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ORIGINAL ARTICLE

# Correlation between serum low-density lipoprotein cholesterol concentration and arterial wall stiffness

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## Abstract

**Background:** Elevated serum low-density lipoprotein cholesterol (LDL-C) concentration is a risk factor for atherosclerosis, which involves remodelling of the arterial walls with their subsequent stiffening.

**Aim:** We sought to evaluate the relationship between serum lipid levels and the elastic properties of the arterial wall.

**Methods:** The study group comprised 315 men and women aged 55.84 ± 9.44 years. Serum glucose and lipid concentrations were determited. All subjects underwent blood pressure (BP) measurement, transthoracic echocardiography, and assessment of vascular compliance of large (C1) and small arteries (C2) using the HDI/Pulse Wave™ CR-2000 Research CardioVascular Profiling System (Hypertension Diagnostics Inc., Eagan, MN, USA). The subjects were divided into three groups: group I — LDL-C < 2.6 mmol/L, group II — LDL-C ≥ 2.6 mmol/L and < 4.0 mmol/L, and group III — LDL-C ≥ 4.0 mmol/L.

**Results:** There were no intergroup differences with regard to smoking status ($p = 0.56$), serum glucose concentration ($p = 0.13$), body mass index ($p = 0.96$), systolic ($p = 0.17$) and diastolic BP ($p = 0.29$), or C1 ($p = 0.09$). However, C2 was higher in groups I and II than in group III (5.12 ± 2.57 vs. 5.18 ± 2.75 vs. 4.20 ± 1.58 mL/mmHg × 100, respectively, $p < 0.01$). Multivariate regression analysis negated the independent associations between C1 and serum lipid levels. In contrast, C2 was independently inversely associated with serum LDL-C concentration ($r = –0.15$, $p < 0.01$).

**Conclusions:** Higher serum LDL-C concentration seems to contribute independently to stiffening of small arterial vasculature in otherwise healthy adults. Screening for dyslipidaemia in the general population and its prompt treatment are highly recommended.

**Key words:** arterial wall compliance, arterial wall elasticity, arterial wall stiffness, large arteries, low-density lipoprotein cholesterol, small arteries

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## INTRODUCTION

Serum low-density lipoprotein cholesterol (LDL-C) is a well-known risk factor for cardiovascular (CV) diseases (coronary artery disease and peripheral arterial diseases) [1, 2]. The prevalence of dyslipidaemia in Poland continues to be high. The highest incidence of increased serum LDL-C levels was found in persons between 40 and 59 years old, similarly as in the case of increased serum total cholesterol [3].

Atherosclerosis is a chronic inflammatory process, initiated by damage to the vascular endothelium, which leads to remodelling of the artery wall. Serum LDL-C plays a significant role in this pathology because oxidised LDL molecules activate a number of cellular responses in macrophages, endothelial cells, T-cells, and smooth muscle cells, which promotes inflammation and atherogenesis [4–6]. The initial phase of atherosclerosis involves arterial wall remodelling and a subsequent decrease of its compliance (or increased stiffness). Arterial stiffness is an early marker of arteriosclerosis and an established predictor of CV morbidity and mortality [7, 8]. Assessment of the elastic properties of the arterial wall is a significant diagnostic tool that enables early detection of the vascular pathology, before the clinical symptoms appear [9–11].

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Diagnosing atherosclerosis at this earliest possible stage can be crucial for treatment outcomes. Assessment of the elastic properties of the arterial wall with a non-invasive technique provides information about the functional and structural changes at the level of the aorta, muscular conduit arteries, their peripheral branches, and the microvascular components [10].

The aim of the study was to evaluate the relationships between serum LDL-C concentration and arterial wall elasticity indices reflective of the large and small artery compliances.

