosis, bartonellosis), and neoplasms. Echocardiography showed persistent, but stable giant coronary aneurysms. Chest X-ray as well as abdominal ultrasound findings were normal. The laboratory tests results are presented in Tab. 1. Multi-joint ultrasound (hips, knees, ankles) showed a small amount of fluid in the left hip joint.

Finally, after excluding the differentials, recurrence of KD with joint inflammation was diagnosed and oral steroids were reinitiated. The symptoms – malaise, leg pain, and recurrent fever – quickly subsided. The boy was then discharged in good general condition with a prescription for a prolonged three-month steroid therapy and continuation of ASA and warfarin.

Laboratory test [unit] / *Local laboratory normal values*	**Result**
Inflammatory markers	
CRP [mg/dL] / *<0.5 mg/dL*	8.9
ESR [mm/1 h] / *<8 mm/1 h*	103
IL-6 [pg/mL] / *<6.9 pg/mL*	37.2
C3 [mg/dL] / *90–180 mg/dL*	199
IgM [mg/dL] / *30–112 mg/dL*	185
Fibrinogen [g/L] / *1.8–3.5 g/L*	7.16
Complete blood count	
WBC [cells/mm^3] / *4.5–10.8 × 10^3/mm^3*	8,240
Hgb [g/dL] / *10.9–14.2 g/dL*	8.6
MCV [fL] / *76–90 fL*	73.2
Neutrophils [%] / *35–45%*	60.9
PLT [cells/µL] / *150–400 × 10^3/µL*	413
Urinalysis	
Erythrocytes [cells/µL] / *<7/µL*	130
Protein [mg/dL] / *<15 mg/dL*	89
Others	
Albumin [g/dL] / *3.8–5.4 g/dL*	3.4
Calcium [mg/dL] / *8.8–10.8 mg/dL*	8
NT-proBNP [pg/mL] / *<125 pg/mL*	127
Normal results	
AST, ALT, creatinine, urea, uric acid, C4, ferritin, IgA, IgG, ASO, phosphates, K, Na, procalcitonin, troponin, ANA, ANCA, aCCP, LDH, RF	Normal
aCCP – cyclic citrullinated peptide antibody; **ALT** – alanine aminotransferase; **ANA** – antinuclear body; **ANCA** – antineutrophil cytoplasmic antibodies; **ASO** – antistreptolysin O; **AST** – aspartate transaminase; **C3** – complement 3 protein; **C4** – complement 4 protein; **CRP** – C-reactive protein; **ESR** – erythrocyte sedimentation rate; **Hgb** – haemoglobin; **IgA** – immunoglobulin A; **IgG** – immunoglobulin G; **IgM** – immunoglobulin M; **IL-6** – interleukin 6; **K** – potassium; **LDH** – lactate dehydrogenase; **MCV**– mean cell volume; **Na** – sodium; **NT-proBNP** – B-type natriuretic peptide; **PLT** – platelets; **RF** – rheumatoid factor; **WBC** – white blood count.	

Tab. 1. Laboratory test results during the third hospital admission (third recurrence of KD)

It has now been three months since the last relapse of KD patient and one month from last follow-up in the cardiology department. The patient remains in good condition and continues to take steroids, with discontinuation planned within the next two weeks. Echocardiography showed persistent giant aneurysms without a tendency to decrease in size. Further follow-up examinations are planned, including CTA one year after the first episode of KD.

DISCUSSION

Kawasaki disease: diagnosis, risk stratification, and treatment

KD is an acute systemic vasculitis with unclear aetiology, usually affecting children under five years of age. Association of various infections, for example *Streptococcus pyogenes* and *Mycoplasma pneumoniae* are taken into consideration in its pathogenesis[5–7] – *Mycoplasma pneumoniae* was diagnosed in the presented boy at the time of the first

