Mid–Coronary Artery Wall Echogenicity Can Contribute to the Initial Diagnosis of Kawasaki Disease: Quantitative Measurements by Transthoracic Echocardiography

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Background: Periarterial echogenicity in the proximal coronary arteries (CAs) increases in the acute phase of Kawasaki disease (KD). However, some studies have questioned the diagnostic value of periarterial echogenicity in differentiating KD from other febrile diseases (non-KD) because of its relatively low specificity. In this study, the authors quantitatively assessed the degree of echogenicity in the proximal and mid segments of both CAs to determine its additional diagnostic value in patients with clinically suspected KD.

Methods: A total of 109 consecutive children (median age, 21 months; interquartile range, 11.0-47.8 months) who underwent transthoracic echocardiography for suspected KD (April 2021 to March 2023) were retrospectively examined. Two-dimensional echocardiographic images in the proximal and mid segments of both CAs were digitally stored and transferred to an offline image analysis system. The mean pixel value of the arterial wall was calculated in grayscale ranging from 0 to 255 (corrected for the intracardiac blood pool adjacent to the target site).

Results: A total of 109 patients were included, 87 (80%) ultimately diagnosed with KD (including 18 with incomplete KD) and 22 (20%) ultimately diagnosed with non-Kawasaki febrile diseases. Although the KD group generally showed higher CA wall echogenicity than the non-KD febrile group, there was no significant difference in the mean pixel values at the proximal segment ($P = .34$ for each). The KD group showed significantly higher echogenicity in the mid segments of both CAs than the non-KD febrile group (mid right coronary artery, $P = .0049$; mid left anterior descending coronary artery, $P = .011$). Similar results were observed in a small prospective cohort of 31 children examined under rigorously standardized ultrasound settings.

Conclusions: CA echogenicity in the mid segments may have potential diagnostic value in the early evaluation of suspected KD, possibly reflecting the characteristic diffuse involvement of the CAs in the acute phase. (J Am Soc Echocardiogr 2026;■:■-■.)

Keywords: Kawasaki disease, Incomplete Kawasaki disease, Perivascular brightness, Coronary artery wall echogenicity, Coronary artery aneurysms, Transthoracic echocardiography

Kawasaki disease (KD) is an acute febrile illness occurring predominantly in childhood. Without treatment, 15% to 25% of patients develop coronary artery aneurysms (CAAs), which can lead to ischemic heart disease and possibly myocardial infarction.[1] A CAA is a serious complication during the course of KD. An early and accurate diagnosis of KD is essential to prevent the development of CAAs. Periarterial brightness in the coronary arteries increases in the acute phase of KD, as shown by conventional two-dimensional images or integrated backscatter.[2-4] Therefore, coronary arterial brightness is clinically considered as an additional diagnostic factor in incomplete KD that does not meet the specific diagnostic criteria.[5,6] However, some researchers have questioned the clinical value of using proximal coronary arterial brightness in the initial stratification of KD from other febrile diseases because of its relatively low specificity.[7-9] Additionally, a definitive initial diagnosis of KD remains elusive. According to previous studies that focused on other febrile diseases,[7] specificity in the diagnosis of KD and reproducibility in the measurements of coronary arterial brightness were limited. Coronary artery brightness is not clearly

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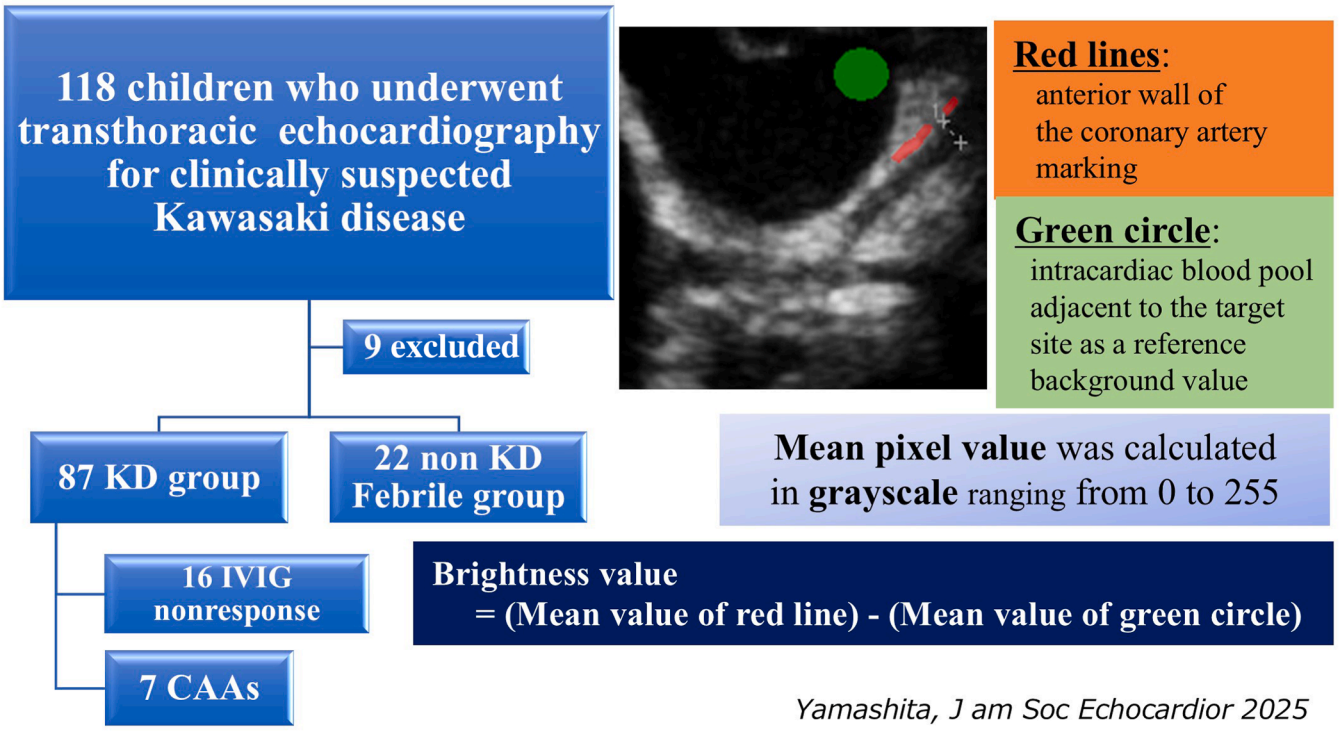