## METHODS

The study was conducted as a part of the Silesian Cardiovascular Study, which is an investigation designed to examine the risk factors underlying CV disorders and which was approved by the Bioethical Committee of the Medical University of Silesia. All subjects (Polish, white) were recruited from the general population in three reference centres for CV diseases in the south of Poland.

Serum fasting levels of total cholesterol, high-density lipoprotein cholesterol (HDL-C), triglycerides, LDL-C, and glucose were measured. The compliance of large and small arteries was assessed non-invasively and automatically using the HDI/Pulse Wave™ CR-2000 Research CardioVascular Profiling Instrument System (Hypertension Diagnostics Inc., Eagan, MN, USA). Subjects were tested after a five-minute rest, in a recumbent position, with their head raised no higher than 30°. Blood pressure (BP) was measured automatically with the oscillometric method. Radial artery pulse wave analysis was performed on the right limb with the use of a piezoelectric sensor (applanation tonometry method). In order to minimise artefacts, the wrist was stabilised in supination with a dedicated tool. The principle of the applied method takes into account BP changes during cardiac diastole and is based on the analogy to an electric current circuit according to a modified Windkessel model [12]. Two vascular systems are distinguished in this model: a high-pressure one, which includes the aorta with its main branches, and a low-pressure one, involving peripheral arteries. As a result, the following parameters are obtained: systolic (SBP) and diastolic BP (DBP), large artery elasticity index reflective of large artery compliance (C1, expressed in mL/mmHg × 10), and small artery elasticity index reflective of small artery compliance (C2, expressed in mL/mmHg × 100) [9].

Echocardiography was performed in all subjects by the same experienced sonographer using a Vivid 4 cardiac system equipped with a 3.5-MHz transducer (Med-Electronics, Beltsville, MD, USA).

Healthy subjects aged ≥ 18 years, who provided written informed consent were included in the study. Exclusion criteria comprised presence of any disease or treatment with any medication. Subjects whose serum fasting glucose level was elevated (≥ 100 mg/dL) in the baseline tests were also excluded from the study.

### *Statistical analysis*

Statistical analysis was performed using the GraphPad InStat software, version 3.05 (GraphPad Software, San Diego, CA, USA). The demographic, anthropometric, clinical, as well as the analysed haemodynamic and biochemical parameters were compared between the groups with Kruskal-Wallis analysis of variance and Mann-Whitney test for the post-hoc comparisons. Simple correlations between serum total cholesterol, HDL-C, triglycerides, LDL-C, and arterial parameters were assessed with Pearson's correlation test. Backward stepwise multivariate regression analysis was used to establish the independent determinants of C1 and C2. The p-value $< 0.05$ was considered statistically significant.

## RESULTS

A total of 315 men and women at the average age of 55.84 ± ± 9.44 years were included in the study and divided into three groups according to their serum LDL-C concentration (group I — LDL-C $< 2.6$ mmol/L, group II — LDL-C $\geq 2.6$ mmol/L and $< 4.0$ mmol/L, and group III — LDL-C $\geq 4.0$ mmol/L). The demographic and clinical characteristics of the study participants are reported in Table 1. There were no differences between the groups in terms of the body mass index (BMI; $p = 0.96$), serum triglycerides ($p = 0.09$), glucose ($p = 0.13$), smoking status ($p = 0.56$), SBP ($p = 0.17$), and DBP ($p = 0.29$). There was no significant intergroup difference with regard to C1 (14.16 ± 4.52 vs. 15.35 ± 4.84 vs. 15.83 ± ± 6.29 mL/mmHg × 10, $p = 0.09$), whereas C2 was higher in groups I and II than in group III (5.12 ± 2.57 vs. 5.18 ± ± 2.75 vs. 4.20 ± 1.58 mL/mmHg × 100, $p = 0.005$; Table 1). Moreover, the subjects did not differ in terms of echocardiographic parameters of the left atrium and left ventricle (Table 2). The correlation test revealed univariate positive associations between C1 and serum total cholesterol ($r = 0.15$, $p < 0.01$), as well as C1 and serum LDL-C ($r = 0.12$, $p = 0.03$), but no significant correlations between C1 and serum HDL-C ($p = 0.08$) or triglycerides ($p = 0.28$). In the multivariate regression analysis, the correlations of C1 with serum cholesterol concentrations were lost, and only female sex, younger age, lower SBP, and higher BMI were independently associated with higher large-artery compliance (Table 3). On the other hand, inverse univariate correlations were found between C2 and serum total cholesterol ($r = –0.13$, $p = 0.02$), as well as LDL-C ($r = –0.15$, $p < 0.01$, Fig. 1). Moreover, lower serum LDL-C concentration, along with younger age, female sex, and higher BMI correlated independently with higher C2 in the multivariate regression analysis (Table 3).

