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ENDOTHELIAL DYSFUNCTION WITH COMORBIDITY OF PATHOLOGIES AS ONE WITH CRITERIA OF HEART FAILURE

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Annotation: It has been shown that high risk factors for endothelial dysfunction (AD) are arterial hypertension (hypertension), hypercholesterolemia, diabetes mellitus, resulting in vascular endothelial damage (changes in levels of nitrates / nitrites, ET-1, sVCAM-1 in the blood). The combination of pathologies, namely ulcerative stomach and duodenal ulcer, arterial hypertension and type 2 diabetes, promotes the development of endothelial dysfunction by increasing the number of desquamation endothelial cells, the level of nitrate / nitrite, ET-1, sVCAM-1, which aggravates diagnosis and treatment the main illness and is one of the triggers for the development of heart failure.

Key words: stomach peptic ulcer, duodenum, diabetes mellitus type II, endothelial dysfunction, desquamation endothelial cells, nitrate / nitrite level, ET-1, sVCAM-1.

The endothelial function remains one of the diagnostic criteria of vascular endothelial pathology [1,2,4,6,8,9]. The comorbidity of pathologies leads to the development of endothelial dysfunction, and the presence of concomitant pathologies, especially diabetes mellitus (DM) and arterial hypertension (AH) provokes the development of heart failure[3,5,7].

Objectives To investigate the condition of endothelial dysfunction in patients with peptic ulcer of the stomach and duodenum combined with arterial hypertension and type 2 diabetes mellitus.

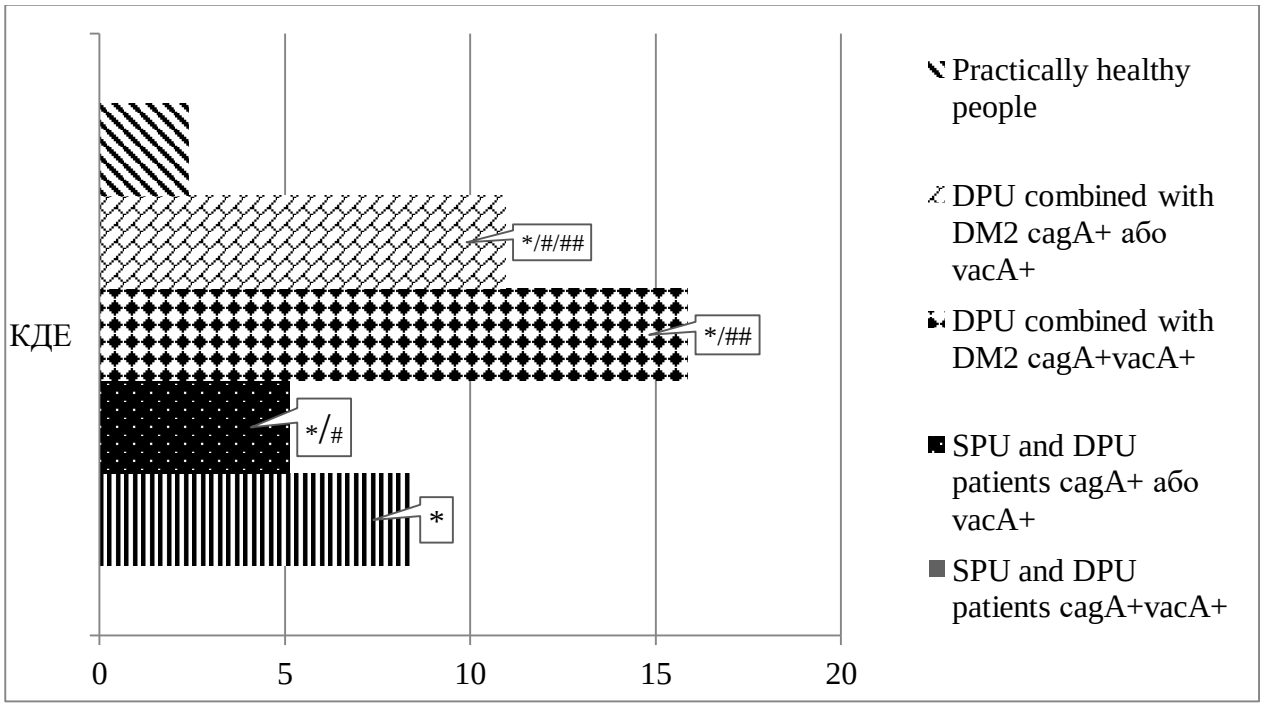
Materials and methods. 108 patients were examined : 28 patients with peptic ulcer of the stomach (PUG) and duodenum (DUK) in the presence of toxigenic strains of CagA + VacA + (group 1), 20 patients with PUG and DUC in the presence of a combination of CagA strains + VacA- / CagA-VacA + (2nd group), 22 patients with PAG and DUC in the presence of toxigenic strains of CagA + VacA + in combination with AH and DM2 (3rd group), 38 patients with PUG and DUC in the presence of a combination of CagA + VacA- / CagA-VacA + in combination with AH and DM2 (4th group) and 30 practically healthy persons (PZL) (5th group).

