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CONCOMITANCE OF ENDOTHELIUM DISORDERS IN THE BRAIN AND HEART VESSELS IN ISCHEMIC HEART DISEASE

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INTRODUCTION & AIM

The beginning of the current millennium has been marked by a substantial increase in the prevalence of cardiovascular diseases, which rank second to third among the leading causes of mortality in industrialized countries [1]. One of the most severe complications, with a mortality rate ranging from 10% to 50% among patients with cardiovascular pathology, is chronic heart failure. It is also well established that the global progression of cardiovascular diseases is associated not only with lifestyle factors, nutrition, harmful habits, and socio-economic disturbances, but also with suboptimal intrauterine conditions.

According to contemporary concepts, endothelial dysfunction (ED) represents one of the principal pathogenetic mechanisms underlying cardiovascular diseases. Results from recent experimental and clinical studies have substantiated the concept of a significant causal relationship between endothelial dysfunction and the progression and/or development of arterial hypertension and hypertensive encephalopathy, atherosclerosis, chronic heart failure, diabetes mellitus, metabolic syndrome, chronic kidney disease and its complications, pulmonary hypertension, dilated cardiomyopathy, obesity, hyperlipidemia, hyperhomocysteinemia, and chronic cerebral ischemia.

The aim of this study was to investigate the structural and molecular features of ED in the myocardial and cerebral vessels in experimental chronic heart failure (CHF) and PH, as well as to evaluate the endothelioprotective potential of pharmacological agents targeting the nitric oxide system.

METHOD

This study was conducted on Wistar rats using experimental models of CHF (doxorubicin administration, cumulative dose 15 mg/kg) and PH (sodium nitrite 50 mg/kg administered to pregnant females on gestational days 16–21).

The endothelial status of cerebral and myocardial vessels was assessed using immunohistochemistry, ELISA, morphometric analysis, and real-time PCR. Key markers of endothelial function, inflammation, nitric oxide metabolism, oxidative stress, and angiogenesis were evaluated. The endothelioprotective potential of pharmacological agents acting on the L-arginine–nitric oxide system—Angiolin, Thiotriazoline, Hypertril, Nebivolol, and Mildronate— was also evaluated.

RESULTS & DISCUSSION

CHF and PH induced pronounced structural and functional endothelial alterations in the microcirculatory and muscular-type vessels of the heart and brain. These changes were characterized by reduced endothelial cell density, suppressed eNOS expression, increased iNOS expression, nitric oxide deficiency, elevated nitrotyrosine levels, and activation of proinflammatory cytokines (Figure 1). VEGF levels were significantly decreased, while apoptotic features of endothelial cells were intensified. Angiolin, a metabolitotropic cardio- and neuroprotective agent [2], and Hypertril, a cardioselective β-blocker with NO-mimetic properties [3], demonstrated the most pronounced endothelioprotective effects (Figure 2).

