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Left ventricular mass and correlated atherosclerotic risk factors in young adolescents: report from Chin-Shan community cardiovascular study in Taiwan

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Abstract

Various subclinical disease indicators can be used as an early stage marker of atherosclerosis. Left ventricular (LV) mass has been related to cardiovascular morbidity and mortality. The distribution of LV mass in Chinese is rarely studied and nothing is known about its relationships with various atherosclerotic risk factors in young teenagers, in particular, aspects of lipid profiles. We performed a community-based survey of 523 males and 555 females, aged 12–15, in Chin-Shan, a suburb area near Taipei, Taiwan. LV mass was calculated from the Penn convention. Normalized LV mass by height with power of 2.7 was defined. LV mass and normalized LV mass were significantly greater in males than in females. There were significant positive correlation coefficients between LV mass and age, blood pressure, body mass index, low density lipoprotein cholesterol (LDL-C), apolipoprotein (Apo) B, fasting insulin levels and significant negative correlation coefficients between LV mass and high density lipoprotein cholesterol (HDL-C) and Apo A1 level in both genders. Multiple linear regression models showed gender and body mass index (BMI) were important factors associated with LV mass or normalized values for adolescents. Age and systolic blood pressure were also significant predictors of LV mass, but not of normalized LV mass values. LV mass values were found to be negatively associated with HDL-C values at marginal statistically significant level. Age and BMI are the most significant factors of echocardiographic LV mass distributions in young adolescent in Taiwan. LV mass may also be associated with atherosclerotic risk factors.

Keywords: Left ventricular mass; Lipid; Risk factors; Population-based

1. Introduction

Prospective studies have recognized that high blood pressure and obesity are related to the degree of aortic atherosclerosis [1]. The clustering of atherosclerotic risk factors in early adult life is associated with cardiovascular disease in middle-aged men [2]. There are conflicts about early onset of atherosclerosis in children. As fatalities due to cardiovascular disease rarely happened in the children and young adults, it is hard to define atherosclerotic risk patterns for children in tissue studies. From the postmortem studies, the characteristics of atherosclerotic lesions in adolescents are similar to that for adults [3,4]. Dyslipidemia, such as high low density lipoprotein cholesterol (LDL-C) and low high density lipoprotein cholesterol (HDL-C) are significantly associated with the severity of the lesion in children and early adulthood [2,5]. Yet, it is of interest to associate dyslipidemia with left ventricular (LV) mass in the population at risk of cardiovascular diseases.

Abbreviations: Apo, apolipoprotein; BMI, body mass index; HDL-C, high density lipoprotein cholesterol; IVS, interventricular septum; LDL-C, low density lipoprotein cholesterol; LV hypertrophy, left ventricular hypertrophy; LV mass, left ventricular mass; LVIDD, left ventricular internal dimension of end-diastole phase; PWT, posterior wall thickness; RWT, relative wall thickness; WHR, waist-to-hip ratio.

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LV hypertrophy, determined by echocardiography, has been recognized as being strongly associated with cardiovascular diseases [6,7]. The Framingham Heart Study found a six to eight-fold increased risk of cardiovascular events for individuals with LV hypertrophy in adults [8]. Previous studies had reported that, in young adolescents, LV hypertrophy was associated with higher blood pressure, obesity and genders [9]. Echocardiogrphy has been recommended as the screening tool for LV hypertrophy [10–14]. To the best of our knowledge, little has been documented about the relationships between lipid profiles and echocardiographic LV mass in the young population.

Morbidity and mortality from cardiovascular diseases in Taiwan are uprising, especially in coronary heart disease [15]. In clinical setting, some of atherosclerotic risk factors have developed in young adults with the advance of Taiwan's economic growth and the westernized life style. This study was designed to examine patterns of atherosclerotic risk factors, especially lipid profiles, and to interpret their associations with echocardiographically determined LV mass for young adolescents in Chin-Shan community, Taiwan.

