JACC: ADVANCES VOL. 1, NO. 4, 2022

ORIGINAL RESEARCH

CONGENITAL HEART DISEASE

Dyslipidemia Among Adults With Congenital Heart Disease

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ABSTRACT

BACKGROUND Atherosclerotic disease is an important cause of morbidity among adults with congenital heart disease (CHD). Prevalence of dyslipidemia in this group is poorly described.

OBJECTIVES This study aimed to describe the prevalence of dyslipidemia among adults with CHD.

METHODS A prospective, outpatient screening study was conducted among adults aged ≥18 years at 4 New England ambulatory congenital cardiology centers. Participants were surveyed regarding cardiovascular risk factors. Nonfasting fingerstick samples were obtained for analysis using a point-of-care lipid analyzer.

RESULTS Lipid screening was completed on 186 participants (median age 30 [range 18-71] years, 50% female). Eighteen (10%) had simple CHD anatomy, and 63 (34%) had complex anatomy. Only 15% of 169 respondents reported history of high cholesterol. Eighty-five (46%) participants met National Cholesterol Education Program definition of dyslipidemia with 60 (32%), 62 (34%), and 37 (20%) having low high-density lipoprotein cholesterol (HDL-C <40 mg/dL), high non-HDL-C (≥130 mg/dL), and high total cholesterol (TC ≥200 mg/dL), respectively. TC was higher among participants with simple CHD than among those with moderate and complex lesions (mean 178.4 ± 48.7 vs 170.1 ± 35.0 vs 157.6 ± 34.5 mg/dL; $P = 0.03$). HDL-C was lower among participants with complex CHD than among those with simple and moderate lesions (mean 44.1 ± 13.5 vs 46.9 ± 12.5 vs 49.8 ± 15.3 mg/dL; $P = 0.05$).

CONCLUSIONS Dyslipidemia is highly prevalent among our cohort of adults with CHD, despite <15% reporting a prior diagnosis. Low HDL-C was more common in complex CHD, and high TC was more common in simple or moderate CHD. Lipid screening should be part of preventive health maintenance for all adults with CHD. (JACC Adv 2022;1:100081)

Advances in medical and surgical care have resulted in a nearly 60% decrease in childhood mortality related to congenital heart disease (CHD) since 1987, with the most dramatic improvement being seen in complex CHD.[1] However, although more than 90% of children born with CHD are now expected to reach adulthood, the mortality rate among adults with CHD is still roughly twice

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received March 22, 2022; revised manuscript received June 14, 2022, accepted June 29, 2022.

ISSN 2772-963X https://doi.org/10.1016/j.jacadv.2022.100081

ABBREVIATIONS AND ACRONYMS

ACHD = adult congenital heart disease

ASCVD = atherosclerotic cardiovascular disease

BMI = body mass index

CHD = congenital heart disease

HDL-C = high-density lipoprotein cholesterol

LDL-C = low density lipoprotein cholesterol

NECCA = New England Congenital Cardiology Association

TC = total cholesterol

that of their peers.[2] The predominant cause of death has shifted over time as well. Before 1990, the most common cause of death among adults with noncyanotic CHD was arrhythmia; since then, myocardial infarction has emerged as the leading cause of mortality.[3]

Studies focusing on atherosclerotic cardiovascular disease (ASCVD) risk factors among the adult congenital heart disease (ACHD) population have shown a higher prevalence of hypertension, obesity, and diabetes compared with age-matched controls.[4,5] The rates of dyslipidemia in ACHD are less well described. On the Gran Canaria Island of Spain, a heterogeneous group of 818 adolescents and adults with CHD was found to have lower levels of low-density lipoprotein cholesterol (LDL-C) (92 vs 104 mg/dL; $P < 0.001$) and triglycerides (84 vs 88 mg/dL; $P = 0.014$) but also lower levels of protective high-density lipoprotein cholesterol (HDL-C; 49 vs 52 mg/dL; $P < 0.001$) compared with age- and sex-matched controls.[6] A Korean study of 135 adults with CHD found that although patients with surgically corrected CHD had significantly higher total cholesterol (TC) (193 vs 185 mg/dL; $P < 0.001$), LDL-C (118 vs 104 mg/dL; $P < 0.001$), and triglycerides (122 vs 112 mg/dL; $P < 0.001$) and lower HDL-C (53 vs 59 mg/dL; $P < 0.001$) compared with age-, sex-, and body mass index (BMI)-matched controls, cyanotic patients (oxygen saturation <93%) had lower lipid values across the board.[7] Regardless, hyperlipidemia remains a strong predictor of ASCVD in adults with CHD, yet fewer patients with CHD are appropriately prescribed statins for primary prevention compared with non-CHD controls.[8,9]

