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АКТИВНОСТЬ ЗАБОЛЕВАНИЯ КАК ФАКТОР РИСКА ПОВЫШЕНИЯ АРТЕРИАЛЬНОЙ ЖЕСТКОСТИ У ПАЦИЕНТОВ С ЯЗВЕННЫМ КОЛИТОМ

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Disease Activity as A Risk Factor for Increased Arterial Stiffness in Patients with Ulcerative Colitis

Резюме

Цель исследования. Изучить показатели артериальной жесткости и ее вариабельности при суточном мониторировании в зависимости от клинической и эндоскопической активности язвенного колита. **Материалы и методы.** В поперечном ретроспективном исследовании участвовали 100 пациентов с язвенным колитом (средний возраст 40 [33; 49] лет, 38 (38 %) мужчин, длительность заболевания — >1 года, без сопутствующих сердечно-сосудистых и метаболических нарушений) и 50 здоровых лиц контрольной группы. Параметры артериальной жесткости оценивались с помощью суточного мониторирования артериального давления с использованием технологии Vasotens. Пациенты были разделены на 3 группы по активности язвенного колита: 1-я группа включала 30 пациентов с клинической и эндоскопической ремиссией, 2-я группа — 22 пациента с клинической ремиссией и эндоскопической активностью, 3-я группа — 48 человек с клиническим и эндоскопическим обострением. Статистическая обработка полученных данных проводилась с помощью пакета компьютерных программ «IBM SPSS Statistics Version 25.0». **Результаты.** У 67 (67 %) пациентов с язвенным колитом выявлена повышенная артериальная жёсткость (скорость распространения пульсовой волны в аорте (PWVao) >10 м/с), что значимо чаще, чем в контрольной группе (OR=4,74; p <0,001). Наиболее выраженные изменения отмечены в группе с клинико-эндоскопическим обострением: повышены скорость распространения пульсовой волны в аорте, приведенная к систолическому артериальному давлению (САД)=100 мм рт.ст. и частоте сердечных сокращений (ЧСС)=60 уд/мин ($PWVao_{100\text{-}60)}$) (p=0,003), индекс аугментации при ЧСС=75 уд/мин (AIx75) (p <0,001) и вариабельность PWVao (p=0,002). При эндоскопической активности без клинических симптомов величины AIx75, $PWVao_{100\text{-}60}$ и вариабельность скорости нарастания артериального давления в аорте (dP/dt вар.) были значимо выше, чем в группе контроля. При регрессионном логистическом анализе выявлено, что возраст старше 40 лет (p=0,001; 95 % ДИ: 2,045 — 15,309), активность язвенного колита (p=0,025; 95 % ДИ: 1,151 — 8,200), отягощенная наследственность по сердечно-сосудистым заболеваниям (p=0,033; 95 % ДИ: 1,131 — 17,312) являются предикторами повышения артериальной жесткости у пациентов с язвенным колитом. Модель демонстрирует хорошую прогностическую способность: площадь под ROC-кривой (AUC) = 0,76 ± 0,051 [95 % ДИ: 0,66 — 0,86], чувствительность — 0,851, специфичность — 0,638, точность — 0,747 (p <0,001). **Заключение.** У пациентов с язвенным колитом наблюдается значимое повышение артериальной жёсткости, коррелирующее с активностью воспалительного процесса. Эндоскопическое обострение в отсутствии симптомов язвенного колита уже ассоциировано с неблагоприятными сосудистыми изменениями. Разработанная модель прогнозирования повышенной артериальной жесткости на основе трёх клинических критериев (возраст >40 лет, обострение язвенного колита, наследственность) может быть внедрена в практику для раннего выявления пациентов с высоким сердечно-сосудистым риском.

Ключевые слова: *язвенный колит, артериальная жёсткость, скорость пульсовой волны, индекс аугментации, суточная вариабельность, сердечно-сосудистый риск*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Objective. We aimed to study the parameters of arterial stiffness and its variability during 24-hour monitoring in relation to the clinical and endoscopic activity of ulcerative colitis. **Materials and methods.** This cross-sectional retrospective study involved 100 patients with ulcerative colitis (mean age 40 [33; 49] years, 38 (38 %) men, disease duration >1 year, without concomitant cardiovascular diseases or metabolic disorders) and 50 healthy control subjects. Arterial stiffness parameters were assessed via 24-hour ambulatory blood pressure monitoring with Vasotens technology. Based on disease activity patients were divided into 3 groups: Group 1 included 30 patients in clinical and endoscopic remission; Group 2 consisted of 22 patients in clinical remission with endoscopic activity; Group 3 included 48 patients with both clinical and endoscopic exacerbation. Statistical analysis was performed using the software package "IBM SPSS Statistics Version 25.0". **Results**. Increased arterial stiffness (aortic pulse wave velocity (PWVao) >10 m/s) was detected in 67 (67 %) of ulcerative colitis patients, which was significantly higher than in the control group (OR = 4.74; p<0.001). The most pronounced alterations were observed in the group with clinical-endoscopic exacerbation, which showed increased aortic pulse wave velocity at systolic blood pressure (SBP)=100 mmHg and heart rate (HR)=60 bpm (PWVao 100-60) (p=0.003), augmentation index adjusted for HR=75 bpm (AIx75) (p<0.001), and PWVao variability (p=0.002). In endoscopically active disease without clinical symptoms, AIx75, PWVao100-60, and variability of the rate of increase in blood pressure in the aorta (dP/dt var.) were significantly higher compared to controls. Logistic regression analysis identified age >40 years (p=0.001; 95 % CI: 2.045 to 15.309), disease activity of ulcerative colitis (p=0.025; 95 % CI: 1.151 to 8.200), and a positive family history of cardiovascular disease (p=0.033; 95 % CI: 1.131 to 17.312) as independent predictors of increased arterial stiffness in patients with ulcerative colitis. The prediction model demonstrated good performance: area under the ROC curve (AUC) = 0.76 ± 0.051 (95 % CI: 0.66–0.86), sensitivity 0.851, specificity 0.638, and accuracy 0.747 (p <0.001) and can be implemented in practice for the early identification of patients with high cardiovascular risk. **Conclusion**. Patients with ulcerative colitis exhibit a significant increase in arterial stiffness, which correlates with the degree of inflammatory activity. Endoscopic activity, even in the absence of clinical symptoms, is associated with adverse vascular changes. The developed predictive model, based on three clinical criteria (age >40 years, active ulcerative colitis, and positive family history), could be implemented clinically for the early identification of high cardiovascular risk in this patient population.