Central Illustration We quantitatively assessed the degree of echogenicity in the proximal and mid segments of both coronary arteries in patients with clinically suspected KD. The KD group showed significantly higher echogenicity in the mid segments of both coronary arteries than the non-KD febrile group.

recommended in the current American Heart Association guidelines, because of the relatively weak scientific evidence.[3] According to previous pathophysiologic evaluation, KD involves coronary branches diffusely and simultaneously in its acute stage.[5,6] The aim of this study was to quantify the echogenicity of the coronary artery wall not only in the proximal segment but also up to the mid segment on the basis of the hypothesis that advanced screening of coronary artery echogenicity contributes to the initial stratification of KD from other febrile diseases.

Abbreviations
CAA = Coronary artery aneurysm
IQR = Interquartile range
IVIG = Intravenous immunoglobulin
KD = Kawasaki disease
LAD = Left anterior descending coronary artery
LMCA = Left main coronary artery
RCA = Right coronary artery

METHODS

Study Population

We retrospectively reviewed the medical records of children who underwent transthoracic echocardiography for clinically suspected KD from April 2021 to March 2023 at Miyazaki Prefectural Miyazaki Hospital in Miyazaki, Japan. This study was conducted with an opt-out consent policy with the approval of the institutional review board of Miyazaki Prefectural Miyazaki Hospital (approved October 26, 2023; reference number 23-53). The KD group was defined as children who met at least five of six diagnostic criteria of KD or those with incomplete KD who met three or four of six criteria but were suspected as having KD on the basis of overall findings by an experienced pediatrician. The non-KD febrile group was defined as children who had been suspected of having KD at their initial presentation but were ultimately diagnosed with non-KD febrile disease on the basis of their clinical course (Central Illustration). In addition, we recruited a prospective validation cohort to confirm the generalizability of the results of retrospective cohort study (approved May 28, 2025; reference number 25-31). The brightness of both the proximal and mid segments of the coronary arteries was compared under standardized conditions in accordance with the guidelines for coronary *Z* score measurements. The examinations were conducted under rigorous settings, including adjustments for dynamic range, postprocessing, and power.

Treatment for KD and Incomplete KD

Intravenous immunoglobulin (IVIG) and oral aspirin treatment was applied for KD and incomplete KD. Initial combination therapy with cyclosporine and prednisolone was administered in addition to IVIG for patients who met either of the following criteria: (1) KD with possible IVIG resistance (Kobayashi score ≥ 5,[10] Egami score ≥ 3,[11] or Sano score ≥ 2[12]) and (2) coronary artery dilation

HIGHLIGHTS

- Mid–coronary artery echogenicity is higher in KD than in non-KD febrile diseases.
- Coronary arterial echogenicity in the proximal segment tends to be higher in KD.
- Scanning coronary arteries in multiple segments helps in initial diagnosis of KD.

on initial echocardiography. Patients were judged as IVIG nonresponders when fever persisted for >24 hours after the initial IVIG treatment and they then required additional IVIG.[13] In accordance with the American Heart Association guidelines,[3] a CAA was defined as a coronary artery diameter with a *Z* score of +2.0 or higher. Small aneurysms were defined as a *Z* score of +2.5 to +5.0. The *Z* score of the coronary arterial internal diameter was calculated using a previously reported *Z* score curve.[14]

