Coronary Wall Structural Changes in Patients With Kawasaki Disease: New Insights From Optical Coherence Tomography (OCT)

Audrey Dionne, MD; Ragui Ibrahim, MD; Catherine Gebhard, MD; Mohamed Bakloul, MD; Jean-Bernard Selly, MD; Mohamed Leye, MD; Julie Déry, MD; Chantale Lapierre, MD; Patrice Girard, RN; Anne Fournier, MD; Nagib Dahdah, MD

Background—Coronary artery aneurysms (CAA) are serious complications of Kawasaki disease (KD). Optical coherence tomography (OCT) is a high-resolution intracoronary imaging modality that characterizes coronary artery wall structure. The purpose of this work was to describe CAA wall sequelae after KD.

Methods and Results—KD patients scheduled for routine coronary angiography underwent OCT imaging between March 2013 and August 2014. Subjects' clinical courses, echocardiography, and coronary angiography examinations were reviewed retrospectively. OCT was performed in 18 patients aged 12.4±5.5 years, 9.0±5.1 years following onset of KD. Of those, 14 patients (77.7%) had a history of CAA (7 with giant CAA and 7 with regressed CAA at time of OCT). Intracoronary nitroglycerin was given to all patients (88.4±45.5 μg/m^2). Mean radiation dose was 10.9±5.2 mGy/kg. One patient suffered from a transitory uneventful vasospasm at the site of a regressed CAA; otherwise no major procedural complications occurred. The most frequent abnormality observed on OCT was intimal hyperplasia (15 patients, 83.3%) seen at both aneurysmal sites and angiographically normal segments amounting to 390.8±166.0 μm for affected segments compared to 61.7±17 μm for unaffected segments (P<0.001). Disappearance of the media, and presence of fibrosis, calcifications, macrophage accumulation, neovascularization, and white thrombi were seen in 72.2%, 77.8%, 27.8%, 44.4%, and 33.3% of patients.

Conclusions—In this study, OCT proved safe and insightful in the setting of KD, with the potential to add diagnostic value in the assessment of coronary abnormalities in KD. The depicted coronary structural changes correspond to histological findings previously described in KD. (***J Am Heart Assoc***. 2015;4:e001939 doi: 10.1161/JAHA.115.001939)

Key Words: coronary disease • imaging • pediatrics

Kawasaki disease (KD) is an acute self-limited vasculitis that primarily affects infants and young children. Coronary artery aneurysms (CAA) are a serious complication of KD and develop in 5% of treated and 30% of untreated patients.[1,2] Conventional cardiac imaging techniques, such as echocardiography, computed tomography, coronary angiography, cardiac magnetic resonance imaging, and invasive angiography are useful to characterize aneurysm size and luminal diameter of the diseased coronary artery segment. However, those modalities are of limited use for visualization of detailed vascular anatomical data. Intravascular ultrasound has been used in interventional cardiology for over 2 decades and provides valuable information on the coronary vascular wall and lumen. Even though it is a cornerstone tool for the assessment of coronary atherosclerotic plaques in adults, its use in a pediatric population suffering from KD is limited by lack of experience in children in general and suboptimal axial resolution (100 to 150 μm).[3–5] Compared to intravascular ultrasound, optical coherence tomography (OCT) provides nearly 10 times higher axial resolution, providing a spatial definition of 10 μm, therefore allowing unique insights into the microstructure of the coronary wall. These features make it an emerging tool in the assessment of coronary pathologies and guiding coronary interventions.[6] The feasibility of OCT in a pediatric population with KD has recently been demonstrated in 5 patients;[7] however, data on microstructural vessel wall abnormalities in KD are scarce. Thus, the aim of our study was to assess microstructural coronary artery abnormalities in both aneurysmal and angiographically normal coronary

segments in patients following KD by OCT and to provide a frequency distribution of the observed changes.

Methods

Population

We included patients diagnosed with KD who underwent clinically indicated cardiac catheterization and coronary angiography between March 2013 and August 2014 at the Children's University Hospital St-Justine, Montreal, Quebec, Canada. Indications for catheterization were either routine follow-up of aneurysms or new cardiac symptoms suggestive of ischemia. Exclusion criteria included concurrent vascular inflammatory disease, evidence of underlying cause of hypercholesterolemia, or diseases known to induce vascular structural changes. Patients' charts were reviewed for demographic and clinical data. Subjects' coronary artery lesion evolution was reviewed by assessing previous echocardiography examinations and coronary angiographies, and compared with OCT findings. Coronary angiography images were independently assessed by 2 radiologists (J.D., C.L.), who were blinded to clinical and OCT findings. This retrospective chart review was approved by the local ethics review board.