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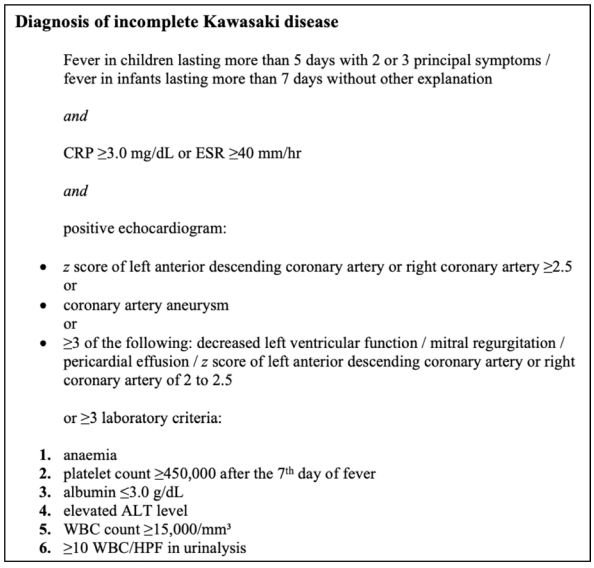

Fig. 4. Algorithm for the diagnosis of incomplete/atypical KD in patients with prolonged unexplained fever, two or three principal symptoms, and typical echocardiographic or laboratory findings according to McCrindle et al.[1]

episode of KD and COVID-19 infection occurred shortly before the second relapse. The most typical symptom is fever lasting more than five days (typically high-spiking, poorly responsive to antipyretics; in the absence of appropriate therapy, fever might persist for even three to four weeks). In addition to fever, four of the following five symptoms must be present to diagnose complete KD[1]: bilateral bulbar conjunctivitis without exudate (painless and usually appears shortly after the onset of fever), oropharyngeal changes (such as erythema and cracking of the lips and/or strawberry tongue), rash (most commonly maculopapular or diffuse erythroderma, typically within three to five days of disease onset), hand and foot erythema and oedema, cervical lymphadenopathy (unilateral and ≥1.5 cm in diameter, usually firm, non-fluctuant, not associated with marked erythema of the overlying skin, and unresponsive to antibiotic therapy). Desquamation of the fingers and toes is observed within two to three weeks after the onset of fever and may extend to the palms and soles.

In patients with prolonged unexplained fever, two or three of the principal symptoms, and typical echocardiographic or laboratory findings, incomplete/atypical KD can be recognised (according to the algorithm shown in Fig. 4)[1]. In some patients, symptoms may be less typical, making diagnosis difficult (e.g. meningitis, seizures, facial paralysis, acute abdomen, acute pancreatitis, cholestatic jaundice, arthritis, kidney damage, pneumonia, etc.)[2].

The patient met the criteria for the diagnosis of complete KD and received first-line treatment according to the Italian and American Heart Association recommendations[1,2]. According to the literature, this treatment applied before the 10th day of illness can reduce the risk of coronary aneurysm from 25% to 5%[8].

According to recommendations, additional treatment (like re-administration of IVIG, corticosteroids, or infliximab) is reserved for patients at high risk of being resistant to intravenous immunoglobulin therapy or at high risk of developing coronary artery aneurysm. Children under one year of age are at particularly high risk as well as patients with an atypical course[8,9]. Several additional factors which may indicate the risk of resistance to IVIG therapy have been identified. To identify this group of patients, several scoring systems have been developed, for example the Kawanet score or the Kobayashi score. In the Kawanet score, hepatomegaly, alanine aminotransferase level >30 IU/L, lymphocyte count <2,400/mm^3, and time to treatment <5 days are identified as risk factors (if the patient has two or more of the above)[8].

In the Kobayasi score, the following aspects are taken into account: sodium ≤133 mmol/L, days of illness at initial treatment ≤4, aspartate transaminase ≥100 IU/L, percentage of neutrophils ≥80%, CRP ≥10 mg/dL, age ≤12 months, platelet count ≤300 × 10^9/L.

The described patient had a score of 0 on both the Kawanet and Kobayasi scales, so he was initially classified as a low-risk patient. Second-line therapy should be implemented to patients resistant to the first dose of IVIG. Unresponsiveness to IVIG (occurring in about 24% patients) is defined as the presence of fever or recurrence of fever at least 36 hours after the end of IVIG infusion[10]. According to a retrospective analysis by Muto[11], the WBC count within 24 hours after IVIG was significantly lower in responders than in non-responders (5,600/μL vs. 9,800/μL) and could potentially be used for early prediction of unresponsiveness to IVIG (thus allowing decisions to initiate second-line therapy earlier). The WBC count in our patient within 24 after IVIG was 7,100/μL, so unfortunately this parameter was not informative for guiding the therapeutic decision.

