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PATHOMORPHOLOGICAL CHANGES IN CEREBRAL VASCULATURE IN ISCHAEMIC STROKE

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ABSTRACT:

The article is devoted to the pathomorphological features of ischemic brain strokes. The aim of the investigation was to establish the morphological peculiarities of the cerebral microcirculatory stroke development. The analysis of 48 cases was carried out, in which an acute cerebral circulation disorder was diagnosed. It was found that at cerebral artery ischemia in the brain tissue in the early period after ischemia initially develops discirculatory and edematous phenomena, then begins the predominance of destructive-necrotic changes in both neural and glial cells. To these destructive changes in the focus of acute ischaemia, an inflammatory-regenerative response develops in the form of vasodilation, migration of leucocytes from the blood to the focus of destruction, and formation of an inflammatory-cell demarcation ramp around the necrosis.

Keywords: acute impairment of cerebral circulation, brain, ischemic stroke, pathomorphological features.

RELEVANCE:

Stroke is the second most frequent cause of death in the world in 2011 (coronary heart disease being more frequent. Stroke kills about 6.2 million people a year (about 11% of all deaths). About 17 million people suffered a stroke in 2010. About 33 million people have previously had a stroke and were still alive in 2010. Between 1990 and 2010, the number of strokes fell by about 10% in developed countries and increased by 10% in developing countries. A higher risk of death from stroke is observed in South Asians, who account for about 40% of deaths from stroke. In 2015, stroke was the second most common cause of death after coronary heart disease, accounting for 6.3 million deaths (11% of the total). In the USA, stroke is the leading cause of disability and was the fourth leading cause of death in the early 2010s. It should be noted that mortality in ischemic stroke reaches 40% or more, and the outcome of stroke depends largely on the size and location of cerebral infarction. Clinical and morphological studies have established that large, large and moderate cerebral infarcts are caused by obstructing atherothrombosis, cardiogenic or arterio-

arterial embolism. Extensive and large infarcts have occasionally been caused by atherostenosis of 70% or more of the extracranial arteries in combination with extracerebral causes of severe blood pressure drop.The mentioned cause, along with tandem atherostenosis, in which stenotic atherosclerotic plaques are located in extra- and intracranial arteries, determines the occurrence of middle, small superficial and small deep infarcts. It should be emphasized that the specific weight of each of these etiological factors in the occurrence of large, large, medium, small infarcts localized in the internal carotid, vertebral, basilar arteries and their branches is not fully understood, and the percentage of infarcts of unknown genesis reaches 30% and more in the clinic. However, the correct interpretation of stroke pathogenesis is crucial, as it determines the treatment strategy. In atherosclerosis of cerebral vessels, multiple infarcts of different size, localization and duration are not uncommon. Infarcts in such cases are usually accompanied by a more complex clinical picture and often lead to severe neurological deficits and mental dysfunction up to the development of dementia. The incidence of multiple cerebral infarcts varies widely, ranging from 30-82% of ischaemic stroke cases. However, few studies have investigated the causes of multiple infarcts, and the findings require further clarification. All of the above points to the need for a comprehensive morphological study with a detailed study of structural changes in the brain and all parts of its vascular system, in order to clarify the main factors of single and multiple strokes of varying magnitude depending on localization.

MATERIALS AND METHODS OF STUDY:

As material, a retrospective analysis of 48 protocols of pathological examination of the units of the Republican Pathological Centre in the period 2019-2020 was carried out. Common research methods were used, i.e. macro- and microscopic examination of the brain and its arterial system at all structural and functional levels, including the main arteries of the head - internal carotid and vertebral arteries, intracranial arteries - the vessels of the villous circle and their branches, as well as intracerebral arteries and vessels of the microcirculatory bed (MCB) were performed. The study of the brain determined the size and localization of intracerebral hematomas, the presence of blood breakthrough into the ventricular system, the severity of cerebral edema, dislocation and compression of the brain stem. Visualized changes in the brain (small hemorrhages, foci of perivascular edema, spongioform state of white matter) were taken into account. Microscopic examination of the brain was performed in histological preparations encased in paraffin. Which were stained with hematoxylin and eosin, according to van Gieson methods (detection of collagen fibers and myocytes in vessels), Weigert (detection of elastic fibers in vessels). Particular attention was paid to microcirculatory vessels within hematomas, in the perifocal zone, as well as at a distance from the hematomas.

