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# SOME INDICES OF CARDIOHEMODYNAMICS, BLOOD PRESSURE AND ACTIVITY OF TUMOR NECROSE FACTOR-ALPHA IN PATIENTS WITH ESSENTIAL HYPERTENSION AND COMORBID DUODENAL ULCER AT THE STAGE OF EARLY REHABILITATION

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The modern stage of studying the genesis and evolution of essential hypertension (EH) determines the important role of the immune system, namely the activation of cells of innate and adaptive immunity, the mediators of which are directly involved in the development of low-intensity chronic inflammation [1]. Data on the role of mediators of intercellular communication in the mechanisms of development of arterial hypertension and, in particular, tumor necrosis factor-alpha (TNF- $\alpha$ ) are being studied and updated.

A feature of this pro-inflammatory cytokine is its ability to influence several risk factors for the development of cardiovascular diseases and, in particular, to



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interact with blood pressure regulatory systems, to increase the expression of inflammatory chemokines, cell adhesion molecules, which contributes to microvascular remodeling [2]. It has been shown that in patients with EH there is an increase in circulating TNF- $\alpha$  content, which has an impact on the formation of vascular dysfunction, as well as on blood pressure parameters [3].

Taking into account the above, it became interesting to find out the features of the content of circulating TNF- $\alpha$  and compare it with blood pressure indices in patients with EH in conditions of syntropia, in particular when combined with duodenal peptic ulcer (DPU) in the early stage (after exacerbation) rehabilitation and treatment.

Circulating TNF- $\alpha$  activity was analyzed in 69 patients (38 men, 31 women; average age  $43.2 \pm 2.9$  years) with II stage of EH and comorbid DPU (within 4-6 weeks after the next exacerbation) in blood serum (immunoenzyme method). Blood pressure (BP) indicators (systolic (BPs) and diastolic (BPd) were determined in these patients using the generally accepted method, pulse pressure (PP) was calculated as the difference between BPs and BPd, and the mean hemodynamic pressure (MHP) was determined according to the formula  $1/3 \times (BPs - BPd) + BPd$ ; assessment of structural and functional indices of the left ventricle was carried out according to echocardiographic research. The obtained data were compared with the data of 33 patients with isolated EH course (comparison group) and 23 practically healthy individuals (reference norm), the composition of which corresponded to patients with comorbid pathology in terms of age and sex.

Processing of the obtained data was carried out using licensed programs Microsoft Office 2003, Microsoft Excel Stadia 6.1/prof. For all indicators, the probability of discrepancies is defined as: \* -  $p < 0.05$ , \*\* -  $p < 0.01$ , \*\*\* -  $p < 0.001$ .

According to the obtained data of observation of patients with comorbid course of EH, an increase in blood pressure was noted, exceeding its target values, as a result of which there was a need to correct antihypertensive treatment. The data of the echocardiographic study revealed an increase in the thickness of the walls of the left ventricle (compared to the control group by more than  $1.5^{***}$  times), which we considered as its hypertrophy; no enlargement of the left chambers was observed, the global contractility (as measured by the ejection fraction) was within the generally accepted norm. Attention was drawn to a significant increase in total peripheral vascular resistance (TPVR), which was almost  $1.4^{***}$  times higher than the reference norm and  $1.14^*$  times the rate of patients with an isolated EH course. It was established that the level of circulating TNF- $\alpha$  in these patients fluctuated within the range from 33.5 to 99.3 pg/ml, and its average value was not only  $2.6^{***}$  times higher than that in practically healthy individuals ( $20.3 \pm 1.4$  pg/ml), but also  $1.6^{***}$  times higher than the corresponding indices in patients of the comparison group (range of fluctuations 23.2–59.7 pg/ml).

For the purpose of further analysis, we arbitrarily formed 3 subgroups of patients with comorbid EH, which had reliable differences in the fold increase in circulating TNF- $\alpha$  both with the group of practically healthy people and among themselves: subgroup "A" - the fold increase in TNF- $\alpha$  up to 2 average values of the reference norm ( $36.1 \pm 2.1$  pg / ml; 26% of patients), subgroup "B" - the fold increase from 2 to 4 average values of the reference norm ( $72.9 \pm 3.6$  pg / ml; 64%) and subgroup "C" - excess of the reference norm by 4 or more times ( $85.2 \pm 2.3$  pg / ml; 10% of patients).