## DISCUSSION

In this study we aimed to define the relationship between serum LDL-C concentration and arterial wall compliance/stiffness. Based on the presented results, we found a negative correlation between serum LDL-C and small-artery compliance.

Table 1. Demographic, biochemical, and clinical characteristics of the study group

| | Group I (serum LDL-C < 2.6 mmol/L) n = 83 | Group II (serum LDL-C ≥ 2.6 and < 4.0 mmol/L) n = 134 | Group III (serum LDL-C ≥ 4.0 mmol/L) n = 98 | p |
|---|---|---|---|---|
| Male/Female sex | 59/24 | 85/49 | 63/35 | 0.48 |
| Age [years] | 58.28 ± 10.49 | 55.01 ± 9.71 | 54.91 ± 7.71 | 0.03 |
| BMI [kg/m$^2$] | 27.36 ± 3.97 | 27.39 ± 3.79 | 27.51 ± 4.13 | 0.96 |
| Serum total cholesterol [mmol/L] | 3.79 ± 0.49 | 5.09 ± 0.54 | 6.83 ± 1.02 | < 0.0001 |
| Serum HDL-C [mmol/L] | 0.87 ± 0.33 | 1.00 ± 0.32 | 1.03 ± 0.31 | < 0.0001 |
| Serum triglycerides [mmol/L] | 1.83 ± 1.02 | 1.64 ± 0.92 | 1.81 ± 0.95 | 0.09 |
| Serum LDL-C [mmol/L] | 2.08 ± 0.40 | 3.35 ± 0.39 | 4.97 ± 0.84 | < 0.0001 |
| Serum glucose [mg/dL] | 83.28 ± 11.62 | 83.36 ± 9.82 | 85.88 ± 9.94 | 0.13 |
| Smokers/Non-smokers | 52/31 (62.65/37.35) | 74/60 (55.22/44.78) | 60/38 (61.22/38.78) | 0.56 |
| SBP [mmHg] | 123.46 ± 13.77 | 124.49 ± 11.69 | 121.30 ± 13.37 | 0.17 |
| DBP [mmHg] | 71.89 ± 9.04 | 72.80 ± 8.78 | 70.87 ± 9.95 | 0.29 |
| C1 [mL/mmHg × 10] | 14.16 ± 4.52 | 15.35 ± 4.84 | 15.83 ± 6.29 | 0.09 |
| C2 [mL/mmHg × 100] | 5.12 ± 2.57 | 5.18 ± 2.75 | 4.20 ± 1.58 | 0.005 |

Data are presented as mean ± standard deviation or number (percentage). BMI — body mass index; C1 — large-artery elasticity index; C2 — small-artery elasticity index; DBP — diastolic blood pressure; HDL-C — high-density lipoprotein cholesterol; LDL-C — low-density lipoprotein cholesterol; SBP — systolic blood pressure

