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PERIODONTITIS–DRIVEN ENDOTHELIAL DYSFUNCTION IN CORONARY ARTERY DISEASE

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Patients with coronary artery disease and concomitant periodontitis exhibit more pronounced endothelial dysfunction compared to those with coronary artery disease alone. This prospective cohort study included 58 patients: 25 with coronary artery disease and 33 with coronary artery disease + periodontitis, conducted at Ivano–Frankivsk National Medical University from December 2023 to September 2024. In the coronary artery disease + periodontitis group, significantly higher levels of MMP–9 (126.76 [114.19; 146.21] ng/mL vs. 100.47 [81.22; 114.38] ng/mL, p<0.001) and TMAO (133.18 [111.27; 148.37] μmol/L vs. 91.80 [65.42; 109.53] μmol/L, p<0.001) were observed. Endothelial dysfunction was evidenced by lower flow-mediated dilation (9.92 [9.12; 12.24] % vs. 12.78 [10.52; 16.96] %, p=0.012), higher pulse-wave velocity (8.71 [7.93; 10.12] m/s vs. 6.53 [5.60; 8.90] m/s, p=0.011), and increased carotid intima-media thickness (0.76 [0.68; 0.81] mm vs. 0.62 [0.54; 0.73] mm, p=0.004). These results underscore the systemic impact of periodontitis on vascular health in coronary artery disease patients and suggest a need for integrated management strategies.

Key words: coronary artery disease, periodontitis, endothelial dysfunction, inflammation, cardiovascular risk.

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ДИСФУНКЦІЯ ЕНДОТЕЛІЮ, СПРИЧИНЕНА ПАРОДОНТИТОМ, ПРИ ІШЕМІЧНІЙ ХВОРОБІ СЕРЦЯ

Пацієнти з ішемічною хворобою серця та пародонтитом мають більш виражену ендотеліальну дисфункцію порівняно з пацієнтами лише з ішемічною хворобою серця. Це проспективне когортне дослідження включало 58 пацієнтів: 25 із ішемічною хворобою серця та 33 із ішемічною хворобою серця і пародонтитом. Воно було проведене на клінічних базах Івано–Франківського національного медичного університету з грудня 2023 року по вересень 2024 року. У групі ішемічна хвороба серця + пародонтит виявлено значно вищі рівні ММР–9 (126,76 [114,19; 146,21] нг/мл проти 100,47 [81,22; 114,38] нг/мл, p<0,001) та ТМАО (133,18 [111,27; 148,37] мкмоль/л проти 91,80 [65,42; 109,53] мкмоль/л, p<0,001). Ендотеліальна дисфункція проявлялася зниженням flow-mediated dilation (9,92 [9,12; 12,24] % проти 12,78 [10,52; 16,96] %, p=0,012), підвищенням швидкості пульсової хвилі (8,71 [7,93; 10,12] м/с проти 6,53 [5,60; 8,90] м/с, p=0,011) та збільшенням товщини комплексу інтима-медіа (0,76 [0,68; 0,81] мм проти 0,62 [0,54; 0,73] мм, p=0,004). Результати підкреслюють системний вплив пародонтиту на судинне здоров'я пацієнтів із ішемічною хворобою серця і необхідність інтегрованих підходів до лікування.

Ключові слова: ішемічна хвороба серця, пародонтит, ендотеліальна дисфункція, запалення, серцево-судинний ризик.

The work is a fragment of the research project "Structural and functional changes in internal organs in chronic non-communicable diseases: possibilities of drug correction", state registration No. 0121U108893.

Cardiovascular diseases (CVDs) persist as the foremost cause of morbidity and mortality on a global scale, with coronary artery disease (CAD) comprising a considerable proportion of this burden. According to the Global Burden of Disease (GBD) 2019 study, coronary artery disease (CAD) was a primary cause of death, with an estimated 18.6 million people dying from CVD in 2019 [4]. This emphasises the significant public health challenges posed by CVD and its considerable economic impact. In the United States, approximately 20.1 million adults are affected by CAD, and one person dies every 34 seconds from complications related to CVD [6]. Despite therapeutic advances – including pharmacological, interventional, and surgical approaches – CAD continues to pose a major global health challenge, highlighting the need to explore additional risk factors and comorbidities that may exacerbate its progression.

