ORAL PRESENTATIONS

Title: Kawasaki Disease — Beyond Coronaries: Clinical and Echocardiographic Spectrum

Authors: Dr Padma Priya K H, Kanchi kamakoti CHILDS trust hospital, Chennai
Dr Janani Sankar, Kanchi kamakoti CHILDS trust hospital, Chennai

Presenting Author: Dr **Kawasaki Disease — Beyond Coronaries: Clinical and Echocardiographic Spectrum**, khpadmapriya@gmail.com, 9677097184

Background: Kawasaki disease (KD) is an acute systemic vasculitis of childhood and the leading cause of acquired heart disease in children worldwide. While coronary artery aneurysms (CAA) are its hallmark, recent evidence and AHA 2024 guidelines recognize KD as a *pancarditis* involving myocardial and pericardial inflammation. Identification of high-risk and refractory phenotypes is crucial for optimizing early therapy and preventing long-term cardiac sequelae.

Methods: Retrospective analysis was conducted on **94 children** diagnosed with KD between **January 2022 and December 2024**. Patients were classified as **complete KD (n=34)**, **incomplete KD (n=49)**, **refractory KD (n=8)**, and **KD/MIS-C(n=3)**. Clinical features, laboratory parameters and echocardiographic findings were compared across subgroups using chi-square and non-parametric tests.

Results: Median age was 1.7 years, with a male predominance (58%). **Rash** and **extremity oedema** were markedly higher in **complete KD** (67.6% and 50%) versus **incomplete KD** (42.9% and 18.4%, $p < 0.05$) **Irritability** was observed in **52.9% of complete KD** and **50% of refractory KD**, compared with **14.3% of incomplete KD** ($p < 0.001$), indicating a strong association with the more inflammatory KD phenotypes. **Gastrointestinal (GI) symptoms** were present in **50% of refractory KD**, compared with **8.8% of complete KD** and **2.0% of incomplete KD** ($p = 0.0005$), highlighting a significant association with treatment resistance. Overall, refractory KD had the **highest CRP (128 mg/L)**, **lowest albumin (2.75 g/dL)**, and **lowest Hb (7.8 g/dL)** — a pattern indicating severe systemic inflammation and vascular leak. Echocardiographic abnormalities were noted in **34**% of cases, significantly higher in complete KD (58.8%, $p = 0.002$), with predominant coronary involvement of the left anterior descending and left circumflex arteries (20.6% each). Beyond CAA, **left ventricular dysfunction (8.5%)**

and **pericardial effusion (21.3%)** were also identified, underscoring broader myocardial involvement. Among patients with initial abnormalities, most **normalized on follow-up ECHO** (86.2% normal at follow-up). **Ferritin**, available in eight cases, was markedly elevated in refractory KD (mean ≈ 914 ng/mL) versus other subtypes (≈ 150–190 ng/mL), indicating heightened inflammation. A clear **seasonal clustering (August–December)** was observed, consistent with regional monsoon-related trends. IVIG was administered to 90% of patients; corticosteroids were used in 23%, and infliximab in 4%, predominantly in refractory KD.

Conclusion: Our cohort highlights that **KD extends beyond coronary involvement**, encompassing **LV dysfunction and pericardial inflammation** as clinically relevant cardiac sequelae. **Elevated CRP, low albumin, and high ferritin** characterize the hyperinflammatory refractory phenotype, reinforcing their value as **risk stratification markers**. Recognition of atypical symptoms—**irritability, gastrointestinal manifestations, and early echocardiographic changes**—should prompt timely escalation to adjunctive therapy. Seasonal clustering supports a possible **infectious or environmental trigger**. These findings align with current AHA 2024 recommendations and strengthen the evolving understanding of KD as a **systemic inflammatory vasculopathy rather than an isolated coronary disease**.

