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CLINICAL PROFILE, TREATMENT RESPONSE, AND SHORT-TERM OUTCOMES IN CHILDREN WITH KAWASAKI DISEASE: A PROSPECTIVE OBSERVATIONAL STUDY AT A TERTIARY CARE CENTRE

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ABSTRACT

Background: Kawasaki Disease (KD) is an acute vasculitis primarily affecting children under five, making it the most common cause of acquired heart disease in this age group. Though its precise cause remains unclear, genetic predisposition and abnormal immune responses to infections or environmental factors are implicated. Early diagnosis and treatment are crucial to prevent coronary artery abnormalities (CAA), the most severe complication. Incomplete or atypical cases, particularly in infants, often result in delayed recognition and adverse outcomes. **Aim and Objective:** To evaluate the clinical profile, treatment response, and short-term follow-up outcomes of children diagnosed with Kawasaki Disease in a tertiary care setting, and to compare clinical and laboratory features between complete and incomplete KD presentations. **Materials and Methods:** This prospective observational study was conducted in the Department of Pediatrics at Jorhat Medical College and Hospital from September 2023 to August 2024. Thirty children diagnosed using AHA 2017 criteria were enrolled. Data were collected on clinical, laboratory, echocardiographic features, and follow-up outcomes. Statistical analyses were performed to identify correlations and risk factors for CAA. **Result:** Complete KD was observed in 80% and incomplete KD in 20% of cases. All patients had fever ≥5 days. Inflammatory markers (WCC, CRP, ESR, platelet count) were significantly higher in incomplete KD. CAA developed in 16% of patients, predominantly among those with incomplete KD. Male sex, prolonged fever, and elevated inflammatory markers were significantly associated with CAA risk. **Conclusion:** Incomplete KD poses a higher risk for coronary complications due to diagnostic challenges and heightened inflammatory response. Early recognition, aggressive management, and structured follow-up are vital to improve outcomes.

INTRODUCTION

Kawasaki disease is a rare but serious illness that primarily affects young children, especially those under five years of age. It is recognized as an acute febrile vasculitis, meaning it causes inflammation in the blood vessels throughout the body. The disease was first described by Dr. Tomisaku Kawasaki in Japan in the late 1967s. Since then, it has gained significant clinical attention due to its potential to cause severe cardiac complications, particularly in the coronary arteries. Kawasaki disease is now acknowledged as the leading cause of acquired heart disease in children in many countries, including both developed and developing regions. Despite extensive global reporting, the exact cause of Kawasaki disease remains unclear. It is hypothesized to result from a combination of genetic susceptibility, environmental factors, and an abnormal immune response, possibly triggered by infectious agents. The highest incidence of this illness has been reported in East Asian countries like Japan, South Korea, and Taiwan, hinting at a strong genetic component. However, cases are reported worldwide, and there has been a steady increase in incidence even in countries with historically low prevalence.[1-3]

The clinical presentation of Kawasaki disease is typically characterized by a prolonged high fever lasting at least five days, along with additional signs such as redness of the eyes without pus, changes in the lips and mouth like cracking or redness, a widespread skin rash, swelling or redness in the hands and feet, and enlarged lymph nodes in the neck. However, not all patients exhibit the full

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spectrum of symptoms, and many children, especially infants under six months or older children, may present with incomplete or atypical forms of the disease. This variability in clinical signs often leads to diagnostic uncertainty and delayed treatment, particularly in regions with limited awareness or resources. Laboratory investigations and imaging, especially echocardiography, are critical in supporting the diagnosis when typical clinical features are absent. The systemic nature of Kawasaki disease means that it can affect several organs, leading to a broad range of non-specific symptoms such as irritability, diarrhea, vomiting, joint pain, or even signs of meningeal irritation, further complicating the diagnosis.[4]