Evaluation of vascular endothelial dysfunction was carried out by the determination of ET-1 with a set of reagents from Bender MedSystems GmbH (Austria), sVCAM-1 - Bender MedSystems GmbH (Austria). The nitrate / nitrite level was determined using the method of determining the final stable metabolites using the Gris reagent. Endothelial cells were determined by the method of N. N. Petrischev, A. A. Berkovich, assessing the number of desquamation of endothelial cells (DEC) in the blood, as an indicator of endothelial dysfunction in patients with various diseases.

Results and discussion The level of DUC in blood was 8.44 ± 0.22 in patients with PUG and DUC CagA + VacA +, which is 3.52 times higher than in the PZL group (2.4 ± 0.23) ($p \leq 0.05$) , and in patients with PUG and DUC, CagA + VacA- / CagA-VacA + - (5.11 ± 0.28) ($p \leq 0.05$), which is 2.12 times higher in comparison with the PZL group. However, in the group of patients with PUG and DUC, CagA + VacA + is 1.65 times higher in comparison with the group of patients with PUG and DUC CagA + VacA- / CagA-VacA +.

In the presence of concomitant pathology, the amount of DEC in patients with PUG and DUC CagA + VacA + (15.86 ± 0.39) ($p \leq 0.05$) increased 6.6 times in comparison with the PZL group, and in patients with PUG and DUC CagA + VacA- / CagA-VacA + (10.94 ± 0.18) ($p \leq 0.001$) - 4.56 times, respectively. However, assessing the effect of toxigenic strains and AH and DM2 on PUG and DUC, it was found that this

index was 1.45 times (p≤0.001), increased in the group of patients CagA + VacA + PUG and DUC in combination with AH and DM 2 in comparison with group of patients with PUG and DUC CagA + VacA- / CagA-VacA + in combination with AH and DM2.



Picture.1 Amount of DEC in the patients with the peptic ulcer of stomach and duodenum combined with the diabetes mellitus type II depending on the availability of cagA and vacA HP genes, M±m

Note. * - accuracy of discrepancies (p<0.05) between the indices in groups IIIa, IIIB, Ib, Ic, Id in comparison with group II;

- accuracy of discrepancies (p<0.05) between the indices in groups Ia and Ib, Ic and Id;

- accuracy of discrepancies (p<0,05) between the indices in groups IIIa and Ib, Ic and Id.