Parameter	Complete KD (n=34)	Incomplete KD (n=49)	Refractory KD (n=8)	p-value	Interpretation
Median Age (years)	1.15	1.70	0.95	0.125	Younger children more prone to refractory KD
Male (%)	58.8	55.1	75.0	0.75	Male predominance across all subgroups
Rash (%)	67.6	42.9	75.0	0.031	Rash significantly higher in complete KD
Extremity Edema (%)	50.0	18.4	50.0	0.017	Extremity changes less common in incomplete KD
Irritability (%)	52.9	14.3	50.0	<0.001	Strong marker of inflammatory and refractory KD

GI Symptoms (%)	8.8	2.0	**50.0**	0.0005	Strong association with hyperinflammatory KD
Mean CRP (mg/L)	117.8	77.7	**128.1**	0.006	CRP significantly higher in refractory KD
Mean ESR (mm/hr)	87.4	73.8	72.0	0.266	ESR elevated but not discriminatory
Mean Albumin (g/dL)	3.19	3.47	**2.75**	0.006	Hypoalbuminemia indicates vascular leak & severity
Mean Hb (g/dL)	9.1	10.8	**7.8**	0.005	Lower Hb supports systemic inflammation
Ferritin (ng/mL)	156	188	**914**	—	Elevated in refractory KD (subset, n=8)
ECHO Abnormalities (%)	**58.8**	20.4	12.5	0.002	Coronary + myocardial involvement higher in complete KD
LAD Involvement (%)	**20.6**	4.1	12.5	0.105	Left-dominant coronary changes
LCx Involvement (%)	**20.6**	0	0	0.004	Most frequently affected coronary vessel
RCA Involvement (%)	14.7	0	0	0.025	RCA less frequent but significant
LV Dysfunction (%)	14.7	2.0	12.5	0.18	Non-coronary myocardial involvement
Pericardial Effusion (%)	17.6	22.4	25.0	0.755	Common in all forms (pancarditis pattern)
IVIG Use (%)	94.1	85.7	100	0.398	Standard first-line therapy
Steroid Use (%)	29.4	4.1	**87.5**	<0.001	Steroid escalation in refractory KD
Infliximab Use (%)	0	0	**50.0**	<0.001	Used exclusively in refractory KD

Clinical Profile and Outcome of 130 patients with Kawasaki Disease from a Tertiary Care Center: A Retrospective Cross-Sectional Study

Uma Tejaswi[1], Jayanth B Nair[1], Bharath A P[2], Chetan Ginigeri[3], Harish Kumar[3], Karthik Arigela[3], Syed MD Naushad Ali[3], Sagar Bhattad[1]

Affiliation:
[1]Division of Pediatric Immunology, Rheumatology and Bone Marrow Transplant, Aster CMI Hospital, Bangalore, India.
[2] Department of Pediatric Cardiology, Aster CMI Hospital, Bangalore, India.
[3] Department of Pediatric Intensive Care Services, Aster CMI Hospital, Bangalore, India.

Background: Kawasaki disease (KD) is a medium vessel vasculitis of childhood with a predilection for coronary arteries, presenting acutely with fever and a typical constellation of clinical findings. This study was conducted to evaluate the clinical and echocardiographic characteristics, treatment received, and outcome of patients diagnosed with Kawasaki disease (KD) during follow up at a tertiary care center in South India.

Methods: Diagnosis of KD was made as per standard AHA criteria. Data of all patients diagnosed with KD between Feb 2017, and September 2025 were collected from inpatient files and outpatient follow-up clinical records.

Results: A total of 130 patients were diagnosed with KD during the study period. The mean age at presentation was 45 months, with 33 patients (25.40%) presenting during infancy. The most common presenting features were fever, oral mucositis, rash, conjunctival injection and extremity changes, followed by cervical lymphadenopathy (Table-1). The mean duration of fever was 9.7 days, with delayed diagnosis (>10 days) in 44 patients (36.10%). Atypical manifestations such as arthritis were noted in two patients, pneumonia in one, Nephrotic Syndrome (congenital NS? Finnish type) in one and hepato-biliary involvement in another. KD shock syndrome was noted in 5.3%(n=7).