2. Methods and materials

2.1. Study design and population

Since 1990, we have established the Chin-Shan community cardiovascular cohort (CCCC) study and aimed to conduct the longitudinal investigation on cardiovascular diseases in Taiwan [16,17]. In 1997, we extended a further survey to include adolescents in the only middle school from this community. Letters describing the purpose of the study were first sent to the parents of those households with children. All the 1124 students, aged 12–15, in this middle school were invited for participation. A clinic was set up at the middle school in an education hall by the study team consisting of four cardiologists and five assistant nurses. Study activity was performed daily, class by class, on weekdays for 3 months. A total of 1078 students (96% response rate) received complete blood and echocardiographic measurements eligible for data analysis. The rest of 46 students were excluded because of poor echocardiographic images. Trained cardiologists took standardized clinical examinations to assess the status of anthropometric measurements. A self-administered questionnaire was used for collecting every subject's basic demographic data, dietary behavior, history of smoking and socioeconomic status of the respective family. Physical examination and 12-lead electrocardiogram were then performed by one of the cardiologists. The student was seated comfortably with the arm supported and positioned at the level of heart, and the measurements were taken with the arm after10-min rest. When measuring blood pressure, the bladder was inflated quickly, and was deflated 2-mmHg every second, and the disappearance of Korotkoff phase V sound was recorded as diastolic pressure. Two blood pressure readings were taken separately by 5 min apart. The average of the two blood pressure readings was used for data analyses.

2.2. Laboratory tests

A blood sample was drawn after a 12-h overnight fast. The serum samples were immediately refrigerated and transported to the National Taiwan University Hospital within 6 h and stored at -70°C for batch assay. Standard enzymatic methods were used to determine serum cholesterol and triglyceride (Merck 14354 and 14366, respectively). HDL-C level was measured in the supernatant after precipitating with magnesium chloride phosphotungstate reagents (Merck 14993). The LDL-C content was measured as 'total cholesterol minus cholesterol in the supernatant' by precipitation method [18,19], since the HDL-C was precipitated using heparin/citrate reagent (Merck 14992). Apolipoprotein (Apo) A1 and B concentrations were measured by turbidimetric immunoassay using commercial kits (Sigma). Blood samples for glucose analyses were drawn into glass test tubes each containing 80 mol/l fluoride/oxalate reagent. After centrifugation at 4°C, $1500 \times g$ for 10 min, glucose levels were measured on supernatant by enzymatic assay (Merck 3389 commercial kit, Germany) in an Eppendorf 5060 autoanalyzer. Plasma insulin level was determined using the ELISA method in which a reagent kit supplied by the Dako Co. was employed. The assay did not measure intact proinsulin, and provided specificity of insulin assay [20]. The level of minimal detection of insulin was 0.02 μU/ml, with CV of 5.0%.

2.3. Echocardiographic measurements

All echocardiographic studies were made with commercially available echocardiographs (HP2500) equipped with each a 2.5/2.0-MHz phase-array transducer and a VHS videotape recorder. All subjects were lying in the left lateral decubitus position to assure the standardized measurement. Observers were not aware of the participant's cardiovascular status during the procedure of echocardiographic examinations and all pictures were taken at the end of expiratory phase.

A two-dimensional parasternal long-axis view of LV was obtained to adjust M-mode cursor position perpendicular to the interventricular septum and posterior wall of LV at the mitral valve chordal level. M-mode measurements were obtained from the long-axis view of LV according to the standard procedure recommended by the American Society of Echocardiography. Gain

control was adjusted to optimize the recording of the endocardial and epicaridal surface and to ensure a continuous line at end diastole and end systole phase [21].

2.4. Determination of LV mass

The LV measurements included the interventricular septal thickness at end diastole (IVS), the posterior wall thickness at end diastole (PWT), as well as the LV internal dimension at end diastole (LVIDD) and end systole (LVIDS). LV mass was calculated from the Penn convention, according to the equation of Devereux and Reichek [11], which was slightly different than measurements based on the American Society of Echocardiography.

$$\text{LV mass} = 1.04\{(\text{IVSD} + \text{LVPWD} + \text{LVEDD})^3 - (\text{LVEDD})^3\} - 13.6 \text{ g}.$$

Normalized LV mass values were calculated by one allometric value height of power 2.7, suggested by de Simone et al. [7], to control for body weight and reduce variability among population. Relative wall thickness was defined as two times LV PWT divided by LV end-diastolic internal dimension.