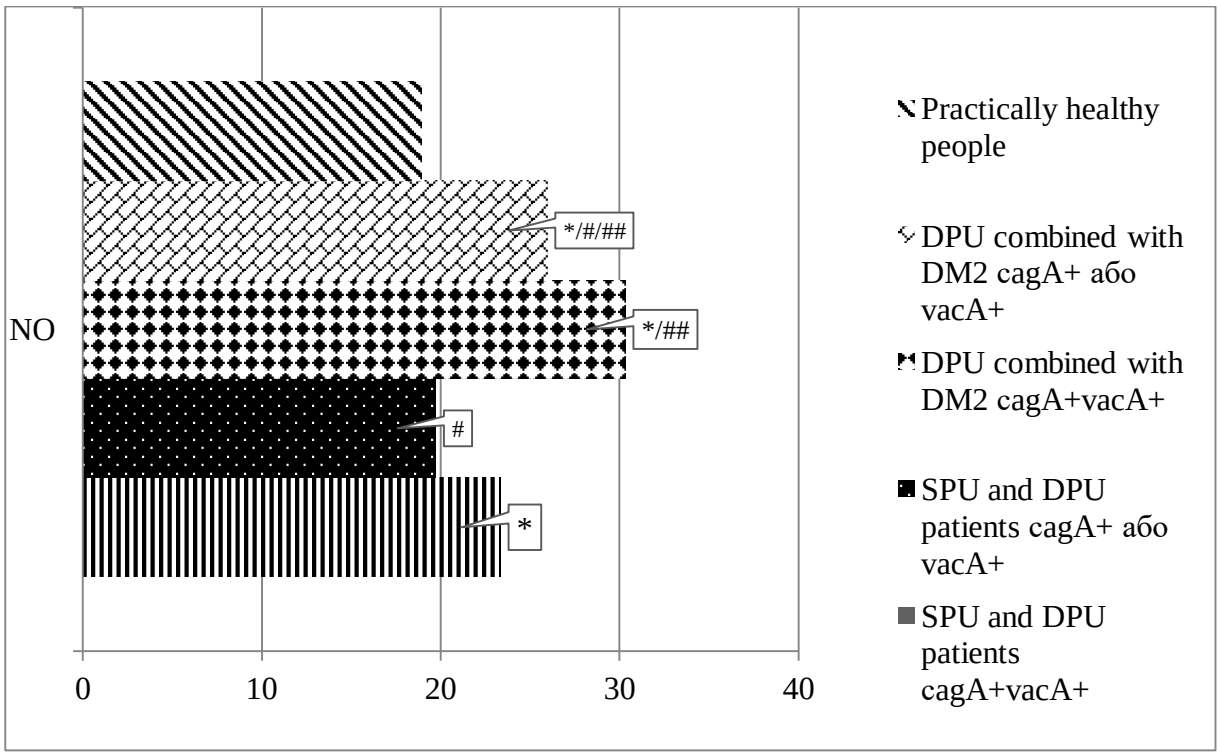
The researchers pay great attention to the risk factors for endothelial dysfunction (DE): arterial hypertension (AH), hypercholesterolemia, diabetes mellitus, lead to damage to the vascular endothelium (changes in the levels of nitrate / nitrite, ET-1, sVCAM-1 in the blood).

Investigating the state of endothelial dysfunction in the blood, it was found that in patients with PUG and DUC with AH and DM2, the level of nitrates / nitrites, the

highest in the group of patients with PUG and DUC CagA + VacA + in combination with AH and DM2.

The level of nitrates / nitrites in the group of patients with PUG and DUC CagA + VacA + is (23.35 ± 0.36) ($p < 0.001$), and in the group of patients with PUG and DUC, CagA + VacA- / CagA-VacA + - (19.74 ± 0.61) ($p < 0.05$), which is 1.23 times and increased 1.04 times in comparison with the group of practically healthy persons (18.92 ± 0.83). However, in the presence of both toxogenous strains in patients with PUG and DUC, the content of this indicator was increased by 1.18 times ($p \leq 0.001$) in comparison with the group of patients with PUG and DUC in the presence of one of the toxicogenic strains.