Table 2. Echocardiographic parameters of the study group

| | Group I (serum LDL-C < 2.6 mmol/L) | Group II (serum LDL-C ≥ 2.6 and < 4.0 mmol/L) | Group III (serum LDL-C ≥ 4.0 mmol/L) | p |
|---|---|---|---|---|
| LA [mm] | 38.85 ± 4.69 | 38.43 ± 4.04 | 38.5 ± 3.76 | 0.75 |
| IVSDD [mm] | 11.04 ± 1.76 | 10.51 ± 1.8 | 11.05 ± 1.63 | 0.07 |
| PWDD [mm] | 10.68 ± 1.55 | 10.22 ± 1.43 | 10.64 ± 1.29 | 0.04 |
| LVESD [mm] | 33.94 ± 8.49 | 33.39 ± 6.54 | 34.16 ± 7.48 | 0.64 |
| LVEDD [mm] | 50.55 ± 6.00 | 50.97 ± 4.96 | 51.15 ± 5.25 | 0.18 |
| LVEF [%] | 54.87 ± 6.40 | 56.10 ± 8.92 | 56.97 ± 7.64 | 0.14 |

Data are presented as mean ± standard deviation. IVSDD — intraventricular septum diastolic diameter; LA — left atrium; LVEDD — left ventricular end-diastolic diameter; LVEF — left ventricular ejection fraction; LVESD — left ventricular end-systolic diameter; PWDD — posterior wall diastolic diameter

Table 3. Multivariate regression analysis with large artery compliance (C1) and small artery compliance (C2) as dependent variables

| Dependent variables | Independent variables | $\beta$-coefficient | 95% CI | p |
|---|---|---|---|---|
| C1 | Male sex | –0.33 | –0.29 to –0.37 | < 0.01 |
| | Age | –0.22 | –0.18 to 0.26 | < 0.01 |
| | BMI | 0.23 | 0.19 to 0.27 | < 0.01 |
| | SBP | –0.37 | –0.32 to –0.42 | < 0.01 |
| C2 | | | | |
| | Male sex | –0.26 | –0.21 to –0.31 | < 0.01 |
| | Age | –0.24 | –0.19 to –0.29 | < 0.01 |
| | BMI | 0.20 | 0.15 to 0.25 | < 0.01 |
| | Serum LDL-C concentration | –0.18 | –0.13 to –0.23 | < 0.01 |

CI — confidence interval; other abbreviations — see Table 1

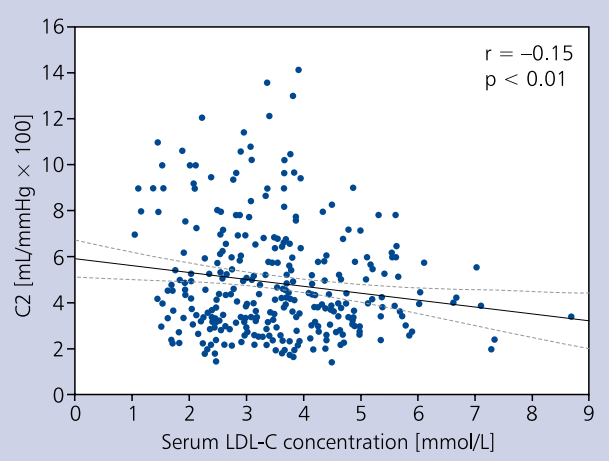


Figure 1. Plot of Pearson's correlation between small-artery elasticity index C2 and serum low-density lipoprotein cholesterol (LDL-C)

