

Pathomorphology of Structural Changes in Brain Neurons of Patients Who Died from Chronic Ischaemic Heart Disease

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Abstract A total of 42 brain tissue sections from patients who died of chronic ischemic heart disease were subjected to pathomorphological examination. It was found that in chronic ischemic heart disease, ischemic and dystrophic changes develop in neurons in the cortex of the cerebral hemispheres. Also, varying degrees of expansion of the pericellular space and lightening of the neuropil are noted. It is emphasized that such changes should be taken into account in the prevention and treatment of chronic ischemic heart disease.

Keywords Chronic ischemic heart disease, Brain, Neuron, Pericellular space, Neuropil, Dystrophy

1. Introduction

On a global scale, ischemic heart disease is still one of the most pressing problems facing medicine today. Despite modern advances in the prevention and treatment of cardiovascular diseases, ischemic heart disease still occupies one of the leading positions [2,5,7]. In developed countries, mortality from this disease accounts for 1/3 of all deaths. More than 1 million people die from this disease every year. Changes in brain structures in chronic ischemic heart disease remain one of the pressing problems facing medicine, which has not been fully elucidated to date and is awaiting its solution [1,3,4,6]. Chronic ischemic heart disease has been a leading cause of death in economically developed countries for many years worldwide [8]. In 2018, it amounted to 17.3 million people and, according to a number of forecasts, will only grow and will amount to 23.6 million people by 2030 [9,10]. Based on 2018 statistics, acute heart attack is a frequent manifestation of cardiovascular diseases, and angina pectoris, the first sign of pathology, occurs in approximately 50% of patients [11].

The purpose of the study: To determine the pathomorphology of structural changes in brain neurons in patients who died from chronic ischemic heart disease.

2. Materials and Methods

Tissue sections taken from the brains of 42 patients who died of chronic ischemic heart disease in the departments of therapy and intensive care of the multidisciplinary clinic of the Samarkand State Medical University were studied in the Department of Pathological Anatomy. Anamnestic, macroscopic, microscopic, morphometric and statistical research methods were used to assess the relationship between morphological and morphometric changes in the neuronal structures of the brain and chronic ischemic heart disease. Sections were taken from the cortex and subcortical areas of the cerebral hemispheres. The size of the sections was 1x1 cm, the thickness did not exceed 0.5 cm, they were fixed in 10% neutral formalin, passed through an alcohol battery, and paraffin blocks were prepared. Histological sections 7-10 microns thick were stained with hematoxylin and eosin, Van Gieson and Nissl (nervous tissue) methods. Microphotographic techniques were conducted.

Histological preparations were studied and photographed using a LeicaGME microscope (Leica, India) coupled with a LeicaEC3 digital camera (Leica, Singapore) and a Pentium IV computer. Photo processing was carried out using Windows Professional applications.

3. Results and Discussion

The results of the study show that ischemic-type changes are observed in neurons in the superficial layers of the cerebral cortex of patients who died of chronic ischemic heart disease (Figure 1).

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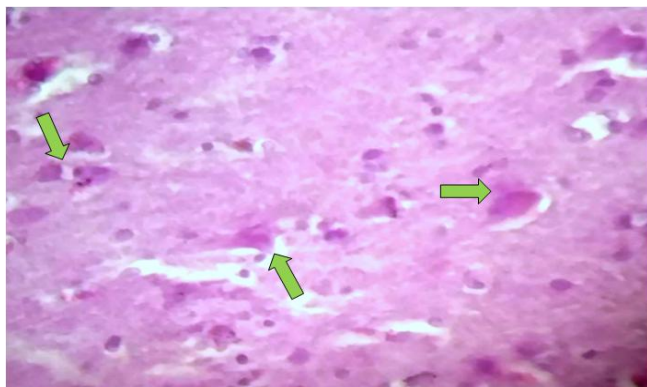