Given the persisting gap in knowledge regarding the prevalence of atherosclerotic risk factors, the relative impact on the health of adults with CHD, and the best management approach, the New England Congenital Cardiology Association (NECCA) sought to describe the burden of atherosclerotic risk in CHD through a pilot screening study for dyslipidemia among adults with CHD. Better descriptive information about the epidemiology of atherosclerotic risk factors in CHD could promote understanding of the relevant physiology, increase awareness and screening by CHD providers, and ultimately support targeted anticipatory guidance and interventions to decrease atherosclerotic morbidity and mortality of this growing patient population.

TABLE 1 Congenital Heart Disease Anatomy by Category

Simple (n = 18)	
Ventricular septal defect	11 (61.1)
Atrial septal defect	4 (22.2)
Patent ductus arteriosus	2 (11.1)
Pulmonary stenosis	1 (5.6)
Moderate (n = 104)	
Tetralogy of Fallot	30 (28.7)
Bicommissural aortic valve	27 (26.0)
Pulmonary stenosis	12 (11.5)
Coarctation of the aorta	8 (7.7)
Atrioventricular septal defect	6 (5.8)
Anomalous coronary artery	5 (4.8)
Subvalvar aortic stenosis	4 (3.8)
Ebstein anomaly	3 (2.9)
Peripheral pulmonary stenosis	2 (1.9)
Anomalous pulmonary veins	2 (1.9)
Ventricular septal defect	2 (1.9)
Complex (n = 63)	
Transposition of the great arteries	25 (39.7)
Fontan procedure	23 (36.5)
Pulmonary atresia	11 (17.5)
Double-outlet right ventricle	2 (3.2)
Cyanotic congenital heart disease, unrepaired	1 (1.6)
Heterotaxy	1 (1.6)

Values are n (%).

METHODS

STUDY DESIGN. This was a prospective, cross-sectional screening study conducted across 4 ambulatory congenital cardiology centers within NECCA, an organization of academic and community-based practices representing all 6 New England states formed in 2009 with a mission to improve the quality, safety, effectiveness, availability, and cost of care for children and adults with congenital or acquired heart disease of childhood. Eligible participants were those aged ≥18 years with CHD being seen in consultation or follow-up at any of the 4 participating outpatient centers: Maine Medical Center (Portland, ME); Boston Children's Hospital (Boston, MA); Patricia Rompf, MD, Inc (Providence, RI); or University of Vermont Medical Center (Burlington, VT). Individuals with impaired decision-making capabilities as determined by their primary ACHD cardiologist were excluded. Individuals who were pregnant or who had had recent myocardial infarction (<3 months) and/or fever within the preceding 2 weeks were also excluded, as these conditions are known to transiently alter lipid levels.

Eligible participants were identified in advance of their appointments by research staff between November 2013 and January 2018. Primary ACHD cardiologists were given the opportunity to request that their patients not be approached. Otherwise, eligible individuals were notified by a known health care provider (eg, a clinic nurse) either while they were waiting for their appointment or immediately after the visit that they were eligible for a research study. A member of the research team was then summoned to speak with those individuals expressing interest in the study. All discussion, obtaining of consent, and blood drawing were conducted in a private room.