Next, the fever persisted after initial therapy – according to the recommendations, the boy had a ninefold increased risk of developing coronary artery anomalies. In such patients, the following therapy should be implemented: a second dose of immunoglobulins, high doses of steroids, and/or infliximab[12]. Although our patient was treated according to these recommendations, and the fever and other symptoms subsided, progressive coronary dilatation was still observed.

Kawasaki disease recurrence and cardiac complications

Liu et al.[13] defined progressive coronary dilatation as a gradual enlargement of the coronary arteries on three consecutive echocardiograms (an increase in dimensions by at least 8% between two subsequent tests). They also observed that an initial maximal coronary *z* score of ≥2.5 and hypoalbuminaemia were associated with progressive coronary dilatation. Only the first of these risk factors occurred

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in our patient (whereas in the retrospective study by Liu et al.[13], in which 169 patients with KD where enrolled, none of the patients with normal serum albumin levels developed progressive coronary dilatation). Terai at al.[14] explained that the pathogenesis of both: coronary aneurysm and hypoalbuminaemia is the same – vascular inflammation and high vascular endothelial growth factor level might lead to vascular leakage and coronary dilatation.

In the presented case, when the size of the aneurysms had stabilised, it was decided to gradually decrease the doses of steroids; however, when small doses of prednisone were still administered, the first relapse of the disease occurred. According to the literature, an episode of symptoms of complete or incomplete KD that occurs after the complete resolution of the previous episode is defined as recurrent KD; most recurrences occur within two years after the initial episode regardless of the occurrence of coronary dilatation during the first episode[5]. Moreover, the likelihood of recurrence is known to be higher among males, children younger than three years old, and those who received intravenous immunoglobulin during the initial episode[15–17]. However, contrary data in the literature can be also found: Maddox et al.[18] described different risk factors for relapse, including older age, atypical presentations, and coronary artery anomalies, regardless of previous IVIG therapy. Generally, recurrent KD is identified in 1.7–3.5% of all KD patients[6] and the course of relapse is usually more severe than that of the first episode[18]. About 0.2% of patients suffer multiple relapses[12]. High-dose pulse steroids, cyclosporine, and infliximab are listed in the American Heart Association recommendations as therapy for the recurrence of KD[1]. Italian guidelines also mention clinical trials for human interleukin-1 receptor antagonist protein[3].

Kawasaki disease recurrence and joint complications

The third recurrence of KD occurred after the next six weeks (one week after discontinuing steroids) and was accompanied by persistent fever and symptoms of arthritis. According to the literature, two types of arthritis associated with KD can be distinguished[19]. The early-onset type, which develops within the first two weeks of illness, is more frequent than that occurring in the subacute phase (53% vs. 47%), affects older patients and usually involves multiple joints (elbows, fingers – especially the proximal interphalangeal joint). This type of arthritis is usually self-limiting[20]. Arthritis during the subacute phase usually appears around the 20th day after KD onset and involves large joints: knees, ankles, hips; in about 50% of cases fever is recorded. Non-steroidal anti-inflammatory drugs (NSAIDs) resistance occurs in about 40% of patients; in such cases, prednisolone is used with rapid improvement – treatment with biological agents is not necessary and generally, the prognosis for arthritis is good. After re-initiation of oral steroids in our patient, the arthritis symptoms subsided. No recurrence of arthritis was observed at the three-month follow-up.

CONCLUSIONS

- The initial typical presentation of KD and treatment implementation may not predict a typical course, response to treatment, recurrence as well as further complications.
- Although the most common and also the most serious consequences of KD are cardiac complications including coronary aneurysms, joint complications in the form of arthritis may also occur among these patients.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study; analysis and interpretation of data; writing of manuscript: AC, JHS. Collection, recording and/or compilation of data: AC, JHS, MF. Critical review of manuscript: BW. Final approval of manuscript: MON, EK, MF, BW.