Echocardiographic Data Processing

In all patients with suspected KD at the initial presentation, transthoracic echocardiography was performed by an experienced pediatric cardiologist. A standard echocardiographic examination, including cardiac function and screening of valve diseases, congenital heart diseases, or pericardial effusion, was performed. Coronary arteries including the left main coronary artery (LMCA), left anterior descending coronary artery (LAD), and circumflex coronary artery were also examined. Additionally, the proximal and mid segments of the right coronary artery (RCA) were carefully examined and visualized to evaluate coronary arterial involvement. In the present study, coronary images were acquired with rigorous settings of the equipment under the following guidance for the measurement of coronary *Z* score, which is generally applied in the investigation of coronary arteries in patients with KD.[14] The diameter of the LMCA was measured at the midpoint between the ostium of the left coronary artery and its bifurcation. The proximal RCA was measured at 3 to 5 mm distal from its origin. The mid segments of the LAD and RCA were defined as follows: mid LAD, 3 to 5 mm distal to the left coronary artery bifurcation; mid RCA, RCA visualized along the tricuspid annulus.[14,15] The right coronary sinus of the aorta was used for analysis of the echogenicity of the aorta according to a previous study.[16] These measurements were obtained using a Vivid i (GE Healthcare) equipped with 6-MHz probe under a sedative procedure, if required. The depth of the two-dimensional images of the coronary arteries was set between 5 and 10 cm, with the gain adjusted as appropriate. All examinations were performed by a single experienced pediatric cardiologist using standardized technical settings as follows: compression, 16 dB; reject, 2; dynamic range, 3 to 5; power, 0; frequency range, 3.5 to 6.9 MHz; use of harmonics; and consistent postprocessing adjustments. In the prospective validation cohort, coronary artery image acquisition was performed using rigorous settings as follows to confirm the generalizability of the retrospective findings: compression, 11 dB; reject, 2; dynamic range, 3; power, 0; frequency range, 2.9-6.9 MHz; use of harmonics; and consistent postprocessing adjustments.

Calculation of the Brightness of the Arterial Wall

Images of the target coronary arteries were digitally stored and transferred for quantitative image analysis, which was performed by an engineer specialized in medical imaging (Y.U.) The images were converted to bitmap format, and the regions of interest of arterial walls were marked using free software (MaZda; https://qmazda.p.lodz.pl/)[17] by an investigator (N.Y.) to calculate arterial

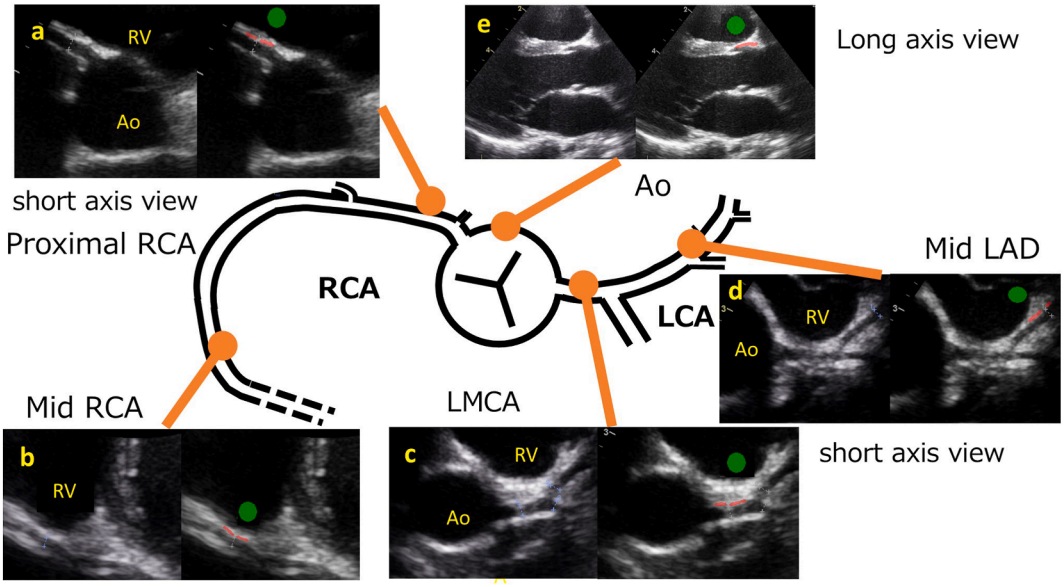

Figure 1 Method of measurement of arterial wall brightness in echocardiographic images. **(A)** Proximal RCA, **(B)** mid RCA, **(C)** LMCA, **(D)** mid LAD in the short-axis view, and **(E)** right coronary sinus of the aorta in the long-axis view. Red lines, approximately 5 to 10 mm in length, were applied to the anterior wall of the coronary artery (thoracic side only) and the right coronary cusp of the aorta. The markings were placed in regions excluding the measured areas present in the original image. To perform correction, the intracardiac blood pool located on the thoracic side within the region of interest was marked with a green circle, which served as the reference background value. *Ao*, Aortic valve; *LA*, left atrium; *LCA*, left coronary artery; *RV*, right ventricle.

Table Baseline profiles of the KD group and non-KD febrile group

	KD (*n* = 87)	Non-KD febrile disease (*n* = 22)	*P*
Male/female	47/40	12/10	.99
Age, mo	20 (11-35)	28 (12-48)	.41
Height, cm	82 (73.0-93.2)	87.5 (77.4-99.6)	.41
Weight, kg	11.0 (9.0-14)	12.2 (9.0-15.6)	.48
Duration of illness at echocardiography, d	5 (4-6)	5 (4-7)	.23
CRP, mg/dL	7.1 (4.1-10)	5.5 (3.2-8.8)	.33
Incomplete KD (≤4 criteria)	16 (18.4)	—	
IVIG nonresponders	16 (18.4)	—	
CAA	7 (8.0)	—	

Data are expressed as number, median (interquartile range), or number (%).

