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RESEARCH ARTICLE

THINK KAWASAKI EARLY: OUR EXPERIENCE FROM A CASE SERIES

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ABSTRACT

Background: Kawasaki disease (KD) is an acute febrile inflammatory vasculitis that mainly affects young children and is now the most common cause of acquired heart disease in the pediatric population globally. It predominantly affects children younger than 5 years of age and carries a significant risk of coronary artery involvement if untreated. Diagnosis remains challenging because of the absence of a specific diagnostic test, variable clinical presentation, and overlap with other inflammatory and infectious illness especially in the Indian context. **Case Series:** We report a series of five pediatric cases of Kawasaki disease, including both complete and incomplete forms in different age groups, youngest being 1 year and oldest of 8 years, presenting with varying clinical manifestations and cardiovascular involvement. Persistent fever, conjunctival congestion, mucosal changes, rash, cervical lymphadenopathy, and extremity changes were the predominant clinical findings. Echocardiography demonstrated coronary artery dilatation and myocardial dysfunction in several cases. Elevated inflammatory markers and NT-pro BNP levels and ECHO findings supported the diagnosis and assessment of disease severity. All patients received timely treatment with intravenous immunoglobulin (IVIG) and aspirin, while selected high-risk patients additionally received corticosteroids. All children showed clinical improvement following treatment, with rapid defervescence and stabilization or resolution of coronary abnormalities on follow-up echocardiography. Some patients exhibited incomplete features of Kawasaki disease making way for diagnostic dilemma and highlighting the need for heightened clinical awareness and suspicion. This case series highlights the diverse clinical spectrum of Kawasaki disease and underscores the importance of early recognition, prompt echocardiographic evaluation, and timely medical intervention in preventing coronary artery complications. It also emphasizes the importance of serial follow-up echocardiography to monitor the evolution or resolution of coronary artery abnormalities and to guide long-term management and prognosis

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INTRODUCTION

Kawasaki Disease (KD) is an acute, systemic vasculitis that primarily affects children under 5 years of age. It is the most common cause of acquired heart disease in children in globally [(1).] First described by Tomisaku Kawasaki in 1967, the disease is most prevalent in East Asian populations [(2,5).] There has been a steady rise in KD incidence in Indian children since the mid 1990s with a male to female predominance of 1.5:1 [[3].] The major concern in Kawasaki disease is coronary artery involvement, including aneurysms, occuring in 20–25% of untreated. [[4,5].] The cause of Kawasaki Disease remains unclear. It results from an abnormal immune response triggered by infectious or environmental factors in genetically predisposed children [[2,5,6]] Theories suggest that immune-mediated vascular injury and excessive cytokine activation involving IL-1, IL-6, TNF-α, and IL-17 contribute to coronary artery inflammation and myocardial involvement [[5].]

The diagnosis of Kawasaki Disease is primarily clinical. Early diagnosis within the first 10 days of fever is essential to reduce the risk of coronary artery complications. Complete Kawasaki disease is diagnosed in the presence of persistent fever with at least four of the five principal clinical features: [(7)]

- Polymorphous erythematous rash
- Bilateral non-purulent conjunctival injection with perilimbal sparing.
- Oral mucosal changes (cracked, dry, red lips; "strawberry tongue")
- Extremity changes (erythema and edema of palms and soles ; periungual desquamation of fingers and toes usually 10–20 days after fever onset)
- Unilateral enlargement of a cervical node ≥1.5 cm diameter

Incomplete Kawasaki Disease should be suspected in children with prolonged unexplained fever and only 2–3 of the principal clinical criteria. 7% of children with KD may present with

cardiovascular collapse (KD shock syndrome) [7], presenting with vasodilatory shock, hypotension, and poor perfusion, with or without myocardial dysfunction [4]. Diagnosis is supported by compatible laboratory markers (elevated inflammatory markers and ALT, thrombocytosis, sterile pyuria) or echocardiographic evidence of coronary involvement [4]. Echocardiography remains the primary imaging modality for assessing coronary artery involvement. However, obtaining an echocardiogram should not delay the initiation of therapy, and normal results do not exclude KD diagnosis.[4] Coronary artery dimensions are assessed using body surface area adjusted Z scores and classified (as per AHA) as : [4]