The results of our research partially coincide with those obtained by Schillinger et al. [13], who showed an association between high serum concentrations of lipoprotein(a) (a modified LDL particle with strong pro-inflammatory and pro-atherogenic effects [14]) and reduced compliance of the small artery walls. In a group of subjects with atherosclerosis, the observed negative correlation between serum lipoprotein(a) and C2 was independent of other risk factors such as sex, smoking status, and diabetes. Moreover, no independent correlation between serum lipoprotein(a) and C1 was shown. On the other hand, Miao et al. [15] did not reveal any independent relationships between serum total cholesterol or LDL-C and the elastic properties of the vascular wall. However, they ascertained associations of low serum HDL-C or elevated triglycerides with reduced compliance of the arterial wall. Moreover, they were able to show a relationship between compliance of the vascular wall and left ventricular diastolic function. In our study, in line with that of Canepa et al. [16], we did not corroborate any correlations between the artery wall elasticity indices and left ventricular end-diastolic diameter. This is in contrast to the majority of clinical studies that showed an association between reduced compliance (or increased stiffness) of the arterial walls and left ventricular diastolic dysfunction [17]. With decreasing arterial wall compliance, cardiac afterload increases because of the accelerated return of the arterial reflection wave from the periphery during systole, which augments the SBP and results in excess stress to the left ventricle [17]. In our work, similarly to Canepa et al. [16], the average age of the studied subjects was lower in comparison to other studies, which may partially explain the discrepancy between the study findings.

The pathophysiology of arterial stiffening involves extracellular matrix remodelling with fragmentation of elastin and deposition of collagen in the arterial walls. Elastin fibres have mechanical properties of elasticity, whereas collagen is significantly stiffer than elastin [18]. Importantly, elastin contains hydrophobic domains, which makes it attractive for interactions with ligands such as cholesterol. Bilici et al. [19] revealed detrimental modifications of elastin due to cholesterol exposure in vitro. It may be assumed that such changes occur in vivo in the course of dyslipidaemia and contribute to arterial stiffening. Biochemical analyses have revealed that lipids accumulating on the membranes of atherosclerotic lesions are bonded with elastin already at the early stages of atherosclerosis [19, 20].

As mentioned above, non-invasive measurement of vascular wall compliance makes it possible to detect pathologies before the clinical symptoms of the CV disease appear. The decreased compliance of small artery walls reflects the disturbed function of the vascular endothelium and has been established as an independent CV risk factor [21, 22]. Panaich et al. [23] showed that both small and large artery compliances were associated with subclinical coronary atherosclerosis in subjects free from symptomatic angina. This association was independent of CV risk factors, i.e. age, sex, race, SBP, diabetes, smoking status, serum total cholesterol, HDL-C, and high-sensitivity C-reactive protein [23]. In a prospective study, Grey et al. [24] demonstrated an association between the increased stiffness of the small arteries and occurrence of CV events. Similarly to our work, the methodology of the measurement of elasticity indices of the small and large arteries was based on the modified Windkessel model. In a long-term observation CV events occurred in 41% of the studied subjects, and both C1 and C2 were their univariate predictors, whereas only C2 was found to predict CV events after taking into account the effect of age [24].

In our study we have shown that higher serum LDL-C appears to contribute independently to small-artery stiffening in otherwise heathy adults. A decreased compliance (or increased stiffness) of these arteries in subjects without diagnosed CV disease can suggest a very early disease course and be the indication to extend patient diagnostics.

Other parameters, such as serum C-reactive protein, aminotransferases, uric acid, creatinine, glomerular filtration rate, and oxidised LDL, which may influence the elastic properties of the arteries, were not taken into consideration in our study.

In conclusion, the small artery compliance is lower among subjects with higher serum LDL-C concentration, and there is an inverse correlation between these two factors.

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***Conflict of interest:*** *none declared*

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## WHAT IS NEW?

Decreased arterial compliance (or increased stiffness) is an early marker of arteriosclerosis and an established predictor of cardiovascular morbidity and mortality. Non-invasive assessment of the elastic properties of the arterial wall enables early detection of the vascular pathology, before the clinical symptoms appear. Diagnosing atherosclerosis at this earliest possible stage can be crucial for treatment outcomes. We demonstrate that higher concentrations of serum low-density lipoprotein cholesterol appear to contribute independently to stiffening of the peripheral arterial vasculature in otherwise healthy adults. The importance of screening for dyslipidaemia in the general population and its prompt treatment are corroborated by the study findings.