Intravenous immunoglobulin (IVIG at 2gm/kg) was administered in all patients and when indicated, IV infliximab (10 mg/kg) and IV Methyl prednisolone (2 mg/kg) were given either as part of primary intensification or as second line therapy. Steroids were tapered over 2-3 weeks. Overall, 28% (n=37) were IVIG-refractory and required additional second line therapy. Three children required cyclosporine as they were refractory to above mentioned therapy. Anakinra was used in three cases {indication – refractory disease, n=2; and KD shock

syndrome, n=1}. Low dose Aspirin was given to all patients, while those with moderate coronary artery aneurysms (CAA) received additional clopidogrel. Patients with giant CAAs were treated with LMWH heparin or direct oral anticoagulants (Apixaban) based on tolerance and compliance. Echocardiography revealed abnormalities in 46.92% (n=61) of the patients, with coronary artery aneurysms being the most common finding. Among the coronary arteries, aneurysms were most frequently associated with the LMCA, followed by the LAD, RCA and LCX in our study. Among the 61 patients with abnormal coronary artery findings, follow-up echocardiography at 6 weeks showed normalization of coronary arteries in 41 patients, while 19 (14.61%) patients continued to have CAAs. At the most recent follow-up, resolution of CAAs was observed in 3 patients within 6 months to 1 year, in another 3 patients between 1.5 and 2 years, and in 2 patients between 4 and 6 years. Nine patients continued to have persistent CAAs, and two were lost to follow-up. A 2-year-old male diagnosed with KD in May 2018 presented with multiple moderate CAAs. During follow-up at 6 months, he developed new-onset severe aortic regurgitation (AR). Over the next four years, the CAAs resolved completely. He subsequently underwent surgical correction for severe AR five years after disease onset and continues to do well on follow up. A 4-month-old male diagnosed with KD in Sept 2022, had bilateral axillary artery aneurysms at presentation along with multiple giant CAA. At latest follow up in June 2025, CAAs along with axillary artery aneurysms are persisting but regressed in size. A 24-year-old man presented with acute MI, and CT Coronary angiography demonstrated giant aneurysmal dilation of LMCA and LAD, along with significant post aneurysmal stenosis and calcification in RCA, consistent with late sequelae of childhood KD at 8 years of age.

Conclusion: This study presents one of the largest cohorts of KD patients with long-term follow-up data from South India. A delay in diagnosis was seen in 36.1%. CAAs were documented in 46.92%(n=61), with giant CAAs in 5.38% (n=7). A few cases were super-refractory warranting multiple immune modulators. On long-term follow-up, most aneurysms showed regression, while 9 patients (6.92%) continued to have CAAs.

Table 1. Characteristics of 130 patients with Kawasaki disease

Total number of KD cases	130
Mean age	45 months

	(2-288)
Gender	
Male (M)	80 (38.4%)
Female (F)	50(61.5%)
M: F	1.6:1
Age -	
< 1 year	33 (25.40%)
1 – 5 years	61 (46.90%)
>5 years	36 (27.70%)
Clinical features	
Complete KD	75 (57.6%)
Incomplete KD	55 (42.31%)
Fever	122 (93.8%)
Oral mucositis	93 (71.54%)
Rash	88 (67.69%)
Conjunctival Injection	80 (61.54%)
Extremity changes	49 (37.69%)
Cervical lymphadenopathy	43 (33.08%)
KD shock syndrome	7 (5.38%)
IVIG Refractory	37 (28.46%)
Echocardiographic changes	
CAAs	61 (46.92%)
Valvular Regurgitation	5 (3.84%)
Pericardial Effusion	4 (3.07%)
LV Dysfunction	2 (1.53%)
Number of CAAs	
LMCA	46 (35.38%)
LAD	37 (28.46%)
RCA	20 (15.38%)
LCX	11 (8.46%)
Based on aneurysm size	
Small CAA	46 (35.38%)

Moderate CAA	22 (16.92%)
Giant CAA	7 (5.38%)
Treatment received	
MPS	58 (45%)
Infliximab	27 (21%)
Cyclosporine	3 (2.31%)
Anakinra	3 (2.31%)

(KD-Kawasaki Disease, IVIG-IV Immunoglobulin, MPS -Methyl Prednisolone, LMWH -low-molecular-weight heparin, CAA-coronary artery aneurysm, LMCA- Left Main Coronary Artery, LAD-left Anterior Descending Artery, RCA- Right Coronary Artery, LCX-Left Circumflex Artery)