In accordance with the device manufacturer's instructions, a fingerstick sample of capillary blood (>10 μL) was obtained from each participant. Participants were not required to be fasting. Each sample was immediately analyzed for TC, HDL-C, triglycerides, and LDL-C using a point-of-care Cholestech LDX analyzer (Abbott), a Clinical Laboratory Improvements Amendments-waived device. While waiting for the results to be reported, participants completed a 13-question survey focusing on cardiovascular risk factors: cholesterol, diabetes, blood pressure, tobacco exposure, family history, and activity level (Supplemental Appendix). On completion of the survey, the participant received the results of the cholesterol screen and additional patient information handouts from the American Heart Association (AHA) explaining the test results and providing guidance on maintaining a healthy lifestyle. The participant's clinic records from the day of the visit were reviewed, and the following clinical data were recorded: age, sex, CHD diagnosis, BMI, blood pressure, and, when available, oxygen saturation. CHD diagnosis was further categorized according to anatomic classification as detailed in Table 4 of the 2018 AHA/ACC Guideline for the Management of Adults with Congenital Heart Disease.[10] The institutional review boards at Boston Children's Hospital and at each participating NECCA site approved this protocol (IRB-P00007537). Written informed consent was obtained from each patient.

VALIDATION COMPONENT. Before enrolling participants outside of Boston Children's Hospital, a validation study was performed using the Cholestech LDX analyzer with adults with CHD. An initial subset of 33 participants enrolled from Boston Children's Hospital underwent venipuncture for lipid analysis at the same time as fingerstick point-of-care cholesterol screening to assess performance of the Cholestech LDX in the presence of CHD.

TABLE 2 Dyslipidemia by CHD Complexity

	Simple (n = 18)	Moderate (n = 104)	Complex (n = 63)
TC ≥200 mg/dL	7 (38.9)	23 (21.2)	7 (11.1)
HDL-C <40 mg/dL	5 (27.8)	26 (25.0)	29 (46.0)
Non-HDL-C ≥130 mg/dL	8 (44.4)	38 (36.5)	16 (25.4)
Triglyceride ≥150 mg/dL	8 (44.4)	37 (35.6)	18 (28.6)
LDL-C ≥130 mg/dL	6 (33.3)	12 (11.5)	5 (7.9)

Values are n (%).
CHD = congenital heart disease; HDL-C = high-density lipoprotein cholesterol; LDL-C = high-density lipoprotein cholesterol; TC = total cholesterol.

STATISTICAL ANALYSIS. For the purposes of statistical analysis, "dyslipidemia" was defined as TC ≥200 mg/dL, HDL-C <40 mg/dL, or non-HDL ≥130 mg/dL. Group characteristics are presented with summary statistics as means and standard deviations. Prevalence is presented as proportions with 95% CIs with additional subdivision based on AHA/ACC anatomic classification. The Kruskal-Wallis test and Wilcoxon signed rank test were used for comparing lipid values between categories of CHD complexity. For the validation study, Wilcoxon signed rank test and Bland-Altman analysis[11] were used for comparing values between testing modalities. The Spearman correlation and Spearman's test were used to assess agreement between the Cholestech LDX analyzer and core values. A "strong" correlation coefficient is defined as ≥0.8, "moderate" as 0.6 to 0.7, "fair" as 0.3 to 0.5, and "poor" as <0.2. The analysis was performed using R 4.0 (R Core Team 2020, R Foundation for Statistical Computing).

RESULTS

DEMOGRAPHICS AND BASELINE CHARACTERISTICS. There were 186 participants recruited across the 4 New England sites: Maine Medical Center (Maine), n = 56; Boston Children's Hospital (Massachusetts), n = 60; Patricia Rompf, MD, Inc. (Rhode Island), n = 49; and University of Vermont Medical Center (Vermont), n = 21. One participant from Boston Children's Hospital was excluded from the analysis due to self-reported fever. In the remaining 185 participants (92 [50%] female, age 34 ± 13 years, BMI 27 ± 6 kg/m^2, 45 [24%] obese, systolic blood pressure 119 ± 13 mm Hg, diastolic blood pressure 69 ± 10 mm Hg), CHD anatomy complexity was classified as simple for 18 (10%) participants, moderate for 104 (56%) participants, and complex for 63 (34%)

TABLE 3 Characteristics of Participants by CHD Complexity (n = 185)