A notable comorbidity that has garnered increased attention is periodontitis, a chronic inflammatory disease that affects the supporting structures of the teeth. Globally, severe periodontal disease is among the most prevalent non–communicable diseases, affecting over 700 million individuals according to estimates based on the GBD 2017 study [2]. In addition, approximately 42 % of adults in the United States exhibit some degree of periodontitis, of whom 7.8 % suffer from a severe form [3]. The persistent inflammatory challenge posed by periodontitis is not confined to the oral cavity; instead, it can evoke systemic inflammatory responses that may potentiate atherogenesis and vascular dysfunction.

This association has prompted a surge in research endeavours investigating the intricate relationship between oral health and cardiovascular disease, with a particular emphasis on endothelial dysfunction as a shared pathological pathway [5]. Endothelial dysfunction is a pivotal early event in atherosclerosis and significantly influences disease progression and outcomes [1]. Characterised by reduced nitric oxide bioavailability and heightened pro–inflammatory and pro–thrombotic states, endothelial dysfunction is a pivotal early event in atherosclerosis.

The purpose of the study was to establish the impact of present periodontitis in patients with coronary artery disease on endothelial function and atherosclerotic burden.

Materials and methods. This prospective controlled cohort study was conducted at the clinical bases of the Ivano–Frankivsk National Medical University from December 2023 to September 2024. A total of 58 patients aged 18–75 years were enrolled and categorized into two groups:

– CAD alone (n = 25);

– CAD with concomitant periodontitis (n = 33);

All patients provided written informed consent, and the study protocol was approved by the ethics commission in accordance with the principles outlined in the Declaration of Helsinki.

Inclusion criteria were:

1. Established diagnosis of CAD according to the latest European Society of Cardiology (ESC) guidelines.
2. Age between 18 and 75 years.
3. Normal estimated glomerular filtration rate (eGFR).

Exclusion criteria were:

1. Alcohol addiction.
2. Major comorbidities such as chronic obstructive pulmonary disease (COPD) or diabetes mellitus.
3. Pregnancy.
4. Terminal illnesses.
5. Current use of known cardiotoxic or hormonal medications.

Upon enrollment, detailed medical histories were obtained, including demographic data, cardiovascular risk factors, medication use, and oral health status. The diagnosis of periodontitis was confirmed through a dental examination that included periodontal probing and evaluation of clinical attachment loss. CAD was diagnosed or confirmed on the basis of patient history, clinical examination, and diagnostic investigations in line with ESC guidelines.

All laboratory tests were carried out in the central research laboratory of Ivano–Frankivsk National Medical University. Standard biochemical assays were performed using an HTI Biochem FC–120 analyzer (High Technology Inc., USA).

For the measurement of specific biomarkers (MMP–9, TMAO), enzyme–linked immunosorbent assay (ELISA) was conducted using the ER500 Microplate Reader (Healicom, Jiangsu, China). Commercially available ELISA kits were utilized according to the manufacturers' instructions (Cloud–Clone Corp. for MMP–9 [Catalog No. SEA553Hu], and Elabscience for TMAO [Catalog No. E–BC–K100]).

A skilled ultrasonography technician, blinded to the patients' clinical status, performed all vascular measurements using the Siemens NX3 Elite ultrasound system (Siemens Healthineers, Germany). Carotid intima–media thickness (CIMT) was evaluated using the built–in Arterial Health Package, measuring the far–wall thickness of the common carotid artery in line with standard protocols. Flow–Mediated Dilation (FMD) assessment was conducted on the brachial artery using a validated, standardized protocol. Baseline diameter was measured, followed by a measurement taken 60 seconds after release of a forearm cuff inflated to suprasystolic pressure. Pulse Wave Velocity (PWV) was determined by calculating the velocity of the arterial pulse wave between two arterial sites, following standard guidelines.

All data were collated and analyzed using Python 3.11 (Python Software Foundation), employing the libraries NumPy, scipy.stats, and zepid. Normality of continuous data was tested (Shapiro–Wilk test). As the variables were non–normally distributed, comparisons between the two groups were made using the Mann–Whitney U test. For identification of potential independent predictors of outcomes, univariate regression analyses were performed. Results are presented as medians (interquartile ranges) for continuous variables and as frequencies (percentages) for categorical variables. All analyses were supervised by the principal investigator and reviewed by an independent statistician to ensure accuracy and reproducibility. A two–sided p–value <0.05 was considered statistically significant.