If not diagnosed and treated promptly, Kawasaki disease can lead to serious cardiovascular complications, the most significant being the development of coronary artery aneurysms. These aneurysms pose a long-term risk of myocardial infarction and sudden cardiac events. However, with early and appropriate treatment, the risk of such complications can be dramatically reduced. The cornerstone of treatment is the administration of intravenous immunoglobulin (IVIG), typically given in a single high dose of 2 g/kg within the first ten days of illness. This therapy is known to significantly lower the risk of coronary artery involvement. Aspirin is also administered during the acute phase for its anti-inflammatory effect and is later continued at a lower dose to prevent clot formation. Although most patients respond well to IVIG, approximately 10–20% exhibit resistance, showing persistent or recurrent fever after treatment. These patients are at increased risk for cardiac complications and may require additional immunosuppressive therapies such as corticosteroids, tumor necrosis factor-alpha inhibitors like infliximab, or other targeted agents.[5,6]

Regular follow-up is essential to monitor the resolution of inflammation and detect any developing coronary complications. Echocardiography is the preferred imaging modality for assessing coronary artery status and is typically performed at diagnosis, two weeks, six weeks, and sometimes later depending on findings. Children who do not show any coronary abnormalities on serial echocardiography generally have a favorable prognosis. However, those with persistent or evolving changes require long-term cardiology follow-up and may need chronic antiplatelet or anticoagulant therapy. In addition to imaging, laboratory markers of inflammation and cardiac function can be helpful in guiding treatment decisions and monitoring disease progression or resolution. Long-term management may also include periodic cardiovascular assessments, especially in patients who developed aneurysms or other cardiac lesions during the acute phase.[7]

In regions like ours, where awareness about Kawasaki disease is still evolving and epidemiological data are limited, the condition is often misdiagnosed or diagnosed late. Many children initially present with symptoms that resemble other common febrile illnesses, such as viral infections or scarlet fever, leading to a delay in appropriate management. The variability in clinical presentation, influenced by geographic, racial, and familial factors, adds to the diagnostic challenge. As a result, some cases remain undetected until complications arise. Studies show that without treatment, around 25% of affected children may develop coronary artery aneurysms. However, with timely administration of IVIG and supportive care, this risk can be reduced to less than 5%. This dramatic difference underscores the importance of clinical vigilance and early recognition.[8]

Given the critical importance of early diagnosis and treatment, there is a growing need for increased awareness among healthcare providers, especially pediatricians and general practitioners, to consider Kawasaki disease in the differential diagnosis of prolonged fever in children. Moreover, since there is no specific diagnostic test for the condition, reliance on clinical judgment supported by laboratory and imaging findings remains essential. Research continues to explore the underlying immune and genetic mechanisms involved in Kawasaki disease, which may eventually lead to more specific diagnostic tools and targeted treatments. Efforts are also underway to identify biomarkers that could predict IVIG resistance or long-term complications, potentially improving outcomes through personalized treatment strategies.[9]

The study aims to evaluate the clinical profile, treatment, and short-term follow-up of patients diagnosed with Kawasaki disease admitted to a tertiary care center. It seeks to describe various clinical manifestations, laboratory findings, treatment modalities, and follow-up outcomes. Additionally, the study intends to compare the clinical and laboratory characteristics between cases of Complete and Incomplete Kawasaki Disease, thereby identifying differences that may aid in early diagnosis and management. This analysis is expected to enhance understanding of disease patterns and guide timely therapeutic interventions in affected pediatric patients.

MATERIALS AND METHODS

This prospective observational study was conducted in the Department of Pediatrics, Jorhat Medical College and Hospital from September 2023 to August 2024. Thirty children diagnosed with Kawasaki Disease (KD) based on AHA 2017 criteria were enrolled using simple random sampling. Inclusion criteria comprised all KD patients treated at JMCH and on regular follow-up, while those treated elsewhere or unwilling to

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participate were excluded. Data on clinical, laboratory, treatment, and echocardiographic findings were collected using a pretested proforma. Ethical clearance was obtained from the Institutional Ethics Committee of Jorhat Medical College prior to study initiation.