Figure 1. Development of ischemic-type changes in brain neurons. Stained with hematoxylin-eosin. Ob.40, ok.10

The nuclei of these cells are visible in a pyknotic and eccentric arrangement. We can see that the axons of neurons are thin and long, extending to a considerable distance from the neuron body. i.e. they can be seen at a great distance from the cell body. Neurons with hydropic dystrophy are detected in the deep layers of the cortex of the cerebral hemispheres. Narrow pericellular spaces are noted around many neurons and gliocytes. In some areas of the visual field, the pericellular space around neurons is enlarged. In this area, the nuclei of neurons are round in shape and hyperchromasia. The nuclei of some neurons are not detected in the visual field due to the development of the karyolysis process (Figure 2).

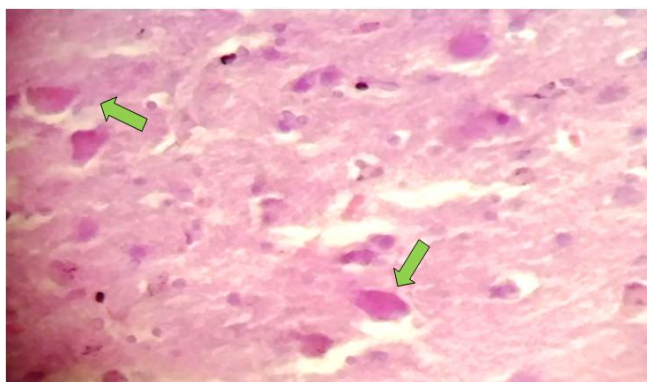


Figure 2. Necrobiotic and necrotic changes in brain neurons. Stained with hematoxylin-eosin. Ob.40, ok.10

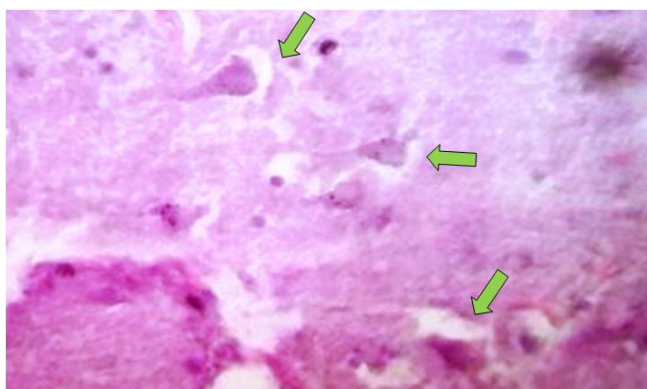


Figure 3. The state of the space around the neurons of the brain (expansion of the pericellular space). Stained with hematoxylin-eosin. Ob.40, ok.10

The neuropil is revealed to be lightened. In these areas, a sharp decrease in the number of gliocytes is noted. In patients who died from chronic ischemic heart disease, karyocytolysis is detected against the background of the development of hydropic dystrophy in the cytoplasm of neurons in the deep layers of the cerebral cortex. An expansion of the pericellular space around many neurons is noted (Figure 3).

In some preparations, dystrophic changes in neurons are detected, the number of gliocytes is reduced. Increased luminosity of the neuropil is observed. Necrotic changes in the form of perinuclear chromolysis and karyolysis are observed in a number of neurons (Figure 4).

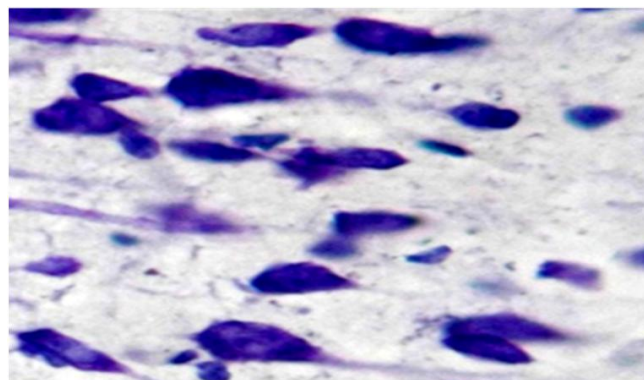


Figure 4. Perinuclear chromolysis and necrotic changes in brain neurons. Stained by Nissl method. Ob.40, ok.10