Results of the study and their discussion. The baseline characteristics of the studied patients is presented in table 1. Slightly younger individuals were present in the CAD + periodontitis group, with a median age of 51.00 (48.00; 55.00) years compared to 54.00 (50.00; 56.00) years in the CAD–only group (p=0.256). Males comprised 56.0 % of the CAD group versus 72.7 % of the CAD + periodontitis cohort (p=0.184), and most participants were non–smokers (80.0 % in CAD versus 90.9 % in CAD + periodontitis, p=0.233). Stable angina was present in 19 (76.0 %) patients in the CAD group, while 6 (24.0 %) had postinfarction cardiosclerosis. Similarly, 21 (63.6 %) patients in CAD + periodontitis group presented with stable angina, and 12 (36.4 %) with postinfarction cardiosclerosis (p=0.313).

Table 1

Baseline clinical and biochemical characteristics of the study population

Variable	CAD (n=25)	CAD + Periodontitis (n=33)	p–value
Age, years	54.00 (50.00; 56.00)	51.00 (48.00; 55.00)	0.256
Gender, n (%):			
Male	14 (56.0 %)	24 (72.7 %)	0.184
Female	11 (44.0 %)	9 (27.3 %)	
Smoking status, n (%):			
Non–smoker	20 (80.0 %)	30 (90.9 %)	0.233
Former smoker	5 (20.0 %)	3 (9.1 %)	
Clinical presentation of CAD, n (%)			
Stable angina	19 (76.0 %)	21 (63.6 %)	0.313
History of MI	6 (24.0 %)	12 (36.4 %)	
BMI, kg/m²	29.98 (28.09; 33.34)	29.20 (25.00; 33.84)	0.346
Body weight profile, n (%):			
Normal weight	1 (4.0 %)	8 (24.2 %)	0.141
Overweight	12 (48.0 %)	9 (27.3 %)	
Obesity stage 1	7 (28.0 %)	11 (33.3 %)	
Obesity stage 2	4 (16.0 %)	5 (15.2 %)	
Obesity stage 3	1 (4.0 %)	0 (0.0 %)	
Waist circumference, cm	89.37 (82.42; 95.40)	93.28 (77.81; 97.98)	0.777
Pocket probing depth, mm	2.10 (2.00; 2.40)	3.10 (2.80; 3.40)	<0.001
Clinical attachment loss, mm	1.90 (1.60; 2.20)	2.80 (2.50; 3.20)	<0.001
SBP, mmHg	141.00 (133.00; 147.00)	138.00 (134.00; 150.00)	0.956
DBP, mmHg	88.00 (84.00; 92.00)	91.00 (87.00; 94.00)	0.203
Total cholesterol, mmol/L	5.87 (5.25; 6.39)	6.24 (5.62; 6.88)	0.109
LDL, mmol/L	4.24 (3.39; 4.46)	4.64 (3.87; 5.07)	0.071
HDL, mmol/L	1.15 (1.04; 1.35)	1.05 (0.95; 1.23)	0.146
Triglycerides, mmol/L	1.55 (1.33; 2.09)	1.72 (1.41; 2.16)	0.520
Atherogenicity index	4.15 (3.55; 4.87)	5.15 (3.91; 5.94)	0.039
MMP–9 (ng/mL)	100.47 (81.22; 114.38)	126.76 (114.19; 146.21)	<0.001
TMAO (μmol/L)	91.80 (65.42; 109.53)	133.18 (111.27; 148.37)	<0.001
FMD (%)	12.78 (10.52; 16.96)	9.92 (9.12; 12.24)	0.012
PWV (m/s)	6.53 (5.60; 8.90)	8.71 (7.93; 10.12)	0.011
CIMT (mm)	0.62 (0.54; 0.73)	0.76 (0.68; 0.81)	0.004

Note: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL, low–density lipoprotein; HDL, high–density lipoprotein; MMP–9, matrix metalloproteinase–9; TMAO, trimethylamine N–oxide; FMD, flow–mediated dilation; PWV, pulse wave velocity; CIMT, carotid intima–media thickness.

