DOI: https://doi.org/10.54393/pjhs.v6i4.2699

PAKISTAN JOURNAL OF HEALTH SCIENCES
(LAHORE)
https://thejas.com.pk/index.php/pjhs
ISSN (E): 2790-9352, (P): 2790-9344
Volume 6, Issue 04 (April 2025)

Case Report

OPEN ACCESS

A Case Report on Two-Month-Old-Infant Atypical Kawasaki Disease

Razia[1*], Shafaat Ullah[1], Kainat Asmat[1], Mohammad Saleem[1], Ihsan Ur Rehman[1] and Jahanzeb[2]

[1]Shifa College of Nursing, Shifa Tameer-e-Millat University, Islamabad, Pakistan
[2]Khyber Medical University, Peshawar, Pakistan

ARTICLE INFO

Keywords:
Atypical Kawasaki Disease, Infant Presentation, Coronary Complications, Early Diagnosis

How to Cite:
Razia, ., Ullah, S., Asmat, K., Saleem, M., Rehman, I. U., & Jahanzeb, . (2025). A Case Report on Two-Month-Old-Infant Atypical Kawasaki Disease: Atypical Kawasaki Disease in Infant. Pakistan Journal of Health Sciences, 6(4), 278-284. https://doi.org/10.54393/pjhs.v6i4.2699

Corresponding Author:
Razia
Department of Clinical Nursing, Shifa Tameer-e-Millat University, Islamabad, Pakistan
razia.ali.baig@gmail.com

Received date: 30th December, 2024
Revised date: 11th March, 2025
Acceptance date: 8th April, 2025
Published date: 30th April, 2025

ABSTRACT

Kawasaki Disease (KD) is a rare vasculitis that predominantly affects children under five, with atypical presentations posing significant diagnostic challenges, especially in infants. This case report describes a two-month-old male who initially presented with high-grade fever and rash, misdiagnosed as subclinical meningitis.Despite multiple hospitalizations and extensive investigations, a definitive diagnosis was delayed.The patient later developed seizures, respiratory distress, and shock, necessitating intensive care.Laboratory findings revealed elevated inflammatory markers, anemia, thrombocytosis, and abnormal echocardiographic findings. Whole-body CT and detailed ECHO ultimately confirmed atypical KD with multiple thromboses and giant coronary aneurysms. Prompt treatment with intravenous immunoglobulin (IVIG), corticosteroids, anticoagulants, and antiplatelet agents was initiated. Although the patient improved clinically, irreversible cardiovascular complications had developed. This case highlights the importance of maintaining a high index of suspicion for atypical KD in febrile infants lacking classic symptoms. Early diagnosis and timely initiation of IVIG are essential to prevent long-term cardiac sequelae. Enhanced clinical awareness and refined diagnostic protocols are critical for improving outcomes in such vulnerable pediatric populations.

INTRODUCTION

Kawasaki Disease (KD), initially described by Tomisaku Kawasaki in Japan, is a rare illness affecting children worldwide, predominantly those under five years old, with a higher incidence in boys [1]. It is the leading cause of acquired cardiac complications, including myocardial ischemia and infarction, in children in developing countries, potentially leading to mortality [2]. Prompt diagnosis is crucial to mitigate complications such as arteritis and aneurysms. Although the etiology remains unknown, typical symptoms include fever, rash, swollen extremities, and conjunctival redness. KD is less common in infants under four months, possibly due to maternal antibody protection. It primarily affects children of Asian descent, particularly Japanese children, and is less prevalent in Caucasians [3]. In the United States, over 5,000 children under 18 were hospitalized with KD in 2019, with 3,693 under five years old [4]. Early detection of KD can be improved by increasing clinical awareness and refining diagnostic approaches. Given that KD diagnosis is primarily clinical, a high index of suspicion, particularly in febrile children with prolonged fever, is essential. Standardized guidelines and diagnostic algorithms, such as those from the American Heart Association (AHA), aid in early identification, especially in incomplete KD. Regular monitoring of inflammatory markers (CRP and ESR) and echocardiography to assess coronary artery involvement are also valuable. Emerging research on biomarkers and genetic susceptibility may further improve diagnostic accuracy [5]. In infants, particularly those two months old, KD often presents atypically, with fewer hallmark

DOI: https://doi.org/10.54393/pjhs.v6i4.2699

symptoms, making diagnosis challenging. Prolonged fever and irritability may be the primary presentations, leading to delayed recognition and increased risk of coronary artery complications[6].