Title: A Comparative Study of Coronary artery diameters on 2D Echocardiography and CT Coronary Angiography: Our experience at Chandigarh, India

Authors: Ripudaman Singh[1], Radhika Semwal[1], Rakesh Kumar Pilania[1], Aarti Goyal[2], Abarna Thangaraj[1], Himanshi Choudhary[1], Pandiarajan Vignesh[1], Deepti Suri[1], Arun Sharma[3], Sanjeev Naganur[4], Manphool Singhal[3], Anju Gupta[1], Shankar Prinja[2], Surjit Singh[1]

Affiliations:
[1]Allergy Immunology unit, Department of Pediatrics, PGIMER, Chandigarh, India, 160012
[2]Department of Community Medicine and School of Public Health, PGIMER, Chandigarh;
[3]Department of Radiodiagnosis and Imaging, PGIMER, Chandigarh, India, 160012
[4]Department of Cardiology, Advanced Cardiac Centre, PGIMER, Chandigarh;

Introduction: 2D echocardiography (2DE) has hitherto been the standard of care for evaluation of coronary artery abnormalities (CAAs) in children with Kawasaki disease (KD). CT coronary angiography (CTCA) is an emerging imaging modality in this field.

Objectives: To compare coronary artery dimensions on CTCA with 2DE in children with KD.

Methods: Records of children with KD were retrieved, wherein both CTCA and 2DE had been carried out within 48 hours of 2DE. Comparison of proximal coronary artery dimensions on both modalities was made. 2DE was carried out by clinical fellows experienced in coronary

artery assessment, while CTCA were performed by an experienced cardiac radiologist blinded to the findings of 2DE. Statistical analysis was done on SPSS software version 23, and a paired t-test was used to compare mean diameters recorded on two imaging modalities.

Results: There were 126 children with KD who fulfilled the inclusion criteria. Fifty-five children were imaged at presentation, while 71 were imaged during convalescence/ follow-up. There was a significant difference in left main coronary artery (LMCA) and right coronary artery (RCA) measurements between CTCA and 2DE in terms of absolute diameter as well as Z scores. For LMCA, the p-value was <0.001 in comparison for both absolute diameters and Z scores, whereas for RCA, it was 0.025 for absolute diameters and 0.021 for Z scores. On average, LMCA measurements on CTCA were 0.466 mm smaller than on ECHO, and RCA measures 0.12 mm more on CTCA as compared to ECHO (95% CI). There was no significant difference between left circumflex artery (LCx) dimensions measured through 2DE and CTCA in terms of absolute diameter(p=0.901) and Z scores (p=0.907) respectively. Similarly, no significant difference was noted between left anterior descending (LAD) with p value = 0.134 and 0.136 in terms of absolute diameters and z scores, respectively. The mean difference between LAD and LCx on 2DE and CTCA was 0.046mm and 0.065mm, respectively. But to make a note that LCX was measured in 66/126 patients via 2D ECHO vs 98 patients with CTCA.

Conclusion: Dimensions of coronary arteries derived from CTCA in proximal segments of LAD and LCx were comparable with 2DE, while LMCA dimensions were significantly lower on CTCA, whereas it was higher for RCA. CTCA has the advantage of evaluation of LCx, which was missed on 2DE in 46.3% patients. CTCA thus has the potential to become the standard of care for diagnostic evaluation of children with KD.

Title: Gut Microbiota Alterations in Children with Kawasaki Disease and the Impact of Treatment: Experience from Chandigarh, North India

Authors: Vaishali Thakur, Rakesh Kumar Pilania, Amit Kumar, Manpreet Dhaliwal, Amit Rawat, Surjit Singh
Affiliation: Allergy Immunology Unit, Department of Pediatrics, Advanced Pediatrics Centre,
PGIMER, Chandigarh
Presenting author with contact details: Vaishali Thakur
Email: vaishalithakur4767@gmail.com

Background: Kawasaki Disease (KD) is an acute vasculitis of childhood associated with systemic inflammation and gut microbiota dysbiosis. Emerging evidence suggests that alterations in gut microbial composition may influence immune activation and disease progression. Data from Indian populations are limited, and regional differences in microbiota may affect disease phenotype and response to treatment.

