

Impaired aortic function in patients with coeliac disease

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Abstract

Background and aim: We aimed to investigate the association between aortic function (aortic stiffness index, aortic strain, and aortic distensibility), which is a predictor of atherosclerosis, and coeliac disease (CD).

Methods: Thirty-six patients with CD and 35 control subjects were included in the study. Serological screening was performed to determine the levels of auto-immune markers, including anti-gliadin immunoglobulin (Ig)A and IgG, and anti-tissue transglutaminase antibodies. Aortic distensibility, aortic strain, and aortic stiffness index were calculated using echocardiography.

Results: Aortic strain and aortic distensibility were significantly lower in patients with CD than in control subjects (0.07 [0.03–0.14] vs. 0.09 [0.06–0.15], $p < 0.001$; 0.0036 ± 0.0012 vs. 0.0051 ± 0.0014 , $p < 0.001$, respectively). However, the aortic stiffness index was significantly higher in patients with CD than in controls (1.14 [0.57–2.69] vs. 0.91 [0.59–1.92], $p = 0.002$). Coeliac disease was the only independent parameter that was correlated with aortic strain, aortic stiffness index, and aortic distensibility ($\beta = -0.427$, $p < 0.001$; $\beta = 0.375$, $p = 0.003$; $\beta = -0.434$, $p < 0.001$, respectively).

Conclusions: In this study, we showed deteriorated aortic functions by echocardiography in CD patients, which predicted subclinical atherosclerosis. Because deteriorated aortic functions is a strong predictor of future cardiovascular events, close cooperation with cardiologists and gastroenterologists is needed in the management of CD patients, and increased awareness of ischaemic heart disease risk factors in these patients and healthcare providers is warranted.

Key words: aortic function, coeliac disease, echocardiography

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INTRODUCTION

Coeliac disease (CD) is characterised by gluten intolerance that particularly affects the gastrointestinal system. The condition causes chronic mucosal inflammation of the proximal small intestine. CD is usually diagnosed during childhood and adolescence, but it can also be diagnosed in adults [1].

Previous studies have demonstrated an increased risk of incident ischaemic heart disease (IHD), mortality, or cardiovascular (CV) disease in CD patients [2–5].

Aortic stiffness (AS) has a direct effect on CV morbidity and mortality [6]. Major CV risk factors such as smoking, hypercholesterolaemia, and diabetes mellitus can increase AS [7–11].

Although some studies investigated the association between CD and CV disease, there was lack of evidence for an association between CD and early predictors of atherosclerosis, such as aortic function (AS index [ASI], aortic strain, and aortic distensibility) [12].

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In this study, we aimed to investigate the association between aortic function (ASI, aortic strain, and aortic distensibility), which are predictors of atherosclerosis, and CD.

METHODS

The study included 36 biopsy-proven CD patients who were admitted to the Department of Gastroenterology and 35 age- and sex-matched control subjects who were admitted to the Department of Cardiology. Exclusion criteria were known history of moderate or severe valvular disease, heart failure (ejection fraction < 50%), rhythm disturbance, structural or congenital heart disease, coronary heart disease, active infection, malignancy, pregnancy, other systemic inflammatory diseases, diabetes mellitus, uncontrolled hypertension, and use of vasoactive drugs.

As part of gastrointestinal endoscopy studies, patients underwent upper gastrointestinal endoscopy with at least six biopsies in the descending duodenum using a Fujinon EG 450PE5 gastroscope (Fujinon, Japan). Histological findings were described using the modified Marsh classification [2]. The patients were enrolled to the study after diagnosis of CD.

During hospitalisation under fasting conditions, blood glucose levels, lipid profile (low-density lipoprotein cholesterol [LDL-C], high-density lipoprotein cholesterol [HDL-C], triglycerides, and total cholesterol), renal and liver function tests, thyroid function tests, ferritin levels, vitamin B₁₂ levels, and folate levels were measured (Beckman Coulter, Fullerton CA, USA). LDL-C was calculated using the Friedewald formula. A complete blood count was also obtained using a Beckman Coulter LH780 Haematology Analyser with the LH SlideMaker and LH SlideStainer (Fullerton CA, USA). Serological screening was performed to determine the levels of auto-immune markers, including anti-gliadin antibody (AGA) immunoglobulin (Ig)A, AGA IgG, and anti-tissue-transglutaminase (anti-tTG) antibodies. Antibodies were detected in CD patients using fluorescence patterns as auto-immune markers using Euroimmun (Medizinische Labordiagnostika AG) immune fluorescence auto-antibody determination kits.