Case Presentation

A previously healthy two-month-old male infant presented to a remote private healthcare facility with a two-day history of recurring high-grade fever and a rash that appeared during fever spikes. Initially diagnosed with subclinical meningitis, empirical antibiotic therapy was initiated. Cerebrospinal Fluid (CSF) analysis was negative, but antibiotics were continued due to persistent symptoms. On the sixth day, a fever spike and seizure activity led to respiratory distress requiring oxygen support. Due to the patient's deteriorating condition, a transfer to a hospital with advanced resources was arranged. The patient was placed on CPAP, and the fever subsided. He was discharged on the fifth day with intravenous antibiotics. However, fever spikes recurred post-antibiotic treatment, leading to readmission. The patient's condition worsened, necessitating transport via ambulance with oxygen support for further management.

Case History

The patient presented to a tertiary care hospital with drowsiness, pallor, and tachypnea. After initial stabilization, diagnostic investigations, including blood tests (CBC, RFTs, LFTs, CRP), imaging (CT head, chest X-ray, abdominal ultrasound), and blood cultures, were initiated. Initial treatment focused on fever and tachycardia management. The patient was transferred to the pediatric ICU due to elevated CRP (>300), indicating severe inflammation, and hepatosplenomegaly. Septic shock prompted elective intubation and infectious disease consultation. Blood transfusions were administered for persistent anemia. Despite extensive workups, including ECHO, EEG, repeated imaging, and multiple blood cultures, no infection source was identified. Post-extubation, CRP decreased, and fever subsided for two days.However, the fever recurred with rising CRP, anemia, and thrombocytopenia, requiring further transfusions. Repeat diagnostic tests were inconclusive, with mild pericardial effusion attributed to infection. The patient's condition remained unclear despite multidisciplinary involvement. On the 14th day, a bone marrow biopsy was requested, but the patient refused and requested discharge. He was transferred to another tertiary care hospital. At the new hospital, atypical KD was diagnosed via whole-body CT and detailed ECHO, revealing multiple thromboses and aneurysms, including coronary artery involvement. Treatment was initiated, and the patient was discharged after a week with continuous IVIG, blood thinners, and follow-up with consultants. In atypical KD, multiple thromboses and coronary aneurysms necessitate intensified IVIG therapy and anticoagulation, guided by echocardiographic monitoring. Corticosteroids may be considered in severe cases[3].

Physical Presentation

Physical examination revealed a GCS of 14/15, pallor, respiratory rate of 50 breaths/minute, temperature of 101 °F, and oxygen saturation of 96% on 4L oxygen. Capillary refill was delayed, pulse rate exceeded 200 beats/minute, and blood pressure was normal. Bruises from multiple pricks and mild peripheral swelling were noted, but the chest examination was clear. The patient's condition necessitates consideration of several differential diagnoses. Meningitis remains a critical possibility due to the presence of neurological symptoms and potential meningeal irritation. Septic shock must also be considered, particularly in the context of systemic signs of infection and hemodynamic instability. Another important diagnosis is COVID-related Multisystem Inflammatory Syndrome in Children (MIS-C), which presents with persistent fever, inflammation, and multiorgan involvement, often following exposure to or infection with SARS-CoV-2. Additionally, Hemophagocytic Lymphohistiocytosis (HLH) should be evaluated, given its association with unremitting fever, cytopenias, and hyperinflammatory response, which can mimic severe infections or autoimmune conditions.