Key words: *ulcerative colitis, arterial stiffness, pulse wave velocity, augmentation index, 24-hour variability, cardiovascular risk*

Conflict of Interest

The authors declare that this work, its topic, subject matter, and content do not affect any competing interests.

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Chita State Medical Academy

Compliance with the principles of ethics

The study was approved by the local ethics committee of Chita State Medical Academy (protocol no. № 125 dated November 23, 2022). Written consent was obtained from the patients for publication of relevant medical information and all of accompanying images within the manuscript.

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AH — arterial hypertension, BP — blood pressure, IBD — inflammatory bowel diseases, SBP — systolic blood pressure, ABPM — 24-hour ambulatory blood pressure monitoring, CVD — cardiovascular diseases, PWV — pulse wave velocity, HR — heart rate, UC — ulcerative colitis

Introduction

Over recent decades, it has become increasingly evident that the systemic inflammation underlying ulcerative colitis (UC) is associated with an increased risk of cardiovascular disease (CVD) [1–3]. At the same time, conventional cardiovascular risk assessment tools have proven to be insufficiently effective in patients with inflammatory bowel disease (IBD), as they tend to underestimate the true risk of cardiovascular events in this population [4].

Arterial stiffness is an early, independent, and powerful predictor of cardiovascular events and may be used to predict CVD risk [5]. Proinflammatory cytokines, oxidative stress, and endothelial dysfunction are the main factors influencing arterial stiffness and contributing to the development of premature atherosclerosis in patients with UC [4, 6]. Recent studies have demonstrated that increased arterial stiffness is associated with both disease duration and the frequency of UC exacerbations [7, 8]. Specifically, the principal marker of arterial stiffness, that is pulse wave velocity (PWV), has been shown to increase by 0.205 m/s for each additional year of IBD duration [9]. In a multicentre prospective study, Zanoli et al. (2019) found that patients with IBD had higher PWV values vs. controls and that indices of arterial stiffness could decrease following long-term treatment with tumour necrosis factor-α (TNF-α) antagonists and with the achievement of remission [10].

Assessment of cardiovascular risk should also take into account increased variability in vascular wall stiffness, which itself has prognostic significance. For example, in patients with hypertension, the greatest variability in arterial stiffness has been observed among those at high and very high cardiovascular risk [11]. According to the available literature, variability in arterial stiffness has not previously been evaluated in patients with UC.

Investigation of arterial stiffness parameters and their variability according to the degree of UC activity may facilitate the identification of clinical predictors of early cardiovascular disease in this patient population.

Aim

The aim of the study was to investigate arterial stiffness parameters and their variability during 24-hour ambulatory monitoring in relation to the clinical and endoscopic activity of UC.

Materials and methods

A cross-sectional, retrospective study of arterial stiffness parameters was conducted in 100 patients with UC (median age 40 [33–49] years; 38 (38 %) were men). The inclusion criteria were age between 20 and 50 years, a duration of UC greater than 1 year (diagnosed in accordance with the 2024 Ulcerative Colitis Clinical Guidelines issued by the Russian Gastroenterological Association and the Russian Association of Coloproctologists) [12], and provision of informed consent to participate in the study. The exclusion criteria included severe UC exacerbation; systemic diseases and malignancies of any localisation, haematologic disorders, endocrine diseases, pregnancy and lactation; grade 2–3 arterial hypertension and other cardiovascular diseases; class II–III alimentary-constitutional obesity; a history of colectomy or proctocolectomy; treatment with glucocorticoids, antihypertensive agents, or lipid-lowering drugs; severe renal or hepatic insufficiency; and other chronic diseases in the stage of exacerbation. The control group consisted of 50 healthy individuals matched to the study group with respect to age and sex. All subjects provided informed written consent. The study protocol (No. 125) was approved by the Local Ethics Committee of the Chita State Medical Academy of the Ministry of Health of the Russian Federation on November 23, 2022. All participants underwent a comprehensive clinical evaluation, including assessment of medical history and physical status, lipid profile analysis, 24-hour ambulatory blood pressure monitoring (ABPM), and video ileocolonoscopy. Cardiovascular risk was assessed using the relative risk charts for individuals younger than 40 years and the SCORE2 scale for those aged 40 years and older. Patients with UC and controls had similar clinical characteristics (Table 1). During ABPM, patients with UC exhibited higher mean 24-hour systolic blood pressure values compared with the control group ($p < 0.05$). It should be noted that, according to medical history data, none of the study participants had a prior diagnosis of arterial hypertension (AH). Labile grade 1 hypertension, characterised by episodic BP elevations during ambulatory monitoring despite normal mean 24-hour blood pressure values, was detected significantly more frequently in patients with UC than in the control group (OR 4.04; 95 % CI: 1.46–11.17; $p = 0.005$). This finding is consistent with previous reports indicating an increased risk of AH in patients with UC [13]. No significant differences were observed between the groups with respect to the magnitude or prevalence of other cardiovascular risk factors.