Coronary Angiography and OCT Acquisition

A BMW (Abbott Vascular) or a Terumo (Terumo Interventional Systems) coronary guidewire was advanced distally in the coronary artery following intracoronary administration of nitroglycerine titrated to patients' body habitus. The Dragonfly® catheter (St-Jude Medical, Inc) was advanced over a guiding coronary catheter (6 Fr for adolescents and young adults, 5 Fr for younger patients). The catheter curve was selected according to the aortic arch curve and length. A low-osmolality contrast agent, Isovue-300® (Bracco Imaging), was hand injected in most cases (power injection in selected older patients) for intracoronary blood clearance. The detection of blood clearance in the lumen triggers automatic pullback recording of the OCT lens within the Dragonfly catheter at a pullback speed of 20 mm/s and a frame rate of 100 frames/s.[6]

OCT Image Analysis

All coronary angiography studies were reviewed for the presence or absence of aneurysm (with giant CAA being defined as a luminal diameter >8 mm), significant coronary artery stenosis (defined as luminal narrowing exceeding 50% in diameter), in stent-restenosis or dissection. Serial measurements of the intima and media were performed along the length of the coronary vessel by using the enabled semiautomatic calliper tool (St-Jude Medical, Inc) and the intima/

media ratio was then calculated. When disappearance of the media was observed, a value of 1 μm was given to allow calculation of the intima/media ratio. Cross-sectional frames containing artifacts or side branches comprising >25% of the image were excluded from the analysis. For segments with persistent CAA, measurements were made in sections adjacent to the CAA. In segments with regressed CAA, measurements were taken at previous CAA sites as well as adjacent sections. In addition, apparent normal sections were quantitatively analyzed. The images were also qualitatively analyzed for the presence of intimal hyperplasia, loss of normal layered structures of the vessel wall (disappearance of the tunica media), medial irregularities, calcifications, macrophage accumulation, white thrombi (platelet-rich), red thrombi (red blood cell–rich) and neovascularization, according to the Consensus standards for acquisition, measurement, and reporting of OCT studies.[5] The measurements of the intima and media were made by the cardiologist performing coronary angiography with OCT imaging (N.D.). Qualitative OCT image analysis was performed by 2 independent blinded interventional cardiologists trained in intra-coronary imaging (R.I., C.G.). For any disagreement in data evaluation between the readers, consensus agreement was achieved. Safety data, including radiation dose, radiation time, amount of contrast dye and nitroglycerine used were documented. Complications related to the procedure, including ischemic events, vasospasm, dissection of the coronary artery, cardiac arrhythmias, and coronary or catheter thrombosis, were reported.

Statistical Analysis

Quantitative variables were expressed as mean±SD or median (range), and categorical variables as frequencies or percentages. The Kolmogorov–Smirnov test was used to test for normal distribution. Comparison of affected and unaffected coronary segments was performed using Student t test for continuous variables with normal distributions and the Mann–Whitney U test for continuous variables with nonnormal distributions. Fisher exact or the χ^2 tests were used for categorical distribution comparison. All analyses were performed with SigmaStat V3.5 (Dundas Software, Germany). A 2-tailed P value of <0.05 was deemed significant.

Results

Study Population

The study comprised 18 patients (10 males, 55.6%) who underwent OCT imaging 9.0±5.1 years after onset of KD (Table 1). Patients were 3.4±2.9 years old at KD diagnosis, with a median of 5 (range 3 to 6) fulfilled diagnostic criteria of KD, with 7/18 (38.9%) having incomplete clinical criteria of

Table 1. Patient Characteristics at Onset of Kawasaki Disease

Basic Characteristics	Mean±SD	Median [Range]
Age at KD diagnosis, y	3.4±2.9	3.2 [0.2 to 10.5]
Male, n (%)	10 (55.6)	—
Diagnostic criteria fulfilled	4.7±1.1	5 [3 to 6]
Incomplete KD, n (%)	7 (38.9)	—
IVIG day from fever, days	9.9±5.2	9 [5 to 21]
On-time IVIG, n (%)	13 (72.2)	—
IVIG resistance, n (%)	6 (33.3)	—
CAA, n (%)	14 (77.8)	—
Giant CAA/CAA, n (%)	7 (50.0)	—

CAA indicates coronary artery aneurysm; IVIG, intravenous immunoglobulin; KD, Kawasaki disease.

KD. Patients received intravenous immunoglobulin treatment 9.9±5.2 days after the onset of fever, among whom 5/18 patients (27.8%) had a delayed therapy (treatment started >10 days after the onset of fever). CAA were present in 14/18 (77.8%) patients on follow up, with 7/14 (50%) having giant CAA, and 7/14 (50%) having regressed aneurysmal lesions at the time of OCT (Table 2). Patients' characteristics at the time of OCT are summarized in Table 3. Indications for

coronary angiography were routine follow-up in order to survey CAA progress (n=14) and evaluation of symptoms suggestive of ischemia (n=4); 3 patients with acute chest pain (among those, 2 patients with abnormal **Technetium (99mTc) sestamibi** (Tc-MIBI) myocardial perfusion scintigraphy, and 1 with a history of D-transposition of the great arteries corrected in the neonatal period), and 1 with fatigue and dyspnea on exercise.