- No involvement: always $Z < 2$
- Dilation only: 2 to <2.5
- Small aneurysm: ≥2.5 to <5
- Medium aneurysm: ≥5 to <10, and absolute dimension <8 mm
- Large or giant aneurysm: ≥10 or absolute dimension ≥8 mm
- Apart from coronaries, cardiac involvement in KD includes- [4,7]
- Aortic root dilatation
- Presence of AV regurgitation
- Myocardial dysfunction: global and regional wall motion abnormalities (RWMA).
- Pericardial effusion

ECHO in a child with KD: [7]

- At diagnosis.
- Uncomplicated patients**:** 1-2 weeks and 4-6 weeks after treatment.
- For significant coronary abnormalities: At least twice per week till luminal dimensions stabilize, then at 2 weeks, 4-6 weeks, 3 months and every 6 -12 months till parameters normalize.
- For giant aneurysm: twice a week while dimensions are expanding and once weekly in the first 45 days of illness, and then monthly until the third month after illness onset.

Studies indicate that Z score ≥2.5 of right CA or left anterior descending at diagnosis and age <6 months are high-risk features [4]..

The differential diagnosis of Kawasaki Disease includes

- Infections: Bacterial (Streptococcal, Leptospirosis, Rickettsia), Viral (measles, adenovirus, Epstein Barr virus). [7]
- Toxin related: Staphylococcal Scalded Skin Syndrome, toxic epidermal necrolysis. [7]
- Multisystem Inflammatory Syndrome in Children (MIS-C) [4].

Management involves first-line therapy with intravenous immunoglobulin (IVIG) at a dose of 2 g/kg administered as a single infusion over 12-24 hours , given within 10 days of illness. Aspirin is given during the acute period of KD at a dose of 30-50 mg/kg/day and continued until the patient is afebrile for 48 hours [7]. Alternatively, low-dose aspirin (3–5 mg/kg per day) can be started in the acute phase as upcoming studies indicate no significant difference in fever duration, CAA incidence, development of IVIG resistance, and hospital stay between low-dose versus high-dose aspirin [11]. Aspirin therapy is continued for 6 to 8 weeks and stopped if CAAs are not detected in the 6th week echocardiography[6]. High-risk patients may additionally benefit from corticosteroids or biological agents [4].

CASE SERIES

CASE REPORT 1: We report a 3-year-old female child, first born to a non-consanguineous couple, developmentally normal and immunized for age, who presented with 4 days of high-grade intermittent fever associated with maculopapular sand paper like rash over her limbs and trunk with itching over bilateral palms and soles along with bilateral redness of eyes. On examination, the child was febrile and irritable. Bilateral non purulent conjunctivitis, erythematous cracked lips, extremity erythema and edema involving the hands and feet, and a perianal rash was noted. Laboratory investigations revealed:

Parameter	Case 1 (3year/F)
Hb (g/dL)	10.4
TLC (/mm^3) Neutrophil	11,200 83%
Platelet count (1,50,000-4,50,000)	3,47,000 ◊6,25,000 (on day 7 of illness)
Serum sodium (136-145 meq/L)	135
SGOT/SGPT (<36/ <40 U/L)	53/20
ESR (0-15 mm/hr)	48
CRP (< 5 mg/L)	70
LDH (120-246 U/L)	297
Ferritin (10-200 ng/mL)	63
NT Pro BNP (<300 pg/mL)	715
Urine routine Urine culture Blood culture	14- 16 pus cells/hpf Negative - Sterile Pyuria Negative

