

Kawasaki Disease Presenting with Fever and Jaundice: Case Report

Xiao-Qin Li¹, Ping Xue^{1,*}, Yan-Mei Zheng^{1,*}, Shuo Liu¹

¹Department of Pediatrics, Taiyuan Children's Hospital & Taiyuan Maternity and Child Health Care Hospital, Taiyuan, Shanxi, China

*Correspondence: ping20050301@163.com (Ping Xue); zyanmei060@sina.com (Yan-Mei Zheng)

Abstract

Kawasaki disease (KD), which is also known as cutaneous mucosal lymph node syndrome, is an acute, self-limiting, necrotizing vasculitis with unclear cause that primarily affects small- and medium-sized blood vessels and most commonly affects children aged 6 months to 5 years. Currently, diagnosis is based primarily on typical clinical symptoms. Approximately 15%–20% of patients are highly suspected of having KD; however, they do not match the diagnostic criteria for typical KD, which is referred to as incomplete Kawasaki disease (IKD), and this has become a major challenge in the diagnosis and treatment of KD. We describe a case of a 7-year-old boy who had a fever and jaundice as his initial symptoms. After a series of clinical laboratory and imaging examinations and marked improvement of symptoms after treatment with intravenous immunoglobulin (IVIG), IKD was considered as the diagnosis. When children present with jaundice and fever, physicians should consider KD as a possible diagnosis to ensure early detection and treatment of the disease.

Key words: Kawasaki disease; incomplete Kawasaki disease; mucocutaneous lymph node syndrome; jaundice; fever

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Introduction

Kawasaki disease (KD) is an acute systemic vasculitis that primarily affects small- and medium-sized blood vessels. Coronary artery disease is the main cause of acquired heart disease in children (McCrindle et al, 2017). The disease usually affects children aged 6 months to 5 years old (Taddio et al, 2012). The diagnosis of KD is based on symptoms and signs, and no specific laboratory diagnostic criteria have been established for KD (Roh et al, 2020). In severe cases, coronary artery lesions and coronary aneurysms can develop in approximately 25% of children with untreated KD, and 5% are treated with high-dose intravenous immunoglobulin (IVIG) (Bajolle and Laux, 2012). The diagnosis of incomplete Kawasaki disease (IKD) is challenging, often leading to diagnostic errors and treatment delays. Histological evidence of liver involvement, inflammatory bile duct injury, and proliferation of bile duct cells (cholangiocytes), has been sporadically reported to be related to KD (Taddio et al, 2012). Cholestasis is uncommon in infants and children with KD.

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Herein, we present a case of a 7-year-old boy with fever and jaundice as the initial symptoms, who was finally diagnosed with KD. This study aims to draw attention to the atypical characteristics of KD to facilitate timely diagnosis and early treatment, prevent misdiagnosis and mistreatment, and avoid the occurrence of serious complications.

Clinical Data

Medical History and Physical Examination

A 7-year-old boy presenting with 5 days of fever and 2 days of jaundiced skin without obvious cause was hospitalized. The maximum temperature was 40 °C, which peaked 3 times per day. The temperature returned to normal after the patient took ibuprofen. He was further treated with oral amoxicillin for 1 day and intravenous cefotaxime for 3 days in the local hospital, but the treatment was ineffective. The outpatient department recommended that the patient be admitted given that “the cause of fever and yellow staining remained to be investigated”. From the onset of fever, the patient occasionally complained of abdominal pain without vomiting, diarrhea, coughing, expectoration, and other symptoms. While he had a poor appetite, his bowel movements and urination frequency were normal.

Physical examination revealed a temperature of 36.3 °C, a pulse rate of 106 beats per minute, a respiration rate of 22 breaths per minute, a blood pressure of 116/58 mmHg, and a weight of 23.5 kg. The patient was conscious with poor reaction and slightly red skin with jaundice (Fig. 1), yellowing of the hands (Fig. 2) and feet, and no obvious rash. The lymph nodes on the right neck, measuring 1.5 × 1.0 cm, were swollen, painful, and hard with clear boundaries. He had throat congestion and a red tongue. Both lungs were clear, and no rales were heard. The heart sounds were audible and rhythmic, and no murmurs were heard. The abdomen was soft, the liver and spleen were not palpable, and bowel sounds were normal. No abnormalities in the nervous system were noted upon examination. No edema and peeling at the extremities were observed. The capillary filling time was 3 seconds. Additionally, it was noted that the patient did not exhibit non-exudative ocular conjunctival congestion, which is often seen in typical cases of KD. Its absence further complicated the initial diagnosis, contributing to the atypical presentation of the disease.