Methods: This study was conducted at a tertiary care centre from **Chandigarh, North India**. Stool samples from 25 children with KD were collected **before and after treatment**. Gut microbial composition was analysed using 16S rRNA sequencing. Alpha diversity was assessed using the Shannon index, and beta diversity was evaluated with Bray–Curtis dissimilarity and principal coordinates analysis (PCoA). Statistical differences were determined using the Kruskal–Wallis test for alpha diversity and PERMANOVA for beta diversity. Differentially abundant genera were identified by comparing pre- and post-treatment
relative abundances. Study was approved by the Institute Ethics Committee. This study was funded by the Department of Science and Technology – Science and Engineering Research Board (DST-SERB), Government of India, under the Start-up Research Grant.

Results: At baseline (pre-treatment), KD patients showed reduced abundance of short-chain fatty acid (SCFA)–producing bacteria, including *Blautia*, *Roseburia*, *Faecalibacterium*, and *Lactobacillus*, along with increased abundance of proinflammatory *Enterococcus*. Following treatment, the relative abundance of *Blautia* (pre: 3.8 ± 1.1%, post: 6.4 ± 1.5%), *Roseburia* (pre: 2.6 ± 0.9%, post: 5.1 ± 1.3%), *Faecalibacterium* (pre: 4.3 ± 1.2%, post: 7.8 ± 1.6%), and
Lactobacillus (pre: 1.9 ± 0.7%, post: 4.2 ± 1.1%) increased significantly, while *Enterococcus* decreased (pre: 6.2 ± 1.8%, post: 2.9 ± 1.0%). Alpha diversity improved after treatment (Shannon index: pre = 2.94 ± 0.32; post = 3.28 ± 0.27; H = 4.03, p = 0.041). Beta diversity analysis demonstrated distinct clustering between pre- and post-treatment samples (PERMANOVA F = 3.54, p = 0.001).

Conclusion: Children with KD from North India exhibit baseline gut microbiota alterations characterized by depletion of SCFA-producing and enrichment of proinflammatory taxa. Treatment leads to partial restoration of beneficial microbes, reduction of proinflammatory bacteria, and improved microbial diversity. These findings suggest that modulation of gut

microbiota may contribute to immune and vascular recovery in KD.

“Early Referral and Intensified Therapy as Game-Changers in High-Risk Kawasaki Disease”

Dr Gopika P Mohan,DM Resident ,Amrita Institute of Medical Sciences
Dr Suma Balan, Professor and HOD,Rheumatology Department Amrita Institute of Medical Sciences
Presenting Author: Dr Gopika P Mohan(9446467250)

BACKGROUND
Kawasaki disease (KD) is an acute, self-limited vasculitis of childhood with a predilection for medium-sized arteries, particularly the coronary arteries. Coronary artery aneurysms (CAA), represent the most serious complication and are strongly associated with delayed or inadequate treatment. Although intravenous immunoglobulin (IVIG) remains the standard of care, a subset of patients,particularly infants, males, and those with early coronary artery dilation remain at high risk. Timely primary intensification of immunosuppressive therapy may mitigate this risk.
METHODS
We conducted a retrospective case series of 53 children with KD managed at our tertiary care center between 2019 and 2025. All patients fulfilled American Heart Association criteria for KD .High-risk features (male sex, infancy, coronary dilation at baseline) and the timing of primary intensification were specifically assessed. Our intensification protocol included infliximab, intravenous methylprednisolone followed by oral prednisolone with cyclosporine or anakinra added for refractory cases.
RESULTS
53 patients were analysed,males predominated, with a median age of 4 years. Thirty patients (56.6%) were referred, while 23 (43.4%) presented directly. Median diagnostic delay exceeded 14 days in 51.7% of referred patients, whereas 85% of directly presenting patients were diagnosed within 5 days (15% within 10 days). All referred patients experienced delays in primary intensification, resulting in progressive coronary dilation. In contrast, all directly presenting patients received intensification within 4 days of high-risk recognition, which effectively prevented giant aneurysm formation. Among referred patients, 45% received intensification only after 14 days, with adverse coronary outcomes. Infants were disproportionately affected, with 62% experiencing delayed intensification and 65% of

patients with baseline coronary dilation had delayed escalation. These findings strongly correlate delays in intensification with the progression to giant CAAs