***Figure 1*. Pathogenetic mechanisms of endothelial dysfunction in CHF and PH**

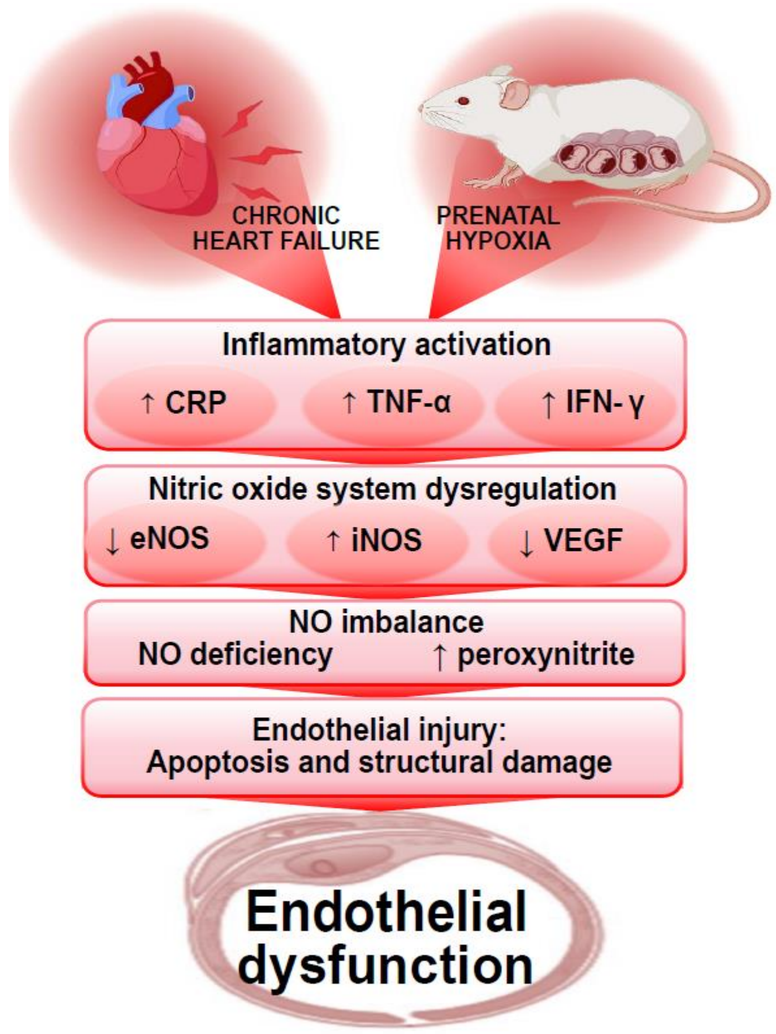
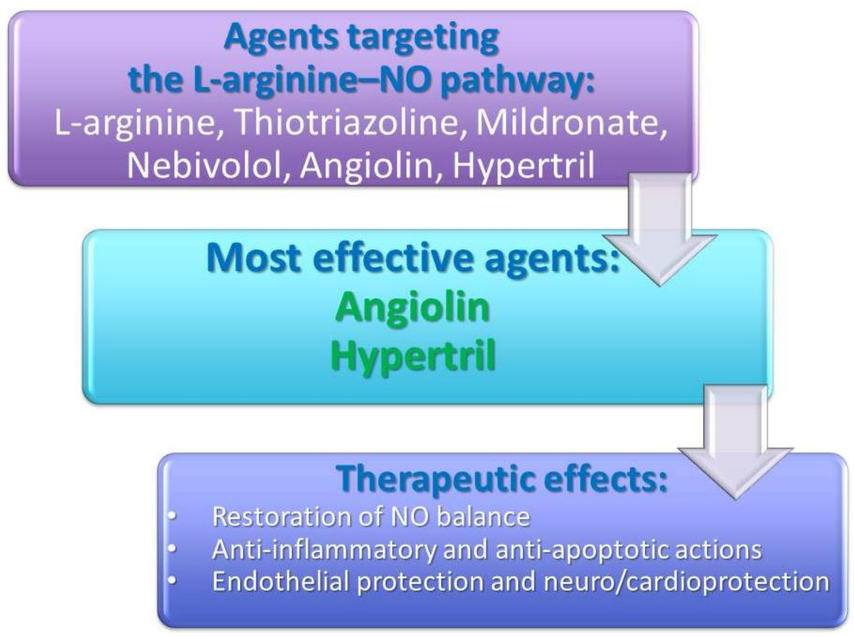


***Figure 2*. Endothelioprotective pharmacological strategies**



CONCLUSION

CHF and PH induce persistent ED in cerebral and myocardial vessels through disruption of the nitric oxide system, oxidative stress, and inflammatory activation. PH may act as an early trigger increasing susceptibility to cardiovascular and cerebrovascular diseases later in life. These findings support the rationale for targeted endothelioprotective therapy in ischemic cardiovascular pathology.

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