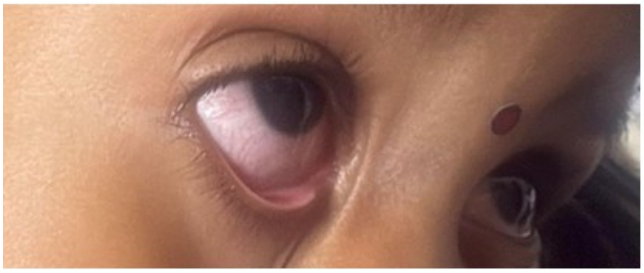

Figure 1. Bilateral non purulent conjuctival congestion

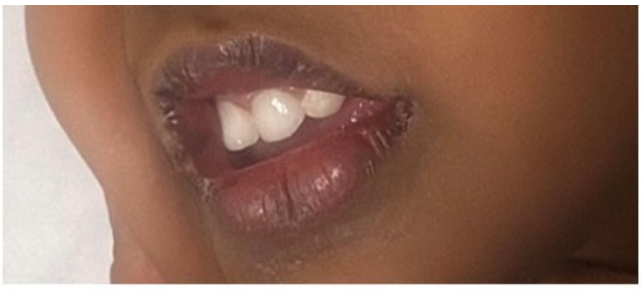

Figure 2. Dry cracked red lips

A diagnosis of Kawasaki disease was made. Echocardiography done on day 5 of fever demonstrated coronary artery dilatation involving the left coronary artery (2.5mm with Z-score of 2 - AHA/Boston model) and right coronary artery (2.25mm with a Z-score of 1.97 - AHA/Boston model) with good biventricular function. The child was treated with IVIG (2 g/kg) (on day 5), low-dose aspirin (5mg/kg/day), and oral corticosteroids (2.5mg/kg/day) as child had coronary

Figure 3. Erythema and edema of palms

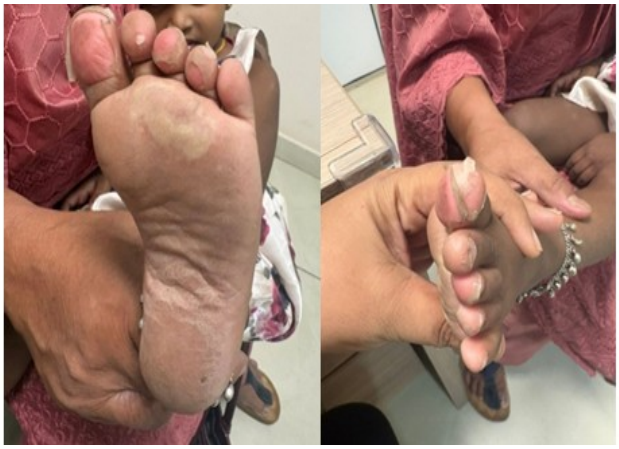

Figure 4,5. Desquamation and skin peeling after 2 weeks of fever onset

artery abnormality and Kobayashi score of 5 indicating high risk for IVIG resistance . Fever subsided within 24 hours of IVIG administration. Follow-up consultation revealed periungual desquamation after 2 weeks. Follow up ECHO at 3 weeks showed coronary artery dimensions :(LMCA: 1.7mm with Z score: -0.67/ RCA: 1.6mm with Z score -0.46; LAD: 1.6mm with Z score +0.11)- normal study with good biventricular function Hence advised to stop aspirin and review after 3 months for follow up.

CASE REPORT 2

An 8-year-old male child, presented with 4 days of high-grade intermittent fever associated with bilateral conjunctival redness , unilateral neck swelling and generalized erythematous rash over his limbs, trunk and neck. Clinical examination revealed bilateral non-purulent conjunctivitis, erythematous dry lips with cheilitis, and polymorphous erythematous rash – targetoid rash resembling erythema multiforme, on neck, trunk, limbs and genitals and redness of palms and soles.