CONCLUSION

This study underscores the critical role of early recognition of high-risk KD and prompt initiation of primary intensification therapy in preventing catastrophic coronary outcomes. Delays in treatment escalation especially in infants ,males and patients with early coronary changes were directly associated with progression to giant aneurysms. In contrast, timely intensification within the first week consistently prevented severe coronary sequelae. These results highlight the urgent need for heightened awareness, streamlined referral pathways, and adoption of standardized intensification protocols in high-risk KD to optimize long-term cardiovascular outcomes.

Rewriting the Timeline: Late New-Onset Coronary Aneurysms in Kawasaki Disease: A Wake-Up Call for Lifelong Vigilance

Aishwarya Susanne M[1], Murugan Sudhakar[1], Shruti Irene Varghese[2], Anu Punnen K[1], Sathish Kumar T[1]

Affiliation:

[1]Division of Pediatric Rheumatology, Department of Pediatrics, Christian Medical College, Vellore.

[2]Pediatric Cardiology, Department of Cardiology, Christian Medical College, Vellore

Background:

Late coronary artery aneurysms (CAAs) in children with Kawasaki disease (KD) is an under-acknowledged entity and has implications for long-term cardiac health. This underscores the necessity for long-term follow-up of children who had documented normalisation of initial CAAs.

Methods:

Analysis of a child with KD with ‘late new-onset CAAs’

Results:

In the past 15 years, our centre reported 150 children with KD, with one patient (0.6%) developing late new-onset CAA. This 8-year-old boy, presented with a seven-day fever, cough, strawberry tongue, red-cracked lips, skin peeling on his fingers, and bilateral non-tender

cervical lymphadenopathy. Laboratory investigations showed elevated inflammatory markers [Figure-1A], anaemia, hypoalbuminemia, and a 2-dimensional echocardiogram showed mild saccular aneurysm in left main coronary artery (LMCA), left anterior descending (LAD) and right coronary arteries (RCA) [done elsewhere; size and Z-scores were not mentioned]. The child was diagnosed with incomplete KD and multiple CAAs and received intravenous immunoglobulin (2 g/kg) and oral aspirin (5 mg/kg/day) on day-9 of illness. Post-treatment, he improved, with no further worsening of CAA. He was discharged on aspirin, and follow-up echocardiogram 2-weeks later showed no progression in CAAs.

He sought a second opinion at our centre during the eighth week of illness, where our echocardiogram revealed normal coronary vessels [Figure-1B]; hence, aspirin was discontinued.

Two-years later, the boy had fever, cough, and cold symptoms lasting two days, but with no systemic features. He then underwent regular echocardiographic screening, which resulted in an abnormal finding, prompting a referral to our facility. A computed-tomography angiogram performed at our centre revealed diffusely ectatic LMCA and LAD [small-aneurysms; 4.6 and 4.1 Z-scores, respectively] [Figure-1B]. He was started on dual antiplatelet therapy (aspirin and clopidogrel). At the 3rd-month follow-up, ECHO showed normalisation of the coronaries; hence, dual antiplatelet therapy was stopped, and he was advised for regular follow-up.

Conclusion

In KD, the risk of developing or worsening CAAs typically lasts until 6 weeks after the onset of illness [subacute phase]. After this period, further deterioration is uncommon, and most aneurysms either decrease in size, transform into stenotic lesions, or remain stable.

Literature documents 21 cases [3% incidence rate] of new or expanding CAAs occurring 1.9 to 19 years after KD diagnosis. Our child had 2 new CAAs [small-sized] after 2 years of diagnosis. This emphasises the probable need for regular screening of patients who have

undergone normalisation of coronaries in follow-up, to timely identify and manage potential life-threatening cardiac events.