Challenges in Diagnosing Atypical KD

The diagnostic complexities inherent in atypical Kawasaki Disease (KD) are particularly pronounced in infants less than four months of age.The increased incidence of incomplete symptomatology in this population significantly impedes clinical recognition, frequently resulting in delayed therapeutic intervention.This delay, even when followed by timely Intravenous Immunoglobulin (IVIG) administration, increases the risk of coronary artery sequelae. Furthermore, the clinical manifestations of KD may be indistinguishable from those of viral and bacterial infections, leading to potential diagnostic errors. While echocardiography serves as a valuable adjunct, the detection of early coronary artery changes may prove challenging, necessitating serial imaging studies. Accordingly, a high index of clinical suspicion and meticulous patient monitoring are essential for the timely and accurate diagnosis of KD in this vulnerable demographic[6].

Laboratory and Diagnostic Test Findings with Rationale

Several diagnostic approaches are used to explore atypical KD and its risk factors. Initial evaluation includes blood tests and imaging studies (chest radiography, head CT, ultrasound, EEG). The patient has several abnormal findings, including hepatosplenomegaly (enlarged liver and spleen) observed on ultrasound, extra pleural fluid likely due to infection on the ECHO, and multiple aneurysms in

DOI: https://doi.org/10.54393/pjhs.v6i4.2699

large and medium-sized vessels along with infarcts in both the kidneys and spleen on the Pan CT. Additionally, a detailed ECHO revealed severely dilated coronary arteries, with a giant aneurysm in the right coronary artery and severe dilation of the left main coronary and circumflex arteries shown in table 1.

Table 1: Summary of Patient's Diagnostic Procedures

Procedures	Patient's Diagnosis
Chest x-ray	Normal
CT head with contrast	Normal
Ultrasound abdomen	Hepatosplenomegaly
EEG	Normal
ECHO	Extra pleural fluid due to infections*3
Pan CT	Multiple aneurysms of large and medium-sized vessels, along with infarcts in both kidneys and spleen
Detailed ECHO (conscious sedation)	Severely dilated right coronary artery with giant aneurysm formation. Severely dilated left main coronary and circumflex artery.

The lab results show anemia (low Hb and Hct), elevated White Blood Cells (WBC) and platelets, indicating possible infection or inflammation. Fibrinogen, D-Dimer, CRP, and ferritin are all elevated, suggesting inflammation. BNP is high, indicating potential cardiac stress. Electrolyte imbalances include low calcium and bicarbonate. INR and PT are elevated, indicating possible coagulation issues. Procalcitonin is normal, ruling out significant bacterial infection as shown in table 2.

Table 2: Summary of Patient's Laboratory Tests first and Last

Labs	Value	Labs	Values
Hb	7.9<9.2	BUN	4<18 mg/dL
Hct	24.8< 29.1	CR	0.30<0.17
WBC	15190< 12850/ µL	Na	136<139mEq
Platelets	698000< 202000	K	4.9<3.1
Serum urea	8.56<38.52mg/dL	Cl	101<98
Fibrinogen level	612.40 mg/dl (82-303)	HCO_3	18<29mEq/ L (13-22)
D-Dimer	7.29 mg/L (upto 0.50)	Ferritin	1914 ng/mL (30-400)
CRP-high sensitivity	331 <116 mg/dl (upto 0.05)	Triglycerides	227<484 (less than 150)
BNP	300	-	-
Blood culture: No growth*5			
S. procalcitonin	0.38 ng/mL	Mg	2.29 mg/dL
BNP	332.3 pg/mL	Ca	7.5 mg/dL
INR	1.38	Albumin	3.1 g/dL
PT	15 sec	-	

The blood gas results show acidosis with a low pH (7.32) and elevated PCO2 (47.8 mmHg), suggesting respiratory acidosis. Bicarbonate (HCO3) is near normal but lower than expected, indicating the body's compensatory response is not fully adequate. The base excess of -1.8 mEq/L suggests mild acidosis. O2 saturation is extremely low (22.9%), which is concerning for severe hypoxia. The elevated lactate (4.93) indicates potential tissue hypoxia or shock detail in table 3.