The higher prevalence of chronic gastrointestinal diseases (including gastritis, cholecystitis, pancreatitis, gastroesophageal reflux disease, peptic ulcer disease, Gilbert syndrome, and cholelithiasis) observed among patients with UC is likely attributable to more intensive and regular follow-up by gastroenterologists, which facilitates the detection of concomitant gastrointestinal pathology.

Patients with UC had various courses of the disease: continuous course was reported in 22 % (22/100), recurrent — in 48 % (48/100), and remission lasting for over one year — in 30 % (30/100). All patients received continuous anti-inflammatory therapy: 57 % (57/100) were treated with 5-aminosalicylic acid derivatives, 20 % (20/100) — with immunosuppressive agents (azathioprine or 6-mercaptopurine), and 23 % (23/100) — with biologic therapies.

Arterial stiffness parameters were assessed using the results of ABPM performed with the BPLabWin device (Petr Telegin LLC, Nizhny Novgorod, Russia) and the Vasotens 24 software package. The following 24-hour indices of arterial stiffness were analysed: mean aortic pulse wave velocity and its variability (PWVao, PWVao var.), ambulatory arterial stiffness index (AASI), augmentation index and its variability (AIx, AIx var.), mean pulse pressure and its variability (mean PP, PP var.), and mean rate of blood pressure rise and its variability (dP/dt, dP/dt var.). Since vascular stiffness parameters are strongly influenced by the current blood pressure (BP) level and heart rate (HR), the analysis used values normalised to a systolic blood pressure (SBP) of 100 mm Hg and a heart rate of 60 beats/min, namely RWTT100-60 and PWVao100-60. In addition, the augmentation index was adjusted to a heart rate of 75 beats/min (AIx75).

Statistical processing of the data was performed using the IBM SPSS Statistics software, version 25.0 (International Business Machines Cation, USA). The distributions of the variables were tested for normality using

the Kolmogorov-Smirnov test (for comparisons between groups) and the Shapiro-Wilk test (for comparisons between subgroups). As the quantitative variables were not normally distributed, they were expressed as the median and interquartile range (Me [Q_1; Q_3]). Comparisons of continuous variables between the ulcerative colitis and control groups were performed using the Mann-Whitney U test, whereas comparisons among multiple groups were conducted using the Kruskal-Wallis H test. To address the issue of multiple comparisons, pairwise comparisons were subsequently performed using the Bonferroni correction, with a significance threshold of p = 0.0083. Qualitative attributes are presented as absolute numbers and percentages (%). Comparisons of qualitative attributes were performed using Pearson's χ^2 test, and, for small sample sizes, the likelihood ratio correction was applied. The significance of differences was assessed using odds ratios (ORs) with corresponding 95 % confidence intervals (95 % CI). Differences were considered statistically significant at $p < 0.05$.

Table 1. *Clinical Characteristics of the Study Participants*

Parameter	Patients with UC (n=100)	Control (n=50)	Test Statistics
Age, years	40 [33; 49]	40[35; 46]	U=2285,0, p=0,391.
Sex male/female, n (%)	38/62 (38/62)	12/38 (24/76)	χ^2=2,94, df=1, p=0,09.
SBP, mm Hg	116 [109; 121]	111 [103; 115]	**U=1835,0, p=0,008.**
DBP, mm Hg	74 [69; 79]	72,5 [67; 77]	U=2123,5, p=0,133.
Labile AG 1st grade, n (%)	31 (31 %)	5 (10 %)	**χ^2=8,059, df=1, p=0,005**
BMI, kg/m²	23,8 [21,0; 27,1]	23,2 [21,0; 24,3]	U=2100,0, p=0,110.
WC, cm	84 [78; 90]	83 [78; 86]	U=2208,5, p=0,244.
Smoking, n (%)	8 (8 %)	6 (12 %)	χ^2=0,20, df=1, p=0,65
FH of CVD, n (%)	27 (27 %)	11 (22 %)	χ^2=0,49, df=1, p=0,49
TC, mmol/L	5,0 [4,2; 5,7]	5,1 [4,3; 5,9]	U=1420,5, p=0,885.
LDL-C, mmol/L	3,2 [2,7; 3,8]	3,5 [2,8; 3,9]	U=1273,5, p=0,934
HDL-C, mmol/L	1,3 [1,1; 1,6]	1,3 [1,2; 1,5]	U=1134,0, p=0,226.
VLDL-C, mmol/L	0,5 [0,3; 0,6]	0,6 [0,3; 0,7]]	U=752,5, p=0,356.
TG, mmol/L	1,0 [0,7; 1,3]	1,2 [0,7;1,6]	U=1150,0, p=0,599.
Cardiovascular risk:			
– Low, n (%)	55 (55 %)	32(64 %)	χ2=2,24, df=2, p=0,33
– Moderate, n (%)	35(35 %)	16 (32 %)	
– High, n (%)	10(10 %)	2 (4 %)	
Comorbidities, n (%):			
– Chronic diseases of the gastrointestinal tract;	28 (28 %)	4 (8 %)	**χ2=18,82, df=7, p=0,009**
– Bronchial asthma;	1 (1 %)	1 (2 %)	
– Nodular goiter;	1 (1 %)	1 (2 %)	
– Deforming osteoarthritis;	4 (1 %)	0	
– osteochondrosis;	5 (5 %)	6 (12 %)	
– Hemorrhoids;	3 (3 %)	10 (20 %)	
– Pyelonephritis	5 (5 %)	1 (2 %)	