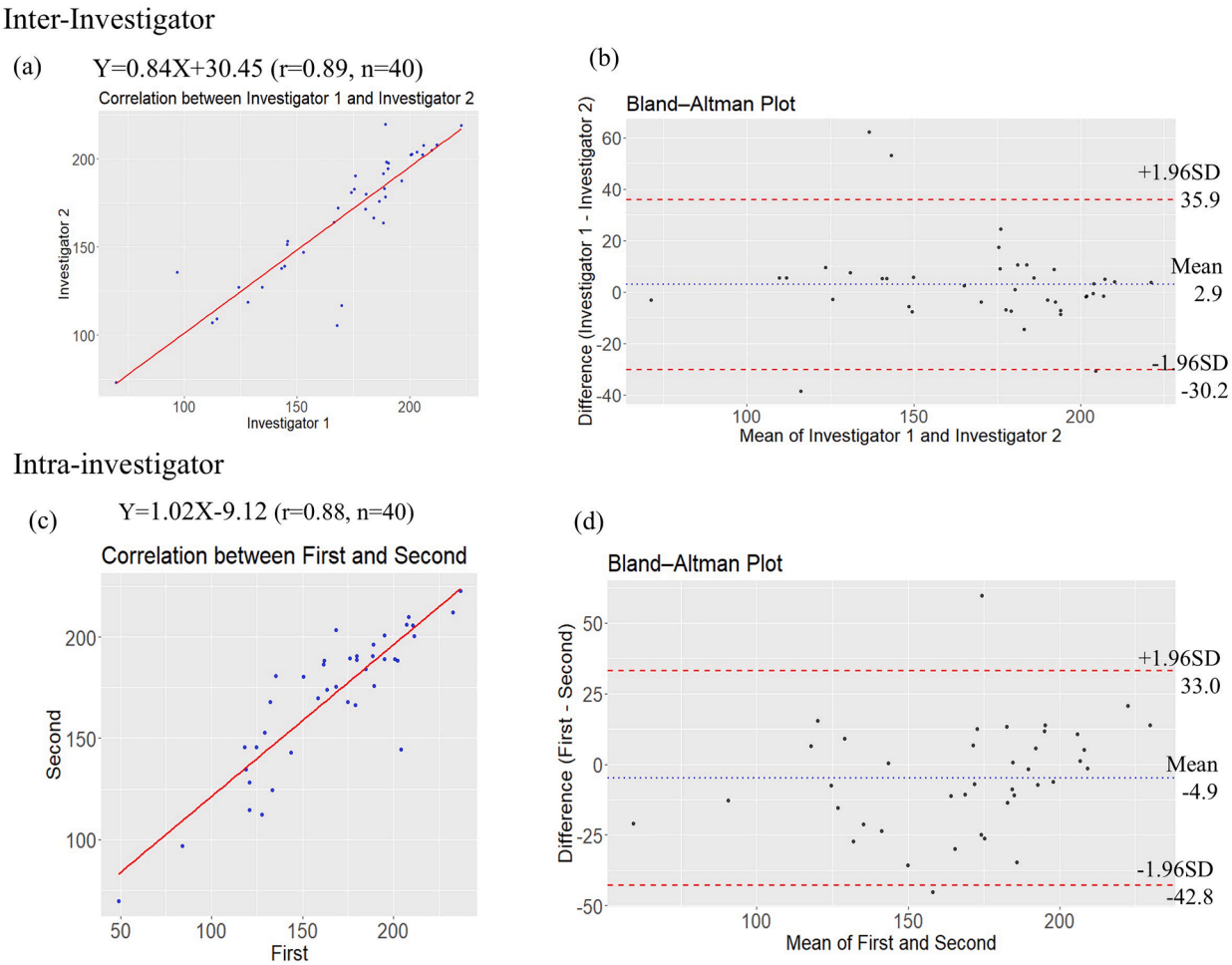

Figure 2 Assessment of interinvestigator and intrainvestigator echogenic brightness values with two different investigators. **(A** and **B)** Interinvestigator assessment of brightness values. **(A)** Linear correlation of brightness values between investigators 1 and 2. There was a strong correlation (*r* = 0.89) between the two investigators. **(B)** Difference in interinvestigator measurement by each value (Bland-Altman plot). **(C** and **D)** Assessment of intrainvestigator brightness values. Two different measurements of the same image were assessed by the same investigator. **(C)** Linear correlation between examinations 1 and 2. These was a strong correlation (*r* = 0.88) between examinations 1 and 2. **(D)** Difference in intrainvestigator measurement by each value (Bland-Altman plot).