Table 3: Summary of Patient's Venous/ Arterial Blood Gases first and Last Report

Variables	Initial Volume	Follow-up Volume
PH	7.32	7.46
PCO_2	47.8mmHg	47.4
HCO_3	24.3mmHg	33.1
Base excess	-1.8 mEq/L	8.4 mmol/L
O2 saturation	22.9%	55.7 %
Lactate	4.93	2.23

Atypical KD can be differentiated from septic shock, meningitis, and Multisystem Inflammatory Syndrome in Children (MIS-C) based on clinical and laboratory findings. Atypical KD presents with prolonged fever, bilateral non-exudative conjunctivitis, polymorphous rash, mucosal changes (strawberry tongue, cracked lips), and extremity changes like erythema and desquamation, with thrombocytosis in later stages. In contrast, septic shock is characterized by acute deterioration, hypotension, multi-organ failure, and Disseminated Intravascular Coagulation (DIC), with thrombocytopenia and markedly elevated inflammatory markers. Meningitis often presents with photophobia, neck stiffness, altered mental status, and Cerebrospinal Fluid (CSF) findings of increased white blood cells and protein with low glucose. MIS-C, associated with SARS-CoV-2, mimics KD but typically affects older children, presents with severe gastrointestinal symptoms, cardiovascular dysfunction (myocarditis, coronary artery abnormalities), and markedly elevated inflammatory markers like CRP, ferritin, and D-dimer. A history of recent COVID-19 infection, severe myocardial involvement, and lymphopenia help distinguish MIS-C from atypical KD [11].

Management

The patient underwent a multifaceted treatment approach, including intravenous antibiotics, antivirals, and antifungals, alongside comprehensive diagnostic testing, including multiple blood cultures. Given the patient's history of seizures, intravenous anticonvulsants were administered. Due to the initial suspicion of meningitis, a standard treatment regimen was initiated. The patient's elevated C - reactive protein (CRP) and lactate levels were addressed through elective intubation for 48 hours. Colistin and solumedrol were added to the therapeutic regimen to aggressively combat the presumed infection, with guidance from Infectious Disease (ID) specialists. A cardiac consultation was conducted due to persistent tachycardia, and an echocardiogram revealed mild pericardial effusion, prompting a specialist to suggest diuretic therapy. A Cerebrospinal Fluid (CSF) analysis was performed to confirm the diagnosis; however, it yielded no evidence of infection. Renal function was meticulously monitored, medication dosages were adjusted accordingly, and hepatosplenomegaly was regularly assessed. By the third day, the patient exhibited clinical

DOI: https://doi.org/10.54393/pjhs.v6i4.2699

improvement, was successfully extubated, and achieved vital stability, with a Glasgow Coma Scale (GCS) score of 15/15. The patient was alert and responsive, and the CRP level decreased from 300 mg/dL to 54. However, on the fifth day in the Intensive Care Unit (ICU), the patient experienced a recurrence of fever. Despite the involvement of a multidisciplinary team, a definitive diagnosis remained elusive. Consequently, the patient was transferred to a private tertiary-care hospital. Detailed echocardiography and whole-body Computed Tomography (CT) scans confirmed the diagnosis of atypical Kawasaki Disease (KD). The medical team promptly initiated a treatment protocol that included injectable and anti-inflammatory medications. The diagnosis and its implications were thoroughly explained to the patient's parents. Unfortunately, the diagnostic delay resulted in the development of irreversible aneurysms. By the 14th day, the patient demonstrated significant improvement, became afebrile with stable hemodynamics, and was discharged with subcutaneous anticoagulant therapy, along with oral medications such as clopidogrel and aspirin. The patient was advised to undergo regular follow-up monitoring of International Normalized Ratio (INR), Prothrombin Time (PT), and Complete Blood Count (CBC).