Note: Data are presented as median [Q_1; Q_3], n (%); U — Mann-Whitney U test, χ^2 — Pearson's chi-square test, df — degrees of freedom, SBP — systolic blood pressure, DBP — diastolic blood pressure, AH– arterial hypertension, BMI — body mass index; WC — waist circumference; CVD — cardiovascular diseases; TC — total cholesterol; LDL-C — low-density lipoprotein cholesterol; HDL-C — high-density lipoprotein cholesterol; VLDL-C — very low-density lipoprotein cholesterol; TG — triglycerides.

Binary logistic regression analysis was used to identify predictors of increased arterial stiffness. A ROC analysis was performed to evaluate the sensitivity, specificity, and overall accuracy of the predictive model.

Results

In clinical practice, increased arterial stiffness is defined by an aortic pulse wave velocity exceeding 10 m/s [5]. According to this criterion, increased arterial stiffness was detected in 67 (67 %) patients with UC, which was 2.2 times more frequent than in controls (OR 4.74; 95 % CI: 2.27–9.87; p < 0.001). Comprehensive assessment of arterial stiffness parameters revealed that patients with UC had significantly higher values of aortic pulse wave velocity normalised to a systolic blood pressure of 100 mm Hg and a heart rate of 60 beats/min (PWVao100-60), which was increased by 1.13 [1.09; 1.15]-fold (p < 0.001), and greater PWV variability, which was elevated by 1.23 [1.09; 1.29]-fold (p = 0.002). The augmentation index (AIx75) was 1.87 [1.29; 1.97]-fold higher (p = 0.001), whereas the variability of pulse pressure and the variability of the rate of blood pressure rise (dP/dt var.) were increased by 1.11 [1.02; 1.14]-fold (p = 0.002) and 1.14 [1.13; 1.25]-fold (p = 0.005), respectively, compared with the control group (Table 2).

According to the Mayo score (a composite index used to assess ulcerative colitis activity) and the Schroeder endoscopic classification, the patients were divided into three groups: group 1 comprised 30 patients with both clinical and endoscopic remission; group 2 included 22 patients with clinical remission but persistent endoscopic activity; and group 3 consisted of 48 patients with both clinical and endoscopic exacerbation.

Table 2. *Arterial stiffness parameters in patients with ulcerative colitis*

Investigated indicators	Controls, n=50	Patients with UC, n=100	Test statistics	
			Mann–Whitney U test	P Value
PP, mm Hg	38,0 [34,0; 42;0]	40,5 [35,0; 44,8]	2023,5	0,084
PP var., mm Hg	9,0 [7,0; 11,0]	10,0 [8,0; 11;2]	**1950,5**	**0,033**
AIx75, %	-78,5 [-100,0; -32,0]	-42,0 [-60,0; -23,5]	**1658,5**	**0,001**
AIx75 var., %	16,0 [15,0; 21,0]	20,0 [16,0; 23,0]	1907,5	0,051
AASI	0,45 [0,30; 0,64]	0,40 [0,27; 0,60]	2216,5	0,258
PWVao100-60, m/s	9,0 [8,8; 9,6]	10,2 [9,6; 11,0]	**1346,0**	**<0,001**
PWVao var., m/s	1,3 [1,1; 1,7]	1,6 [1,2; 2,2]	**1720,0**	**0,002**
RWTT100-60, ms	145,5 [138,8; 171,3]	150,5 [137,3; 167,0]	2444,0	0,823
RWTT var., ms	18,5 [15,8; 24,0]	19,0 [15,0; 23,5]	2435,5	0,797
dP/dT, mm Hg/s	465,0 [403,0; 518,0]	468,5 [403,8; 570,5]	2203,5	0,237
dP/dT var., mm Hg/s	134,0 [102,5; 158,8]	152,5 [128,0; 188,8]	**1799,0**	**0,005**

Note: data are presented as median [Q1; Q3]; PP — pulse pressure, PP var. — pulse pressure variability, AIx75 — augmentation index adjusted for heart rate 75 bpm; AIx75 var. — augmentation index variability; AASI — ambulatory arterial stiffness index, PWVao100-60 — aortic pulse wave velocity at SBP=100 mmHg and HR=60 bpm., PWVao var. — aortic pulse wave velocity variability, RWTT100-60 — reflected wave transit time in the aorta at SBP=100 mmHg and HR=60 bpm, RWTT var. — reflected wave transit time variability, dP/dT — rate of increase in blood pressure in the aorta., dP/dt var. — variability of the rate of increase in blood pressure in the aorta.