Lab investigations showed:

Parameter	Case2 (8year/M)
Hb (g/dL) (11.5 to 15.5 gm/dl)	12
TLC (/mm^3) (4,500 to 14,500) Neutrophil	8,400 89%
Platelet count (1,50,000- 4,50,000)	2,72,000
Serum sodium (136-145 meq/L)	127(hyponatremia)
Serum albumin (3.5-5.0 gm/dl)	2.9(hypoalbuminemia)
SGOT/SGPT (<59/ <52 U/L)	25/17
ESR (0-15 mm/hr)	36
CRP (< 5 mg/L)	85
LDH (120-246 U/L)	254
Ferritin (17.4 - 464 ng/mL)	265
Procal (<1 ng/mL)	5.24
D dimer (<500 ng/ml	2217
NT Pro BNP (<300 pg/mL)	10,500
Trop I (<0.034 ng/ml)expected pediatric upper limit for his age is 0.0055ng/ml)[10]	0.066
Blood and urine culture	Negative
Scrub typhus/ infectious mononucleosis/ dengue fever / mycoplasma	Negative

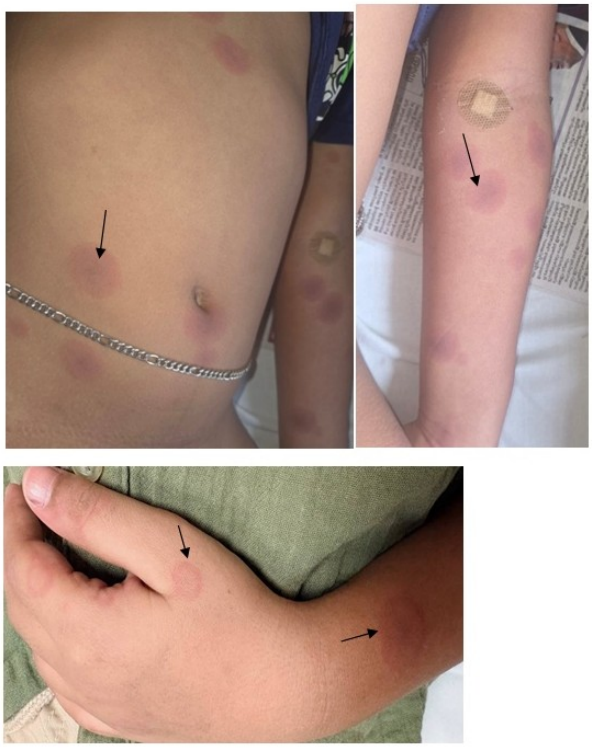

Figure 6,7,8. Targetoid rash resembling erythema multiforme

A diagnosis of Kawasaki disease was made and echocardiography was taken which revealed right coronary artery dilatation of 3.7 mm (Z score: 2.84 -AHA/ Boston model) and left coronary artery diameter of 2.5 mm(Z score of 1.93- AHA/Boston model)along with mild global hypokinesia and left ventricular dysfunction. The patient received IVIG (2g/kg over 16 hours), low-dose aspirin (3mg/kg/day), and oral corticosteroids (2mg/kg/day)- Kobayashi score of 7 indicating high risk of IVIG resistance. Steroids were started as the inflammatory markers were high and early cardiac involvement [8]. The child became defervescent following IVIG therapy and remained afebrile thereafter. Child was advised to continue aspirin (75 mg/day) for 3 more months and review after 3 months for evaluation of coronaries.

CASE REPORT 3: An 8-year-10-month-old female child presented with fever for 4 days , redness of eyes for 3 days,

reddish lips and tongue and cervical swelling. She had initially received oral antibiotics elsewhere with no improvement. Examination revealed bilateral non-purulent conjunctivitis with limbal sparing, strawberry tongue, red lips, cervical lymphadenopathy.