However, in the presence of concomitant pathology, the level of nitrates / nitrites in patients with PUG and DUC CagA + VacA + (30.33 ± 5.07) ($p < 0.001$), 1.6 times increased in comparison with the PZL group, and in patients PUG and DUC CagA + VacA- / CagA-VacA + (25.96 ± 0.97) ($p < 0.05$), - 1.37 times, respectively. However, assessing the effect of toxigenic strains and AH and DM2 on PUG and DUC, it was found that this index was increased by 1.17 times ($p < 0.05$) in the group of patients CagA + VacA + PUG and DUC in combination with AH and DM2 in comparison with a group of patients with PUG and DUC CagA + VacA- / CagA-VacA + in combination with AH and DM2.



PICTURE 2. A level of nitrates / of nitritis is in blood (mcmol/l) in the patients with the peptic ulcer of stomach and duodenum combined with the diabetes mellitus type II depending on the availability of cagA and vacA HP genes, M±m

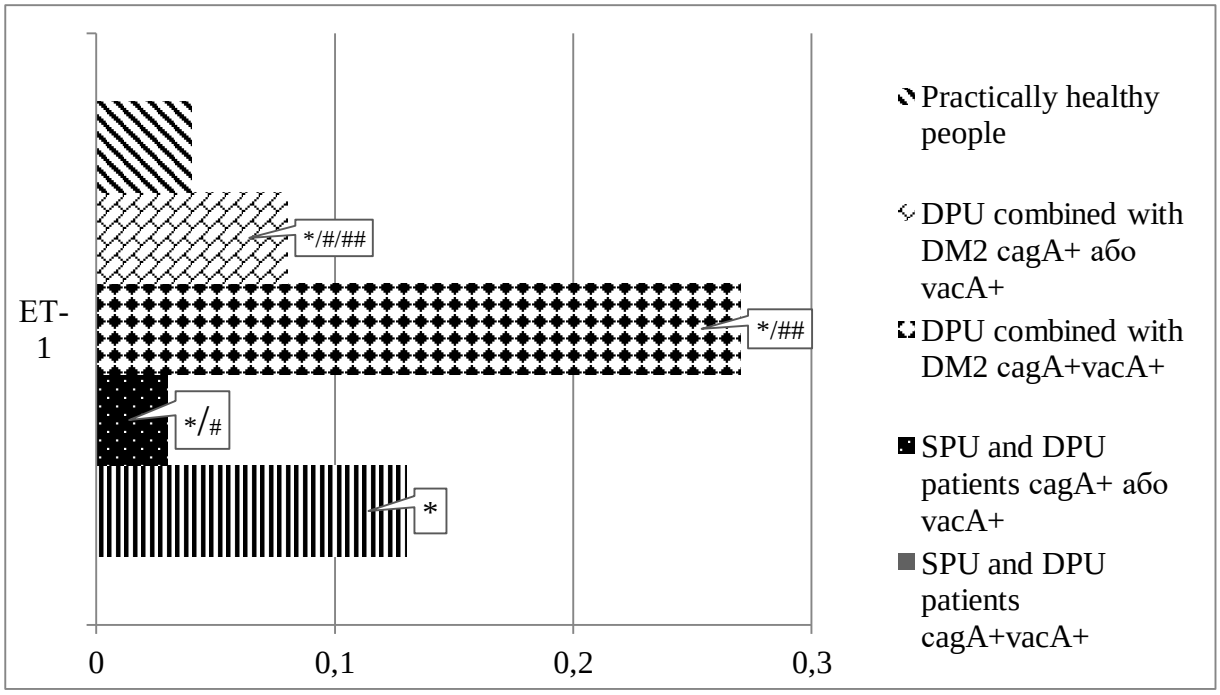
Note. * - accuracy of discrepancies ($p<0.05$) between the indices in groups IIIa, IIIB, Ib, Ic, Id in comparison with group II;

##- accuracy of discrepancies ($p<0.05$) between the indices in groups Ia and Ib, Ic and Id;

##- accuracy of discrepancies ($p<0,05$) between the indices in groups IIIa and Ib, Ic and Id.