Pharmacological Management

A range of medications was prescribed to manage the patient's condition. Specific medications and their respective classifications, mechanisms of action, and dosages are detailed in Table 4.

Table 4: Medications Prescribed to the Patient during Treatment

Medications	Classification	Mechanism of Actions
Ceftriaxone	Antibiotic (3rd generation cephalosporin)	Inhibits bacterial cell wall synthesis by binding to penicillin-binding proteins, leading to cell lysis and death.
Acetaminophen	Analgesic, Antipyretic	Paracetamol acts centrally to inhibit prostaglandin synthesis for pain relief and lowers fever by affecting the hypothalamus to promote heat loss.
Amikacin	Antibiotic (Aminoglycoside)	Inhibits bacterial protein synthesis by binding to the 30S ribosomal subunit
Vancomycin	Antibiotic (Glycopeptide)	Inhibits cell wall synthesis by binding to D-alanyl-D-alanine portion of cell wall precursors
Meropenem	Antibiotic (Carbapenem)	Inhibits bacterial cell wall synthesis by binding to penicillin-binding proteins
Colistin	Antibiotic (Polymyxin)	Disrupts the bacterial cell membrane by interacting with phospholipids
Solumedrol	Corticosteroid	Suppresses inflammation and immune response by inhibiting the release of pro-inflammatory cytokines mediators.
Levetiracetam	Anticonvulsant	Binds to synaptic vesicle protein 2A (SV2A), reducing neurotransmitter release and modulating synaptic transmission
Dexamethasone	Corticosteroid	Reduces inflammation by inhibiting multiple inflammatory cytokines and suppressing the immune response.
Enoxaparin	Anticoagulant	Inhibits factor Xa and IIa (thrombin), reducing blood clot formation.
Omeprazole	Proton pump inhibitor (PPI)	Inhibits the H+/K+ ATPase enzyme in the gastric parietal cells, reducing gastric acid secretion.
Syrup Pyridoxine	Vitamin	Acts as a coenzyme in amino acid metabolism, neurotransmitter synthesis, and hemoglobin production.
Syrup propranolol	Beta-blocker	Blocks beta-adrenergic receptors, reducing heart rate
Syrup Warfarin	Anticoagulant	Inhibits vitamin K reducing the synthesis of vitamin K-dependent clotting factors (II, VII, IX, X).
Tab Aspirin	Antiplatelet	Inhibits cyclooxygenase-1 (COX-1) enzyme, reducing thromboxane A2 production, which prevents platelet aggregation.
Clopidogrel	Antiplatelet	Inhibits ADP receptor (P2Y12) on platelet surfaces, preventing platelet aggregation and thrombus formation.

The delayed diagnosis of atypical KD resulted in severe cardiovascular complications, including giant coronary aneurysms, infarcts in multiple organs, and pericardial effusion, necessitating an aggressive, multidisciplinary treatment approach. Initial management involved broad-spectrum antibiotics, antivirals, corticosteroids, and supportive therapies, including elective intubation to manage respiratory distress and metabolic abnormalities. Due to persistent cardiovascular involvement, anticoagulation with warfarin and enoxaparin, along with dual antiplatelet therapy (aspirin and clopidogrel), was initiated to prevent thrombotic events. Despite clinical improvement by day 14, the presence of irreversible vascular damage significantly increases the risk of long-term complications such as thrombosis, myocardial ischemia, and sudden cardiac events. Lifelong follow-up with cardiology is essential, requiring serial echocardiograms, CT angiography, and continuous monitoring of inflammatory markers, coagulation status, and cardiac function. Additionally, long-term anticoagulation

DOI: https://doi.org/10.54393/pjhs.v6i4.2699

therapy and potential interventional procedures, such as stent placement or coronary artery bypass grafting, may be required if ischemic complications develop. While the patient's condition stabilized with treatment, the guarded prognosis underscores the importance of early diagnosis and prompt intervention in Kawasaki Disease to prevent severe and irreversible cardiovascular outcomes [12].