Safety Measures

The estimated radiation dose of a complete examination including angiography and OCT imaging was 597.0±678.0 mGy, or 10.9±5.2 mGy/kg. Average fluoroscopy time was 18.6±6.2 minutes. Importantly, the safety data represent the entire angiography procedure, not only OCT imaging, including therapeutic procedures in some patients. Patients received a total contrast amount of 1.8±0.8 mL/kg. Intracoronary nitroglycerin was administered to all patients to prevent coronary artery spasm at a dose of 83.9±44.3 µg/kg (right coronary artery) and 93.0±46.7 µg/kg (left coronary artery), respectively. No major complications, such as ischemic event, coronary artery dissection, or arrhythmia occurred during the procedure. One patient had a transitory uneventful right coronary artery

Table 2. Detailed Patient Characteristics at Onset of Kawasaki Disease

Case No.	Age at KD Diagnosis (Y)	Sex	Diagnostic Criteria	IVIG Day From Fever (Days)	IVIG Resistance	CAA	Giant CAA
1	4.0	M	3	9.0	0	x	X
2	3.2	M	3	9.0	0	x	x
3	6.5	M	6	11.0	0	x	x
4	6.0	M	6	10.0	1	x	x
5	0.3	M	5	19.0	0	x	
6	4.4	F	3	7.0	0	x	
7	5.3	F	4	10.0	1	x	x
8	1.0	M	4	17.0	1	x	x
9	0.5	F	6	6.0	1	x	x
10	10.5	M	5	5.0	0		
11	0.9	M	5	5.0	1	x	
12	0.4	F	4	21.0	1	x	
13	1.5	M	5	17.0	0		
14	3.1	F	5	5.0	0		
15	6.0	F	6	5.0	0	x	
16	0.2	M	3	10.0	0	x	
17	7.1	F	5	5.0	0		
18	1.4	F	6	7.0	0	x	

CAA indicates coronary tortery aneurysm; IVIG, intravenous immunoglobulin; KD, Kawasaki disease.

Table 3. Patient Characteristics at the Time of OCT

Characteristics	Mean±SD	Median [Range]
Age, y	12.4±5.5	12.8 [3.5 to 21.0]
Weight, kg	50.5±29.0	52.3 [17.0 to 125.0]
Height, cm	147.7±23.6	155.1 [102.0 to 182.0]
Body surface area, m^2	1.41±0.5	1.51 [0.7 to 2.4]
Time from diagnosis, y	9.0±5.1	7.6 [2.8 to 18.1]
Abnormal echogram, n (%)	10 (55.6)	—
Abnormal angiography, n (%)	9 (50.0)	—

OCT indicates optical coherence tomography.

Table 5. Frequency of Abnormal OCT Findings

OCT Findings	n (%)
Intimal hyperplasia	15/18 (83.3)
Partial disappearance of media	13/18 (72.2)
Fibrosis	14/18 (77.8)
Calcifications	5/18 (27.8)
Macrophage accumulation	8/18 (44.4)
Neovascularization	6/18 (33.3)
White thrombus	3/18 (16.7)

OCT indicates optical coherence tomography.

vasospasm at the site of a regressed aneurysm where intimal hyperplasia with preserved media as well as fibrosis was observed on OCT. Safety data are summarized in Table 4.

Analysis and Interpretation of OCT Images

Cardiac catheterization and selective coronary angiography were performed including OCT of all 3 coronary vessels in 44.4% of patients, while 38.9% and 16.7% of patients underwent OCT imaging of 2 or 1 coronary arteries, respectively. A total of 42 coronary artery segments were evaluated. Significant coronary artery wall changes were identified in aneurysmal sites as well as angiographically normal segments (Table 5). Normal coronary artery wall structure was defined as a trilaminar, well-delineated pattern of the arterial wall with the intima being characterized by a highly backscattering signal-rich layer, the media by a homogeneous layer that has low backscattering, and the adventitia by highly backscattering layer (Figure 1). Intimal hyperplasia was the most frequent finding in our study cohort and was observed in 15 (83.3%)