2.5. Reliability study of intra- and inter-rater variability

A sub-sample of 15 participants was selected to receive echocardiography by the four physicians to estimate the inter-observer variability of measurement parameters. The intra-class correlation reliability was calculated by a simple replication one-way analysis of variance test [22]. The values of correlation reliability were between 0.70 and 0.85 in the various measurements.

2.6. Statistical analysis

We first compared distributions of LV mass and normalized LV mass values by gender. Means and S.D. were presented for various continuous anthropometric and atherosclerotic risk factors. Because of skewed distributions, we analyzed triglyceride and fasting glucose levels using the logarithm transformation and their geometric means. Mean values were compared between genders using Students's t-test for continuous variables. Pearson correlation coefficients were calculated to detect the relationships between echocardiographic parameters and various risk factors, and Fisher's Z-tests were used to detect the significance level of variables for each gender. We used multiple linear regression models to estimate the parameters and related S.E. of various atherosclerotic risk factors on the outcome of LV mass, with or without normalized values, respectively. The most suitable models were chosen by Mallows' Cp statistics to select risk factors [23]. The Cp statistic is an estimate of the standardized total mean squared error of estimation for the current dataset, and the subset models with small Cp and close to the numbers of parameters are best fitted model. Mallows' Cp statistic was considered the most favored criterion for subset size selection [24]. P-values less than 0.05 were significant. All data analyses were performed using the SAS [25].

3. Results

The basic anthropometric risk profiles are presented by gender in Table 1. Males and higher systolic and diastolic blood pressure, waist-to-hip ratio (WHR) and higher fasting glucose levels than females. Lipid profiles, including total cholesterol, tiglyceride, LDL-C, Apo B and fasting insulin levels were higher in females than in males. The prevalence of hyperglycemia, defined as fasting glucose greater than 126 mg/dl, was significantly higher in boys than in girls (11.8 vs. 8.0%, respectively, χ^2-test P value = 0.009, data not shown).

Table 2 shows the echocardiographically determined LV mass and related measurements for both genders. LV mass and normalized LV mass were significantly higher in males than in females, as were IVS, PWT and LVIDD values. However, females had higher relative wall thickness (RWT) than males. When gender-specific normalized LV mass was plotted in graph, the distribution showed higher values for boys than for girls after the summit of frequency (Fig. 1).

The Pearson's correlations coefficients showed significant positive correlations between LV mass and age, blood pressure, body mass index (BMI), WHR, triglyceride, LDL-C, Apo B, and fasting insulin levels, and significant negative correlations between LV mass and HDL-C and Apo A1 level for both genders (Table 3). The correlations found, albeit significance, were generally modest. Most of these correlation coefficients remained significant for normalized LV mass.

When a multiple linear regression model was applied to predict LV mass values, Mallows' Cp criteria was used to select the most parsimonious model (Table 4). After adjusting for other variables, LV mass for girls was 24.8 g lower than that for boys. BMI was also a significant variable predicting both LV mass and normalized values. Age and systolic blood pressure were significant in predicting LV mass, but not normalized LV mass values. HDL-C levels had the effect of lowering LV mass values at a modest significance level, while LDL-C levels had a marginal significant positive effect on LV mass. The adjusted R^2 values by selected variables in the best-fitted model were 0.354 in LV mass and 0.230 in normalized LV mass (data not shown).

4. Discussion

The results clearly demonstrated several atherosclerotic risk factors, such as age, sex, BMI, blood pressure and lipid profiles, on the echocardiographically determined LV mass for adolescents. To the best of our knowledge, this is the first report of the distribution and its associated factors of LV mass for community-based Chinese adolescents.

Age has a strong and persistent effect on LV mass throughout life. The associations between LV mass and gender or age have been well-established [26–29]. There are gender differences on LV mass during adolescents with respect to blood pressure and body size [26]. These effects were clearly demonstrated in our Chin-Shan young adolescents. All studies as mentioned have the implication that age is influential on LV mass at an early age. Furthermore, LV mass is greater in men than in women in adults, after controlling for age, BMI, blood pressure, and lipid profiles [27–29]. Etiologically, increasing sympathetic activity or sex hormone effect was assumed to be crucial for the difference of LV mass between genders [30]. It is of value to note that increasing sympathetic nerve activity in males and adiposity in females as plausible explanations for LV hypertrophy [31].