RESULTS OF THE STUDY AND THEIR DISCUSSION:

All were diagnosed with acute cerebral circulation disorder (ACD) of the ischaemic type. Among them 34 males (70,8%) aged from 20 to 72 years and 14 females (29,2%) aged from 33 to 65 years. The results of microscopic examination of the brain at cerebral artery ischemia showed that in the early period of ischemia widespread dyscirculatory changes in the form of paralytic dilatation of vessels, especially vessels of the microcirculatory barrier, were observed in all parts of the brain. At the same time, more pronounced dilation of

blood vessels was observed in the area of the nuclear structures of the intermediate brain and in the deep layers of the cerebral cortex. The arterial walls were somewhat thickened due to edema and loosening of the endothelium and basal membrane with the formation of a light edematous zone in the perivascular zone (Fig. 1**).** In later periods of ischaemia, more pronounced and widespread dyscirculatory and oedematous phenomena were observed in the brain tissue, which were also particularly pronounced in the nuclear structures of the intermediate brain and in the cerebral cortex. Most of the vessels were paralytically dilated, but the lumen together with blood elements showed a pink mass (Fig. 2) and peripherally located leucocytes. In the cerebral cortex, the nerve cells lose their typical arrangement, their borders are indistinct, and pronounced pericellular oedema is determined in the circumference of both nerve cells and glial cells. At this time, some nerve cells show signs of autolytic necrosis in the form of cytoplasm vacuolization, membrane disintegration, disappearance of tiger substance, lysis or pycnosis of nuclei (Fig. 3).

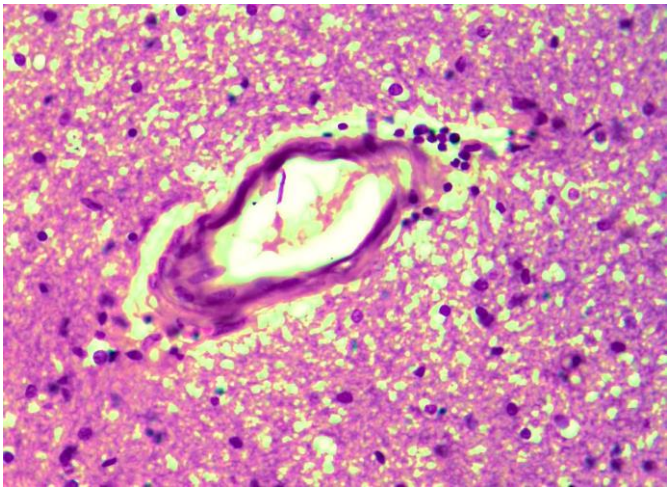

Figure 1. The arterial wall is thickened due to edema and loosening of the endothelium and basal membrane with the formation of a light edematous zone in the perivascular zone. Staining: Haematoxylin-Eosin. Magnification: 10x40.

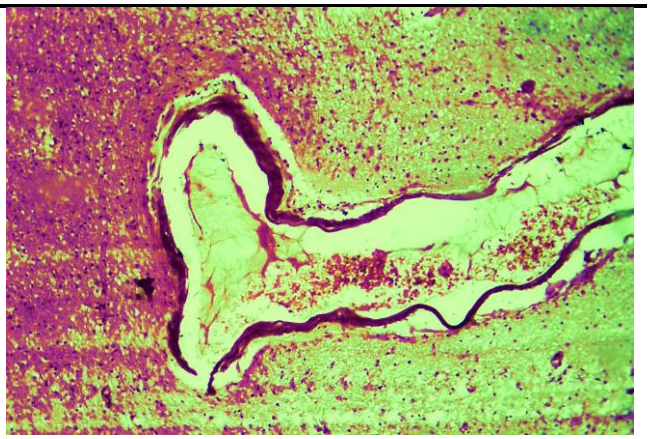

Figure 2. Paralytic vein dilation, pink and fibrillar masses are identified in the lumen together with blood elements: Staining: Haematoxylin-Eosin. Magnification:10x10