RESULTS

The Kawasaki Disease (KD) predominantly affects males (63.33%) and children aged 1–5 years (80%). No statistically significant difference was found in sex distribution between complete and incomplete KD ($p = 1.0$). However, age distribution showed a significant difference ($p = 0.0072$), suggesting age may influence KD presentation. Notably, no cases were seen in children >5 years.

Table 1: Clinical characteristics of patients with Kawasaki disease at the time of diagnosis

Characteristics	Complete KD	Incomplete KD	Total	Total Percent (%)
Fever Lasting for at least 5 days	24	6	30	100
Changes in extremities (erythema of palms and soles, extremity oedema and periungual peeling)	19	2	21	70
Oral mucosal changes	19	5	24	80
Polymorphous rash	17	4	21	70
Conjunctivitis	15	2	17	56.7
Cervical lymphadenopathy	12	0	12	40.0

The table reveals that all patients with Kawasaki Disease (KD) presented with fever ≥5 days (100%), a hallmark diagnostic feature. Oral mucosal changes (80%) and extremity changes (70%) were common, especially in complete KD. Conjunctivitis (56.7%) and cervical lymphadenopathy (40%) were less frequent, more often absent in incomplete KD. This highlights the diagnostic variability, particularly in incomplete cases.

Table 2: Laboratory parameters of the patients in our cohort

Parameters	Complete KD (n=24)	Incomplete KD (n=6)	Total (n=30)	t-test	p-value
Haemoglobin, g/dL	10.00±1.68 (7.3-13.2)	9.55±1.09 (7.6-11.3)	10.01± 1.53 (7.3-13.2)	0.7601	0.46
WCC, ×109/L	16.48±3.70 (10.4-23.3)	22.03±1.8 (19.5-24.3)	16.81± 4.0 (9.9-23.3)	-4.970	0.00015
Platelet count, ×109/L	368.62±98.34 (80-501)	671±122.43 (545-885)	401.7± 150.4 (80-856)	-5.171	0.0016
ESR, Westergren, mm/h	60.2±20.23 (30-105)	85±18.25 (60-105)	67.33± 22.61 (30-120)	-2.6975	0.027
CRP, mg/L	65.975±27.7 (19.5-129.6)	127.78±33.98 (83.89-180.8)	87.3± 39.02 (32.1-195)	-3.8016	0.0076
Urine R/E (>10 pus Cells per HPF)	1 (3.33)	3 (10)	4 (13.33)	0.00	1

The table shows that incomplete Kawasaki Disease (KD) cases had significantly higher WCC, platelet counts, ESR, and CRP levels compared to complete KD ($p < 0.05$), indicating a more intense inflammatory response. Hemoglobin levels and abnormal urine findings were not significantly different between groups ($p > 0.05$). The most notable differences were in platelet count and CRP, suggesting their utility in distinguishing KD variants. These lab parameters reinforce systemic inflammation as a hallmark of KD, especially in incomplete forms.

Table 3: Non-cardinal symptoms in the patients

SYMPTOMS	n	(n%)
Gastrointestinal	0	0.00
Abdominal pain	1	3.33
vomiting	2	6.67
diarrhoea	2	6.67
Gall bladder hydrops	1	3.33
hepatomegaly	1	3.33
genitourinary	0	0.00
Sterile pyuria	4	13.33
Central nervous	0	0.00
Perineal desquamation	1	3.33
BCG Reactivation	1	3.33
Total	13	43.33

The table indicates that 43.33% of patients with Kawasaki Disease exhibited non-cardinal symptoms, with sterile pyuria (13.33%) being the most common. Gastrointestinal and genitourinary symptoms were absent, while vomiting and diarrhea were each seen in 6.67% of cases. Other symptoms like BCG reactivation, hepatomegaly, and perineal

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desquamation occurred in 3.33% of patients,