	Simple		Moderate		Complex		
	Median	IQR	Median	IQR	Median	IQR	*P* Value
Demographic factors							
Age (y)	25.5	16.8	30.0	21.1	32.0	16.5	0.57
Height (cm)	162.3	16.9	167.8	14.0	165.7	11.3	0.14
Weight (kg)	78.0	22.6	73.1	25.4	70.6	17.2	0.21
BMI (kg/m^2)	27.4	11.3	25.7	7.4	25.6	6.6	0.37
SBP (mm Hg)	123.4	14.8	120.5	13.0	115.0	15.5	0.002
DBP (mm Hg)	74.0	9.5	71.0	14.0	66.0	13.0	0.003
Lipids							
TC (mg/dL)	162.5	88.8	171.0	54.5	155.0	40.5	0.03
HDL-C (mg/dL)	44.5	16.0	48.5	17.5	41.0	16.5	0.02
Triglyceride (mg/dL)	133.0	118.0	116.5	90.5	111.0	103.0	0.52
LDL-C (mg/dL)	107.0	67.3	94.5	42.5	88.5	33.8	0.24
Non-HDL-C (mg/dL)	121.5	78.3	118.5	45.8	109.0	39.5	0.15
TC/HDL-C ratio	3.7	1.9	3.4	1.6	3.5	1.2	0.60

BMI = body mass index; CHD = congenital heart disease; DBP = diastolic blood pressure; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; SBP = systolic blood pressure; TC = total cholesterol.

participants (Table 1). Of those who responded to the survey question (n = 156, 84%), 68% recalled having their cholesterol levels checked previously. Only 15% of respondents reported a known history of high cholesterol or triglycerides.

VALIDATION OF POINT-OF-CARE CHOLESTEROL SCREENING. Thirty-three of the Boston Children's Hospital participants (46% female) were enrolled in the validation study of the Cholestech LDX. The median age among this subset was 33 years (range 21-69 years). The median BMI was 26.2 kg/m^2 (range 19.2-52.2 kg/m^2), and the median blood pressure was 121/71 (95-148/52-86) mm Hg. The mean fingerstick lipid values from the Cholestech LDX point-of-care analyzer are generally lower than those from venipuncture (Supplemental Table 1). The lipid values are all highly correlated and statistically significant (TC 0.92, HDL 0.92, triglyceride 0.94, LDL 0.91, non-HDL 0.86, and TC/HDL ratio 0.94, all $P < 0.05$). Overall, the correlation between fingerstick lipid measurements and lipid measurements from venipuncture samples was within published international standards (Supplemental Figure 1A).[12,13]

SCREENING CHOLESTEROL VALUES FOR ADULTS WITH CHD. Eighty-five of the 185 (46%) participants met the National Cholesterol Education Program definition of dyslipidemia on nonfasting screening, with HDL-C <40 mg/dL found in 60 (32%) participants, non-HDL-C ≥130 mg/dL in 48 (26%) participants, and TC ≥200 mg/dL in 37 (20%) participants (Table 2).

In the overall study sample, the median (IQR) TC, HDL-C, non-HDL-C, and LDL-C was 162 (53) mg/dL, 46 (18) mg/dL, 116.5 (47.5) mg/dL, and 92 (44.5) mg/dL, respectively. There was a statistically significant difference in TC among the groups ($P = 0.03$); the median TC was highest in the simple CHD group and lowest in the complex CHD group. HDL-C was also statistically significant among the groups ($P = 0.02$); the median HDL-C was significantly higher in the moderate CHD group compared with the complex CHD group ($P = 0.02$ for pairwise comparison), but differences between the simple CHD group and moderate CHD group and between the simple CHD group and complex CHD group were not statistically significant. There was no significant difference in LDL-C, non-HDL-C, or triglycerides between CHD groups (Table 3).