Table 3. *Arterial stiffness parameters in patients with ulcerative colitis depending on the inflammatory activity*

Investigated indicators	Control group, n=50	Study groups			Test statistics		
		Group 1 (patients with clinical and endoscopic remission of UC) n=30	Group 2 (patients with clinical remission and endoscopic exacerbation of UC) n=22	Group 3 (patients with clinical and endoscopic exacerbation of UC) n=48	The Kruskal-Wallis test, df=3	Mann-Whitney U Test	
						Comparison with the control group	Comparison of the studied groups
	c	1	2	3			
PP, mm Hg	38,0 [34,0; 42,0]	40,0 [35,8; 45,8]	41,5 [34,0; 43,5]	40,0 [34,3; 45,0]	H=3,11, p=0,38	$U_{c\text{-}1}$=509,5, $p_{c\text{-}1}$=0,016; $U_{c\text{-}2}$=429,0; $p_{c\text{-}2}$=0,134; $U_{c\text{-}3}$=999,0, $p_{c\text{-}3}$=0,236.	$U_{1\text{-}2}$=321, $p_{1\text{-}2}$=0,867; $U_{1\text{-}3}$=689, $p_{1\text{-}3}$=0,75; $U_{2\text{-}3}$=511,5, $p_{2\text{-}3}$=0,834.
PP var., mm Hg	9,0 [7,0; 11,0]	10,0 [9,0; 12,0]	10,0 [9,0; 10,0]	9,0 [8,0; 11,0]	H=6,78, p=0,08	$U_{c\text{-}1}$=589,5, $p_{c\text{-}1}$=0,141; $U_{c\text{-}2}$=435,0, $p_{c\text{-}2}$=0,194; $U_{c\text{-}3}$=1012,0, $p_{c\text{-}3}$=0,236.	$U_{1\text{-}2}$=283,5, $p_{1\text{-}2}$=0,381; $U_{1\text{-}3}$=564,5, $p_{1\text{-}3}$=0,138; $U_{2\text{-}3}$=459,5, $p_{2\text{-}3}$=0,454.
AIx75, %	-78,5 [-100,0; -32,0]	-49,5 [-65,0; -31,3]	-35,5 [-51,3; -26,0]	-32,5 [-50,3; -16,5]	**H=18,07, p<0,001**	$U_{c\text{-}1}$=953,5, $p_{c\text{-}1}$=0,073; **$U_{c\text{-}2}$=319,0, $p_{c\text{-}2}$=0,004; $U_{c\text{-}3}$=386,0, $p_{c\text{-}3}$<0,001.**	$U_{1\text{-}2}$=298,0, $p_{1\text{-}2}$=0,553; **$U_{1\text{-}3}$=461,0, $p_{1\text{-}3}$=0,008;** $U_{2\text{-}3}$=402,0, $p_{2\text{-}3}$=0,11.
AIx75 var., %	16,0 [15,0; 21,0]	18,0 [15,0; 22,3]	20,0 [16,0; 23,0]	19,5 [16,0; 23,0]	H=3,54, p=0,32	$U_{c\text{-}1}$=625, $p_{c\text{-}1}$=0,264; $U_{c\text{-}2}$=425; $p_{c\text{-}2}$=0,154; $U_{c\text{-}3}$=946,5, $p_{c\text{-}3}$=0,097;	$U_{1\text{-}2}$=312, $p_{1\text{-}2}$=0,738; $U_{1\text{-}3}$=688, $p_{1\text{-}3}$=0,742; $U_{2\text{-}3}$=521,5, $p_{2\text{-}3}$=0,934;
AASI	0,45 [0,30; 0,64]	0,42 [0,29; 0,63]	0,45 [0,31; 0,64]	0,35 [0,26; 0,50]	H=3,45, p=0,33	$U_{c\text{-}1}$=694,5, $p_{c\text{-}1}$=0,581; $U_{c\text{-}2}$=541,0, $p_{c\text{-}2}$=0,912; $U_{c\text{-}3}$=963,0, $p_{c\text{-}3}$=0,092.	$U_{1\text{-}2}$=298,5, $p_{1\text{-}2}$=0,528; $U_{1\text{-}3}$=629,0, $p_{1\text{-}3}$=0,35; $U_{2\text{-}3}$=422,0, $p_{2\text{-}3}$=0,18.
PWVao100-60, m/s	9,0 [8,8; 9,6]	10,0 [8,8; 10,5]	10,6 [10,0; 11,2]	10,4 [9,8; 11,7]	**H=33,19, p<0,001**	$U_{c\text{-}1}$=894,0, $p_{c\text{-}1}$=0,03; **$U_{c\text{-}2}$=327,5, $p_{c\text{-}2}$=0,006; $U_{c\text{-}3}$=451,5, $p_{c\text{-}3}$=0,003.**	$U_{1\text{-}2}$=329,5, $p_{1\text{-}2}$=0,993; **$U_{1\text{-}3}$=454,5, $p_{1\text{-}3}$=0,006;** $U_{2\text{-}3}$=487,5, $p_{2\text{-}3}$=0,608.
PWVao var., m/s	1,3 [1,3; 1,7]	1,5 [1,2; 2,2]	1,7 [1,4; 2,0]	1,8 [1,4; 2,2]	**H=11,92, p=0,008**	$U_{c\text{-}1}$=949,0, $p_{c\text{-}1}$=0,074; **$U_{c\text{-}2}$=328,0; $p_{c\text{-}2}$=0,006; $U_{c\text{-}3}$=443,0, $p_{c\text{-}3}$=0,002**.	$U_{1\text{-}2}$=308,5, $p_{1\text{-}2}$=0,689; $U_{1\text{-}3}$=598,5, $p_{1\text{-}3}$=0,211; $U_{2\text{-}3}$=454,0, $p_{2\text{-}3}$=0,348.
RWTT100-60, ms	145,5 [138,8; 171,3]	148,5 [129,0; 160,8]	150,0 [141,8; 158,0]	156,5 [139,3; 171,5]	H=3,56, p=0,31	$U_{c\text{-}1}$=652,5, $p_{c\text{-}1}$=0,332; $U_{c\text{-}2}$=513,0, $p_{c\text{-}2}$=0,651; $U_{c\text{-}3}$=1083,5, $p_{c\text{-}3}$=0,408.	$U_{1\text{-}2}$=295,5, $p_{1\text{-}2}$=0,266; $U_{1\text{-}3}$=524,5, $p_{1\text{-}3}$=0,045; $U_{2\text{-}3}$=456, $p_{2\text{-}3}$=0,362.
RWTT var., ms	18,5 [15,8; 24,0]	19,0 [15,8; 21,0]	21,5 [17,0; 24,3]	18,0 [15,0; 25,0]	H=1,88, p=0,60	$U_{c\text{-}1}$=724,5, $p_{c\text{-}1}$=0,799; $U_{c\text{-}2}$=463,0, $p_{c\text{-}2}$=0,287; $U_{c\text{-}3}$=1197,0, $p_{c\text{-}3}$=0,983.	$U_{1\text{-}2}$=246,0, $p_{1\text{-}2}$=0,119; $U_{1\text{-}3}$=710,0, $p_{1\text{-}3}$=0,918; $U_{2\text{-}3}$=447,5, $p_{2\text{-}3}$=0,307.
dP/dT, mm Hg/s	465,0 [403,0; 518,0]	472,5 [412,8; 591,3]	477,0 [402,0; 565,3]	464,5 [384,0; 580,8]	H=1,55, p=0,67	$U_{c\text{-}1}$=647,5, $p_{c\text{-}1}$=0,308; $U_{c\text{-}2}$=469,0; $p_{c\text{-}2}$=0,322; $U_{c\text{-}3}$=1087,0, $p_{c\text{-}3}$=0,422.	$U_{1\text{-}2}$=328,5, $p_{1\text{-}2}$=0,978; $U_{1\text{-}3}$=687,5, $p_{1\text{-}3}$=0,739; $U_{2\text{-}3}$=504,5, $p_{2\text{-}3}$=0,766.
dP/dT var., mm Hg/s	134,0 [102,5; 158,8]	148,0 [128,0; 181,8]	159,0 [146,5; 181,0]	147,0 [114,0; 194,5]	**H=9,82, p=0,02**	$U_{c\text{-}1}$=544,5, $p_{c\text{-}1}$=0,041; **$U_{c\text{-}2}$=311,0, $p_{c\text{-}2}$=0,003;** $U_{c\text{-}3}$=943,5, $p_{c\text{-}3}$=0,068.	$U_{1\text{-}2}$=270,0, $p_{1\text{-}2}$=0,266; $U_{1\text{-}3}$=690,5, $p_{1\text{-}3}$=0,762; $U_{2\text{-}3}$=424,5, $p_{2\text{-}3}$=0,19.