Echocardiographic recordings were obtained by standard ultrasonography (Vivid 7, GE, Horten, Norway) and a 2.5–3.5-Hz transducer at parasternal and apical windows by a single experienced observer who was blinded to clinical and laboratory data. Apical four-chamber and parasternal views of the left ventricle were obtained at end-expiratory apnoea. The left ventricular ejection fraction was measured with M-mode recordings using the Teichholz method [13].

Aorta (Ao) diameters were measured from the same view on the M-mode tracing at a level of 3 cm above the aortic valve. The systolic diameter (AoSD) was measured at the maximum anterior motion of the Ao, while the diastolic diameter (AoDD) was measured at the peak of the QRS complex on a simultaneously recorded electrocardiogram. Blood pressure (systolic [SBP] and diastolic [DBP]) was simultaneously measured by sphygmomanometry during echocardiography.

Aortic strain is the deformation (percentage change in diameter) of an aorta due to pulse pressure as a stress force. Aortic stiffness is the resistance to deformation, and it is dependent on the complex interaction between the vascular smooth muscle cells with the extracellular matrix containing elastin, collagen, and fibrillin. By contrast, aortic distensibility is defined as the relative compliance or relative change in diameter as pressure increases.

The following indices of aortic function were calculated.

- aortic strain (%) = $100 \times \frac{(AoSD - AoDD)}{AoDD}$
- aortic distensibility = $2 \times \frac{(AoSD - AoDD)}{(AoDDPP)} \text{ [cm}^2 \times \text{dyn}^{-1} \times 10^{-6}]$ [14, 15]
- aortic stiffness index (ASI) = $\ln \frac{(SBP/DBP)}{(AoSD - AoDD)/AoDD}$
- pulse pressure (PP) was calculated as SBP – DBP [16]
- mean blood pressure (MBP) was calculated as $[\frac{2DBP + SBP}{3}]$.

The study protocol was approved by the local ethics committee, and informed consent was obtained from each subject before study enrolment.

Statistical analysis

The International Business Machines Statistical Package for the Social Sciences for Windows 15.0 was used for all statistical analyses. Continuous variables are presented as means ± standard deviations or medians (minimum–maximum). Categorical variables are summarised as frequencies and percentages. Normality of the continuous variables was evaluated by the Shapiro–Wilks test. Differences between groups according to continuous variables were determined by an independent samples t-test or the Mann–Whitney U-test as appropriate. Categorical variables were compared by Pearson's χ^2 or Fisher's exact test. Factors affecting aortic function were verified by multiple linear regression analysis. A p-value < 0.05 was considered statistically significant.

RESULTS

Clinical and demographic variables of the groups are summarised in Table 1. There were no significant differences in gender, age, body mass index, hyperlipidaemia, smoking status, and blood pressure. LDL-C, serum triglyceride, and serum thyroid stimulating hormone levels were significantly higher in the CD group than in controls: 103.65 ± 25.63 vs. 89.82 ± 32.23 , $p = 0.049$; 109.88 ± 39.28 vs. 83.14 ± 41.22 , $p = 0.007$; $2.1 (0.74–17.9)$ vs. $1.44 (0.39–4.65)$, $p = 0.001$, respectively. HDL-C and free tri-iodothyronine levels were significantly higher in controls than in the CD group: 52.08 ± 13.16 vs. 45.28 ± 10.34 , $p = 0.002$; 3.40 ± 0.39 vs. 3.07 ± 0.76 , $p = 0.026$, respectively.

Echocardiographic variables of the groups are shown in Table 2. The left atrial diameter and aortic root diameter were significantly greater in CD patients than in controls: $3.25 (2.5–3.6)$ vs. $3.0 (2.3–3.9)$, $p = 0.005$; $2.30 (2.0–3.3)$ vs. $2.10 (2.0–2.6)$, $p = 0.006$, respectively.