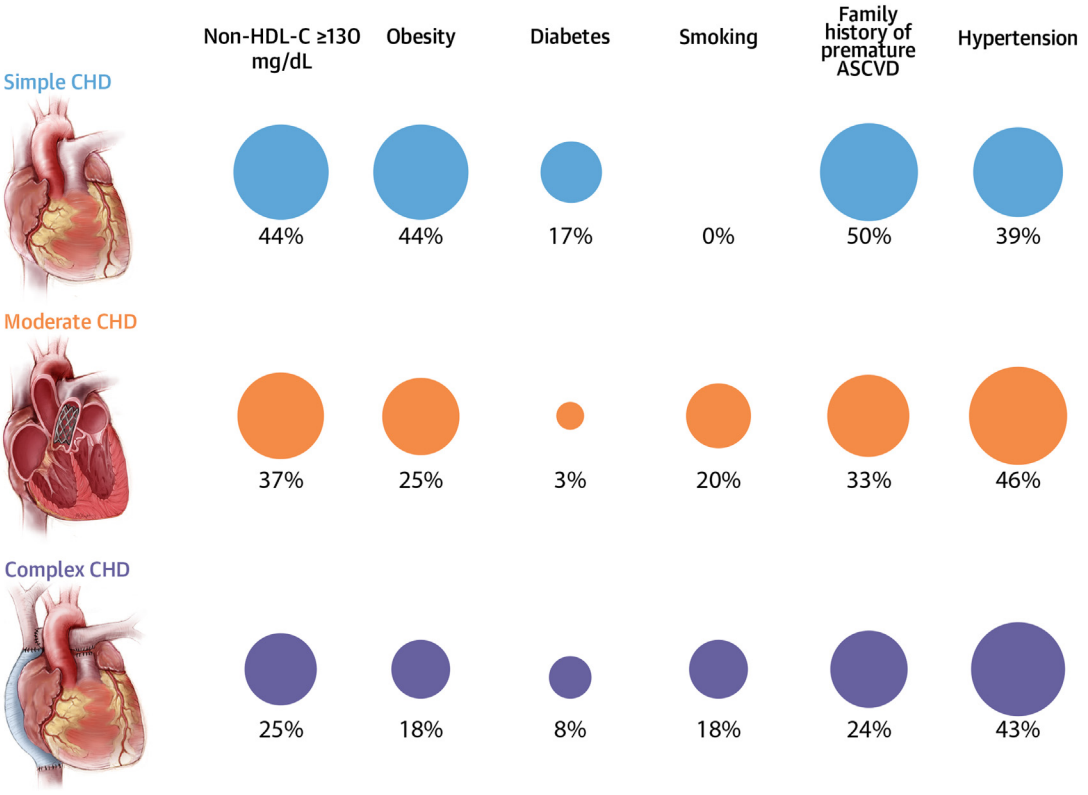
In multivariable models adjusted for age, sex, and BMI, we examined the associations between CHD group and each lipid parameter separately to determine if the difference in lipid parameters between CHD complexity groups was due to differences in age, sex, and BMI. TC remained lower in the complex CHD group in the multivariable-adjusted model compared with the simple CHD group by 20.6 mg/dL on average ($P = 0.03$). Non-HDL-C was significantly lower in the complex CHD group vs the simple CHD group by 19.3 mg/dL on average ($P = 0.03$) (Supplemental Table 2).

CHOLESTEROL SCREENING VALUES AND OTHER REPORTED CARDIOVASCULAR RISK FACTORS. As expected, participants with dyslipidemia (TC ≥200 mg/dL and/or HDL-C <40 mg/dL) were more likely to be obese compared with those without dyslipidemia (35% vs 15%; $P = 0.002$). Sex, CHD complexity, diabetes, tobacco use, family history of ASCVD, reported physical activity, and blood pressure (Central Illustration) did not differ significantly between those with and without dyslipidemia.

SELF-REPORTED CARDIOVASCULAR HEALTH BEHAVIORS. Modifiable cardiovascular health behaviors were also high in this population of adults with CHD. Twenty percent of adults with moderate CHD and 18% with complex CHD reported current cigarette smoking. The number reporting regular physical activity was also low, with fewer than one-half of the participants with moderate CHD and complex CHD reporting 5 or more hours of moderate physical activity per week (Table 4).

OXYGEN SATURATION AND CHOLESTEROL SCREENING VALUES AMONG ADULTS WITH CHD. Finally, we examined the relationship between oxygen saturation and lipid measurements. Of the 185 participants, oxygen saturation data were available for 87 participants (53% female, 41% with

CENTRAL ILLUSTRATION **Prevalence of Abnormal Lipids and Nonlipid ASCVD Risk Factors According to AHA/ACC CHD Anatomic Classification**



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ASCVD = atherosclerotic cardiovascular disease; CHD = congenital heart disease.

dyslipidemia). The median oxygen saturation for the group was 99% (range 80%-100%, IQR: 97%-100%). Oxygen saturation was <92% in 3 participants and <90% in only 2 participants. HDL-C was higher among participants with higher oxygen saturation (correlation coefficient 0.30; $P < 0.01$). The correlation between oxygen saturation and TC was not statistically significant (correlation coefficient 0.21; $P = 0.051$) (Supplemental Figure 2).