Note: data are presented as median [Q1; Q3]; df — degrees of freedom; PP — pulse pressure, PP var. — pulse pressure variability, AIx75 — augmentation index adjusted for heart rate 75 bpm; AIx75 var. — augmentation index variability; AASI — ambulatory arterial stiffness index, PWVao100-60 — aortic pulse wave velocity at SBP=100 mmHg and HR=60 bpm., PWVao var. — aortic pulse wave velocity variability, RWTT100-60 — reflected wave transit time in the aorta at SBP=100 mmHg and HR=60 bpm, RWTT var. — reflected wave transit time variability, dP/dT — rate of increase in blood pressure in the aorta., dP/dt var. — variability of the rate of increase in blood pressure in the aorta

Table 4. *Significance of predictors in the prognostic model, df = 1*

Variables	B	Std. Error	Wald	P-value	Exp(B)	95 % Confidence Interval for Exp(B)
Age >40 years	1.722	0.514	11.246	0.001	5.596	2.045 — 15.309
UC activity	1.123	0.501	5.024	0.025	3.073	1.151 — 8.200
Family history of CVD	1.487	0.696	4.565	0.033	4.425	1.131 — 17.312
Constant	-1.000	0.433	5.342	0.021	0.368	

Note: B — unstandardized logistic regression coefficient; Стд. ошибка — standard error of the coefficient; Вальд — Wald statistic; Знач. — significance level (p-value); Exp(B) — odds ratio; 95 % Дов. интервал для EXP(B) — 95 % confidence interval for the odds ratio; ЯК — ulcerative colitis; ССЗ — cardiovascular diseases