Table 1. Demographic and laboratory characteristics of subjects by study groups

	Celiac patients (n = 36)	Controls (n = 35)	P
Gender (male/female)	6/30	5/30	0.783
Age [years]*	28.5 (17–53)	27 (18–52)	0.682
Body mass index [kg/m ²]	22.52 ± 4.13	21.42 ± 3.51	0.235
Pulse [bpm]	72.08 ± 6.83	75.51 ± 8.32	0.062
Hyperlipidaemia	5 (13.9%)	3 (8.6%)	0.710
Smoking	5 (13.9%)	8 (22.9%)	0.503
Systolic blood pressure [mm Hg]*	110 (90–120)	110 (90–130)	0.489
Diastolic blood pressure [mm Hg]*	69 (60–80)	70 (60–80)	0.675
Antiendomysium ab. (+/-)	19/17		
Antigliadin IgA (+/-)	14/23		
Antigliadin IgG (+/-)	11/25		
Total cholesterol [mg/dL]	164.25 ± 34.16	159 ± 37.98	0.542
HDL cholesterol [mg/dL]	45.28 ± 10.34	52.08 ± 13.16	0.019
LDL cholesterol [mg/dL]	103.65 ± 25.63	89.82 ± 32.23	0.049
Triglyceride [mg/dL]	109.88 ± 39.28	83.14 ± 41.22	0.007
Haemoglobin [g/dL]	12.97 ± 1.71	13.16 ± 1.62	0.633
Vitamin B ₁₂ [pg/mL]	353.05 ± 125.07	320.68 ± 181.26	0.383
Ferritin [mg/mL]*	59 (5-214)	69 (11-251)	0.154
Folate [ng/dL]	6.77 ± 3.80	6.40 ± 2.61	0.637
Free T ₃ [pmol/L]	3.07 ± 0.76	3.40 ± 0.39	0.026
Free T ₄ [pmol/L]	1.98 ± 1.75	1.12 ± 0.21	0.376
TSH [mU/mL]*	2.1 (0.74–17.9)	1.44 (0.39–4.65)	0.001

*Median (minimum–maximum), variables without normal distribution; mean ± standard deviation, variables with normal distribution; HDL — high density lipoprotein; LDL — low density lipoprotein; TSH — thyroid-stimulating hormone

Table 2. Echocardiographic characteristics of subjects by study groups

	Celiac patients (n:36)	Controls (n:35)	P
LV end-diastolic diameter [cm]	4.52 ± 0.41	4.51 ± 0.37	0.909
LV end-systolic diameter [cm]	2.83 ± 0.28	2.73 ± 0.31	0.174
Ejection fraction [%]	67 ± 4.27	67.97 ± 3.82	0.317
Left atrium diameter [cm]*	3.25 (2.5–3.6)	3.0 (2.3–3.9)	0.005
Septal wall thickness [cm]*	0.9 (0.7–1.0)	0.90 (0.7–1.1)	0.672
Posterior wall thickness [cm]*	0.90 (0.7–1.10)	0.90 (0.6–1.1)	0.908
Aortic root [cm]*	2.30 (2.0–3.3)	2.10 (2.0–2.6)	0.006
Aortic velocity [m/sn]*	1.22 (1.0–1.6)	1.20 (1.0–1.5)	0.169
Pulmonary velocity [m/sn]*	0.90 (0.7–1.1)	0.90 (0.6–1.0)	0.447
Mitral E wave [m/sn]*	0.90 (0.5–1.3)	0.90 (0.6–1.3)	0.338
Mitral A wave [m/sn]*	0.60 (0.5–1.0)	0.60 (0.4–0.8)	0.174
Aortic strain [%]*	0.07 (0.03–0.14)	0.09 (0.06–0.15)	< 0.001
Aortic stiffness index*	1.14 (0.57–2.69)	0.91 (0.59–1.92)	0.002
Aortic distensibility	0.0036 ± 0.0012	0.0051 ± 0.0014	< 0.001