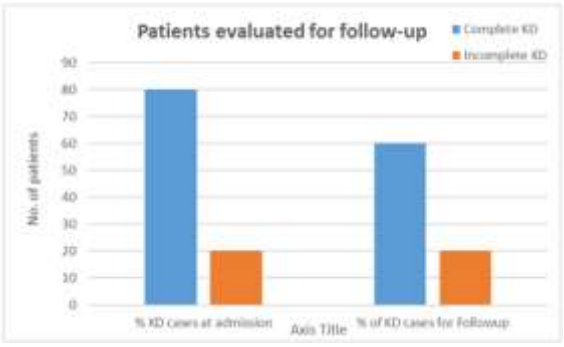

Figure 1: Patients evaluated for Follow up

The chart shows that a higher proportion of complete Kawasaki Disease (KD) cases were both admitted and followed up compared to incomplete KD cases. Follow-up rates dropped for both groups, but more markedly for complete KD. This suggests better continuity of care in complete KD, potentially due to clearer clinical presentation. [Figure 1]

Patients with complete Kawasaki Disease (KD) had a significantly longer mean fever duration (6 days) compared to incomplete KD (1.5 days), with the t-test p-value (0.0488) indicating statistical significance. However, the chi-square test did not show a significant association (p = 0.2766). This reflecting variable systemic involvement. suggests fever duration is more distinctly different by mean comparison than by categorical distribution. [Table 4]

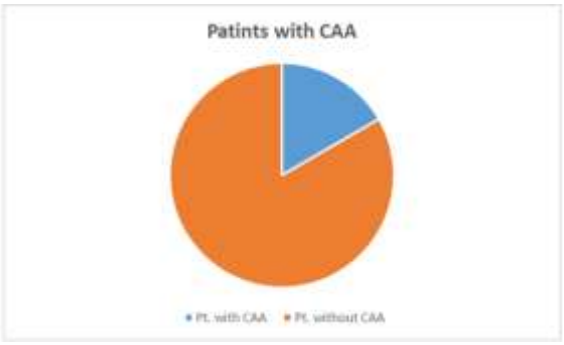

Figure 2: Patients with CAA

The pie chart illustrates the distribution of patients with and without coronary artery abnormalities (CAA) among Kawasaki Disease cases. A majority of patients did not develop CAA, while a smaller proportion—depicted in blue—did. This highlights that although CAA is a known complication, it affects only a subset of KD patients. [Figure 2]

Table 4: Total duration of fever among the patients with Kawasaki disease

Fever	Complete KD	Incomplete KD
<10 days	9	0
>10 days	4	1
<15 days	8	3
>15 days	3	2
Total	24	6
Total %	80	20
Mean	6	1.5
Min	3	0
Max	9	3
Chi-square	3.864	
p-value	0.2766	
t-test	2.800	
p-value	0.0488	

Table 5: Multivariate analysis for risk factors associated with CAA in patients with KD

Variables	With CAA	Without CAA	χ^2	p-value	z- value	p-value
N (%)						
Male n%	3 (10)	16 (53.33)	8.7328	0.0031	-3.1862	0.0014
Female	2 (6.67)	9 (30)	3.8247	0.0505	-2.2585	0.0239
Age n%						
<1 year	3 (10)	3 (10)	0	1	-0.1021	0.9187
1-5 year	2 (6.67)	22 (73.33)	16.9576	0	-4.3239	0
Urine R/E (>10 pus Cells per HPF)	1 (3.33)	3 (10)	0.3415	0.5589	-1.0855	0.2777
Changes in extremities (erythema of palms and soles, extremity edema and periungual peeling)	2 (6.67)	19 (63.33)	13.7695	0.0002	-3.9306	0.0001
Oral mucosal changes	4 (13.33)	20 (66.67)	10.8523	0.001	-3.5002	0.0005
Polymorphous rash	3 (10)	18 (60)	10.6987	0.0011	-3.4908	0.0005
Conjunctivitis	2 (6.67)	14 (46.67)	8.6095	0.0033	-3.1857	0.0014
Cervical lymphadenopathy	1 (3.33)	11 (36.67)	7.5916	0.0059	-3.0453	0.0023
Non-cardinal symptoms	2 (6.66)	10 (10)	3.8692	0.0515	-2.2581	0.0588