Table 4. Safety Data During Procedure

Characteristics	Mean±SD	Median [Range]
X-ray exposure, mGy	597.0±678.0	409.0 [92 to 3136]
X-ray exposure, mGy/m^2	375.0±261.7	318.4 [110.1 to 1303.5]
X-ray exposure, mGy/kg	10.9±5.2	9.6 [4.4 to 25.0]
Fluoroscopy time, min	18.6±6.2	18.5 [7.7 to 31.6]
Contrast, mL	73.6±30.3	66.5 [31.6 to 154]
Contrast, mL/kg*	1.8±0.8	1.8 [0.4 to 3.3]
RCA nitroglycerin, μg	104.3±59.4	87.5 [35 to 200]
RCA nitroglycerin, μg/m^2	83.9±44.3	72.1 [31.5 to 208.1]
LCA nitroglycerin, μg	130.6±64.5	150 [25 to 250]
LCA nitroglycerin, μg/m^2	93.0±46.7	83.37 [29.1 to 260.1]

LCA indicates left coronary artery; RCA, right coronary artery.
*Includes ≤1 mL/kg (up to 40 mL) for left ventriculography.

patients (Figure 2). The thickness of the intima was significantly different between diseased and normal coronary artery segments (390.8±166.0 μm versus 61.7±17.0 μm, $P<0.001$). Eccentric intimal hyperplasia, defined as focal (as opposed to circumferential) intimal thickening, was observed in 52.4% of segments, while concentric intimal hyperplasia was seen in 47.6% of segments. Similarly, media thickness differed significantly between aneurysmal and angiographically normal segments (30.2±56.9 μm versus 61.4±16.7 μm, $P<0.001$). Consequently, the calculated intima/media ratio was statistically different between diseased and normal segments (320.4±236.2 versus 1.0±0.3, $P<0.001$) (Table 6).

Partial disappearance of the tunica media was found in 13/18 (72.2%) patients (Figure 3). Fibrosis was identified in 14/18 (77.8%) patients (Figure 4). Calcifications, and calcified nodules were detected in 5/18 (27.8%) patients (Figure 5). Light attenuation consistent with macrophage accumulation was found in 8/18 (44.4%) of patients (Figure 6). White thrombus was identified in 3/18 (16.7%) patients (Figure 7) and neovascularization in 2/18 (11.1%) patients (Figure 8), while no red thrombus could be seen. The above findings were found in both aneurysmal and angiographically normal coronary artery segments. However, pathological structural changes were observed more often in segments with persistent CAA (18/18, 100%), compared to 13/24 (54.2%) for angiographically normal segments (regressed CAA, and no history of CAA). Out of 3 cases of previous stent implantation (all of which were drug-eluting stents), 2 cases showed neointima proliferation measuring 80 to 160 μm, while in 1 case no proliferation was detected. Detailed imaging results are presented in Table 7.

Discussion

Our study shows significant structural changes of the arterial wall in patients with KD as assessed by OCT imaging. The most frequent finding in this population was intimal
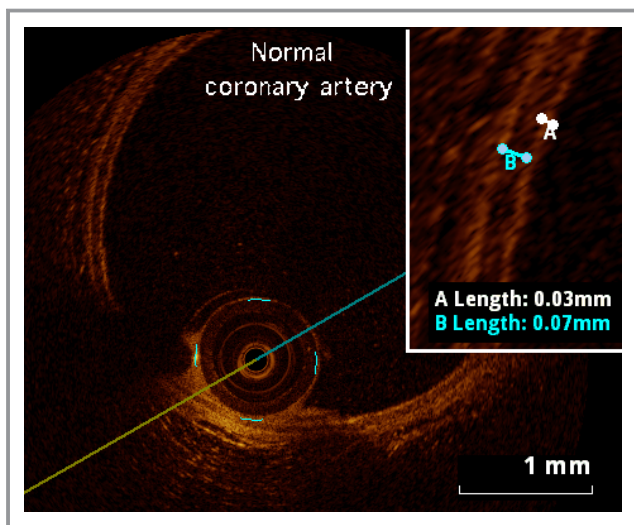
Figure 1. Normal coronary artery with intima (white measure) and media (blue measure) being well delineated and of normal thickness.

hyperplasia, followed by fibrotic vessel wall changes, partial disappearance of media, macrophage accumulation, and calcifications. Interestingly, these structural arterial wall changes were not only seen at aneurysmal sites, but also in coronary artery segments that appeared angiographically normal, suggesting that OCT imaging may add incremental diagnostic value to conventional imaging techniques in
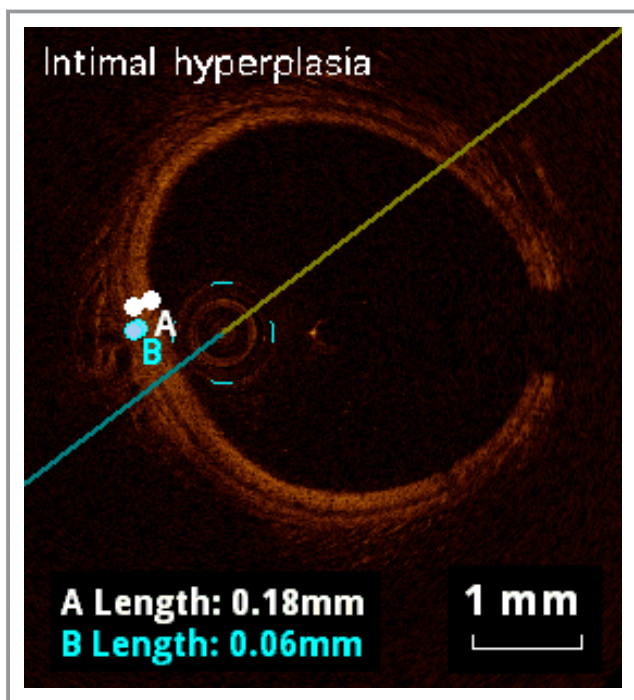