Admission Examination

Complete blood count (CBC) revealed a leukocyte count of $12.39 \times 10^9/L$, a hemoglobin level of 138 g/L, a neutrophil ratio of 82.2%, a lymphocyte ratio of 9.1%, a platelet count of $251 \times 10^9/L$, and an abnormal lymphocyte percentage of 3%. C-reactive protein (CRP) and procalcitonin levels were 76.18 mg/L and 0.75 ng/mL, respectively. The erythrocyte sedimentation rate (ESR) was 74 mm/h. The patient was positive for *Mycoplasma pneumoniae*, and the titer was 1:320. The results of the anti-*Streptococcus* “O” test were <200 IU/mL. The results of liver function tests were as follows: serum alanine aminotransferase (ALT), 174.8 U/L; serum aspartate aminotransferase, 46.9 U/L; serum total protein, 51.40 g/L; serum



Fig. 1. Slightly red skin with jaundice.



Fig. 2. Yellow skin of hands.

albumin, 35.0 g/L; serum total bilirubin, 72.6 $\mu\text{mol/L}$; serum direct bilirubin, 42.9 $\mu\text{mol/L}$; serum alkaline phosphatase, 255.3 U/L; and serum total bile acid, 168.9 $\mu\text{mol/L}$.

The results of other routine tests, including renal function, electrolytes, and coagulation profile, were within normal limits. The patient was negative for hepatitis B, hepatitis C, hepatitis A, syphilis, human immunodeficiency virus (HIV), and various other infections.

Chest radiograph revealed multiple lesions on both lungs. Ultrasonography of the lymph nodes showed multiple visible and partially enlarged lymph nodes in the bilateral neck. The largest lymph nodes on the right and left sides measured 1.4×0.8 cm and 1.1×0.6 cm, respectively. Several lymph nodes could be imaged in the left inguinal area, with the largest one measuring 0.8×0.3 cm, and several lymph nodes could be viewed in the abdominal mesentery, with the largest one measuring 0.9×0.4 cm. These lymph nodes had clear boundaries, oval shapes, and complete lymph node structures, and no abnormal lymph nodes could be found in the right inguinal area. Abdominal ultrasonography did not find abnormalities in the liver, gallbladder, pancreas, spleen, and kidney. No enlargement of the appendix was noted in the right lower abdominal appendix area. By colour Doppler echocardiography, the foramen ovale (3.3 mm) was patent, bilateral coronary arteries were not wide (3.0 and 2.3 mm on the left and right, respectively), and the left ventricular systolic function was normal. The patient's left coronary artery had a Z-score of +2.5, indicating mild dilation. The electrocardiogram revealed sinus tachycardia, which is generally normal.

Course of Diagnosis and Treatment

The patient was initially diagnosed with sepsis and treated with cefotaxime sodium, azithromycin, glycyrrhizin, and rehydration therapy. However, after 2 days of treatment, the fever could not be controlled, with no significant change in the peak temperature or the interval between fevers. Upon admission, the patient did not exhibit skin flushing or bayberry tongue. These symptoms developed during treatment, with skin flushing and bayberry tongue (Fig. 3) noted after the initial antibiotic therapy was ineffective. CBC indicated further increases in leukocyte count, CRP levels, and procalcitonin levels, along with worsening liver function, suggesting a more severe inflammatory response.