4. Conclusions

Thus, in chronic ischemic heart disease, ischemic and dystrophic changes develop in neurons in the cortex of the cerebral hemispheres. Also, varying degrees of expansion of the pericellular space and lightening of the neuropil are noted. Dystrophic changes in neurons are detected, and a decrease in the number of gliocytes is noted. Increased lightening of the neuropil is observed. Necrotic changes in a number of neurons and in the form of karyolysis are observed. Such changes should be taken into account in the prevention and treatment of chronic ischemic heart disease.

REFERENCES

- [1] Kaprin A.D., Starskiy V.V., Petrova G.V. Zlokachestvenno novoobrazovaniya v Rossii v 2017 godu (zabolevaemost va smertnost). M.: MNIIO im. P.A. Gertsena; 2018.
- [2] Koblyakov V.A. HIFa kantserogeneze kabi ob'ektlarni turli xil onkobelkov. Uspexi molekulyarnoy onkologii. 2018; 5(4): 64-71.
- [3] Kurman RJ, Carcanglu ML, Herrington CS, Young RH JSST Ayol jinsiy a'zolarining o'smalari tasnifi. 4-nashr. IARC. Jahon sog'liqni saqlash tashkiloti o'smalari tasnifi. Lion: IARC Press; 2014 yil.
- [4] Levakov S.A., Sheshukova N.A., Kedrova A.G. va boshqalar.

Molekulyar-biologik profil giperplaziyasi endometriya va endometrial intraepitelnoy neo-plaziyasi. Opuxoli jenskoj reproduktiv sistemy. 2018; 2: 76-81.

- [5] Orazov M.R., Radzinskiy V.E., Arakelov S.E., Xamoshina M.B., Nosenko Ye.N., Duxin A.O. i dr. Faktori riska giperplasticheskix prosessov endometriya u jenshin v reproduktivnom vozraste // Trudniy pasient. 2019. T. 17, № 5.
- [6] Tkachenko L.V., Sviridova N.I. Dvuxetapnyy metod lecheniya xronicheskogo endometrita u jenshchin va giperplastik jarayonlar endometriya v period-pauze. Gineologiya. 2016; 1: 40-44.
- [7] Radzinskiy V.E., Ordiyants I.M., Dobretsova T.A. Endometriy v ogne. Ostroe i xronicheskoe vospalenie endometriya: ot novykh vzglyadov k novym strategiyam. Status Praesens. Ginekologiya, akusherstvo, besplodnyy brak. 2016; 2: 126-132.
- [8] Rush CJ, Berry C, Oldroyd KG, Rocchiccioli JP, Lindsay MM, Touyz RM, Murphy CL, Ford TJ, Sidik N, McEntegart MB, Lang NN, Jhund PS, Campbell RT, McMurray JJV, Petrie MC. Prevalence of Coronary Artery Disease and Coronary Microvascular Dysfunction in Patients With Heart Failure With Preserved Ejection Fraction. JAMA Cardiol. 2021 Oct 1; 6(10): 1130-1143. doi: 10.1001/jamacardio.2021.1825. PMID: 34160566; PMCID: PMC8223134.
- [9] Wong ND. Epidemiological studies of CHD and the evolution of preventive cardiology. Nat Rev Cardiol. 2014; 11: 276-289.
- [10] World health statistics 2018: monitoring health for the SDGs, sustainable development goals. Geneva: World Health Organization, 2018. Licence: CC BY-NC-SA 3.0 IGO.
- [11] World health statistics 2018: monitoring health for the SDGs, sustainable development goals.