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CLINICAL AND PROGNOSTIC ROLE INTENSIFICATION OF APOPTOSIS IN PATIENTS WITH ARTERIAL HYPERTENSION AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Tyaglaya O. S.

*Postgraduate Student at the Department of Clinical Pharmacology,
Pharmacy and Pharmacotherapy with Course of Cosmetology
Zaporizhzhia State Medical University
Zaporizhzhia, Ukraine*

Introduction. It is known that both chronic obstructive pulmonary disease (COPD) and arterial hypertension (AH) are multifactorial diseases, and develop as a result of a complex interaction of genetic and environmental factors.

The purpose of this study was to study the metabolism of caspase-7 and caspase-9 in patients with chronic obstructive pulmonary disease (COPD) in combination with arterial hypertension (AH).

Materials and methods. In 23 patients were diagnosed stage II hypertonic disease (HD) and stage II COPD without clinically significant concomitant pathology, with an average age of 51.72 ± 1.22 (49.33-54.09 years) (gender composition: 22 men and 1 woman), smoking status is comparable to COPD patients, 18 patients with HD of both sexes aged from 33 to 67 (mean age 50.74 ± 1.49 (47.81-53.76), male / female ratio 17 / 83%), stage II of HD with the level of hypertension I-III degree, different cardiovascular risk, without adequate systematic antihypertensive therapy, and 18 patients with COPD stage II, mean age 50.32 ± 0.99 (48.22-52.16) years (gender composition: 14 males and 4 females), duration of the disease 7.52 ± 1.14 . 80% of active smokers, the index of pack-years 17.23 \pm 2.69, the harmful professional factor (industrial) indicated 23.53%. All patients expressed their consent to participate in the study.

The plasma level of caspase-7 and caspase-9 was determined using the appropriate ELISA test systems (manufacturer – Bender Medsystems, Austria) at the Medical and Laboratory Training Center of ZSMU in accordance with the instructions attached to the kit.

Results. Patients with hypertension had the lowest values of caspase-7 among the study groups – 0.11 ± 0.02 ng / ml, while in COPD patients this indicator was statistically significantly ($p < 0.05$) higher in 72.73% – 0.19 ± 0.04 ng / ml. Patients with comorbid pathology as COPD + AH demonstrated a statistically significant ($p < 0.05$) elevation of the proapoptotic marker of caspase-7 to 0.41 ± 0.09 ng / ml, which is in 272.73% higher compared with the group of patients with AH, and in 115,79% more in comparison with a group of patients with COPD.

The dynamics of caspase-9 was quite similar to the dynamics of caspase-7 in experimental groups. The presence of hypertension in patients led to increase in the level of this cysteine-dependent aspartate-specific protease to 1.45 ± 0.23 ng / ml, in the examination of patients with COPD, the value of this marker was 1.27 ± 0.29 ng / ml, it was in 12.41% higher. The highest level of caspase-9 was observed in the third group of patients with COPD + AH – 2.16 ± 0.29 ng / ml, exceeding the

similar index of the first group of patients with hypertension by almost 50%, and the second group with COPD more than 70%, respectively statistically significant ($p < 0,05$). The comorbid pathology contributed to a higher level of activation of apoptotic processes in the body of patients compared with monopathology groups.

It is important to note that the relative risk of development of ventilation disorders in patients with comorbidity in combination with caspase-7 and 9 levels is higher Q75 is almost in 3.5 times (RR = 3.47 at 1.08-11.29 95% CI), with this ratio of odds is in 6.7 times higher (OR = 17.4 at 1.22-15.64 95% CI), compared with the cohort of patients with COPD without hypertension.

Conclusion. The obtained results require further clarification of the nature of the relationship between changes in the metabolism of cysteine proteases and the primacy of pathogenetic processes in the mechanisms of risk formation for complications and progression of ventilation disorders in patients with COPD in combination with hypertension. The reliable predictive value of elevated expression of caspase-7 and caspase-9 in relation to the severity of respiratory disturbances in patients with chronic obstructive pulmonary disease combined with arterial hypertension is shown.

АЛЕЛЬНИЙ СТАН ГЕНА АРО-В ЯК ЧИННИК РИЗИКУ ДОДАТКОВОЇ СУПУТНЬОЇ ПАТОЛОГІЇ У ХВОРИХ НА ХРОНІЧНИЙ ПАНКРЕАТИТ, ПОЄДНАНИЙ З ОЖИРІННЯМ ТА ЦУКРОВИМ ДІАБЕТОМ ТИПУ 2

Ферфецька К. В.

*кандидат медичних наук,
асистент кафедри внутрішньої медицини та інфекційних хвороб
ВДНЗ «Буковинський державний медичний університет»*

Кравчук Н. Д.

*викладач внутрішньої медицини
Чернівецького медичного коледжу
Буковинського державного медичного університету*

Бабій Ю. Є.

*викладач внутрішньої медицини
Чернівецького медичного коледжу
Буковинського державного медичного університету
м. Чернівці, Україна*

Актуальність. Отримані дані щодо асоціації *Ins/Del*-поліморфізму гена *Аро-В* із ліпідним обміном, атеросклерозом у різних популяціях та етнічних групах суперечливі, а особливості експресії даної мутації залежать від сукуп-