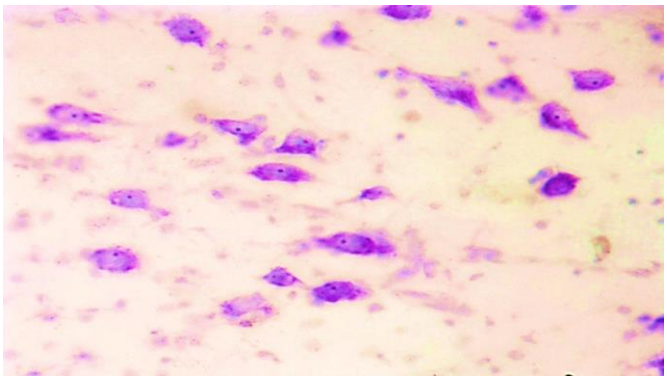

Figure 3. Vacuolisation of cytoplasm, disintegration of membranes, disappearanceof tiger substance, lysis or pycnosis of nerve cell nuclei. Staining: Nissl. Magnification: 10x40.

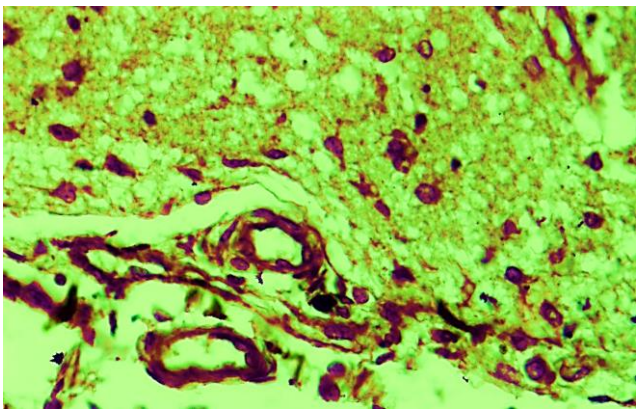

Figure 4. Perivascular and pericellular oedema is noted in the immediate vicinity of the ischaemic areas of the brain. Staining: Haematoxylin-Eosin. Magnification: 10x40.

In acute ischaemia, destructive-necrotic and reactive changes already predominate in the tissue of the brain and intermediate brain over dyscirculatory disturbances. Immediately in the areas of ischaemia the nerve cells are in a state of complete autolytic decay with the formation of large vacuolised cavities together. Where cerebral substance and cytoplasm of nerve cells completely disintegrated, only nuclear structures remained in the form of cellular shadows. Perivascular and pericellular edema is also noted in areas of the brain immediately adjacent to the ischaemic areas (Fig. 4). Where the nerve cells lose their normal orientation with tiger substance decay and some nuclei wrinkling. On the glial cell side, there is some activity in the form of hyperchromasia of the nuclei and an increase in their number. Vessels are sharply dilated, full of blood, lumen has increased number of leukocytes with peripheral location and migration through wall into perivascular space.

At prolonged ischemia in the ischemic area of the brain the development of complete necrosis of nerve cells and pronounced infiltration by polynuclear leukocytes and macrophages is noted. At the same time the vessels are paralytically dilated, their walls are torn, in the lumen there is a pink squirrel mass and polynuclear leucocytes. The entire necrotic nervous tissue is diffusely infiltrated with leucocytes, glial cells and isolated macrophages, with destructive and dying forms predominating among these inflammatory cells, with the formation of small foci of decay and non-structive foci (Figure 5). Dyscirculatory, reactive and oedematous phenomena in the form of marked perivascular and pericellular oedema persist in the surrounding ischaemic areas of the brain tissue. Foci of inflammatory cellular infiltration of polynuclear leukocytes and macrophages are identified around the vessels. In the nerve tissue itself, there is pronounced focal and, in places, widespread gliosis.