Figure 2. Concentric intimal hyperplasia (white measure) with preserved 3-layered structure of the vessel wall.

patients with KD. Furthermore, OCT imaging proved to be feasible and safe in our pediatric population.

The findings seen by OCT imaging in the present study are consistent with histopathologic observations after KD. The acute stage is characterized by necrotizing arteritis, responsible for saccular aneurysm formation at risk of rupture or thrombosis. The subacute/chronic phase is marked by an inflammatory process associated with luminal fibroblastic proliferation, with destruction of the internal elastic lamina, external elastic lamina and media in the most severe cases. The chronic phase is marked by intraluminal stenosing caused by myofibroblast proliferation, with progressive luminal narrowing and intimal hyperplasia (Figure 9).[8]

The observation that striking changes, in particular intimal hyperplasia, are observed in coronary artery segments where aneurysmal dilatation had regressed on angiography are in line with 2 previous case reports.[7,9] The first series included KD and post-transplant pediatric patients. It focused on feasibility and safety, describing 4 KD patients with normal concurrent angiography and 1 with mild irregularities of the proximal right coronary artery. Their qualitative findings were comparable to ours, with intimal hyperplasia, absence of media, thrombosis, calcification, and neovascularization being reported.[7] Kakimoto et al in 2014 described 2 adolescents with a history of KD in whom OCT showed intimal hyperplasia and disruption of the media, not only on segments with CAA but also on normal angiographic segments.[9] OCT was also used for stent positioning guidance in a patient with a severely calcified coronary plaque presenting with acute coronary syndrome and a history of KD coronary artery complications.[10]

Our findings are of particular importance when considering that these persistent vascular wall abnormalities, which are not evident on angiography, may contribute to the increased risk of myocardial ischemia observed in young adults who suffered from KD earlier in life.[11–14] Another important perspective is the fact that there are no current guidelines for the evaluation and treatment of adult patients who had KD. Thus, identifying more precisely a population at risk may have the potential to tailor treatment and improve outcomes. Furthermore, OCT imaging may help to choose the proximal and distal stent landing zones during percutaneous interventions, owing to a better depiction of coronary wall abnormalities in KD patients that cannot be readily appreciated on coronary angiography. Of note, however, the retrospective design of our study does not allow addressing the prognostic value of our findings, which needs to be assessed by a prospective study with adequate power.

Furthermore, the pathophysiological relevance of our findings cannot be estimated by the current study since histopathological correlates are lacking. Intimal thickening or

Table 6. Quantitative Analysis of OCT Data

	Mean±SD	Median [Range]
Normal segments		
Intima thickness, µm*	61.7±17.0	60.0 [40.0 to 110.0]
Media thickness, µm*	61.4±16.7	60.0 [40.0 to 111.0]
Intima/media ratio*	1.0±0.3	1.0 [0.7 to 2.5]
Diseased segments, RCA		
Intima, µm*	347.1±173.4	315.0 [140.0 to 670.0]
Media, µm	63.4±77.3	55.0 [1.0 to 270.0]
Intima/media*	190.2±253.2	4.4 [1.2 to 670.0]
Diseased segments, LAD		
Intima, µm*	435.0±158.1	455.0 [150.0 to 810.0]
Media, µm*	14.2±31.6	1.0 [1.0 to 100.0]
Intima/media*†	403.4±209.7	440.0 [2.0 to 810.0]
Diseased segments, LCx		
Intima, µm*	360.0±165.5	360.0 [150.0 to 640.0]
Media, µm*	1.0±0.0	1.0 [1.0 to 1.0]
Intima/media*†	360.0±165.5	360.0 [150.0 to 640.0]

LAD indicates left anterior descending artery; LCx, left circumflex artery; OCT, optical coherence tomography; RCA, right coronary artery.
*P<0.001 diseased vs normal segments.
†Mann–Whitney U test.