*Median (minimum–maximum), variables without normal distribution; mean ± standard deviation, variables with normal distribution; LV — left ventricle

Table 3. Multivariate linear regression analysis according to factors which effects aortic functions

Variables	Aortic strain		Aortic stiffness index		Aortic distensibility	
	β	P	β	P	β	P
Age	-0.129	0.242	0.112	0.347	-0.097	0.380
Smoking	-0.171	0.114	-0.017	0.886	-0.108	0.319
BMI	-0.144	0.195	-0.203	0.094	-0.170	0.129
TSH	-0.094	0.393	0.015	0.898	-0.098	0.371
Celiac disease	-0.427	< 0.001	0.375	0.003	-0.434	< 0.001
LDL-C	-0.969	0.001	0.541	0.067	-0.913	0.002
TC	1.000	0.001	-0.711	0.017	0.885	0.003

BMI — body mass index; TSH — thyroid-stimulating hormone; LDL-C — low density lipoprotein cholesterol; TC — total cholesterol

The degree of aortic strain and aortic distensibility was significantly smaller in CD patients than in controls: 0.07 (0.03–0.14) vs. 0.09 (0.06–0.15), $p < 0.001$; $0.0036 \pm \pm 0.0012$ vs. 0.0051 ± 0.0014 , $p < 0.001$, respectively, but the ASI was significantly higher in CD patients than in controls: 1.14 (0.57–2.69) vs. 0.91 (0.59–1.92), $p = 0.002$.

As shown in Table 3, although we found that age and celiac disease were correlated both with aortic strain ($p = 0.036$, $p < 0.001$, respectively), and aortic distensibility ($p = 0.028$, $p < 0.001$, respectively), celiac disease was the only parameter that correlated with ASI ($p = 0.002$). After multivariate analysis, celiac disease was the sole independent factor for aortic strain, aortic distensibility, and ASI ($\beta = -0.437$, $p < 0.001$; $\beta = 0.369$, $p = 0.002$; $\beta = 0.451$, $p < 0.001$, respectively).

DISCUSSION

Celiac disease is characterised by life-long intolerance to gluten found in dietary cereals. The prevalence of CD is 1% in the general population, but the prevalence in patients with autoimmune disorders is higher than that in the normal population (8–20%) [1, 17]. In CD patients, regular ingestion of wheat, rye, and barley induces T-cell-mediated inflammation in the gut and an autoimmune response to self proteins, mainly tissue type-2-transglutaminase, leading to the production of anti-tTG antibodies [18].

The aetiopathological mechanisms of CV outcomes in CD patients can be chronic inflammation or augmented levels of plasma homocysteine because of the malabsorption of folic acid and vitamin B₁₂ [19, 20]. In our study, the underlying mechanism of CD may be chronic inflammation because there were no significant differences in folate and vitamin B₁₂ levels between groups.

AS has a strong effect on CV prognosis. This association can be explained by three primary mechanisms [21]. First, increased AS may contribute to atherosclerotic progression to other arteries such as the coronary and carotid arteries. Second, in addition to AS, SBP and pressure on vital organs is

increased, contributing to the risk of atherosclerotic complications. Last, endothelial dysfunction can lead to AS and atherosclerotic progression. Therefore, AS plays an important role in CV events [22–24].

Previous studies have shown a higher CD prevalence among patients with idiopathic dilated cardiomyopathy and myocarditis [25–27]. Moreover, Makhdoom et al. [28] demonstrated the association between gluten restriction and improvement of cardiac function in patients with CD. In our study, there were significant differences in the diameter of the left ventricle between the CD patients and controls. Left atrial diameters were greater in the CD group; this condition could be related to left ventricular diastolic dysfunction.

Ludvigsson et al. [29] found a positive association between CD and IHD; they also found an elevated risk of IHD that was independent of small intestinal histopathology in CD patients. Also, Wei et al. [5] reported a 2.5-fold increased risk of CV disease in celiac patients without prior CV disease.

Endothelial dysfunction is a marker of vascular involvement in any disease affecting the vascular structures that can be linked with vascular involvement in atherosclerosis [30]. In our previous study, we demonstrated macrovascular endothelial dysfunction in CD patients using a flow-mediated dilatation method [31].