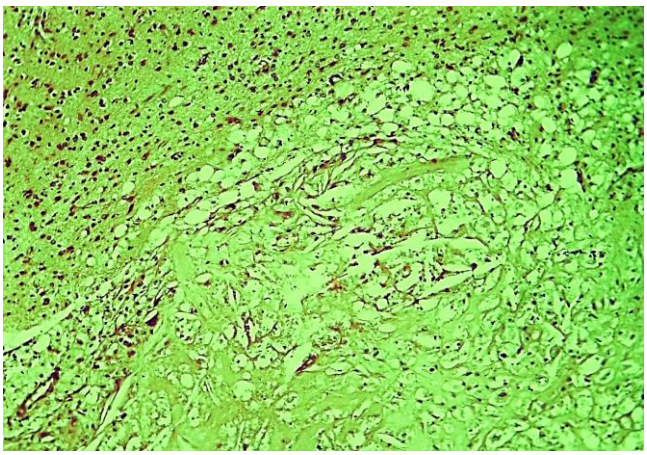

Figure 5. Formation of small foci of decay and destructive foci in the ischaemic zone. Staining: Haematoxylin-Eosin. Magnification:10x10

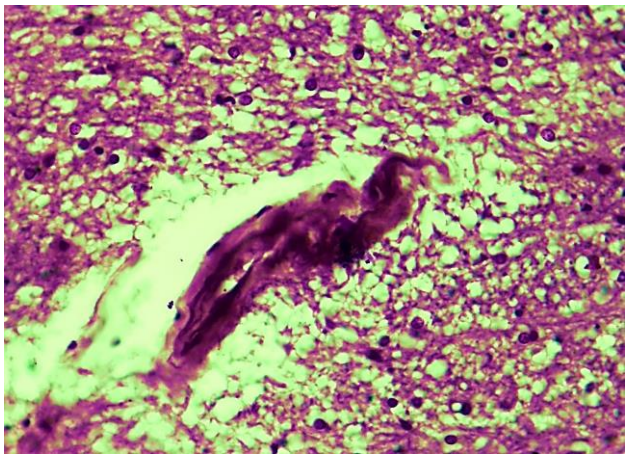

Figure 6. Narrowing of the lumen of the arterioles, due to thickening and compaction of the structural elements of all layers of the wall. Haematoxylin-Eosin. Magnification: 10x40.

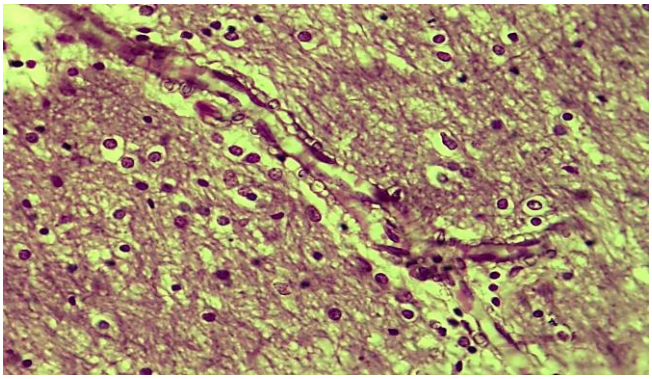

Figure 7. Dilation of postcapillary venules and brain tissue veins with vacuolisation of the adventitia and perivascular space. Haematoxylin-Eosin. Magnification: 10x40.

The results of the morphological study of the blood-brain barrier during acute cerebral ischemia showed that the main pathomorphological changes characterizing the restructuring of the structural elements of the GEB occurred in the walls of the microcirculatory channel vessels. Arterioles initially responded to acute ischemia in the form of pronounced vasoconstriction with increasing capillary blood flow, which morphologically manifested as narrowing of arteriolar lumen, thickening and compaction of structural elements of all their wall layers (Fig. 6). Pre-capillaries and capillaries of brain tissue are dilated, filled with blood elements, including the presence of leucocytes. Their wall is due to stretching of endothelial and pericytic cells and thinning of the basal membrane. The postcapillary venules and veins of brain tissue are particularly dilated with vacuolization of the adventitia and perivascular space (Fig. 7), indicating that blood flow in the venous part of the angioarchitecture of brain tissue slowed during acute ischemia. In the subsequent terms of acute ischemia, pronounced pericellular edema or expansion of the Virchow-Robin space was observed in all sections of the cerebral microcirculatory bed vessels, indicating that due to ischemia, biological active substances were released in the microcirculatory bed areas, leading to expansion of the vessel wall, increased permeability and release of liquid part and blood plasma proteins into the surrounding tissue, which is the beginning of the GEB patency disruption. On the neural tissue side, activation of pericytic cells of the vascular wall and microglia localised around the vessels occurs under the influence of biological inflammatory factors on the damage (Figure 8). When morphologically manifested by the proliferation of these cells and the transformation of microglia into macrophages, which participate in the process of resorption of damaged necrotic tissue in the focus of acute ischaemia. Due to this, widespread perivascular and intracerebral inflammatory infiltrates from haematogenous, histiogenic cells as well as from macrophages of microglial origin develop in all parts of the peri-ischaemic areas of the brain tissue. These changes end with the development of secondary complications in the form of perivascular sclerosis and diffuse and focal gliosis of the neural tissue.