Differential Diagnosis

The differential diagnosis for a child presenting with fever and jaundice includes several severe conditions. Infectious hepatitis, caused by viruses such as hepatitis A, B, or C, often presents with jaundice and elevated liver enzyme levels, but our patient tested negative for these infections. Sepsis, a severe bacterial infection, can also cause fever and jaundice. Despite initial antibiotic treatment, the patient's persistent symptoms made sepsis unlikely. Autoimmune hepatitis, characterized by jaundice and elevated liver enzyme levels, was considered. However, the absence of other autoimmune markers and the patient's rapid response to IVIG and aspirin suggested KD instead. Hemolytic-uremic syndrome (HUS), which can present with jaundice, elevated bilirubin levels, and anemia, was ruled out due to the absence of hemolytic anemia and renal impairment. Biliary atresia, a condition typically seen in neonates, causes jaundice and liver dysfunction. The patient's age and clinical presentation were inconsistent with this diagnosis. Additionally, drug-induced liver injury was considered, but the patient had no history of drug exposure that could account for the symptoms. All indices were higher than before they were at the time of admission. Based on the child's clinical manifestations and



Fig. 3. Bayberry tongue.

laboratory examinations, the following observations were made: (1) the child had a fever for more than 5 days, (2) the antibiotic treatment was ineffective, (3) the child developed skin flushing and red bayberry tongue, (4) the child had swollen neck lymph nodes, and (5) the child developed anemia, along with increases in CRP and ESR levels. Based on these observations, the child was diagnosed with IKD, and the original diagnosis was revised.

Subsequently, the child was treated with human immunoglobulin (1 g/kg/day) for 2 days, and 30 mg/kg/day of aspirin was given orally three times at the same time of the day. The body temperature returned to normal on the day following treatment. After 3 days, the aspirin dosage was reduced to 10 mg/kg/day orally. CBC showed a decrease in leukocyte count and neutrophil ratio, with CRP and procalcitonin levels significantly declining. Liver function tests indicated a reduction in alanine aminotransferase (ALT) and bilirubin levels. The D-dimer was 2819 ng/mL, lower than it was at the time of admission. The urinalysis results were normal. The patient's symptoms resolved, and he was discharged after 1 week of treatment.

Follow-up and Outcomes

After 1 week, the child was followed up in the outpatient department. At this time, fingertip peeling was observed (Fig. 4), and follow-up echocardiography showed Z-scores of +1.8 for the left coronary artery and +1.5 for the right coronary artery, both indicating normalization. CBC showed significant decreases in leukocyte count and CRP levels, with further improvement in liver function tests. Oral aspirin therapy was continued at 10 mg/kg/day. Two weeks later, the child was followed up by telephone. No toe peeling was reported (Fig. 5), and aspirin was continued at the original dosage. After 4 weeks, the patient was followed up in the

outpatient department. CBC and CRP levels continued to decrease, liver function tests returned to normal, and the platelet count normalized. After two months, all laboratory indicators, including leukocyte count, neutrophil ratio, CRP, procalcitonin, ESR, ALT, and bilirubin levels, were within normal ranges. The child was advised to stop taking aspirin. Follow-up showed normalization of laboratory indices and resolution of symptoms (Table 1).



Fig. 4. Fingertip peeling.



Fig. 5. Toe peeling.

Table 1. Laboratory results.

Test	Admission	Day 2	Day 5	Follow-up (1 week)	Follow-up (4 weeks)	Follow-up (8 weeks)
Leukocyte count ($\times 10^9/L$)	12.39	14.6	9.6	8.6	8.2	7.88
Hemoglobin (g/L)	138	109	102	112	126	132
Neutrophil ratio (%)	82.2	87.7	71.0	70.0	71.0	71.6
Lymphocyte ratio (%)	9.1	4.8	13.6	14.8	16.2	17.1
Platelet count ($\times 10^9/L$)	251	293	326	512	516	350
CRP (mg/L)	76.18	168.81	50.64	10.4	10.7	<5
Procalcitonin (ng/mL)	0.75	1.50	0.49	-	-	-
ESR (mm/h)	74	120	75	36	22	7
ALT (U/L)	174.8	224.9	42.7	42.7	24.7	24.7
Serum aspartate aminotransferase (U/L)	46.9	49.9	24.7	24.7	-	-
Serum total protein	51.40	46.40	66.4	72.4	-	-
Serum albumin (g/L)	35.0	30.0	26.8	42.2	-	-
Total bilirubin ($\mu\text{mol/L}$)	72.6	92.7	14.9	10.9	-	-
Direct bilirubin ($\mu\text{mol/L}$)	42.9	48.3	6.59	4.12	-	-
Alkaline phosphatase (U/L)	255.3	312.3	138	170	-	-
Total bile acid ($\mu\text{mol/L}$)	168.9	224.8	14.6	8.6	-	-

Note: “-” indicates undetected. CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; ALT, alanine aminotransferase.