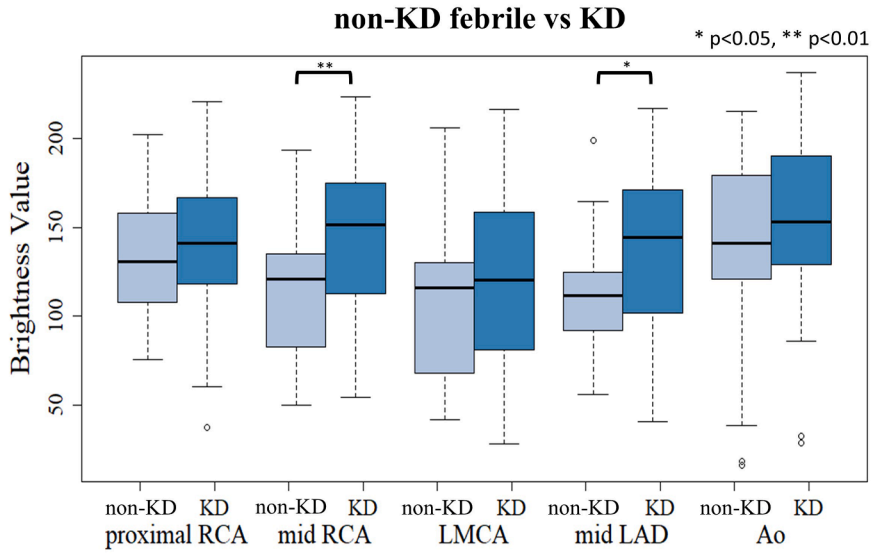

Figure 3 Comparison of brightness values between the KD and non-KD febrile groups in the retrospective cohort. The KD group showed significantly higher coronary artery brightness in the mid RCA and mid LAD than the non-KD febrile group. *Ao*, Aorta.

wall brightness. The regions of interest were at the (1) proximal RCA, (2) mid RCA, (3) LMCA, (4) mid-LAD, and (5) right coronary sinus of the aorta in the long-axis view. The coronary artery wall and the right coronary cusp of the aorta were linearly marked at intervals of three pixels each for a length of approximately 5 to 10 mm, and the mean pixel value was calculated in grayscale ranging from 0 to 255 (Figure 1). The intracardiac blood pool adjacent to the target site was measured as a reference background value, and the difference was recorded as the brightness value. To assess the reproducibility of the corrected brightness value, 40 images were randomly selected from recorded images by two separate investigators (interinvestigator measurement variability), and echogenicity was assessed twice in the same images by a single investigator (intrainvestigator measurement variability).

Statistical Analysis

Statistical analyses were performed using R version 4.3.2 (R Foundation for Statistical Computing). Quantitative image processing was conducted in Python version 3.11 (using public libraries cv2 and numpy). The scripts used for brightness quantification and subsequent analyses are available in a publicly accessible GitHub repository (https://github.com/UchiLab-Miyazaki/code-for-paper2025). The clinical variables are reported as median (interquartile range [IQR]). Comparisons of variables between the KD and non-KD febrile groups and between any two subgroups in the KD group were made using the Mann-Whitney *U* test. The sex ratio between the two groups was analyzed using Pearson's χ^2 test. The reproducibility of inter- and intrainvestigator brightness values was compared using Pearson's correlation coefficient and Bland-Altman analysis. *P* values <.05 were considered to indicate statistical significance.

RESULTS

Patient Characteristics

During the study period, 118 children underwent echocardiography for suspected KD. Patients with KD without appropriate digital echocardiographic images ($n = 6$) and those who had not required IVIG treatment because of resolution of their fever ($n = 3$) were excluded. Ultimately, 109 patients were included in the present study: 87 in the KD group (including 18 with incomplete KD) and 22 in the non-KD febrile group. In the KD group, coronary image acquisition was achieved for the proximal RCA and LMCA in 87 patients (100%), for the mid RCA in 82 patients (94.4%), for the mid-LAD in 86 patients (97.8%), and for the aorta in 83 patients (93.1%). In the non-KD febrile group, coronary image acquisition was achieved for the proximal RCA, LMCA, and aorta in 22 patients (100%), and it was achieved for the mid RCA and mid LAD in 21 patients (95.5%). The baseline profiles of the two groups were similar (Table). There were no significant differences in sex, age, or body size between the KD and non-KD febrile groups. The duration of fever at the time of the echocardiographic examination and C-reactive protein concentrations were not different between the two groups. Among the patients, 16 (18.4%) were classified as having incomplete KD with three or four diagnostic criteria. Sixteen patients (18.4%) had initial treatment resistance to IVIG (IVIG nonresponders), and seven (8.0%) had CAAs. All patients with KD received oral aspirin (30 mg/kg/d) and 2 g/kg IVIG as initial treatment. High-risk patients with IVIG resistance were initially treated with a combination of IVIG and cyclosporine ($n = 23$) or prednisolone ($n = 2$), and the absence of suspicious concomitant bacterial or fungal infection was confirmed. The final diagnoses in the non-KD febrile group were viral infection ($n = 12$), bacterial infection ($n = 7$; urinary tract infection, cervical lymphadenitis, pneumonia, bacteremia, and hepatic abscess), and other infectious diseases ($n = 3$).

Reproducibility of Measurements of Brightness Values

Measurements of coronary artery echogenicity were performed on 40 randomly selected echocardiographic images by two investigators. The interinvestigator reproducibility was $r = 0.89$ (95% CI, 0.80-0.94; mean difference, 2.9 ± 16.8; Figure 2). The intrainvestigator reproducibility, as assessed by investigator 1, who conducted measurements twice at different time points, was $r = 0.88$ (95% CI, 0.78-0.93; mean difference, −4.9 ± 19.3; Figure 2). The inter- and intrainvestigator reproducibility were strongly correlated.