Figure 4. Intimal hyperplasia (white measure) with medial irregularities most likely due to fibrosis (red arrow) with diffuse medial thickening.

hyperplasia on OCT may be the result of destruction of the internal elastic layer, proliferation of vascular smooth-muscle cells, and fibrous tissue in the intima following the inflammatory process of KD. Macrophage accumulation, however, seen in 44.4% of our study population, points towards an inflammatory process and raises the question whether (and for how long) the inflammatory process outlasts the acute arteritis phase and the development of aneurysms in KD. Notably, previous histopathology studies provided conflicting results regarding macrophage involvement in the late disease process of KD,[15,16] and OCT imaging may help to clarify the natural course of inflammatory coronary artery changes in KD. Whether the vasculopathy observed after KD is a distinct
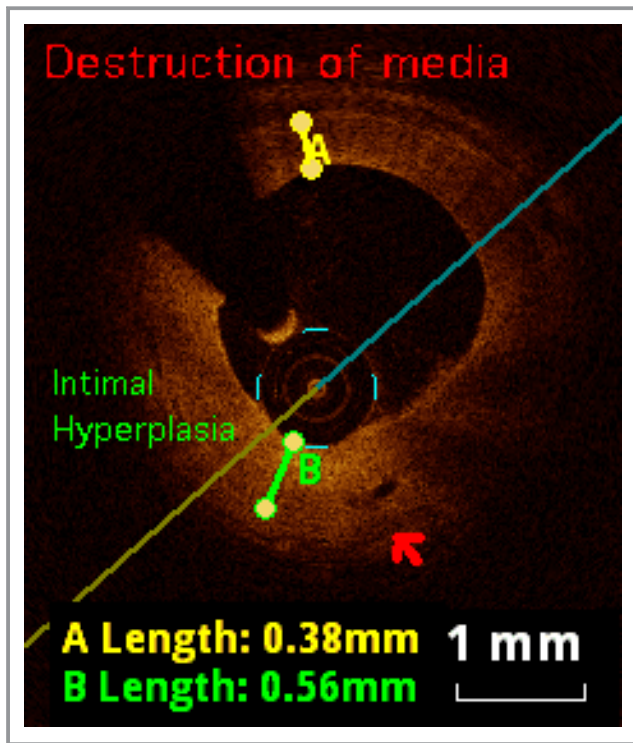

Figure 3. Severe concentric intimal thickening (yellow and green measures) with loss of the normal structure of the vessel wall, disappearance of media (red arrow), and nonobstructive luminal narrowing.
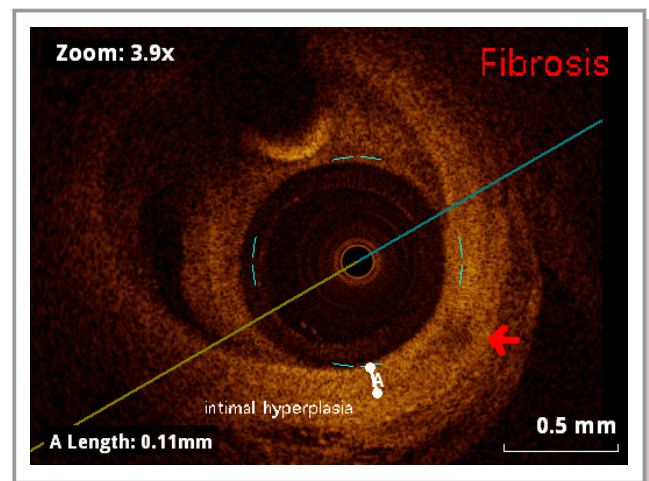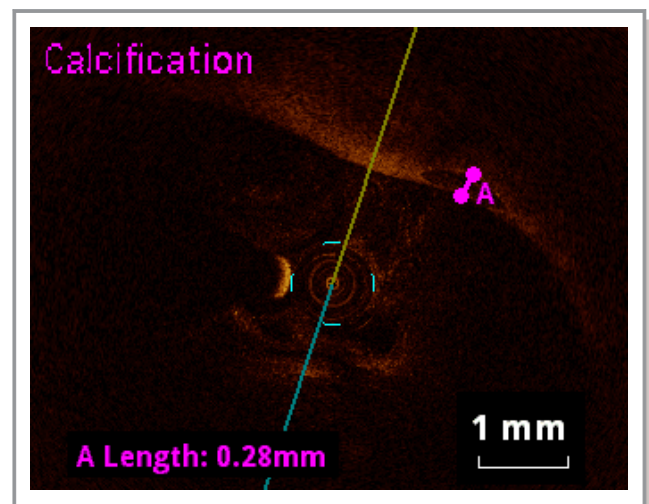