When comparing blood pressure indices between the patients of the subgroups formed above, it was found that the patients of subgroup "A" mainly had an increase in BPs, the value of which was within the limits of the I degree of arterial hypertension ( $148.7 \pm 4.5$  mm Hg). In subgroup "B" patients the increase in arterial pressure was more significant (BPs  $166.4 \pm 4.3$  mm Hg, BPd  $95.1 \pm 4.3$  mm Hg), which in terms of systolic arterial pressure corresponded to stage II arterial hypertension. The most pronounced arterial hypertension was observed in patients of subgroup "C" (BPs  $175.6 \pm 4.1$  mm Hg, BPd  $103.4 \pm 3.6$  mm Hg), the average values of which were within the limits of stage II arterial hypertension in terms of both systolic and diastolic BP. It should be noted that the probability of discrepancies was observed between patients of all studied subgroups in terms of BPs level, and in terms of BPd level - only between patients of subgroup "A" and subgroups "B" and "C" ( $p < 0.001$ ). Patients of all studied subgroups were characterized by a probable ( $p < 0.001$ ) increase in PP (compared to the control group by 1.63-1.67 times), however, no probable differences were observed between the subgroups. It should be emphasized that the value of the MHP indices in patients of subgroup A was on the border of the upper norm, while in patients of subgroups "B" and "C", its value probably exceeded the value of patients in subgroup A (by 1.18\* and 1.27\*\* times respectively).

A repeated examination of patients with comorbid pathology (after a month of observation) revealed a decrease in TNF- $\alpha$  activity to the level of the values of the comparison group, however, its values were 1.6\*\*\* times higher compared to practically healthy individuals.

In our opinion, the increase in TNF-alpha activity and its changes in the dynamics of observation of patients with comorbid course of EH should be considered in the context of the inflammatory process of the duodenum. The correspondence between the degree of increase in TNF- $\alpha$  activity and the unidirectional increase of BP indices with reliable dynamics of increase in vascular stiffness (PP, MHP) and TPVR allows us to take this fact into account as a criterion for aggravation of arterial hypertension in conditions of syntropy. The above indicates the need to correct antihypertensive and antiulcer therapy taking into

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account *Helicobacter pylori* infection [4, 5] and an assessment of the effectiveness of eradication therapy.

**REFERENCES:**

- [1] Wenzel, U. O., Ehmke, H., & Bode, M. (2021). Immune mechanisms in arterial hypertension. Recent advances. *Cell and tissue research*, 385(2), 393–404. <https://doi.org/10.1007/s00441-020-03409-0>
- [2] Zhang, Z., Zhao, L., Zhou, X., Meng, X., & Zhou, X. (2023). Role of inflammation, immunity, and oxidative stress in hypertension: New insights and potential therapeutic targets. *Frontiers in immunology*, 13, 1098725. <https://doi.org/10.3389/fimmu.2022.1098725>
- [3] Rodríguez-Iturbe B. (2020). The participation of immunity in the pathogenesis of arterial hypertension. La participación de la inmunidad en la patogenia de la hipertensión arterial. *Nefrología*, 40(1), 1–3. <https://doi.org/10.1016/j.nefro.2019.04.006>
- [4] Hassan, A. A., Ahmed, B. E., Osman, O. E., & Adam, I. (2023). Association between *Helicobacter pylori* seropositivity and hypertension among adults in Northern Sudan: a community-based case-control study. *The Journal of international medical research*, 57(6), 3000605231182545. <https://doi.org/10.1177/03000605231182545>
- [5] Fang, Y., Xie, H., & Fan, C. (2022). Association of hypertension with *helicobacter pylori*: A systematic review and meta-analysis. *PloS one*, 17(5), e0268686. <https://doi.org/10.1371/journal.pone.0268686>