The index of obesity, such as high body mass index and WHR, has been related to increased LV mass in adult population [27,32,33]. Kono et al. also have found obesity is closely related to LV mass in Japanese children [34]. In our study, obesity is an independent predictor of LV mass. Obesity had its pathophysiological mechanisms, such as hemodynamic and sympathetic, on the development of hypertension and LV hypertrophy [35]. It is particularly associated with dyslipidemia and insulin resistance syndrome. Thus, the clusterings of metabolic risk factors increase the likelihood of cardiovascular events.

Table 1
Differences between boys and girls in basic anthropometric and atherosclerotic risk factors[a]

Variable	Boys ($n = 523$)		Girls ($n = 555$)		P value
	Mean	S.D.	Mean	S.D.	
Age (years)	14.4	1.00	14.4	0.97	0.920
SBP (mmHg)***	107.5	12.2	103.1	11.4	0.0001
DBP (mmHg)*	66.2	8.2	65.0	8.3	0.023
BMI (kg/m^2)	19.4	3.2	19.6	3.1	0.192
WHR***	0.78	0.06	0.74	0.05	0.0001
Cholesterol (mg/dl)***	157.5	27.8	165.6	29.6	0.0001
Triglyceride (mg/dl)***	70.9	33.5	78.0	31.3	0.0004
HDL-C (mg/dl)	45.5	10.9	45.8	9.8	0.705
LDL-C (mg/dl)***	65.5	26.1	74.1	27.9	0.0001
Fasting glucose (mg/dl)***	112.2	16.4	108.7	14.2	0.0002
Apolipoprotein A1 (mg/dl)	110.1	18.0	110.8	18.7	0.563
Apolipoprotein B (mg/dl)***	41.2	12.0	44.8	11.8	0.0001
Insulin (μu/ml)*	7.73	6.79	8.25	4.60	0.014
Triglyceride (mg/dl)†***	4.175	0.404	4.287	0.362	0.0001
Fasting glucose (mg/dl)†***	4.713	0.122	4.681	0.118	0.0001
Insulin (μu/ml)†***	1.81	0.57	1.98	0.49	0.0001

[a] S.D., standard deviation; †, geometric mean; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; HDL-C, high-density-lipoprotein cholesterol; LDL-C, low-density-lipoprotein cholesterol; WHP, waist-to-hip ratio; *, $P<0.05$; **, $P<0.01$; ***, $P<0.001$.

Table 2
Echocardiographically determined LV mass and associated measurements in study adolescents by gender ($n = 1078$)[a]

Variable	Boys ($n = 523$)		Girls ($n = 555$)		P value
	Mean	S.D.	Mean	S.D.	
LV mass (g)***	123.0	38.20	98.90	28.30	0.0001
Normalized LV mass (g/m$^{2.7}$)†***	32.50	8.40	29.00	8.20	0.0001
Interventricular septum thickness (mm)*	7.57	1.19	7.38	1.27	0.013
LV posterior wall thickness (mm)*	7.70	1.34	7.54	1.24	0.035
End-diastolic internal dimension (mm)***	45.24	4.37	41.20	3.78	0.0001
Relative wall thickness (%)††***	34.3	6.65	36.9	7.13	0.0001

[a] †, LV mss normalized by allometric parameter of height, with power of 2.7; ††, relative wall thickness — two times LV posterior wall thickness divided by LV end-diastolic internal dimension; *, $P<0.05$; ***, $P<0.001$.