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Fever n%						
<10 days	1 (3.33)	8 (26.67)	4.549	0.0329	-2.4674	0.0136
>10 days	4 (13.33)	17 (56.67)	8.0148	0.0046	-3.051	0.0023
Haemoglobin, g/dL	9.14±1.3(9.4)	10.072±1.6(10.1)	0.0282	0.8667	-0.3977	0.6909
WCC, ×109/L	20.88±2.0(19.8)	16.936±4.06(17.4)	0.0511	0.8211	0.3909	0.6959
Platelet count, ×109/L	513.4±256.5(545)	412.24±124.81(385)	10.416	0.0012	3.2797	0.001
ESR, Westergren, mm/h	98±6.0(95)	58.6±18.03(60)	7.2466	0.0071	2.7764	0.0055
CRP, mg/L	126.72±20.4(121.4)	68.6596±33.20(67.4)	14.0758	0.0002	3.8284	0.0001

Patients with incomplete clinical presentations and elevated inflammatory markers are at a higher risk for developing coronary artery abnormalities (CAA). Statistically significant risk factors include male sex, increased platelet count, elevated CRP and ESR levels, and the absence of cardinal Kawasaki Disease features. These findings emphasize the need for early diagnosis and prompt, aggressive management, particularly in atypical or incomplete KD cases.

Table 6: Risks of CAL among subjects with Kawasaki disease

	Univariate Analysis			Multivariate Analysis		
Variable	**OR**	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**
< 1 year of age at admission	2.8	0.66 - 4.95	0.0104	1.87	-0.56 - 4.29	0.1312
Males	19.23	-19797.52 - 19835.98	0.9985	19.11	-25746.62 - 25784.83	0.9988
> 10 days Fever	0.98	-0.94 - 2.89	0.3176	-0.65	-3.14 - 1.84	0.6113
White blood cell count>15 000/mm^3	34.26	-29050294.24 - 29050362.77	1	35.54	-82166701.6 - 82166772.67	1
Platelet count >450000/mm^3	2.34	0.03 - 4.65	0.0469	1.43	-1.27 - 4.13	0.2982
ESR ≥40mm1st h	24.53	-322831.64 - 322880.71	0.9999	-11.76	-82169681.48 - 82169657.97	1
CRP ≥30mg/l	20.73	-44859.62 - 44901.08	0.9993	12.22	-1861.28 - 1885.71	0.9898

The table shows the results of both univariate and multivariate analyses evaluating potential risk factors for coronary artery abnormalities (CAA) in Kawasaki Disease. In univariate analysis, age <1 year and platelet count >450,000/mm³ were significantly associated with CAA (p = 0.0104 and 0.0469, respectively). However, in multivariate analysis, none of the variables, including age, sex, fever duration, WBC count, platelet count, ESR, or CRP, remained statistically significant, indicating that these factors may not independently predict CAA when adjusted for others.

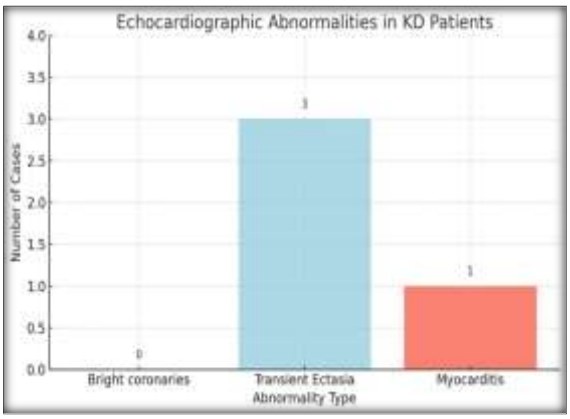

Figure 3: Echocardiographic Abnormalities in KD Patients

The chart shows that transient ectasia was the most common echocardiographic abnormality observed in Kawasaki Disease patients, occurring in 3 cases. Myocarditis was seen in 1 case, while no cases of bright coronaries were detected. This indicates that coronary involvement is variable, with transient ectasia being more prevalent.