This is the first study to investigate aortic function using echocardiography in CD patients, which revealed deteriorated aortic function. These results suggest that the consumption of gluten-free diets from childhood may play a preventive role in the development of further atherosclerotic diseases in CD patients.

Limitations of the study

This study has some limitations. This study is a single-centre, nonrandomised study. The sample size was relatively small. Other limitations of the study were the fact that no inflammation markers were used and that the blood pressure was measured from the brachial artery instead of aorta.

CONCLUSIONS

In conclusion, our findings suggest that CD is associated with impaired aortic function. This finding helps to explain why celiac subjects have increased CV risk. Because deteriorated aortic function is a strong predictor of future CV events, close cooperation with cardiologists and gastroenterologists is needed in the management of CD patients, and increased awareness of IHD risk factors in these patients and healthcare providers is warranted.

Conflict of interest: none declared

References

- Green PH, Cellier C. Celiac disease. *N Engl J Med*, 2007; 357: 1731–1743. doi: [10.1056/NEJMra071600](https://doi.org/10.1056/NEJMra071600).
- Ludvigsson JF, Montgomery SM, Ekblom A et al. Small intestinal histopathology and mortality risk in celiac disease. *JAMA*, 2009; 302: 1171–1178. doi: [10.1001/jama.2009.1320](https://doi.org/10.1001/jama.2009.1320).
- Ludvigsson JF, de Faire U, Ekblom A et al. Vascular disease in a population-based cohort of individuals hospitalised with coeliac disease. *Heart*, 2007; 93: 1111–1115. doi: [10.1136/hrt.2006.097097](https://doi.org/10.1136/hrt.2006.097097).
- Peters U, Askling J, Gridley G et al. Causes of death in patients with celiac disease in a population-based Swedish cohort. *Arch Intern Med*, 2003; 163: 1566–1572. doi: [10.1001/archinte.163.13.1566](https://doi.org/10.1001/archinte.163.13.1566).
- Wei L, Spiers E, Reynolds N et al. Association between coeliac disease and cardiovascular disease. *Aliment Pharmacol Ther*, 2007; 27: 514–519. doi: [10.1111/j.1365-2036.2007.03594.x](https://doi.org/10.1111/j.1365-2036.2007.03594.x).
- Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness. *J Am Coll Cardiol*, 2010; 55: 1318–1327. doi: [10.1016/j.jacc.2009.10.061](https://doi.org/10.1016/j.jacc.2009.10.061).
- Nichols WW, O'Rourke MF. McDonald's blood flow in arteries: theoretical, experimental and clinical principles. 5th Ed. Hodder Arnold, London 2005.
- Sassalos K, Vlachopoulos C, Alexopoulos N et al. The acute and chronic effect of cigarette smoking on the elastic properties of the ascending aorta in healthy male subjects. *Hellenic J Cardiol*, 2006; 47: 263–268.
- Cruickshank K, Riste L, Anderson SG et al. Aortic pulsewave velocity and its relationship to mortality in diabetes and glucose intolerance: an integrated index of vascular function? *Circulation*, 2002; 106: 2085–2090. doi: [10.1161/01.CIR.0000033824.02722.F7](https://doi.org/10.1161/01.CIR.0000033824.02722.F7).
- Laurent S, Boutouyrie P, Asmar R et al. Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. *Hypertension*, 2001; 37: 1236–1241. doi: [10.1161/01.HYP.37.5.1236](https://doi.org/10.1161/01.HYP.37.5.1236).
- Laurent S, Cockcroft J, Van Bortel L et al. European Network for Noninvasive Investigation of Large Arteries. Expert consensus document on arterial stiffness: methodological issues and clinical applications. *Eur Heart J*, 2006; 27: 2588–2605. doi: [10.1093/eurheartj/ehl2542588-2605](https://doi.org/10.1093/eurheartj/ehl2542588-2605).
- Rostami K, Kerckhaert J, Tiemessen RV et al. Sensitivity of antiendomysium and anti gliadin antibodies in untreated celiac disease: disappointing in clinical practice. *Am J Gastroenterol*, 1999; 94: 888–894. doi: [10.1111/j.1572-0241.1999.983.f.x](https://doi.org/10.1111/j.1572-0241.1999.983.f.x).
- Teichholz LE CM, Sonnenblick EH, Gorlin R. Study of left ventricular geometry and function by B-scan ultrasonography in patients with and without asynergy. *N Engl J Med*, 1974; 291: 120–128. doi: [10.1056/NEJM197412052912304](https://doi.org/10.1056/NEJM197412052912304).