An essential diagnostic criterion of endothelial dysfunction is endothelin - 1 (ET - 1). Estimating the level of ET-1 in patients of the 1 st group, this indicator is (0.13 ± 0.02), which is 3.25 times higher than the content in the PZL group (0.04 ± 0.01) ($p\leq0.001$), and in patients of the 2 nd group - (0,03 ± 0,01) ($p\leq0,05$), which is 1,33 times higher in comparison with the PZL group. However, in the patients of the 1st group this index is 3.25 times ($p <0.05$) higher in comparison with the 2 nd group. In the presence of concomitant pathology, the content of ET-1 in patients of the 3rd group is (0.27 ± 0.67) ($p <0.001$), which is 6.75 times higher in comparison with the 5th group, and in patients with 4- th group - (0,08 ± 1,61) ($p <0,05$), which is twice

higher with the 5th group, respectively. However, assessing the effect of toxigenic strains and AH with DM2 on PUG and DUC revealed that this index was 3.38 times (p <0.001) elevated in group 3 of patients compared with group 4 of patients. Estimating the content of the adhesion molecule (sVCAM-1) in patients without concomitant pathology, it was found that in patients with PUG and DUC CagA + VacA + the index was (1912.96 ± 231.31), which is 3.83 times higher in the PZL group (493 , 87 ± 119.72) (p≤0.001), and in patients with PUG and DUC CagA + VacA- / CagA-VacA + - (1504,00 ± 360,30) (p≤0,001), which is 3.05 times increased in comparison with the PZL group. However, the content of this indicator in patients with PUG and DUC CagA + VacA + is 1.27 times (p≤0.001) higher in comparison with the group of patients with PUG and DUC CagA + VacA- / CagA-VacA +.



PICTURE 3. ET-1 (pmol/l) in the patients with the peptic ulcer of stomach and duodenum combined with the diabetes mellitus type II depending on the availability of cagA and vacA HP genes, M±m

Note. * - accuracy of discrepancies (p<0.05) between the indices in groups IIIa, IIIB, Ib, Ic, Id in comparison with group II;

##- accuracy of discrepancies (p<0.05) between the indices in groups Ia and Ib, Ic and Id;

- accuracy of discrepancies (p<0,05) between the indices in groups IIIa and Ib, Ic and Id.

In the presence of concomitant pathology, the content of sVCAM-1 in patients with PUG and DUC CagA + VacA + (3394.55 ± 299.4) (p≤0.05) was increased by 6.87 times in comparison with the PZL group, and in patients with PUG and DUC CagA + VacA- / CagA-VacA + (1664.00 ± 145.56) (p <0.05) - 3.37 times, respectively. However, assessing the effect of toxigenic strains and AH and DM2 on the PNG and PDC, it was established that this index was increased by 2.04 times (p <0.001) in the group of patients CagA + VacA + PUG and DUC in combination with AH and DM2 in comparison with the group patients with PUG and DUC CagA + VacA- / CagA-VacA + in combination with AH and DM2.

PICTURE.4 . VCAM-1 (ng/ml) in the patients with the peptic ulcer of stomach and duodenum combined with the diabetes mellitus type II depending on the availability of cagA and vacA HP genes, M±m

Note. * - accuracy of discrepancies (p<0.05) between the indices in groups IIIa, IIIB, Ib, Ic, Id in comparison with group II;

##- accuracy of discrepancies (p<0.05) between the indices in groups Ia and Ib, Ic and Id;

- accuracy of discrepancies (p<0,05) between the indices in groups IIIa and Ib, Ic and Id.

If studying the oxidative-antioxidative system (Table 1), it was found out that content of MA in blood plasma of patients with SPU and DDPU cagA+vacA+ HP was 1.38 times higher (p<0.05) in comparison with PHP group. However, it considerably differed in case of available cagA+vacA-/cagA-vacA+ genotypes (p>0.05).