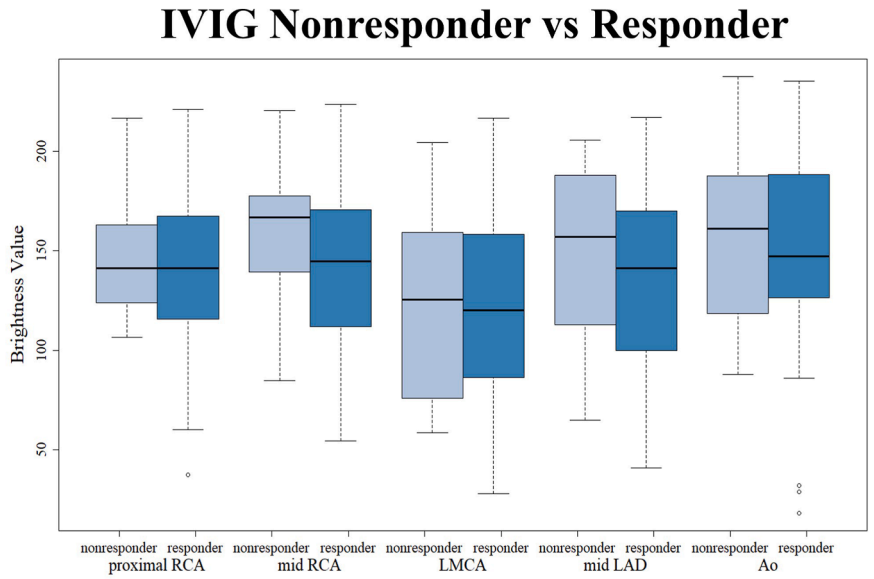

Figure 4 Comparison of echogenic brightness values between IVIG responders and nonresponders in the retrospective cohort. There was no significant difference in echogenicity in the coronary artery or aorta between IVIG nonresponders and responders. *Ao*, Aorta.

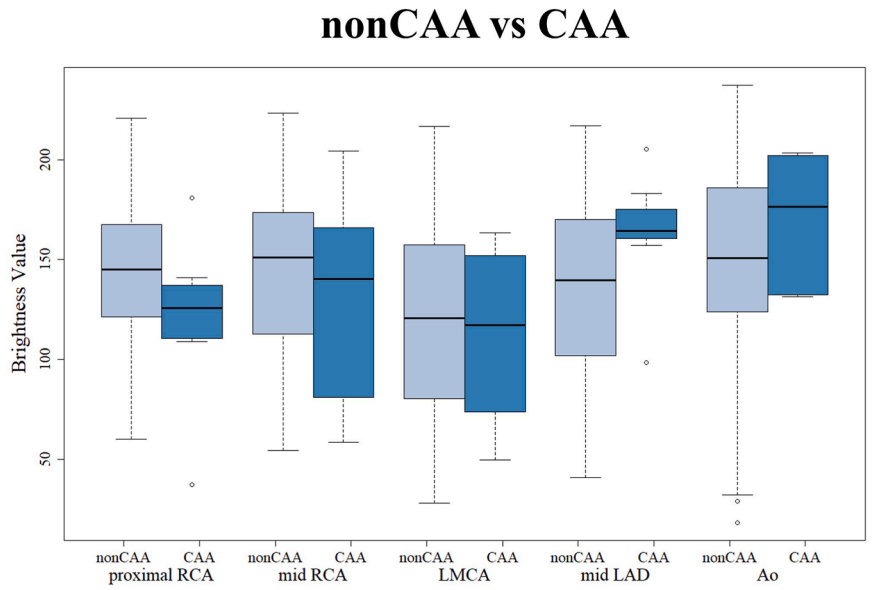

Figure 5 Comparison of echogenic brightness values between patients with and without CAAs in the retrospective cohort. There was no significant difference in echogenicity between these two groups. *Ao*, Aorta.

Comparison of Coronary Artery Brightness Values Between the KD and Non-KD Febrile Groups

Overall, although the KD group showed higher coronary artery wall brightness values than the non-KD febrile group, there was no significant difference at the proximal RCA or LMCA between the two groups. The KD group showed significantly higher coronary artery echogenicity in the mid segments of the RCA and LAD than the non-KD febrile group (mid RCA, 139.1 [IQR, 109.7-174.5] vs 121.1 [IQR, 82.9-134.8] [$P = .0049$]; mid LAD, 136.0 [IQR, 87.9-168.1] vs 111.7 [IQR, 92.0-124.5]; $P = .011$). The aorta showed no significant difference in echogenicity between the KD and non-KD febrile groups (Figure 3).

Comparison of Coronary Artery Brightness Values Between IVIG Nonresponders and Responders and Those With and Without CAAs

There was no significant difference in echogenicity of the proximal and mid coronary arteries between IVIG nonresponders and responders. Similarly, no significant difference in echogenicity was observed in the aortic wall between the two groups (Figure 4).