DISCUSSION

This prospective outpatient lipid screening study found high cholesterol values in almost one-half of the study sample, despite only 15% of participants reporting previous abnormal cholesterol values. Nonideal modifiable cardiovascular health behaviors were also highly prevalent. Surprisingly, ~20% of adults with complex CHD reported current cigarette smoking, and regular physical activity was reported to be low across all CHD complexity groups.

There were significant differences in screening cholesterol values across CHD complexity group. Dyslipidemia was predominantly due to high TC among adults with simple and moderate CHD lesions and due to low HDL-C among adults with complex CHD. Elevated non-HDL-C was also commonly found, with non-HDL-C approximately 20 mg/dL higher among adults with simple defects compared with complex CHD in age-, sex-, and BMI-adjusted models. In general, the differences in abnormal lipid values were not completely explained by differences in age, sex, or BMI across complexity groups. Compared with the general population, adults with CHD had a lower prevalence of abnormal TC and abnormal LDL-C but a higher prevalence of abnormal HDL-C.[14] Finally, we found a positive correlation between oxygen saturation and both TC and HDL-C that was driven by 2 cyanotic patients with Fontan physiology.

TABLE 4 CHD Complexity and Self-Reported Risk Factors

	Simple	Moderate	Complex	*P* Value
Obesity	8 (44.4)	26 (25.0)	11 (17.5)	0.07[a]
Diabetes	3 (16.7)	3 (2.9)	5 (7.9)	0.04[a]
Smoking	0	21 (20.2)	11 (17.5)	0.09[a]
Family history	13 (72.2)	55 (52.9)	33 (52.4)	0.29[b]
Premature cardiovascular disease	9 (50.0)	34 (32.7)	15 (23.8)	0.10[b]
Regular physical activity	12 (66.7)	48 (46.2)	30 (47.6)	0.27[b]
Hypertension	7 (38.9)	48 (46.2)	27 (42.9)	0.81[b]

Values are n (%). [a]Using Fisher exact test due to small number of patients. [b]Using Pearson's chi-square test.

As the number and life expectancy of adults living with CHD continue to increase, cardiologists caring for ACHD patients have had to shift their clinical focus from the sequelae of congenital heart defects and their associated surgeries to also include acquired ASCVD. However, studies provide conflicting data on the relative frequency of dyslipidemia, probably due in part to the heterogeneous nature of the ACHD population. It was for this reason that we chose to study the relative rates of dyslipidemia among ACHD patients in the context of the Anatomic and Physiological classification system proposed in the 2018 AHA/ACC Guideline for the Management of Adults with Congenital Heart Disease.[10]

Our data suggest differing dyslipidemia risk among patients with CHD may be related to differing levels of obesity, which may also underlie some of the higher rates of hypertension and diabetes seen in ACHD. The rates of obesity and overweight are, in general, as high in children with CHD, as they are in the general population.[15] A shift from neonatal and early childhood underweight to adolescent overweight has been described in a cohort with CHD.[16] These types of changes are associated with increased risk for sustained obesity in non-CHD populations. As many as 59.5% of ACHD patients were classified as overweight to morbidly obese in 1 recent study.[17] The root of obesity in this population is likely multifactorial; however, exercise restriction has been shown to be an important predictor.[18] Programs to promote greater physical activity among both children and adults with CHD may play an important role in reducing ASCVD risk later in life.

Among individuals with complex forms of CHD, previous studies have shown that both cyanotic patients and those with Fontan physiology have lower lipid levels, although the difference is driven primarily by low HDL-C levels.[19-22] This may also be related in part to exercise restriction and generally low levels of physical activity. Chronic inflammation could also explain the lower HDL-C levels in complex CHD. In chronic inflammatory disorders such as systemic lupus erythematosus and rheumatoid arthritis, inflammatory marker levels correlate inversely with HDL-C.[23] As studies have demonstrated elevated markers of systemic inflammation in Fontan circulation, transposition of the great arteries with systemic right ventricle, and cyanotic heart disease, similar mechanisms could be causing the lower HDL-C observed in these patients.[24] Finally, at least among those with Fontan circulation, lower lipid levels could reflect altered liver metabolism resulting from lifelong circulatory derangements.[19,22]