Figure 5. Calcified nodule (pink measure), characterized by a well-demarcated border and heterogeneous, low-signal composition.
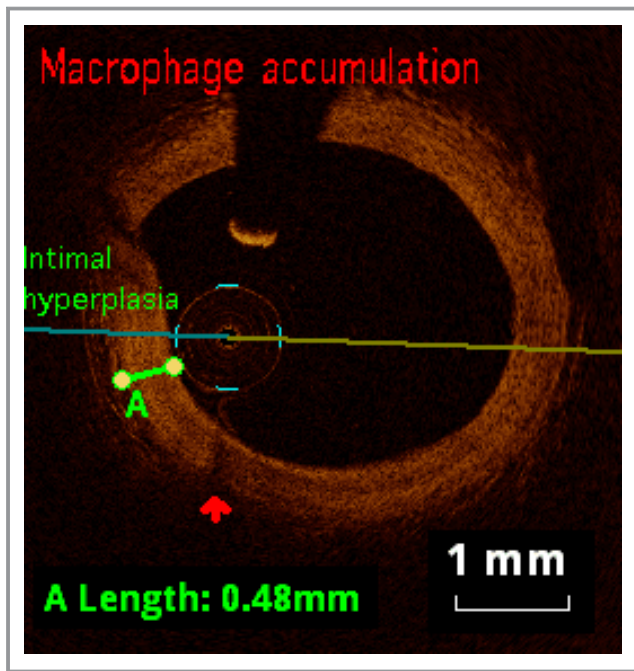
Figure 6. Intimal hyperplasia (green measure) with macrophage accumulation (red arrow): signal-rich band, strongly light-attenuating and causing an underlying signal-poor region.

process or accelerated atherosclerosis has been controversially discussed in the past. Importantly, in line with previous histopathological studies, no typical features of
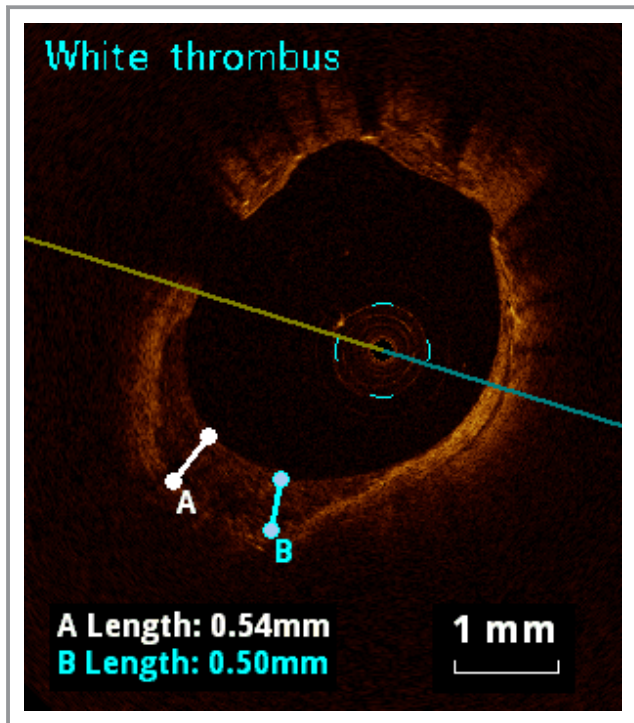

Figure 7. White thrombus (white and blue measures), characterized by a signal-rich, irregular mass protruding into the lumen with mild light attenuation (as opposed to red thrombus).
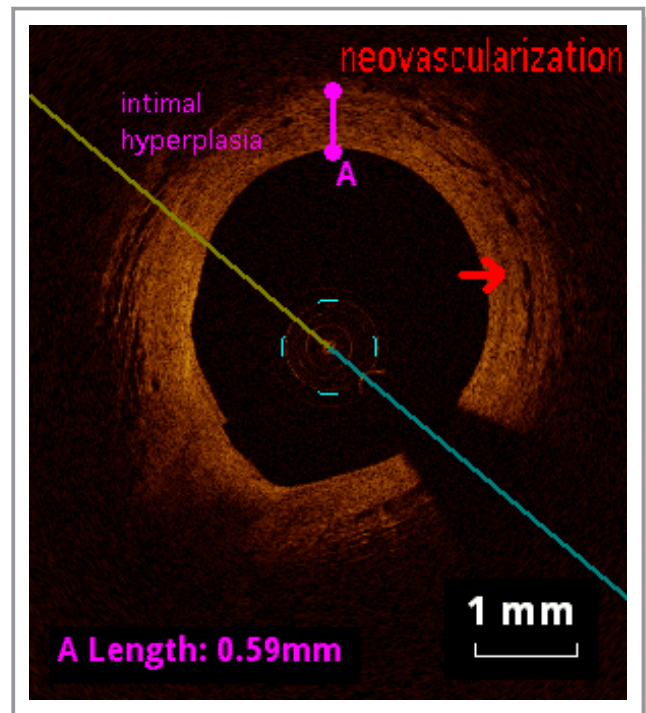