- Stefanadis C, Stratos C, Vlachopoulos C et al. Pressure diameter relation of the human aorta: a new method of determination by the application of a special ultrasonic dimension catheter. *Circulation*, 1995; 92: 2210–2219. doi: [10.1161/01.CIR.92.8.2210](https://doi.org/10.1161/01.CIR.92.8.2210).
- Stefanadis C, Dernellis J, Vlachopoulos C et al. Aortic function in arterial hypertension determined by pressure-diameter relation: effects of diltiazem. *Circulation*, 1997; 96: 1853–1858. doi: [10.1161/01.CIR.96.6.1853](https://doi.org/10.1161/01.CIR.96.6.1853).
- Stefanadis C, Dernellis J, Tsiamis E et al. Aortic stiffness as a risk factor for recurrent acute coronary events in patients with ischemic heart disease. *Eur Heart J*, 2000; 21: 390–396. doi: [10.1053/euhj.1999.1756390-396](https://doi.org/10.1053/euhj.1999.1756390-396).
- Tommasini A, Not T, Kiren V et al. Mass screening for coeliac disease using anti-human transglutaminase antibody assay. *Arch Dis Child*, 2004; 89: 512–515. doi: [10.1136/adc.2003.029603](https://doi.org/10.1136/adc.2003.029603).
- Koning F. Celiac disease: caught between a rock and hard place. *Gastroenterology*, 2005; 129: 1294–1301.
- Lee SK, Lo W, Memeo L et al. Duodenal histology in patients with celiac disease after treatment with a gluten-free diet. *Gastrointest Endosc*, 2003; 57: 187–191.
- Lim PO, Tzemos N, Farquharson CA et al. Reversible hypertension following coeliac disease treatment: the role of moderate hyperhomocysteinaemia and vascular endothelial dysfunction. *J Hum Hypertens*, 2002; 16: 411–415. doi: [10.1038/sj/jhh/1001404](https://doi.org/10.1038/sj/jhh/1001404).
- Jankowski P, Kawecka-Jaszcz K, Czarnecka D et al. Pulsatile but not steady component of blood pressure predicts cardiovascular events in coronary patients. *Hypertension*, 2008; 51: 848–855. doi: [10.1161/HYPERTENSIONAHA.107.101725](https://doi.org/10.1161/HYPERTENSIONAHA.107.101725).
- Giannattasio C, Failla M, Emanuelli G et al. Local effects of atherosclerotic plaque on arterial distensibility. *Hypertension*, 2001; 38: 1177–1180. doi: [10.1161/hy1101.095994](https://doi.org/10.1161/hy1101.095994).
- Silacci P, Desgeorges A, Mazzolai L et al. Flow pulsatility is a critical determinant of oxidative stress in endothelial cells. *Hypertension*, 2001; 38: 1162–1166. doi: [10.1161/hy1101.095993](https://doi.org/10.1161/hy1101.095993).
- Ikonomidis I, Stamatelopoulou K, Lekakis J et al. Inflammatory and non-invasive vascular markers: the multimarker approach for risk stratification in coronary artery disease. *Atherosclerosis*, 2008; 199: 3–11. doi: [10.1016/j.atherosclerosis.2008.02.019](https://doi.org/10.1016/j.atherosclerosis.2008.02.019).
- Curione M, Barbato M, De Biase L et al. Prevalence of coeliac disease in idiopathic dilated cardiomyopathy. *Lancet*, 1999; 354: 222–223. doi: [10.1016/S0140-6736\(99\)01501-9](https://doi.org/10.1016/S0140-6736(99)01501-9).
- Not T, Faleschini E, Tommasini A et al. Celiac disease in patients with sporadic and inherited cardiomyopathies and in their relatives. *Eur Heart J*, 2003; 24: 1455–1461. doi: [10.1016/S0195-668X\(03\)00310-5](https://doi.org/10.1016/S0195-668X(03)00310-5).
- Frustaci A, Cuoco L, Chimenti C et al. Celiac disease associated with autoimmune myocarditis. *Circulation*, 2002; 105: 2611–2618. doi: [10.1161/01.CIR.0000017880.86166.87](https://doi.org/10.1161/01.CIR.0000017880.86166.87).
- Makhdoom ZA, Randall NW. Dilated cardiomyopathy due to anticardiolipin syndrome in association with celiac sprue [letter]. *J Clin Gastroenterol*, 2000; 31: 91–92.
- Ludvigsson JF, James S, Askling J et al. Nationwide cohort study of risk of ischemic heart disease in patients with celiac disease. *Circulation*, 2011; 123: 483–490. doi: [10.1161/CIRCULATIONAHA.110.965624](https://doi.org/10.1161/CIRCULATIONAHA.110.965624).
- Hadi HA, Carr CS, Al Suwaidi J. Endothelial dysfunction: cardiovascular risk factors, therapy, and outcome. *Vasc Health Risk Manag*, 2005; 1: 183–198.
- Sari C, Bayram NA, Dogan FE et al. The evaluation of endothelial functions in patients with celiac disease. *Echocardiography*, 2012; 29: 471–477. doi: [10.1111/j.1540-8175.2011.01598.x](https://doi.org/10.1111/j.1540-8175.2011.01598.x).