DISCUSSION

This case report underscores the diagnostic challenges inherent in atypical Kawasaki Disease (KD), particularly in infants. The two-month-old patient initially presented with high-grade fever and a rash, leading to a misdiagnosis of subclinical meningitis. Despite antibiotic therapy, the patient's condition deteriorated, culminating in seizures and respiratory distress. A diagnosis of atypical KD was only established after transfer to a tertiary care facility and comprehensive investigations, including Echocardiography (ECHO) and whole-body Computed Tomography (CT) scan. Regrettably, the diagnostic delay resulted in severe complications, including thrombosis and aneurysms in multiple arteries [7]. This case emphasizes the critical importance of considering KD in pediatric patients with prolonged fever, even in the absence of typical symptoms, to prevent long-term cardiovascular damage [8].The atypical presentation of KD in infants, especially those under six months, significantly impacts treatment and prognosis.The absence or attenuation of classic symptoms often leads to delayed diagnosis, increasing the risk of coronary artery complications. Early initiation of Intravenous Immunoglobulin (IVIG) therapy is crucial for reducing inflammation and preventing coronary artery aneurysms; however, delays can diminish treatment effectiveness. Prognosis worsens with delayed treatment, as younger infants are more prone to severe cardiac sequelae. Therefore, heightened clinical awareness, early echocardiographic screening, and consideration of incomplete KD in febrile infants are essential for improved outcomes [6]. Differentiating atypical KD from septic shock, meningitis, and MIS-C requires careful assessment of clinical and laboratory features. Atypical KD presents with prolonged fever, mucocutaneous symptoms (bilateral conjunctivitis, lip swelling, strawberry tongue), and potential coronary artery involvement. In contrast, septic shock features systemic infection signs (tachycardia, hypotension, altered mental status), meningitis presents with fever, neck stiffness, and neurological symptoms, and MIS-C exhibits significantly elevated inflammatory markers with cytotoxic profiles. Echocardiographic findings are also instrumental, as atypical KD may reveal cardiac involvement not observed in septic shock or meningitis [9, 5].This case also highlights the management challenges of KD in young infants, who are less frequently affected and often present with atypical symptoms that complicate timely diagnosis [10].In this instance, the patient's condition improved only after receiving IVIG therapy, standard KD treatment, anticoagulants, and anti-inflammatory medications.However, the late diagnosis resulted in irreversible aneurysms, underscoring the need for increased awareness among healthcare providers regarding atypical KD presentations, particularly in infants lacking classic symptoms [7].This case underscores the need for improved diagnostic protocols to facilitate earlier KD identification, thereby preventing severe cardiac outcomes and enhancing patient prognosis. Ureteral stent encrustation remains a significant urological challenge, influenced by multiple clinical, procedural, and biochemical factors.In recent years, numerous studies have expanded our understanding of both the risk factors and management strategies for this complication.A thorough review and updated treatment algorithm provided by Yi XX *et al.*, emphasized the need for early detection and tailored endourological interventions, especially in severely encrusted stents.Their guidance from the European Association of Urology Young Academic Urology Group is vital for clinical decision-making [13]. Li C *et al.*, developed and validated a nomogram-based model using a large Chinese pediatric cohort to predict coronary artery lesions in KD.Their model provides a practical, individualized risk prediction tool that may assist clinicians in early identification of patients at high risk for CALs, supporting precision medicine in KD care [14].One of the persistent clinical challenges in KD is resistance to intravenous immunoglobulin (IVIG) therapy.The KIDCARE trial by Burns JC *et al.*, compared infliximab to a second IVIG dose in resistant cases, demonstrating that infliximab is a viable and possibly superior alternative, especially for rapid symptom resolution and reduced hospitalization duration [15]. Similarly, He L *et al.*, conducted a randomized trial comparing various initial IVIG regimens, highlighting the need for optimal dosing strategies tailored to patient profiles [16].In refractory cases, corticosteroid therapy has been explored.Ogata S *et al.*, found that pulse corticosteroid therapy in combination with IVIG was effective in reducing inflammation and coronary involvement, reinforcing its role in managing IVIG-resistant KD [17].Risk stratification tools continue to evolve. Kobayashi T *et al.*, proposed a scoring system to predict IVIG resistance, which remains a cornerstone in assessing high-risk patients.Their scoring system is widely used, especially in East Asian populations, for tailoring initial treatment [18].McCrindle BW *et al.*, further explored the progression of coronary artery involvement by analyzing serial echocardiographic measurements.They identified specific risk factors associated with aneurysm development, stressing the importance of longitudinal monitoring of coronary dimensions in KD patients [19].On the genetic front, Kuo HC *et al.*, identified the ITPKC gene polymorphism (rs7251246) as a significant marker associated with both KD susceptibility and coronary artery lesion formation.This finding highlights the potential of pharmacogenomic profiling in predicting disease course