Figure 8. Intimal hyperplasia (pink measure) with ellipsoid structures of varying size with a dark appearance suggesting microvessels, a typical picture seen in neovascularization (red arrow).

atherosclerosis, such as necrotic cores or lipid pools, have been identified in patients after KD in our study.[11,15,16]

OCT cannot image through blood because it attenuates the infrared light before reaching the arterial wall, hence the need for blood clearance before acquisition of images. In the early stages of OCT development, blood clearance was performed with an occlusive balloon installed proximally.[6] Lately, OCT technology does not require coronary artery occlusion. Instead, nonocclusive flush with contrast dye is detected by the lens and pullback is triggered automatically.[6,17,18] While the risks of OCT imaging have been well characterized in the early stages of its utilization in the adults, transient chest pain (47.6% of patients), transient ECG changes (45.5% of patients), and ventricular fibrillation were mostly associated with the coronary balloon occlusion technique for lumen clearance. Other associated complications included deep guide catheter intubation (0.6%), air embolism (0.6%), and coronary artery dissection (0.2%).[19] In the current era of OCT imaging, proximal balloon occlusion is no longer in use due to procedural ischemic events.[17] The nonocclusive clearance technique, applied to our series, is rarely a cause of major complications such as death, myocardial infarction, emergency revascularization, embolization, life-threatening arrhythmia, coronary dissection, prolonged and severe vessel spasm, and contrast-induced nephropathy.[20] In our series, only 1 patient had a transient uneventful vasospasm, most likely

Table 7. Summary of OCT Imaging Results

Case No.	CAA	Giant CAA	Intimal Hyperplasia	Destroyed Media	Fibrosis	Calcification	Macrophage Infiltration	White Thrombus	Neovascularization
1	x	x	x	x			x		x
2	x	x	x	x	X				x
3	x	x	x	x	X	x	x	x	
4	x	x	x	x	X	x		x	
5	x		x	x	x				x
6	x		x	x	x				
7	x	x	x	x	x	x	x		
8	x	x	x	x	x	x	x		x
9	x	x	x	x	x	x	x	x	x
10					x		x		
11	x		x	x	x		x		x
12	x		x		x		x		
13									
14			x	x	x				
15	x		x	x					
16	x		x		x				
17									
18	x		x	x	x				

CAA indicates coronary artery aneurysm; OCT, optical coherence tomography.

owing to the fact that the OCT technique (eg, hand injection of contrast dye instead of power injection) and material (small catheter size) were adapted for appropriate use in children. The presence of mural thrombi by angiography was not considered an absolute exclusion criterion for the OCT procedure, and the decision to perform OCT was left to the discretion of the interventional cardiologist (N.D.) considering the risk of distal embolization. Nevertheless, at present data regarding safety measures of OCT imaging in a pediatric population are lacking and will have to be provided by adequately powered studies.

Our study has limitations, which have to be taken into account. First, the most important limitation of this study is the absence of comparison to the "gold standard" of histopathology. This is due to the rarity of preserved cardiac specimen. Nonetheless, our findings are consistent with histopathological specimen reports from patients with KD. Second, the penetration depth of OCT amounts only 0.1 to 2 mm and therefore limits the analysis of giant CAA.[6] Third, the prognostic value of our findings and the course of CAA as evaluated by OCT cannot be assessed by the present study due to its retrospective design and its small population.
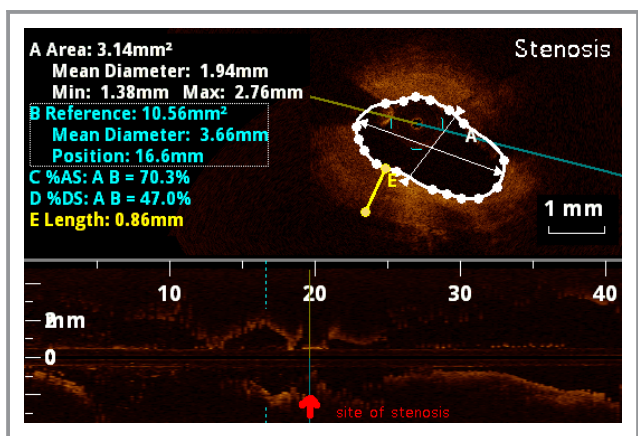

Figure 9. Coronary artery showing severe intimal hyperplasia (yellow measure) with resulting stenosis (red arrow).

Conclusions

In our experience, OCT proved to be safe, providing new insights in the setting of KD. The observed coronary structural changes correspond to published histological findings. These findings should be further validated in larger cohort studies, and if possible with pathologic correlation.

Acknowledgments

Disclosures

None.

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