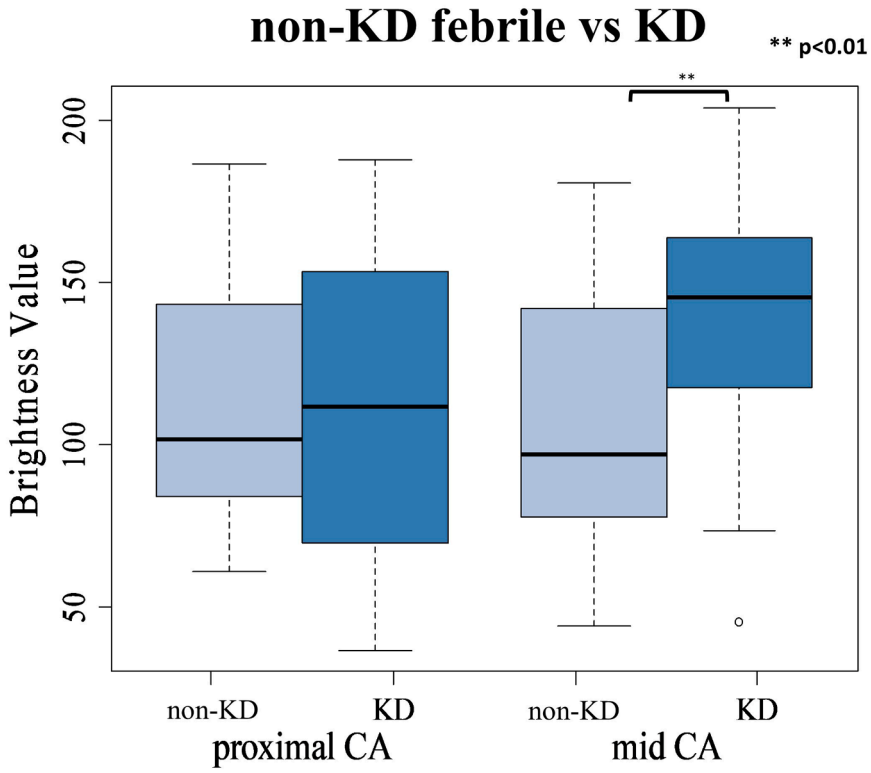

Figure 6 Comparison of brightness values between the KD and non-KD febrile groups in the prospective validation cohort. This analysis includes a total of 42 coronary arteries in the KD group and 20 coronary arteries in the non-KD febrile group, and the KD group showed significantly higher coronary artery brightness in the mid-CA. *CA*, Coronary artery.

Among the seven patients with CAAs, four showed transient dilation that normalized after 1 month, and three coronary diameters had regressed by 1 year after the onset. None of these patients showed residual aneurysms as sequelae. There was no significant difference in echogenicity in the proximal or mid coronary arteries between those with and those without CAAs (Figure 5).

Prospective Study to Support the Generalizability of Quantitative Coronary Artery Wall Echogenicity Using Rigorous Machine Settings for Data Acquisition

We prospectively enrolled 31 children clinically suspected of having KD (21 patients with KD and 10 non-KD febrile control subjects). Echocardiographic brightness was evaluated in both the proximal and mid segments of the left coronary artery and RCA, resulting in a total of 42 coronary arteries in the KD group and 20 coronary arteries in the non-KD febrile group. There was no significant difference in age, sex distribution, height, body weight, or the timing of echocardiography between the KD and non-KD febrile groups and no difference in the timing of examinations between the retrospective and prospective cohorts.

Although no significant difference was observed in the proximal coronary arteries ($P = .86$) between the two groups, brightness in the mid segments was significantly higher in the KD group ($P = .002$; Figure 6).

DISCUSSION

Previous studies of KD focused primarily on analyzing the brightness of the proximal left coronary artery or RCA.[2,4,8] However, to the best of our knowledge, this study is the first to investigate coronary artery echogenicity not only in the proximal coronary arteries but also in the mid segments of the coronary arteries in patients who were suspected of having KD in the acute phase. The main finding of the present study is that echogenicity in the mid segments of the coronary arteries was significantly higher in children with KD than in those who were ultimately diagnosed with non-KD febrile disease. Coronary arterial brightness values in the proximal segment alone tended to be higher in children with KD than in those with non-KD febrile group, but this finding was not significant. Our results might reflect the pathophysiologic characteristics of coronary vasculitis in KD that simultaneously and diffusely involves the coronary territories from the proximal to distal branches.[5,6] Interestingly, these findings were found in the KD group, including incomplete KD, which is often difficult to judge simply from the initial findings and carries a risk for CAA because of delayed diagnosis.[18,19] Scanning coronary arteries not only in the proximal segments but also up to the mid distal segments may support the initial diagnosis of KD that requires IVIG treatment.[8] A previous study showed that CAAs can occur not only in the proximal segments but also in the mid segments in patients with KD.[20] Our results suggest the importance of observing the entire coronary arteries when evaluating aneurysms and subclinical CAAs in suspected KD. Initial screening of KD can also contribute to avoid unnecessary IVIG administration to patients without KD.

Diagnostic Value of Coronary Artery Echogenicity in KD

Increased coronary artery brightness in KD was first reported by Newburger *et al.*[21] at the 7th International Kawasaki Disease

Symposium in 2001, and their findings were incorporated into the American Heart Association/American Academy of Pediatrics KD diagnostic guidelines in 2004.[2] Previous studies have shown mixed results regarding coronary arterial brightness in KD. Positive findings were reported by Abe *et al.*[4] and Nagata *et al.*,[16] who used integrated backscatter to evaluate proximal coronary segments, although both studies were limited by small sample sizes and nonfebrile control subjects. In contrast, Yu *et al.*,[9] McCulloch *et al.*,[8] and Rabinowitz *et al.*[7] found no consistent differences, using integrated backscatter or visual assessment, often including congenital heart disease or healthy control subjects. These discrepancies may be explained by differences in control selection and the reliance on vendor-specific, advanced imaging techniques, which limit generalizability. Our study differs by using conventional echocardiography and febrile control subjects, demonstrating for the first time that mid segment coronary brightness may distinguish KD from other febrile illnesses. In clinical practice, on the basis of the knowledge that coronary brightness increases in KD, visual judgment of echogenicity is commonly used among physicians as a key factor in deciding whether patients require treatment.