DISCUSSION

Kawasaki disease (KD) is an acute vasculitis that primarily affects medium-sized arteries, especially the coronaries, and is the leading cause of acquired heart disease in children globally. First described by Dr. Tomisaku Kawasaki in 1967, the disease is thought to result from an aberrant immune response to an unknown infectious or environmental trigger in genetically predisposed individuals.[10] The highest incidence is reported from East Asian countries—Japan (309/100,000), South Korea (194.7/100,000), and Taiwan (82.8/100,000)—suggesting genetic and environmental influences. In India, epidemiological data are limited, but studies such as one from Chandigarh reported an incidence of 5.35/100,000 among children under five, highlighting underdiagnosis and lack of awareness.[11]

In our study, all patients presented with fever lasting at least five days, establishing it as a universal symptom. Common accompanying features were oral mucosal changes and extremity changes (both around 70–80%), while conjunctivitis and cervical lymphadenopathy were seen in 56.7% and 40%, respectively. These findings are consistent with Bressieux-Degueldre et al. (2023), who reported less frequent classical features in incomplete KD (iKD) compared to complete KD (cKD), especially in

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younger children. Similarly, Perrin et al. (2009) and Yu (2012) noted lower frequencies of cardinal features in iKD, with risks for coronary artery abnormalities (CAAs) persisting regardless of clinical completeness.[12-14]

Patients with incomplete KD in our study exhibited significantly elevated inflammatory markers—WCC, ESR, CRP, and platelet counts—compared to those with complete KD. This aligns with Ha et al. (2018), who reported heightened inflammatory response in iKD and associated it with greater coronary dilation risk. However, Perrin et al. (2009) found no significant differences in inflammatory markers between KD types. The younger age of iKD patients was significant ($p = 0.0072$), supporting studies by Peng et al. (2021) and de La Harpe et al. (2019), who found iKD more prevalent in children <1 year and linked it with diagnostic delays.[13,15-17]

Non-cardinal symptoms were observed in 43.3% of patients, including sterile pyuria (13.3%), vomiting, and diarrhea (6.67% each), consistent with Shenoy et al. (2020) who emphasized that such symptoms can complicate diagnosis and delay treatment. Of the 30 patients, 24 returned for follow-up; coronary artery abnormalities were observed in 5 patients (16%), more common in iKD (50%) than in cKD (8.3%). This difference was statistically significant ($p = 0.0143$) and aligns with findings by Sonobe et al. (2007) and Bressieux-Degueldre et al. (2023).[12,18,19]

Hematologic abnormalities such as leukocytosis and thrombocytosis peaked in the subacute phase, with normalization by 4–8 weeks. IVIG was effective in most patients; IFX was used successfully in IVIG-resistant cases, consistent with Kuo (2023) and Morana et al. (2024). Univariate analysis showed age <1 year and high platelet count as significant predictors of CAA, though multivariate analysis was not significant—likely due to small sample size. Similar findings were reported by Niu et al. (2024) and Chen et al. (2024), reinforcing the role of inflammation and age in coronary risk.[20-23]

CONCLUSION

This study highlights the diverse clinical presentations and laboratory profiles of Kawasaki Disease in children, emphasizing the importance of early diagnosis and prompt treatment to prevent serious cardiovascular complications such as coronary artery aneurysms. Incomplete KD cases showed higher inflammatory markers and a greater risk of coronary involvement, underlining the need for heightened clinical vigilance. The majority of patients responded well to intravenous immunoglobulin therapy, with only a few requiring additional treatments. Regular follow-up with echocardiography proved essential in monitoring cardiac outcomes. Overall, timely intervention and systematic follow-up significantly improve prognosis and reduce long-term cardiovascular risks in affected children.

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