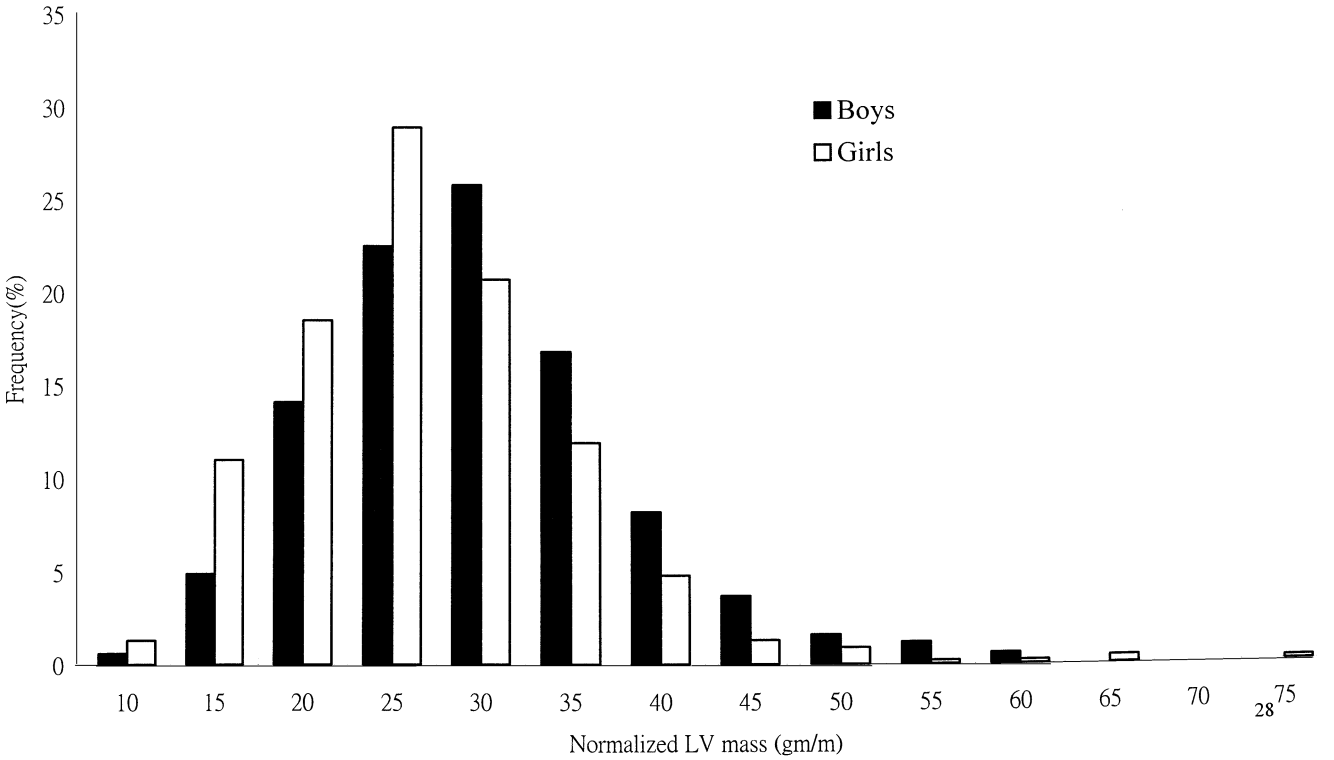

Fig. 1. The distribution of sex-specific normalized LV mass in the study population ($n = 1078$).

Table 3
Pearson correlation coefficients between LV mass and demographic and atherosclerotic risk factors in study adolescents by gender ($n = 1078$)[a]

Variable	Boys ($n = 523$)		Girls ($n = 555$)	
	LV mass	Normalized LV mass	LV mass	Normalized LV mass
Age (years)	0.32***	0.16***	0.10*	0.05
SBP (mmHg)	0.33***	0.18***	0.17***	0.13**
DBP (mmHg)	0.29***	0.13**	0.13**	0.11*
BMI (kg/m^2)	0.54***	0.43***	0.47***	0.48***
WHR	0.19***	0.17***	0.19***	0.19***
Cholestero (mg/dl)	0.02	0.05	0.08	0.06
Triglyceride (mg/dl)	0.19***	0.14**	0.09*	0.10*
HDL-C (mg/dl)	−0.21***	−0.16***	−0.18***	−0.17***
LDL-C (mg/dl)	0.15***	0.14**	0.18***	0.17***
Fasting glucose (mg/dl)	0.09*	0.03	0.05	0.04
Apolipoprotein A1 (mg/dl)	−0.15***	−0.12**	0.06	0.09*
Apolipoprotein B (mg/dl)	0.07	0.11*	0.10*	0.11*
Triglyceride (mg/dl)	0.19***	0.13**	0.77	0.08*
Fasting glucose (mg/dl)	0.10*	0.04	0.64	0.04
Insulin (μu/ml)	0.10*	0.08	0.13**	0.11*
Insulin (μu/ml)	0.11*	0.10*	0.12**	0.10*

[a] †, Geometric mean; abbreviation — BMI, body mass index; WHR, waist-to-hip-ratio; HDL-C, high-density-lipoprotein cholesterol; LDL-C, low-density-lipoprotein cholesterol; Lp(a). ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; *, $P<0.05$; **, $P<0.01$; ***, $P<0.001$.