The level of MA in blood plasma is 2 times higher in the group of patients with SPU and DDPU combined with DM2 in case of available cagA+vacA+ if compared with PHP group (p<0.05). At the same time, it is 1.32 times higher (p<0.05) if compared

with the group of patients suffering from SPU and DDPU cagA+ or vacA+ combined with DM2.

The analogical situation is observed if analyzing the concentration of malonaldehyde in the blood erythrocytes. The considerable changes are found in the group of patients with SPU and DDPU cagA+ or vacA+ combined with DM2 that is proved by the increase of index in 1.83 times ($p<0.05$) and in 1.22 times ($p<0.05$) if compared with the group of patients suffering from SPU and DDPU cagA+ or vacA+ combined with DM2.

Table 1

The state of oxidative-antioxidative homeostasis in the patients with the peptic ulcer of stomach and duodenum combined with the diabetes mellitus type II depending on the availability of cagA and vacA HP genes, M±m.

Index	Test groups				
	SPU and DDPU patients		Patients with SPU and DDPU combined with DM2		Practically healthy people,
	cagA+ vacA+ n =8	cagA+ or vacA+ n =7	cagA+ vacA+ n =9	cagA+ or vacA+ n =6	n =15
Malonaldehyde in blood erythrocytes, mcmol/l	8.32±0.34 *	5.78±0.52 */#	9.75±0.37 */##	8.49±0.3 */#/##	4.97±0.22
Malonaldehyde in blood plasma, mcmol/l	1.47±0.14 *	2.27±0.23 *	3.45±0.05 */##	2.83±0.09 */#//##	1.52±0.09
Renewed glutathione, mmol/l	0.50±0.01 *	0.67±0.01 */#	0.33±0.01 */##	0.35±0.01 */##	1.04±0.06
Glutathione	215.46±	148.6±	293.46±	261.19±	111.08±

peroxidase, nmol RG / 1 g Hb per min.	12.22 *	3.17 */#	7.95 */##	2.91 */#/##	8.04
Glutathione-S-transferase, nmol RG / 1 g Hb per min.	112.69± 2.34 *	98.85± 0.99 */#	135.00± 0.92 */##	115.66± 3.33 */#/##	84.09± 3.29

Note. * - accuracy of discrepancies ($p<0.05$) between the indices in groups IIIa, IIIB, Ib, Ic, Id in comparison with group II;

##- accuracy of discrepancies ($p<0.05$) between the indices in groups Ia and Ib, Ic and Id;

- accuracy of discrepancies ($p<0,05$) between the indices in groups IIIa and Ib, Ic and Id.

When analyzing the glutathione system (Table 1), it was found out that patients with SPU and DDPU and available cagA+vacA+ HP genes have the reduced amount of renewed glutathione (RG) (in 2.04 times) ($p<0.05$) that is caused by the increased activity of glutathione peroxidase (GP) (in 1.86 times) ($p<0.05$) and glutathione-S-transferase (GT) (in 1.3 times) ($p<0.05$) if compared with PHP group. At the same time, the level of RG is lower in patients with SPU and DDPU cagA+ or vacA+ HP (in 1.53 times) ($p<0.05$) because of the increased activity of GP (in 1.31 times) ($p<0.05$) and GT (in 1.16 times) ($p<0.05$) if compared with PHP group respectively.

The combination of SPU, DDPU and DM2 with the available cagA+vacA+ causes the considerable reduction of RG (in 3.03 times) ($p<0.05$) in the setting of increased activity of glutathione peroxidase (GP) (in 2.5 times) ($p<0.05$) and glutathione-S-transferase (GT) (in 1.54 times) ($p<0.05$) if compared with PHP group.

Conclusions.

The combination of pathologies, namely peptic ulcer of the stomach and duodenum, arterial hypertension and type 2 diabetes mellitus, promotes the development of endothelial dysfunction by increasing the number of desquamation endothelial cells,

nitrate / nitrite level, ET-1, sVCAM-1, which burdens the diagnosis and treatment of the underlying disease and is one of the triggers for the development of heart failure.

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