DOI: https://doi.org/10.54393/pjhs.v6i4.2699

and guiding early interventions [20]. Collectively, these studies underscore the multi-dimensional nature of Kawasaki Disease, advocating for a combination of clinical scoring, advanced risk modeling, biologic therapies, and genetic markers to improve diagnosis, treatment, and long-term outcomes. Future research must focus on integrating these elements into routine clinical practice and adapting them across diverse populations.

CONCLUSIONS

In conclusion, this case report emphasizes the complexities and critical need for early recognition of atypical KD in infants. The delayed diagnosis in this patient, due to atypical presentation and initial misdiagnosis, resulted in severe cardiovascular complications. This highlights the significance of maintaining a high index of clinical suspicion for KD in cases of persistent fever with unexplained inflammatory markers, even in the absence of typical features.Timely and accurate diagnosis, followed by prompt administration of IVIG and anticoagulation therapy, is essential for preventing life-threatening complications. Enhancing awareness and diagnostic protocols among healthcare providers can significantly improve patient outcomes, particularly in high-risk pediatric populations.

Authors Contribution

Conceptualization: R
Methodology: R, SU, MS
Formal analysis: KA
Writing, review and editing: R, SU, KA, MS, IUR, J
All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

Source of Funding

The author received no financial support for the research, authorship and/or publication of this article.

REFERENCES

[1] Jone PN, Tremoulet A, Choueiter N, Dominguez SR, Harahsheh AS, Mitani Y *et al.* Update on Diagnosis and Management of Kawasaki Disease: A Scientific Statement From the American Heart Association. Circulation.2024Dec;150(23):e481-500.doi:10.1161/CIR.0000000000001295.

[2] Owens AM. Kawasaki Disease [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cited on: 15th Apr 2025]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK537163.

[3] Tutic E and Evey K. Kawasaki Disease: Recent Diagnostics, Treatment, and Predictors for Treatment Failure and Coronary Artery Involvement. Current Emergency and Hospital Medicine Reports. 2023 Dec; 11(4): 158-64. Doi: 10.1007/s40138-023-00273-4.

[4] Jain B, Agarwal H, Bhatia N, Yadav V. Atypical Kawasaki Disease.MaxMedJ.2024Mar;1(1):102-4.doi:10.62830/MMJ1-4-17C.

[5] Scherler L, Haas NA, Tengler A, Pattathu J, Mandilaras G, Jakob A. Acute phase of Kawasaki disease: a review of national guideline recommendations.European Journal of Pediatrics.2022Jul;181(7):2563-73.doi:10.1007/s00431-022-04458-z.

[6] Kuo HC. Diagnosis, progress, and treatment update of Kawasaki disease.International Journal of Molecular Sciences.2023Sep;24(18):13948.doi:10.3390/ijms241813948.

[7] Navidi N, Najibi B, Dinarvand N, Zamani A, Fathi MR. Atypical Kawasaki disease with giant coronary artery aneurysms in a 2-month-old boy:a case report. Journal of Medical Case Reports.2024Dec;18(1):630.doi:10.1186/s13256-024-04987-1.