Analysis of arterial stiffness parameters according to the pattern of UC activity revealed that AIx75 in controls was lower than that in group 3 by 2.41 [1.94; 2.98]-fold ($p < 0.001$) and lower than that in group 2 by 2.21 [1.23; 2.95]-fold ($p = 0.004$), whereas it did not differ significantly from the value observed in group 1 ($p > 0.0083$). Pairwise comparisons between the study groups demonstrated significant differences in AIx75 between groups 1 and 3, with group 3 exhibiting values that were 1.52 [1.29; 1.89]-fold higher ($p < 0.0083$). The PWVao100-60 value in the control group was lower than that in group 2 by 1.18 [1.07; 1.87]-fold ($p = 0.006$) and lower than that in group 3 by 1.16 [1.11; 1.22]-fold ($p = 0.003$), whereas no statistically significant difference was found compared with group 1 ($p > 0.0083$). Pairwise comparisons among the UC groups revealed significant differences in PWVao100-60 between groups 1 and 3, with the value being 1.04 [1.11; 1.12]-fold higher in group 3 ($p = 0.006$). The variability of PWVao100-60 in the control group was lower than that in group 3 by 1.38 [1.08; 1.64]-fold ($p = 0.002$) and lower than that in group 2 by 1.31 [1.08; 1.59]-fold ($p = 0.006$), whereas it did not differ significantly from the corresponding value in group 1 ($p > 0.0083$). Pairwise comparisons among the UC groups did not reveal any statistically significant differences in PWVao100-60 variability. The variability of dP/dt in the control group was 1.19 [1.15; 1.43]-fold lower than that in group 2 ($p = 0.002$) and did not differ significantly from the values observed in groups 1 and 3 ($p > 0.0083$). Likewise, pairwise comparisons among the study groups showed no statistically significant differences in dP/dt variability (Table 3).

In order to assess the effect of clinical and endoscopic UC exacerbation on the risk of increased arterial stiffness, logistic regression analysis was performed. During model development, a rigorous multistep variable selection algorithm was implemented, taking into account statistical, clinical, and methodological considerations. Initially, the following variables were considered as potential predictors: demographic characteristics (age and sex), medical history data (duration of UC and a family history of CVDs), clinical characteristics (smoking status and body mass index), disease activity (clinical and endoscopic exacerbation according to the Mayo score and the Schroeder classification), and morphological features (extent of colonic involvement). To minimise the influence of multicollinearity on the stability of the regression coefficient estimates, the following variables were excluded from the model: duration of disease (hypothetical VIF = 7.3), the presence of extensive colonic involvement (VIF = 8.1), and smoking status (VIF = 5.8). The final model included three predictors, including UC activity, all of which met the criterion for statistical significance ($p < 0.05$) and demonstrated independent prognostic value (Table 4).

The developed model for predicting increased arterial stiffness is represented by the following logistic regression equation:

$$y = \frac{1}{1 + e^{1 - 1{,}722 \times age - 1{,}123 \times UC\ activity - 1{,}487 \times familiy\ hostory}},$$

where y is the probability coefficient for the presence of increased arterial stiffness; e is the base of the natural logarithm ($e \approx 2.72$); –1 is the constant term (regression coefficient b0); 1.722, 1.123, and 1.487 are the unstandardised regression coefficients (b). Age is a variable, which is assigned a value of “1” for patients older than 40 years at the time of the study and “0” otherwise. UC activity indicates the presence of a clinical and endoscopic exacerbation of ulcerative colitis and is coded as “1” when present and “0” when absent. Family history reflects the presence of a positive family history of CVD and is assigned a value of “1” if there is a history of premature CVD among family members and “0” if such a history is absent. When the value of the coefficient y exceeds 0.646, the likelihood of increased arterial stiffness rises by 7.368-fold (95 % CI: 2.486–21.843; $p < 0.001$). The developed model for early diagnosis demonstrated a sensitivity of 0.851, a specificity of 0.638,

and an overall accuracy of 0.747. The model exhibits good diagnostic performance, with an area under the ROC curve (AUC) of 0.76±0.051 (95 % CI: 0.66–0.86; $p < 0.001$) (Figure 1) [14].

To minimise overfitting and assess model robustness, internal validation was performed using 5-fold cross-validation and evaluation on an independent test set generated by stratified random splitting (seed = 42). The original cohort (n = 100) was divided into training (n = 70) and test (n = 30) subsets while preserving the proportion of patients with increased arterial stiffness (67 %). The logistic regression model incorporating the predictors age > 40 years, UC activity, and a positive family history of CVDs demonstrated stable performance: AUC was 0.77 (95 % CI: 0.65–0.89) in the training set, 0.74 (95 % CI: 0.55–0.92) in the test set, and 0.73±0.04 in cross-validation. Sensitivity was 86.7 %, 80.0 %, and 82.1 %, specificity was 64.3 %, 60.0 %, and 61.5 %, and overall accuracy was 75.7 %, 70.0 %, and 72.3 %, respectively. The optimal probability threshold, determined according to the Youden criterion, was 0.646 in all cases. A slight decrease in AUC (Δ = 0.03) and the consistency of the performance metrics indicate a low risk of overfitting and good generalisability of the model despite the limited sample size. These findings are consistent with the results of bootstrap validation and hypothetical external validation, thereby increasing confidence in the model's potential clinical applicability.

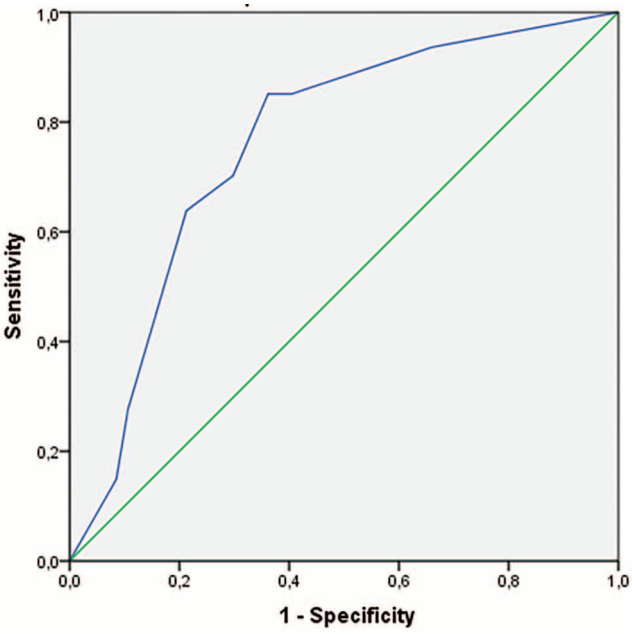

Figure 1. *ROC curve of the early diagnostic model for increased arterial stiffness in patients with ulcerative colitis*