Leukocyte count and CRP levels: Highlighted significant changes and their correlation with treatment interventions.

Liver function tests: Annotated trends in ALT and bilirubin levels, indicating liver inflammation resolution.

Discussion

The etiology of KD is unclear. At present, genetic susceptibility factors may be the root cause based on the induction of infection. Given the lack of specific diagnostic tests, the diagnosis of KD mainly depends on clinical symptoms and signs. Typical KD occurs in children younger than 5 years, manifesting as fever for more than 5 days. At least four of the five diagnostic criteria must be met (de Graeff et al, 2019; Zhu and Ang, 2016; McCrindle et al, 2017; Ramphul and Mejias, 2018): (1) oral lesions, such as erythema of the oropharyngeal mucosa, cracked lips, and strawberry tongue; (2) bilateral conjunctivitis without exudate; (3) pleomorphic rash; (4) edema or erythema of the limbs and progressive peeling of hands and feet; and (5) cervical lymphadenopathy, usually unilateral (≥ 1.5 cm). In the early stage, our patient did not present with these symptoms. The patient mainly presented with fever, jaundice, skin flushing, and cervical lymph node enlargement. Due to a lack of evidence, we could not determine whether these symptoms were caused by hepatobiliary disease, and clear guidance could only be provided through clinical experience and relevant literature. IKD is relative to complete KD (cKD). It occurs in those with fever for more than 5 days and only meets 2–3 of the other 5 key symptoms, that is, patients whose clinical manifestations do not fully meet the diagnostic criteria of KD. The clinical manifestations of IKD do not follow a clear pattern or an established standard during onset, resulting in differences among children. According to the 2017 American Heart Association (AHA) guidelines (McCrindle et al, 2017), IKD should be considered if a child meets the requirements of fever for more than 5 days, CRP ≥ 40 mg/L, ESR ≥ 40 mm/h, anemia, elevated ALT levels, and white blood cell counts of $15 \times 10^9/L$.

The pathogenesis of KD involves widespread inflammation of small and medium-sized blood vessels, leading to vasculitis. This inflammation is thought to result from an immune-mediated response triggered by an unknown antigen that is potentially infectious. The resulting immune activation leads to endothelial damage and inflammatory cytokine release, which contributes to the vascular and systemic manifestations of KD (Zhu et al, 2021).

KD typically presents with prolonged fever, conjunctival injection, oral mucosal changes, rash, extremity changes, and cervical lymphadenopathy. However, atypical presentations, such as jaundice and gastrointestinal symptoms, complicate diagnosis. IKD presents a significant diagnostic challenge as it does not fulfill all the classic criteria. This underscores the need for heightened clinical suspicion and consideration of KD in the differential diagnosis of prolonged fever and jaundice in children (Cavalcante et al, 2021).

IKD is the systemic inflammation of small and medium blood vessels in the whole body. Cardiac involvement is the most common manifestation, but the disease can also involve various organs and tissues in the body, such as the respiratory system, urinary system, hematopoietic system, digestive system, and central nervous system to varying degrees of involvement (Arora et al, 2019; Arslanoglu Aydin et al, 2019; Karunakar et al, 2020; Osman et al, 2021; Singh et al, 2018; Watanabe, 2015; Zhang et al, 2019; Zhuang et al, 2017). In addition to fever, jaun-

dice, which is relatively rare in clinical practice, was the main manifestation of our patient. The patient was 7 years old, suggesting he falls outside of the KD population. Treatment with antibiotics could not resolve the symptoms. Moreover, the relevant inflammatory indices were higher than at the time of admission, suggesting a stronger inflammatory reaction. After consulting relevant literature reports and re-evaluating laboratory test results, the child was found to have IKD. After treatment with human immunoglobulin, all inflammatory indices recovered quickly, which confirmed that our diagnosis was correct. After 2 weeks, the patient's fingertips were peeling, which further confirmed the initial diagnosis. Therefore, the experience of diagnosing, treating, and following up on this case has provided valuable insights.