It must be stated that while the ultimate goal of understanding dyslipidemia risk among ACHD patients is to reduce the risk of ASCVD, traditional risk factors alone may underestimate risk in certain ACHD populations. For example, individuals with coarctation of the aorta have higher rates of subclinical atherosclerotic disease on computed tomographic imaging compared with those without coarctation.[25] Premature ASCVD in this population is likely multifactorial and influenced by endothelial dysfunction and abnormal arterial stiffness rather than due to hypertension alone. In addition, patients who have undergone coronary artery manipulation, whether due to a congenital coronary artery anomaly or during an arterial switch operation for transposition of the great arteries, may develop coronary lesions late after surgery even in the absence of traditional atherosclerotic risk factors.[26]

A few cross-sectional studies have applied existing ASCVD risk scores to estimate risk for acquired cardiovascular disease in ACHD but without longitudinal outcomes.[27,28] We considered calculating a 10-year risk score for our patients, but fewer than one-third fell within the valid age range for the Pooled Cohort Risk Assessment Equations, which was developed for individuals without pre-existing cardiovascular disease between 40 and 79 years. Furthermore, given that these scores were neither derived from nor validated in adults with CHD, their applicability in this population remains speculative. Risk models for ASCVD specific to adults with CHD are sorely needed to guide strategies for optimizing cardiovascular risk.

STUDY LIMITATIONS. We acknowledge that there are some limitations to our study. Because participants were not limited to those who were in a fasting state, we are unable to draw clear conclusions about triglyceride levels. Similarly, not all the participating centers routinely recorded oxygen saturation, limiting our power to detect correlations between

oxygen saturation and lipid levels. Although we were able to enroll 186 participants, they comprised at least 20 different forms of CHD. Although these were reasonably well distributed based on degree of complexity, specific lesions were mostly represented in small numbers that are insufficient for performing individual analyses.

Because of the cross-sectional design of the study, we are unable to comment on whether measured lipid levels predict future ASCVD risk. In addition, we did not examine related risk-enhancing factors such as Lp(a) or apoB, which may merit investigation in future studies on ASCVD risk in ACHD. We were not able to ascertain whether measuring lipid profiles in individuals with ACHD can lead to improvements in lifestyle or initiation of statins to lower lipid levels. Finally, Anatomic and Physiological classification was assigned based on information available from the medical record. Appropriately classifying patients sometimes requires more detailed criteria than provided in the 2018 AHA/ACC Guideline; while inter-observer agreement for anatomic classification is generally good, differences in the methodology individual cardiologists use to assign anatomic class could conceivably affect our results to some degree.[29]

CONCLUSIONS

Dyslipidemia is highly prevalent among adults with CHD, occurring in nearly one-half of our cohort despite <15% reporting a prior diagnosis of high cholesterol. As is true in individuals without ACHD, ACHD patients with dyslipidemia were more likely to be obese than those without dyslipidemia. Low HDL-C was most common and predominated in those with complex CHD, whereas high TC more often affected individuals with simple and moderate CHD. The high prevalence of lipid disorders in patients with ACHD (~50%) strongly supports the recommendation for regular screening for cardiovascular risk factors, particularly dyslipidemia, as part of routine ACHD care.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

This study was supported in part by the Boston Adult Congenital Heart Disease Program Dunlevie Fund and by the New England Congenital Cardiology Research Foundation. Dr. Mendelson became an employee of Novartis after completion of study data collection. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: Dyslipidemia was present in nearly one-half of the ACHD patients screened despite <15% of the cohort having a prior diagnosis of such. The pattern of lipid abnormalities differed, with low HDL-C being the predominant pattern among patients with complex CHD and high non-HDL-C being the predominant pattern among patients with simple and moderate CHD.

TRANSLATIONAL OUTLOOK: These data strongly support the recommendation for regular screening for cardiovascular risk factors, particularly dyslipidemia, as part of routine ACHD care.

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KEY WORDS adult congenital heart disease, cholesterol, congenital heart disease, dyslipidemia

APPENDIX For supplemental tables and figures, please see the online version of this paper.