[1A]

Age of the child	**8-years**		**10-years**
Date	**01-Apr-23**	**26-May-23**	**07-Feb-25**
Haemoglobin (g/dL) [N: 11-15]	**10.0**	11.6	13
Total leukocyte counts [x10^9/L, N: 4-11]	11.67	8.4	7.4
Differential counts (%) N/L/E/M	84/7/4/3	68/21/1/10	51/38/4/1
Platelets [x10^9/L, N: 150-450]	263	285	247
ESR (mm/1st hour) [N: <10]	**87**		6
CRP (mg/L) [N: <6]	**26.4**	12	<0.53
Serum Albumin (g/dl) [N: 3.5-5]	**2.3**		
ALT (U/L) [N: <45]	28	19	13
Urine WBC (Cells/ HPF, N: <5)	4		

[1B]

Timeline	At diagnosis	2-week	8-week		**CT angiogram [1y 10month]**		2y 1month	
			26-May-23		**11-Feb-25 [CT angio]**		**14-May-25 [ECHO]**	
ECHO	**02-Apr-23**	**18-Apr-23**	**Size [mm]**	**Z-score**	**Z-score**	**Description**	**Size [mm]**	**Z-score**
LMCA	Mild saccular aneurysm in LMCA, LAD, RCA. LCx: Normal	Mild dilatation of LMCA, LAD. LCx and RAD normal	2.6	0.21	**4.662**	**5.3mm**	3.5	1.57
LAD			2.1	0.42	**4.152**	**4.2mm**	2.8	1.55
LCx			1.5	-0.91	1.632	3.2mm	2.2	0.11
RCA			2.2	-0.08	0.39	3mm	2.3	-0.51

Figure [1A]: Laboratory investigations at diagnosis and at follow-up. [1B]: Serial echocardiograms from initial diagnosis and computed tomography angiogram at 1y 10months

Novel Biomarkers in Long-term Surveillance of Coronary Artery Abnormalities in Kawasaki Disease Patients with Giant Coronary Aneurysms: a Comparative Study with Healthy Controls

Authors & Affiliations
Taranpreet Kaur1, Rakesh Kumar Pilania2, Amit Rawat2, Ankur Kumar Jindal2, Manpreet Dhaliwal2,
Saniya Sharma2, Priyanka Srivastava2, Arun Sharma1, Surjit Singh2, Manphool Singhal1,
1Department of Radiodiagnosis, Nehru Hospital, PGIMER, Chandigarh, India
2Department of Paediatrics, PGIMER, Chandigarh, India
Presenting Author:

Taranpreet Kaur, Research Scientist, Department of Radiodiagnosis, PGIMER, Chandigarh, India. Email:

Background

Kawasaki Disease (KD) is a childhood vasculitis that can cause giant coronary aneurysms (GCA), predisposing patients to late cardiovascular sequelae. Although cardiac magnetic resonance (CMR) allows detailed assessment of myocardial structure and function, circulating
biomarkers may reveal subtle inflammatory or fibrotic processes not captured by imaging. Galectin-3, a β-galactoside-binding lectin implicated in fibrosis and extracellular matrix remodeling, has emerged as a potential marker of low-grade myocardial injury. This study evaluated CMR findings and plasma levels of Galectin-3, Growth Differentiation Factor-15 (GDF-15), and S100A8/A9 in KD patients with GCA compared with healthy controls.

Methods

Twenty KD patients with documented GCA underwent CMR on a 3T Siemens Vida scanner. Sequences included cine imaging for ventricular function, native T1/T2 mapping for tissue characterization, and late gadolinium enhancement (LGE) for fibrosis detection. Plasma samples from KD patients and 20 matched healthy controls were analyzed for Galectin-3, 2GDF-15, and S100A8/A9 using ELISA. Results were expressed as mean ± SD, and intergroup comparisons were performed using appropriate statistical tests.