Laboratory investigations demonstrated

Parameter	Case3 (8year/F)
Hb (g/dL) (11.5 to 15.5 gm/dl)	11.8
TLC (/mm^3) (4,500 to 14,500) Neutrophil	15,500 64%
Platelet count (1,50,000- 4,50,000)	4,47,000-->5,81,000 (on day 14 of illness)
Serum sodium (136-145 meq/L)	140
Serum albumin (3.5- 5.0 gm/dl)	4.3
SGOT/SGPT (<36/ <40 U/L)	15/18
ESR (0- 15 mm/hr)	51
CRP (<5 mg/L)	47
LDH (120-246 U/L)	216
Ferritin (10- 200 ng/mL)	80.9
NT Pro BNP (<300 pg/mL)	91.3
Urine routine	2-3 pus cells/hpf
Blood and urine culture	Negative
Monospot test	Negative

Echocardiography showed dilatation of the left main coronary artery(3.4 - 3.7mm) with a Z-score ranging from 2.37 to 3.11 - AHA/Boston model and normal right coronary artery (2.5 mm) with a Z score of 1.32. A diagnosis of incomplete Kawasaki disease disease was established. The child was treated with continuos IVIG (2g/kg) infusion and low-dose aspirin (3mg/kg/day). Her fever subsided post IVIG infusion. 2D echo done after 4 days showed normal study with good biventricular function. Follow up ECHO after 3 months revealed normal study with normal coronaries.

CASE REPORT 4: A 1-year-3 month old female child presented with high-grade fever for 2 days, bilateral conjunctival congestion, diffuse polymorphous rash, and cervical lymphadenopathy. Laboratory investigations demonstrated:

Parameter	Case4 (1year/F)
Hb (g/dL) (11 to 14 gm/dl)	10.8
TLC (/mm^3) (5,000 to 17,000) Neutrophil	22,500 61%
Platelet count (1,50,000- 4,50,500)	4,32,000
Serum sodium (136-145 meq/L)	136
Serum albumin (3.5-5.0 gm/dl)	4.5 g/L
SGOT/SGPT (<36/<40 U/L)	148/141
ESR(mm/hr)	110
CRP (< 5 mg/L)	55
NT pro BNP (<300 pg/ml)	1270
Urine routine	3-4 pus cells/hpf
Blood and urine culture	Negative
IgM mycoplasma	Negative

Although echocardiography did not demonstrate coronary artery dilatation (prominent RCA 2.2mm with Z score 1.8 and LCA 1.8mm with Z score 0.9) , the child fulfilled the clinical criteria for Kawasaki disease. She received prophylactic IVIG (2g/kg) and low dose aspirin therapy (4mg/kg/day) and showed excellent clinical response and was discharged on maintenance low-dose aspirin (4mg/kg/day for 30 days) with advice to follow up after 1 month. Follow up echo at 1 month showed normal study and at 8 months showed normal ventricular function with prominent RCA (LMCA (2.1 mm with Z score 0.5), LAD (1.8mm with Z score 0.9), proximal RCA (2.4mm with Z score 1.9) and distal RCA (1.5mm with Z score 0.5)).