[8] Raza AA, Khan W, Khan AA, Mahrukh SK, Balasubramanian K.Atypical Kawasaki Disease Presentation in a Previously Healthy Infant:A Diagnostic Challenge. Cureus.2023Oct;15(10).doi:10.7759/cureus.46748.

[9] Tsujioka Y, Handa A, Nishimura G, Miura M, Yokoyama K, Sato K *et al.* Multisystem imaging manifestations of Kawasaki disease.Radiographics.2022Jan;42(1):268-88.doi:10.1148/rg.210070.

[10] Garrido-García LM, Gutiérrez-Alanis JH, Ramírez-Perea AI, Tremoulet A, Yamazaki-Nakashimada MA. Kawasaki disease in infants in the first 3 months of age in a Mexican population: a cautionary tale.Frontiers in Pediatrics.2020Jul;8:397.doi:10.3389/fped.2020.00397.

[11] Marzano AV, Cassano N, Moltrasio C, Verdoni L, Genovese G, Vena GA. Multisystem inflammatory syndrome in children associated with COVID-19: a review with an emphasis on mucocutaneous and kawasaki disease-like findings.Dermatology.2022 Jun; 238(1): 35-43. doi: 10.1159/000515449.

[12] Lee JJ, Lin E, Widdifield J, Mahood Q, McCrindle BW, Yeung RS *et al.* The long-term cardiac and noncardiac prognosis of Kawasaki disease:a systematic review. Pediatrics.2022Mar;149(3):e2021052567.doi:10.1542/peds.2021-052567.

[13] Yi XX, Zhang WR, Wang DM, Wang XP, Zhang FX. Research perspective in the clinical management of Kawasaki disease.Frontiers in Pediatrics.2024Jul;12:1415941.doi:10.3389/fped.2024.1415941.

[14] Li C, Zhang H, Yin W, Zhang Y. Development and validation of a nomogram-based prognostic model to predict coronary artery lesions in Kawasaki disease from 6847 children in China.Computer Methods and Programs in Biomedicine.2025Mar;260:108588.doi:10.1016/j.cmpb.2024.108588.

[15] Burns JC, Roberts SC, Tremoulet AH, *et al.* Infliximab versus second intravenous immunoglobulin for

DOI: https://doi.org/10.54393/pjhs.v6i4.2699

treatment of resistant Kawasaki disease in the USA (KIDCARE): a randomised, multicentre comparative effectiveness trial.The Lancet Child & Adolescent Health.2021 Dec;5(12):852–861.doi:10.1016/S2352-4642(21)00270-4.

[16] He L, Liu F, Yan W, *et al*. Randomized trial of different initial intravenous immunoglobulin regimens in Kawasaki disease. Pediatric International.2021Jul; 63(7): 757–763. doi: 10.1111/ped.14656.

[17] Ogata S, Ogihara Y, Honda T, *et al*. Corticosteroid pulse combination therapy for refractory Kawasaki disease: a randomized trial.Pediatrics.2012Jan;129(1): e17–e23. doi: 10.1542/peds.2011-0148.

[18] Kobayashi T, Inoue Y, Takeuchi K, *et al*. Prediction of intravenous immunoglobulin unresponsiveness in patients with Kawasaki disease.Circulation.2006 Jun;113(22):2606–2612.doi:10.1161/CIRCULATIONAHA .105.592865.

[19] McCrindle BW, Li JS, Minich LL, *et al*. Coronary artery involvement in children with Kawasaki disease: risk factors from analysis of serial normalized measurements.Circulation.2007Jul;116(2):174–179. doi: 10.1161/CIRCULATIONAHA.107.690875.

[20] Kuo HC, Lo MH, Hsieh KS, *et al*. Single-nucleotide polymorphism rs7251246 in ITPKC is associated with susceptibility and coronary artery lesions in Kawasaki disease.PLOS One.2014Mar;9(4):e91118.doi:10.1371/ journal.pone.0091118.