Hypertension was found to be strongly associated with LV mass reported elsewhere [28,36–38]. Increased vascular tone and arterial wall stiffness due to hypertension may place a greater load on the heart. In Chin–Shan adolescents, 1.0 mmHg increase in systolic blood pressure may increase LV mass by 0.18 g. LV hypertrophy can regress if blood pressure is controlled. Strategies on blood pressure management are undertaking at this community.

In this study, we have attempted to define the relationships between various lipid profiles and LV mass. Indeed, this relation between normal LV mass and lipid

Table 4
The most parsimonious models selected by Mallows' Cp criteria to predict left ventricular mass and normalized left ventricular mass in the study adolescents[a]

Variable	LV mass			Normalized LV mass		
	Parameter	S.E.	*P* value	Parameter	S.E.	*P* value
Intercept	−19.11	16.16	0.238	13.56***	2.22	0.0001
Sex (girls/boys)	−24.84***	1.83	0.0001	−3.407***	0.459	0.0001
Age (years)	4.363***	0.917	0.0001	–	–	–
Systolic blood pressure (mm/Hg)	0.178*	0.081	0.029	–	–	–
Body mass index (kg/m^2)	4.517***	0.318	0.0001	1.129***	0.077	0.0001
HDL-C (mg/dl)	−0.141†	0.088	0.111	−0.047†	0.024	0.052
LDL-C (mg/dl)	0.063†	0.033	0.059	–	–	–
Cholesterol (mg/dl)	–	–	–	0.014	0.008	0.101

[a] †, $0.05<P<0.1$; *, $P<0.05$; **, $P<0.01$; ***, $P<0.001$. The adjusted variables were included sex, age, systolic and diastolic blood pressure, BMI, cholesterol, triglyceride, HDL-C, LDL-C, Apo A1, Apo B, fasting glucose, and insulin levels.

levels is weak. Only marginally significant levels showed in HDL-C and LDL-C. The roles of HDL-C in the pathogenesis of atherosclerosis have been shown in several studies [39,40], including a Chinese population [41]. It was implied that adolescents with low HDL-C and high LDL-C levels tend to have high LV mass values. Metabolic disorder such as insulin resistance syndrome is assumed alternatively to associate with dyslipidemia and LV hypertrophy. Triglyceride, Apo A1 and Apo B, on the other hand, did not have any significant association with LV mass in the fitted linear model. It was possibly due to the shared effects of other variables, especially such as obesity, age and blood pressure.

Fasting hyperinsulinemia, a marker for atheroscelerosis, has been associated with LV hypertrophy [37,42]. Lind et al. [37] have demonstrated the relation to LV wall thickness and to LV mass, but not to LV mass index. In this study, we did not find this association for adolescent. Hyperinsulinemia, per se, is probably not a determinant for LV mass among youth.

The normalized LV mass by allometric parameter of height, with power of 2.7, was one choice for adjusting LV mass [7]. Several other anthropometric variables such as body surface area and height are also indices of LV mass in adult population [28]. In adolescent population, normalizing height with power 2.7 may be more suitable [7]. Sex and BMI are the only significant parameters which influence normalized LV mass. The normalization procedure by height seemed to decrease the effects of age and blood pressure on LV mass.

This study was a cross-sectional study based on community middle school students. The results may not be generalized to all adolescents in Taiwan nor extended to other Chinese populations. This study was highly appraised for its remarkable response rate through the enthusiastic cooperation among community leaders and school teachers. The data excluded for analysis was minor and would not affect causal inference. The potential bias in this study is rather minimal. It is worthwhile to mention that this study has shown LV hypertrophy is associated with atherosclerotic risk factors in youth, especially with various lipid risk factors. Thus life style modifications, such as weight reduction, good dietary behavior and physical activity, should be started at childhood for the primary prevention of cardiovascular diseases and to prevent further cardiovascular events.

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