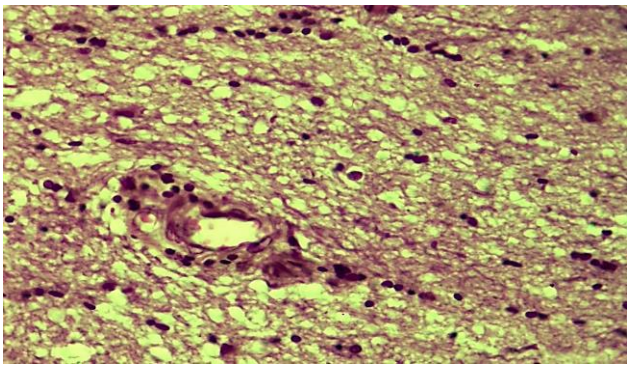

Figure 8. Proliferation around the vessels of lymphoid cells and macrophages. Haematoxylin-Eosin. Magnification: 10x40.

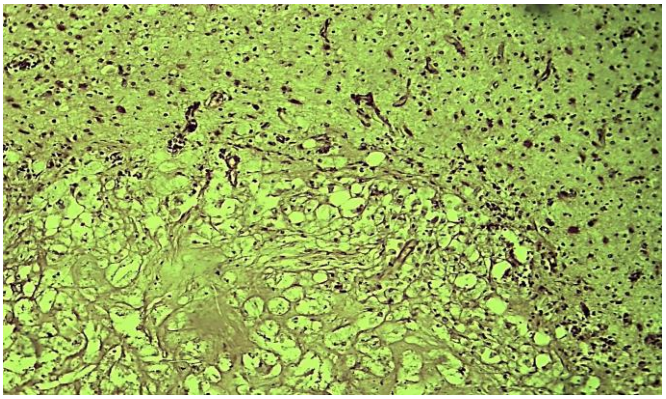

Figure 9. Cell-free homogenous necrotic mass in the ischaemic focus. Haematoxylin-Eosin. Magnification: 10x40.

In prolonged cerebral artery ischaemia in the cerebral hemisphere and in the intermediate brain, three distinct lesion areas are clearly defined. In the centre of the ischaemic cerebral tissue, a huge field consisting of a cell-free, homogeneous necrotic mass is defined (Fig. 9). A second zone consisting of densely arranged cellular infiltration densely surrounds the central

necrotic zone. The cellular infiltration is formed around the necrosis as a demarcation line of inflammatory cells dominated by polynuclear leucocytes and macrophages. The third zone is represented by the surrounding nervous tissue with marked dyscirculatory and reactive changes. Where vessels are significantly dilated, full of blood, some of them form cavernous cavities. Their walls are thinned and in some places torn, endothelial cells are hypertrophied. The concentration of leucocytes and blood monocytes around the vessel wall indicates their increased migration to the focus of ischemia and necrosis. The nerve tissue itself is swollen and stained pale, with glial cells and polynuclear leukocytes predominating in its cellular composition.

CONCLUSION:

The morphological studies have established the diagnostic features of ischemic strokes. In particular, in cerebral artery ischemia, initially dyscirculatory and edematous phenomena develop in the brain tissue early after ischemia, followed by the predominance of destructive-necrotic changes in both nerve and glial cells. In response to these destructive changes, an inflammatory-regenerative response develops in the circumference of the acute ischaemic focus in the form of vasodilation, migration of leucocytes from the blood to the focus of destruction, and formation of an inflammatory-cellular demarcation ramp around the necrosis.

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