References

1. McCrindle BW, Rowley AH, Newburger JW 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–e99.
2. Marchesi A, Tarissi de Jacobis I, Rigante D et al.: Kawasaki disease: guidelines of the Italian Society of Pediatrics, part I – definition, epidemiology, etiopathogenesis, clinical expression and management of the acute phase. Ital J Pediatr 2018; 44: 102.
3. Marchesi A, Tarissi de Jacobis I, Rigante D et al.: Kawasaki disease: guidelines of Italian Society of Pediatrics, part II – treatment of resistant forms and cardiovascular complications, follow-up, lifestyle and prevention of cardiovascular risks. Ital J Pediatr 2018; 44: 103.
4. Pettersen MD, Du W, Skeens ME et al.: Regression equations for calculation of *z* scores of cardiac structures in a large cohort of healthy infants, children, and adolescents: an echocardiographic study. J Am Soc Echocardiogr 2008; 21: 922–934.
5. Medaglia AA, Siracusa L, Gioè C et al.: Kawasaki disease recurrence in the COVID-19 era: a systematic review of the literature. Ital J Pediatr 2021; 47: 95.
6. Hamada H, Sekizuka T, Oba K et al.: Comprehensive pathogen detection associated with four recurrent episodes of Kawasaki disease in a patient during a single year using next-generation sequencing. JMM Case Rep 2016; 3: e005019.
7. Guo Y, Yang L, Shao S et al.: Coronary artery dilation in children with febrile illnesses other than Kawasaki disease: a case report and literature review. Heliyon 2023; 9: e21385.
8. Koné-Paut I, Piram M: Importance of predicting non-response to intravenous immunoglobulin therapy in non-Asian patients with Kawasaki disease. Lancet Reg Health Eur 2022; 22: 100507.
9. Goswami N, Marzan K, De Oliveira E et al.: Recurrent Kawasaki disease: a case report of three separate episodes at >4-year intervals. Children (Basel) 2018; 5: 155.
10. Dionne A, Le CK, Poupart S et al.: Profile of resistance to IVIG treatment in patients with Kawasaki disease and concomitant infection. PLoS One 2018; 13: e0206001.
11. Muto T, Masuda Y, Numoto S et al.: White blood cell and neutrophil counts and response to intravenous immunoglobulin in Kawasaki disease. Glob Pediatr Health 2019; 6: 2333794x19884826.
12. Osman NM: Recurrent Kawasaki disease resistant to initial treatment with intravenous immunoglobulin. Sudan J Paediatr 2012; 12: 65–69.
13. Liu MY, Liu HM, Wu CH et al.: Risk factors and implications of progressive coronary dilatation in children with Kawasaki disease. BMC Pediatrics 2017; 17: 139.
14. Terai M, Honda T, Yasukawa K et al.: Prognostic impact of vascular leakage in acute Kawasaki disease. Circulation 2003; 108: 325–330.
15. Sudo D, Nakamura Y: Nationwide surveys show that the incidence of recurrent Kawasaki disease in Japan has hardly changed over the last 30 years. Acta Paediatr 2017; 106: 796–800.
16. Guleria S, Pilania RK, Jindal AK et al.: Recurrent Kawasaki disease at a tertiary care center in Chandigarh, North West India: 24 years of clinical experience. Int J Rheum Dis 2019; 22: 1183–1187.
17. Kang SJ, Jin BK, Hwang SJ et al.: Sequential changes in left ventricular systolic myocardial deformation mechanics in children with recurrent Kawasaki disease. J Cardiovasc Imaging 2018; 26: 147–154.
18. Maddox RA, Holman RC, Uehara R et al.: Recurrent Kawasaki disease: USA and Japan. Pediatr Int 2015; 57: 1116–1120.
19. Hattori S, Nozawa T, Nishimura K et al.: Characteristics of persistent arthritis with refractory Kawasaki disease: a single-center retrospective study. Sci Rep 2023; 13: 9890.
20. Go E, van Veenendaal M, Manlhiot C et al.: Kawasaki disease and systemic juvenile idiopathic arthritis – two ends of the same spectrum. Front Pediatr 2021; 9: 665815.

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