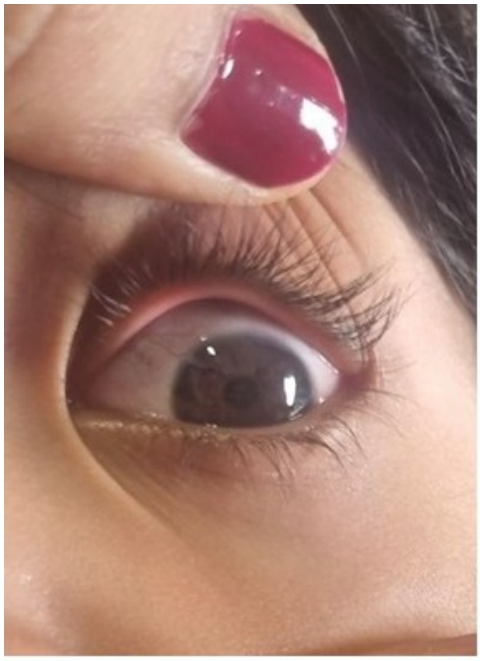

Figure 9. Conjunctival congestion with perilimbal sparing

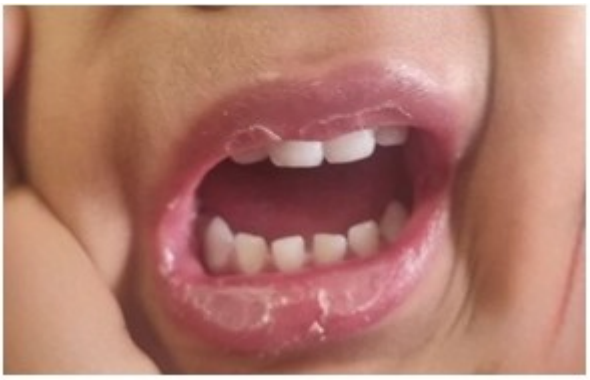

Figure 10. Red dry cracked lips

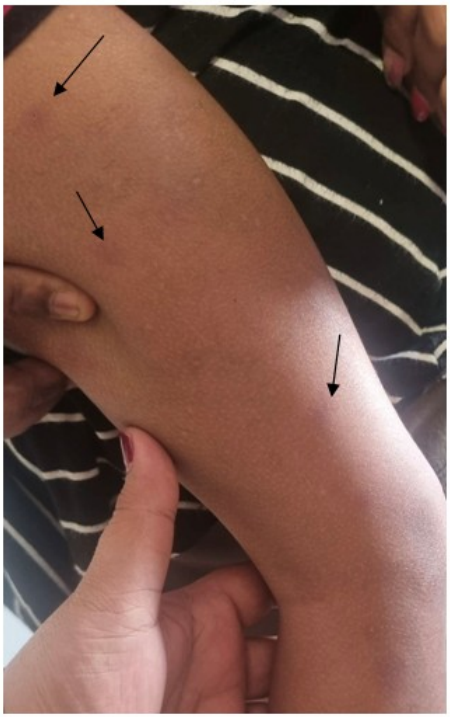

Figure 11. Polymorphous rash in KD

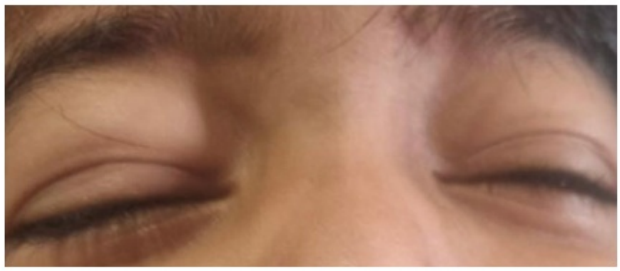

Figure 12. Peri orbital edema

CASE REPORT 5

A 3-year 11 month old female child presented with high-grade fever for 2 days, throat pain, poor oral intake. By day 4 of fever she developed macular rashes over lower limbs, conjunctival injection, red cracked lips and strawberry tongue . Laboratory investigations:

Parameter	Case 5 3year/F)
Hb (g/dL) (11.5 to 15.5 gm/dl)	13.1
TLC (/mm^3) (4,500 to 14,500) Neutrophil	22,400 89%
Platelet count (1,50,000- 4,50,500)	1,92,000
Serum sodium (136-145 meq/L)	133(hyponatremia)
Serum albumin (3.5-5.0 gm/dl)	3.3(hypoalbuminemia)
SGOT/SGPT (<36/<40 U/L)	46/21
ESR (0-15 mm/hr)	46
CRP (<5 mg/L)	236
LDH (120-246 U/L)	316
NT Pro BNP (<300 pg/mL)	>30,000
Trop I (<0.034 ng/ml) expected pediatric upper limit for her age is 0.0055ng/ml)[10]	0.056
Urine routine Urine culture	8-10 pus cells/hpf Negative- Sterile pyuria
Blood culture	Negative
Dengue , Influenza , scrub typhus, typhoid	Negative

Echocardiography demonstrated mild global hypokinesia and left ventricular dysfunction, mild MR, mild TR. Coronaries were normal. The child received IVIG (2g/kg) , low dose aspirin (4mg/kg/dose) and supportive care . After 36 hours post IVIG transfusion , child was afebrile , repeat Echo after 48 hours of IVIG infusion showed normal cardiac function . Coronaries were normal . Low dose aspirin was continued for 6 weeks . Repeat echo at 3 months showed normal study .