Results

All KD patients demonstrated preserved left ventricular systolic function (mean LVEF 62 ± 4.5%) with normal native T1 (1045 ± 28 ms) and T2 (48.3 ± 3.1 ms) values. No diffuse myocardial fibrosis or edema was noted, and only one patient (5%) showed focal endocardial enhancement associated with aneurysmal thrombus. Galectin-3 levels were higher in KD patients (11.37 ± 1.60 ng/mL) compared to controls (9.63 ± 1.20 ng/mL), though not statistically significant ($p = 0.286$). GDF-15 levels were modestly lower (467.4 ± 95.4 pg/mL vs. 606 ± 102 pg/mL; $p = 0.155$), while S100A8/A9 levels were comparable (0.398 ± 0.016 ng/mL vs. 0.414 ± 0.014 ng/mL; $p = 0.365$).

Conclusion

KD patients with GCA showed preserved myocardial function and no imaging evidence of diffuse fibrosis on CMR. Galectin-3 levels tended to be higher in KD patients than in controls,
suggesting possible mild or residual myocardial matrix activity. This finding, while

preliminary, indicates that Galectin-3 may reflect subtle myocardial alterations in long-term KD survivors and warrants investigation in larger, longitudinal studies.

Exercise capacity in children with Kawasaki disease and MIS-C (Multi-system Inflammatory Syndrome in Children) with and without coronary artery aneurysms in comparison to healthy controls and the effect of a structured exercise program

Authors:
Dr. Shahid H Shali, Senior Resident, Cardiology, JIPMER, Puducherry.
Dr. Avinash Anantharaj, MD, DM Cardiology, Associate Professor, JIPMER, Puducherry.
(Presenting author, Ph- 7728050413, E-mail: avinboxer@gmail.com)

Background

Kawasaki Disease (KD) and Multi-system Inflammatory Syndrome in Children (MIS-C) are pediatric inflammatory disorders linked to long-term cardiovascular complications, primarily coronary artery aneurysms (CAA). Despite normal resting cardiac evaluations, concerns exist regarding reduced aerobic exercise capacity in this population. Evidence on objective functional assessments and the benefits of exercise interventions in this group remains limited. This study primarily compared the baseline aerobic exercise capacity of children with prior KD or MIS-C with that of age, gender, and weight-matched controls. Secondary objectives included assessing changes in exercise capacity after a 6-month structured exercise program and screening for inducible myocardial ischemia in those with CAAs.

Methods

This single-center study at JIPMER, Pondicherry, enrolled 15 patients ≥7 years (mean age 11.1 □} 3.3 years) with documented KD or MIS-C, with or without mild-to-moderate CAAs based on 2D Echo (Z-score ≥2.5 to <10 as per Dallaire and Dahdah, JASE 2011) or CT coronary angiography, and 15 matched healthy controls. Baseline aerobic capacity was evaluated by symptom-limited cardiopulmonary exercise testing (CPET) on a bicycle ergometer using the Modified Godfrey protocol. Patients with CAAs (n=3) underwent myocardial perfusion imaging (MPI) using 99mTc-MIBI. The patient group then completed a 6-month exercise program (30

minutes of aerobic activity, thrice weekly). A follow-up CPET was performed 6 months after the initial test. Independent and paired t-tests were applied for group comparisons.

Results

All subjects (patients and controls) had significantly lower peak VO_2/kg compared to Western data. At baseline, the patient group had significantly reduced aerobic capacity compared to controls, with lower mean peak VO_2/kg (24.77}5.99 vs. 31.54}6.83 ml/kg/min; p=0.007) and METS (7.07□}1.71 vs. 8.99□}1.95; p=0.007). Out of three patients with CAAs (LMCA

Z score +7.46; RCA Z score +7.74; and the third patient with LMCA Z score +3.74, RCA Z score

+4.66), none showed inducible ischemia on stress MPI. After 6 months, the patient group demonstrated statistically significant improvement in exercise capacity, with VO_2/kg rising to 28.10□}3.87 ml/kg/min (p=0.022) and METS to 7.94□}0.96 (p=0.040

Conclusion

Children with KD or MIS-C show impaired aerobic capacity compared with healthy peers, likely reflecting deconditioning rather than ischemia in mild-to-moderate CAAs. A structured 6-month aerobic program was safe and effective, significantly improving exercise tolerance. These findings highlight the need to integrate functional assessment and the role of regular aerobic exercises in improving long-term cardiovascular outcomes for this population.

The limitations our study were small sample size, single center data and smaller number of patients with CAAs.