Discussion of results

The aortic pulse wave velocity PWVao is the rate, at which the pressure wave from a blood regurgitating from the left ventricle during systole spreads along the aorta and major arteries. This value depends primarily on arterial wall elasticity. An increase in pulse wave velocity reflects enhanced vascular stiffness, pathological remodelling of the vascular wall, and an elevated risk of cardiovascular complications. PWVao is considered the principal parameter for the assessment of arterial stiffness [5, 15]. The augmentation index (AIx) is a measure of vascular wall distensibility that correlates positively with aortic stiffness. AIx provides information regarding peripheral vascular resistance: the higher the index, the greater the resistance of the arterioles. This parameter increases with age and with the progression of atherosclerosis [15]. According to a large meta-analysis by Lu Q. et al. (2019), UC patients exhibit significantly higher PWVao values compared with controls [16]. Similarly, in our study, both PWVao and AIx were elevated in patients with UC. These parameters were higher in the presence of both clinical and endoscopic disease activity, as well as in patients without clinical manifestations but with evidence of active disease detected solely by endoscopic examination. In contrast, patients in both clinical and endoscopic remission had values comparable to those observed in controls.

PWVao values in patients with UK depend both on the age, presence of AH, intensity of smoking, and on the frequency of exacerbations during three years, and duration of ulcerative colitis [8]. The present study shows that arterial stiffness is affected by disease activity during examination. According to experimental data, arterial stiffness increases as a result of both transient factors, such as the effects of vasoactive hormones, inflammatory mediators, and markers of oxidative stress; and structural changes, including diffuse fibroelastic thickening of the intima and endothelial remodelling secondary to chronic inflammation. Endothelial dysfunction plays a central role in this process. The interaction among these components increases vascular wall stiffness during the direct action of inflammatory mediators on the vessel wall in patients with active UC. Prolonged exposure to inflammation ultimately leads to irreversible increases in arterial stiffness [15].

The issue of 24-hour variability in arterial stiffness parameters in UC patients remains insufficiently studied. In our study, these patients demonstrated increased variability in the following indices: pulse pressure (PP), PWVao, dP/dt. Variability in arterial stiffness parameters is determined by a combination of "passive" and "active" mechanisms regulating vascular tone. "Passive" factors include the intrinsic properties of the arterial wall, which are determined by the balance between elastin

and collagen, as well as the haemodynamic parameter of heart rate. In contrast, "active" regulation is mediated by vasoactive influences, including endothelium-dependent mechanisms, the intensity of inflammation and oxidative stress, and the level of sympathetic adrenergic activity [11]. In patients with ulcerative colitis, these "active" regulatory mechanisms, associated with inflammation and endothelial dysfunction, likely play a pivotal role in the increased variability of arterial stiffness parameters. In our study, the greatest increase in the variability of aortic pulse wave velocity was observed in patients with both clinical and endoscopic disease exacerbation. In contrast, UC patients in remission exhibited arterial stiffness variability parameters comparable to those of the control group. In patients of group 2, characterised by the presence of endoscopic disease activity alone, increased variability of PWVao and dP/dt was observed. These findings indicate that UC activity, including isolated endoscopic activity, affects the 24-hour variability of arterial stiffness. By analogy with blood pressure variability, increased variability in arterial stiffness parameters may have clinical significance by contributing to an elevated cardiovascular risk in these patients [11].

Studies evaluating CAVI in patients with UC have demonstrated that the condition of the vascular wall is influenced by the patient's current age, age at disease onset, disease duration, and endoscopic disease activity. Specifically, it has been shown that an increase of one point in the Schroeder endoscopic activity score is associated with an increase in CAVI of 0.11 above the age-adjusted normal value [17]. Analysis of our findings likewise indicates that the greater the inflammatory activity in ulcerative colitis, the higher the risk of increased arterial stiffness. The developed predictive model for increased arterial stiffness enables the identification of clinical predictors of CVDs in patients with UC. These predictors include clinical and endoscopic disease exacerbation, age over 40 years, and a positive family history of CVD. Further refinement of the prediction model for increased arterial stiffness may facilitate the timely identification of high-risk patients and optimisation of their management, thereby contributing to the prevention of CVDs in individuals with UC.

Conclusions

Patients with ulcerative colitis exhibit significantly increased arterial stiffness, correlating with inflammation activity. Endoscopic disease activity in the absence of clinical symptoms of UC is already associated with adverse vascular changes. The developed prediction model for increased arterial stiffness in patients with UC, based on three clinical criteria (age > 40 years, UC exacerbation, and a positive family history of cardiovascular disease), may be implemented in clinical practice for the early identification of individuals at high cardiovascular risk.

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Zhigula Z.M.: important contribution to the concept and design of the study, collection, analysis and interpretation of the data, writing of the article

Zhilina A.A.: important contribution to the concept and design of the study, critical revision and editing of the article, final approval of the version of the article for publication

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