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Nieprawidłowa czynność aorty u pacjentów z chorobą trzewną

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Streszczenie

Wstęp i cel: Badanie przeprowadzono w celu oceny powiązań między czynnością aorty (wskaźnik sztywności aorty i rozszerzalność aorty), będącą czynnikiem predykcyjnym miażdżycy, a chorobą trzewną (CD).

Metody: Do badania włączono 36 pacjentów z CD i 35 osób stanowiących grupę kontrolną. W celu określenia stężeń wskaźników autoimmunologicznych, w tym miana przeciwciał przeciw gliadynie IgA i IgG oraz przeciw transglutaminazie tkankowej, przeprowadzono badania serologiczne. Rozszerzalność aorty, odkształcenie aorty i wskaźnik sztywności aorty obliczono na podstawie badania echokardiograficznego.

Wyniki: Odkształcenie i rozszerzalność aorty były istotnie mniejsze u pacjentów z CD niż u osób z grupy kontrolnej [odpowiednio 0,07 (0,03–0,14) vs. 0,09 (0,06–0,15), $p < 0,001$; $0,0036 \pm 0,0012$ vs. $0,0051 \pm 0,0014$, $p < 0,001$]. Z kolei wskaźnik sztywności aorty był istotnie wyższy u pacjentów z CD niż w grupie kontrolnej [1,14 (0,57–2,69) vs. 0,91 (0,59–1,92), $p = 0,002$]. Choroba trzewna była jedyną niezależną zmienną skorelowaną z odkształceniem aorty, wskaźnikiem sztywności aorty i rozszerzalnością aorty (odpowiednio $\beta = -0,427$; $p < 0,001$; $\beta = 0,375$, $p = 0,003$; $\beta = -0,434$; $p < 0,001$).

Wnioski: Na podstawie badania echokardiograficznego wykazano, że u pacjentów z CD występuje nieprawidłowa czynność aorty, która jest czynnikiem predykcyjnym subklinicznej miażdżycy. Ze względu na fakt, że upośledzenie czynności aorty jest silnym czynnikiem predykcyjnym wystąpienia w przyszłości zdarzeń sercowo-naczyniowych, w leczeniu osób z CD konieczna jest ścisła współpraca między kardiologami a gastroenterologami. Potrzebne jest również zwiększenie wśród pacjentów i lekarzy wiedzy na temat związanych z tym schorzeniem czynników ryzyka choroby niedokrwiennej serca.

Słowa kluczowe: czynność aorty, choroba trzewna, echokardiografia

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