DISCUSSION

This case series highlights the clinical spectrum and cardiovascular involvement of Kawasaki disease in children presenting with persistent fever and mucocutaneous manifestations. The series included children ranging from 1 year 3 months to 8 years 10 months, demonstrating that although Kawasaki disease predominantly affects children younger than 5 years of age [1,2], it should also be considered in older children. This issupported by a study conducted by Jindal AK et al where 5.3% of patients with KD in their study were aged 10 years or older[8].

All five children presented with fever and mucocutaneous manifestations including bilateral non-purulent conjunctivitis, oral mucosal changes, extremity involvement, rash, and cervical lymphadenopathy [7]. Rash morphology varied considerably, ranging from maculopapular and sandpaper-like rash to targetoid lesions resembling erythema multiforme. Erythema multiforme has been reported as an uncommon cutaneous manifestation of Kawasaki disease[13].Cutaneous manifestations can be less conspicuous in Indian children with darker skin tone making this diagnostic criteria a challenge to identify. Laboratory investigations like elevated CRP and ESR were consistently observed in all children. Leukocytosis with neutrophilic predominance was common. Other findings included thrombocytosis, hypoalbuminemia, hyponatremia, elevated transaminases and sterile pyuria[4]. Thrombocytosis, though a well-recognized feature of Kawasaki disease (sub acute phase), its significance is questionable in Indian children, among whom anemia is highly prevalent. The coexistence of anemia and reactive thrombocytosis can confound clinical assessment. Cardiac involvement was a major finding in this series. Four of the five children had coronary artery dilatation on echocardiography, while Cases 2 and 5 also demonstrated myocardial dysfunction with elevated NT-pro BNP levels and ventricular hypokinesia. These findings emphasize the importance of early echocardiographic evaluation in all suspected cases. Serial follow-up echocardiography showed improvement of coronary abnormalities in most children following treatment, highlighting the benefit of early diagnosis and intervention. We excluded various infectious etiologies before establishing the diagnosis reflecting the diagnostic challenge in regions where tropical and infectious illnesses are common differentials [7] . In addition, parental reluctance to accept the diagnosis and understand the potential severity of the disease posed a significant obstacle. As many children appeared relatively well despite the risk of serious cardiovascular complications, some caregivers could not comprehend the need for extensive investigations, echocardiographic monitoring, and timely administration of intravenous immunoglobulin. All patients received timely administration of IVIG and low dose aspirin therapy[11], resulting in rapid clinical improvement and defervescence. Adjunctive corticosteroids were used in selected high-risk patients with coronary involvement and severe inflammatory response [9,12]. Follow-up echocardiography demonstrated resolution or stabilization of coronary abnormalities , highlighting the importance of early diagnosis and prompt initiation of therapy to prevent long-term cardiovascular sequelae .

CONCLUSION

This case series highlights the clinical spectrum and degrees of cardiovascular involvement in pediatric patients diagnosed with acute Kawasaki disease. Both complete and incomplete forms were encountered, with several children demonstrating early coronary artery abnormalities and myocardial dysfunction. Persistent fever associated with mucocutaneous manifestations and elevated inflammatory markers should prompt consideration of Kawasaki disease, even when all classical diagnostic criteria are not fulfilled. Early echocardiographic evaluation remains essential for identifying coronary artery involvement and guiding management.

Early recognition, prompt treatment, and careful cardiac follow-up are crucial in reducing morbidity and preventing long-term cardiovascular complications associated with Kawasaki disease.

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