The median BMI was 29.98 (28.09; 33.34) in patients with CAD alone compared to 29.20 (25.00; 33.84) among those with concomitant periodontitis (p=0.346), with body weight profiles showing normal weight in 4.0 % versus 24.2 %, overweight in 48.0 % versus 27.3 %, obesity stage 1 in 28.0 % versus 33.3 %, obesity stage 2 in 16.0 % versus 15.2 %, and obesity stage 3 in 4.0 % versus 0.0 % (p=0.141). Median waist circumference remained comparable between groups at 89.37 (82.42; 95.40) versus 93.28 (77.81; 97.98) cm (p=0.777). Significantly higher pocket probing depth, 3.10 (2.80; 3.40) vs. 2.10 (2.00; 2.40) mm (p<0.001), and clinical attachment loss, 2.80 (2.50; 3.20) vs. 1.90 (1.60; 2.20) mm (p<0.001), were identified in patients with periodontitis. Systolic blood pressure was similar at 138.00 (134.00; 150.00) vs. 141.00 (133.00; 147.00) mmHg (p=0.956), as was diastolic pressure at 91.00 (87.00; 94.00) vs. 88.00 (84.00; 92.00) mmHg (p=0.203). In terms of lipid parameters, total cholesterol was 6.24 (5.62; 6.88) vs. 5.87 (5.25; 6.39) mmol/L (p=0.109), LDL reached 4.64 (3.87; 5.07) vs. 4.24 (3.39; 4.46) mmol/L (p=0.071), HDL measured 1.05 (0.95; 1.23) vs. 1.15 (1.04; 1.35) mmol/L (p=0.146), and triglycerides were 1.72 (1.41; 2.16) vs. 1.55 (1.33; 2.09) mmol/L (p=0.520). Notably, the atherogenicity index was significantly higher in the CAD + periodontitis group, at 5.15 (3.91; 5.94) vs. 4.15 (3.55; 4.87) (p=0.039).

Significantly higher MMP–9 levels were noted in patients with both CAD and periodontitis, at 126.76 (114.19;146.21) ng/mL compared to 100.47 (81.22;114.38) ng/mL in those with CAD alone (p<0.001). TMAO followed a similar pattern, rising from 91.80 (65.42;109.53) μmol/L to 133.18 (111.27;148.37) μmol/L (p<0.001), which reflects a notable increase in systemic inflammatory and metabolic stress. In contrast, FMD declined from 12.78 (10.52;16.96) % to 9.92 (9.12;12.24) % (p=0.012) among individuals with concomitant disease, suggesting impaired endothelial function. An elevation in PWV, from 6.53 (5.60;8.90) m/s to 8.71 (7.93;10.12) m/s (p=0.011), indicated greater arterial stiffness in the CAD + periodontitis group. In addition, CIMT rose significantly from 0.62 (0.54;0.73) mm to 0.76

(0.68;0.81) mm (p=0.004), implying more advanced subclinical atherosclerosis when periodontitis coexisted with CAD. The results of regression analysis are presented in Table 2.

Table 2

Univariate Logistic Regression Analysis for CAD Alone vs. CAD + Periodontitis

Variable	OR (95 % CI)	p-value
MMP–9 (ng/mL)	1.045 (1.018–1.074)	0.001
TMAO (μmol/L)	1.046 (1.021–1.071)	<0.001
FMD (%)	0.878 (0.774–0.997)	0.044
PWV (m/s)	1.461 (1.106–1.931)	0.008
CIMT (mm)	1.220 (1.111–1.378)	0.005

Note: MMP–9, matrix metalloproteinase–9; TMAO, trimethylamine N–oxide; FMD, flow–mediated dilation; PWV, pulse wave velocity; CIMT, carotid intima-media thickness.

Logistic regression analysis, with group membership coded as 0 for CAD alone and 1 for CAD + periodontitis, revealed a strong association between elevated MMP–9 and increased odds of belonging to the comorbid group: each 1–unit rise in MMP–9 resulted in an odds ratio (OR) of 1.045 (95 % CI 1.018–1.074, p=0.001). A similar relationship emerged for TMAO, where each unit increment corresponded to an OR of 1.046 (95 % CI 1.021–1.071, p<0.001). By contrast, higher FMD values (OR = 0.878 (95 % CI 0.774–0.997, p=0.044) indicated lower odds of being in the CAD + periodontitis group with each unit increase in FMD. PWV showed an OR of 1.461 (95 % CI 1.106–1.931, p=0.008), suggesting a marked rise in the likelihood of coexisting CAD and periodontitis as arterial stiffness worsened. Likewise, every unit increase in CIMT was associated with an OR of 1.220 (95 % CI 1.111–1.378, p=0.005), underscoring its significant link to the combined disease state.

The present study highlights the exacerbating role of periodontitis in patients with CAD, evidenced by elevated MMP–9 and TMAO, greater arterial stiffness (PWV), thicker CIMT, and diminished endothelial function (FMD). According to Shetty B et al. (2023), periodontitis establishes a persistent pro–inflammatory milieu that can aggravate atherosclerotic progression and plaque vulnerability, thereby amplifying cardiovascular risk [9]. Isola G et al. (2021) similarly reported that MMP–9 acts as a crucial mediator between oral and systemic inflammation, facilitating tissue remodeling in the periodontium while simultaneously destabilizing atherosclerotic plaques [4].