To strengthen the persuasiveness of our case report, we have included descriptions of recent cases from the literature that highlight similar presentations of KD with fever and jaundice. These cases emphasize the importance of considering KD in the differential diagnosis for such symptoms. A 4-year-old boy presented with fever, abdominal pain, vomiting, jaundice, and mild liver enlargement, gradually developing other KD symptoms and signs. He was treated with aspirin, IVIG, and methylprednisolone, with no sequela (Talebian et al, 2015). In another case, a six-year-old boy presented afebrile but with recent fevers and jaundice, along with an erythematous tongue with papules. Subsequently, he developed continuous fever, a maculopapular rash, and desquamation of hands and feet. Diagnosed with IKD and treated with IVIG, he avoided cardiac complications. This case highlights the rare presentation of IKD with hyperbilirubinemia when the patient is afebrile at the initial presentation (Bylund et al, 2020).

In a study by Taddio et al (2012), children with acute febrile cholestatic jaundice later diagnosed with KD showed similar initial symptoms but had varying responses to treatment. The study indicated that early treatment with IVIG and aspirin significantly improves outcomes, similar to our case. However, our patient presented with higher initial levels of CRP and ESR, indicating a more severe inflammatory response, which resolved rapidly after treatment with IVIG and aspirin. This supports the importance of early intervention in preventing severe complications.

Furthermore, Bajolle and Laux (2012) evaluated different treatment regimens, including corticosteroids and other immunoglobulin doses, for refractory KD cases. The patient responded well to the standard IVIG and aspirin regimen without the need for additional interventions, highlighting the effectiveness of this approach in typical IKD presentations.

Future research should focus on identifying the precise etiological agent or agents responsible for triggering KD. Advances in genomic and proteomic technologies may help elucidate the genetic predispositions and molecular pathways involved in KD pathogenesis. Additionally, research into the development of specific biomarkers for early and accurate diagnosis is crucial, particularly for atypical and incomplete presentations.

Immunological studies are also vital to better understanding the aberrant immune response in KD. Investigating the role of specific cytokines, immune cells,

and their interactions can provide insights into targeted therapies. Furthermore, long-term studies on the cardiovascular outcomes of KD patients are needed to optimize management strategies and improve prognostic assessments.

Conclusion

KD involves multiple systems that trigger an inflammatory response. Its diagnosis is complex and lagging, which can easily result in misdiagnosis and delay the optimal time of treatment. In cases of fever of unknown cause, the possibility of KD must be considered. Early treatment can prevent the occurrence of serious complications.

Learning Points

- Because of the variety of symptoms of Kawasaki disease, it is important to perform a thorough history and physical examination and to analyse the symptoms and signs thoroughly.
- The possibility of incomplete KD should be considered when the cause of the fever is unclear.
- Early diagnosis and prompt treatment with IVIG and aspirin are crucial in KD and IKD to prevent severe complications such as coronary artery aneurysms. The rapid improvement in our patient's symptoms following treatment emphasizes the importance of early intervention.
- Elevated inflammatory marker levels (e.g., CRP, ESR) and liver function abnormalities can support the diagnosis of KD when clinical symptoms are atypical. Our case demonstrates the importance of a thorough laboratory evaluation in guiding diagnosis and treatment decisions.

Availability of Data and Materials

All the data of this study are included in this article.

Author Contributions

SL was the patient's attending doctor. YMZ and PX reviewed the literature. YMZ, PX, SL and XQL analyzed the data. YMZ, PX and XQL drafted the manuscript. All authors contributed to the important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the principles of the Helsinki Declaration. The study protocol was approved by the Ethics Commission of Taiyuan Children's Hospital & Taiyuan Maternity and Child Health Care Hospital (Approval number: 2024-21). Additionally, we confirm that the guidelines for case

reports as outlined by the EQUATOR Network were followed. The guidelines can be found on the EQUATOR Network's website. Written informed consent was obtained from the parent(s)/guardian(s) of the patient.

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Conflict of Interest

The authors declare no conflict of interest.

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