Our study had three novel approaches. First, an experienced engineer performed quantitative measurements using a Python program created specifically for this research dedicated to determining coronary echogenicity. Second, the coronary arteries were observed not only at the proximal sites but also at the mid coronary segments. The quantitative algorithm that we used is based on a simple 0-to-255 black-and-white gradation analysis that can be generally applied. Third, by adding a small prospective cohort of 31 children examined using rigorously standardized ultrasound settings, our intention was to minimize variability in image acquisition and to prospectively validate the association observed in the retrospective study. This design allowed us to verify whether the segment-specific difference in coronary brightness, particularly the greater echogenicity in the mid segments, would persist when images were obtained under consistent technical conditions.

Possible Mechanisms and Clinical Implications of Increased Coronary Artery Brightness in KD

Although the increased coronary artery brightness during the acute phase of KD have been reported by some research groups,[2,4,21] the mechanism remains unclear. In KD, coronary arteritis typically presents with edema, which results in separation of the tunica media, along with degeneration of smooth muscle cells, occurring at approximately the sixth to eighth day of illness.[22] The vasculitis progresses synchronously throughout the body, typically with a monophasic course, which is a characteristic feature of KD.[2,5,6] Cellular infiltration and edematous changes are generally thought to be a possible reason for this finding, on the basis of pathophysiologic findings of the coronary arteries in KD. Coronary vasculitis in KD is more robust and rapidly progresses to systematic vasculitis compared with other types of febrile illnesses that commonly do not cause coronary complications.[2,5,6] We speculate that the differences in the pathologies between KD and non-KD were responsible for our finding that echogenicity in the mid segment showed significant diagnostic value, not just in the proximal sites alone. Our study suggests that coronary arterial echogenicity not only in the proximal portion but also in the mid segment of the left coronary artery and RCA may have potential diagnostic value in the early evaluation of suspected KD. This can be a new recommendation for the routine coronary imaging in the initial screening of the children who are suspected of having KD, and further prospective studies to clarify the temporal changes in coronary artery echogenicity and to explore their associations with important clinical outcomes, including IVIG resistance, the development of CAAs, mitral regurgitation, pericardial effusion, and cardiac function, are needed.

Identifying High-Risk Patients by Coronary Echogenicity in the Acute Phase

Abe *et al.*[4] and Nagata *et al.*[16] reported that using calibrated integrated backscatter to measure coronary luminosity may be beneficial in assessing IVIG resistance and possible future progression of coronary artery lesions. However, we did not find any difference in echogenicity between IVIG responders and nonresponders in the present study. This lack of findings might be because of the use of cyclosporine as an initial treatment, on the basis of the currently recommended IVIG resistance score in this study.[23] In addition, we did not find significantly increased echogenicity in patients with KD who developed CAAs compared with those who did not. We note that the number of patients with CAA (defined as having a Z score of +2.0 or higher) was small, and all of the coronary dilation and aneurysms in this cohort showed regression within 1 year.

Limitations

There are several limitations to this study. First, the study was conducted at a single center using a single ultrasound system (Vivid i) used in daily clinical practice. Second, variations in gain settings and patient-specific factors, including thoracic thickness, may have influenced coronary brightness values. However, examinations were standardized and performed by an experienced pediatric cardiologist, and gain and depth were appropriately optimized for each patient.

Third, the regions of brightness measurements were limited to four specific coronary segments (proximal RCA, mid RCA, LMCA, and mid LAD). Fourth, the small number of patients with CAAs limits the ability to assess the prognostic value of coronary echogenicity. Although the findings suggest that mid segment brightness may aid in differentiating KD from other febrile illnesses, the real-world applicability of this parameter requires further evaluation.

Fifth, although a small prospective cohort was incorporated to validate the segment-specific differences under standardized imaging conditions, a larger prospective study integrating coronary echogenicity with other clinical information, biomarkers, and echocardiographic indices is needed to determine the diagnostic and prognostic utility of this approach, particularly for predicting IVIG resistance and CAA development. Our present findings, including both retrospective and initial prospective observations, demonstrate the diagnostic potential of mid segment coronary brightness for KD and provide a foundation for future multicenter prospective studies.

CONCLUSION

Coronary arterial echogenicity in the mid segments of the left coronary artery and RCA may have potential diagnostic value in the early evaluation of suspected KD. Therefore, examining not only the proximal segment but also up to the mid segment of the coronary arteries may be useful for differentiating KD. In terms of generalizability, the results were verified to persist when images were obtained under consistent technical conditions.

This study was a simple investigation that quantified data using grayscale imaging. In the future, we aim to conduct a prospective study and incorporate this measurement as an additional parameter in routine echocardiographic examinations for KD, with the goal of improving the diagnostic accuracy of KD.

CONFLICTS OF INTEREST

None.

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