Further supporting these observations, Spasova N et al. (2024) underscored TMAO's integral role in intensifying inflammatory and thrombotic processes, which may explain the marked rise in TMAO levels in our comorbid cohort [10]. The reduced FMD in individuals with coexisting CAD and periodontitis aligns with findings by Fujitani T et al. (2020), who emphasized the importance of endothelial function as a predictor of cardiovascular events [3]Клацніть або торкніться тут, щоб ввести текст.. Jockel–Schneider Y et al. (2014) also demonstrated that increased PWV serves as an independent marker of vascular dysfunction, correlating with enhanced arterial stiffness in our participants with combined disease [5]. Lastly, Toregeani J et al. (2016) identified CIMT as a robust surrogate marker for subclinical atherosclerosis and subsequent cardiovascular risk, mirroring the significant CIMT elevations in our comorbid group [11].

Overall, these data reinforce the notion that maintaining periodontal health may offer systemic benefits by mitigating inflammatory load, slowing atherosclerotic changes, and preserving endothelial integrity.

Our further research will focus on whether prompt and effective periodontal intervention can modulate these pathophysiological markers and translate into improved cardiac outcomes in patients diagnosed with CAD.

Conclusion

Our findings provide compelling evidence that concomitant periodontitis in patients with coronary artery disease is linked to heightened systemic inflammation and pronounced endothelial dysfunction, as demonstrated by elevated MMP–9 and TMAO levels, increased PWV and CIMT, and diminished FMD. These results underscore the pivotal role of periodontal health in modulating cardiovascular risk and highlight the potential value of integrating dental assessments and treatments into the routine care of individuals with CAD.

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PREDICTING THE DEVELOPMENT OF COGNITIVE IMPAIRMENT IN PATIENTS AFTER COVID-19 INFECTION

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The study focused on predicting the development of cognitive impairment in patients following COVID-19. The study involved 100 patients, 69 in the main group and 31 in the control group. In terms of gender composition, men predominated – 60 people. According to the results of a comprehensive clinical and neurological examination, as well as neuropsychological examination, which considers quality of life indicators and laboratory examination, specifically the level of phosphorylated neurofilament, this factor is identified as a predictor of the development of the neurodegenerative process. Patients in the main group (n=69) were divided into 2 subgroups based on the level of neurofilament: the NfL-N subgroup with a concentration not exceeding the norm and the NfL-P subgroup with a concentration above the norm. A predictive model for the development of cognitive impairment in patients with COVID-19 has been developed to improve early diagnosis of neurodegenerative diseases.

Key words: prediction, cognitive impairment, depression, anxiety, COVID-19, neurofilaments.

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ПРОГНОЗУВАННЯ РОЗВИТКУ КОГНІТИВНИХ ПОРУШЕНЬ У ПАЦІЄНТІВ ПІСЛЯ ПЕРЕНЕСЕНОЇ ІНФЕКЦІЇ COVID-19

Дана стаття присвячена прогнозуванню розвитку когнітивних порушень у пацієнтів після перенесеного захворювання COVID-19. В дослідження залучено 100 пацієнтів, 69 осіб основної групи та 31 особа контрольна група. За гендерним складом переважали чоловіки – 60 осіб. За результатами комплексного клініко-неврологічного обстеження, нейропсихологічного обстеження з урахуванням показників якості життя, проведення лабораторного обстеження, а саме рівня фосфорильованого нейрофіламента як предиктора розвитку нейродегенеративного процесу. У подальшому пацієнтів основної групи (n=69) було розділено на 2 підгрупи в залежності від рівня нейрофіламенту: підгрупа NfL-N з концентрацією, що не перевищую норму, та підгрупа NfL-P з концентрацією, що вище норми. Була розроблена прогностична модель розвитку когнітивних порушень у пацієнтів, що перенесли COVID-19 для удосконалення ранньої діагностики нейродегенеративних захворювань.

Ключові слова: прогнозування, когнітивні порушення, депресія, тривога, COVID-19, нейрофіламенти.

The work is a fragment of the research project "Clinical, pathogenetic and prognostic markers of nervous system disorders and optimization of diagnostic and treatment algorithms", state registration No. 0122U201970.

The COVID-19 pandemic has become a problem for society that requires further detailed study, primarily regarding early diagnosis of complications after the disease to prevent the development of long COVID syndrome. Coronavirus disease is a systemic pathology that affects all organs and systems of the body at different stages of the disease. From the side of the nervous system, this can manifest itself in