THE MORPHOLOGICAL PECULIARITIES OF THE CEREBRUM ARTERIES UNDER THE HEMORRHAGIC AND ISCHEMIC STROKE IN THE PATIENTS WITH THE METABOLIC SYNDROME

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ABSTRACT

In this article, the results of the morphological study of the cerebral arteries in the patients with the hemorrhagic ischemic stroke on the background of metabolic syndrome were submitted. We established that under hemorrhagic stroke on the background of metabolic syndrome one could observe the atherosclerotic damages in the form of plaques, hyalinosis of vessels walls, destructive and necrotic changes of the middle coat of vessel wall, which are, in our opinion, the main reason in morphogenesis of hemorrhagic stroke. The atherosclerotic affection of the cerebral vessels with the lipidosis and fibrotic plaques in the vessel wall, which caused in disorganization of the vessel wall in the form intimae proliferation, structural reconstruction of the internal elastic membrane which resulted in narrowing of the vessel lumen and development of ischemia is the morphological basis for the ischemic stroke with the metabolic syndrome.

Also diabetic microangiopathy with the development of the hyalinosis, vessels walls sclerosis, which provoked the microcirculatory and metabolism processes injury in the cerebral substance that complicate the disease course is the important factor of cerebral blood circulation damage.

UDC CODE & KEYWORDS

■ UDC: 616 - 008.9 – 06:616.381 – 005.4 – 091.8 ■ Cerebral arteries ■ Hemorrhagic stroke ■ Ischemic stroke ■ Metabolic syndrome

INTRODUCTION

Cardio-vascular diseases, including cerebral blood circulation disorders, are the leading mortality reason in the world. They are also the main cause of the disability of the essential part of the employable population, and therefore represent important medical and social problem (Kolotilov, 2011, Vaartjes et al, 2013). Combination of several hazards considerably raises the probability of the development of the most serious cardio-vascular diseases (Tanashian et al, 2007, Orlov et al, 2008, Mychka, Chazova, 2009). Metabolic syndrome (MS) is the combination of the factors (arterial hypertension, hyperinsulinemia, dislipidemia, obesity), which increase the risk of the appearance of the cardio-vascular diseases, favour their earlier development, rapid progress and the possibility of serious complications. Due to the last classification, the diagnosis “metabolic syndrome” is considered reliable when at least thereof the following factors are available (European guidelines on cardiovascular disease prevention on clinical practice, 2003). MS is the complex of metabolic disorders, which are pathogeneticaly connected, and therefore it is very difficult to identify the correlation of the causes and effects of its manifestations and complications. The investigation of the MS components in correlation with its main display of the cardio-vascular pathology, including cerebral stroke, can help to understand the pathogenesis of this syndrome.

The main manifestation of the MS is carbohydrate and lipid metabolism disturbance as well as disorder in the processes of regulation of the arterial blood pressure and endothelium function, the main reason for which is the reduction of insulin sensibility of the tissues. The direct glucose effect on the matrix proteins of the vascular walls is accompanied with the formation of the cross-relations between collagen fibers, and results in increase of the arteries rigidity, arouse release of the oxygen radicals that provokes endothelial dysfunction (Melvin, 2006). As a rule, hemorrhagic stroke develops in patients with arterial hypertension, which is one of the MS components. Metabolic disorders give rise to the disorganization of the vascular wall that is the main reason of the ischemic stroke (Bays, 2003, Sitia et al, 2010).

Although clinical and pathophysiological investigations of the acute disorders of the cerebrum blood circulation are carried out actively in recent years, the phatomorthological basis of this widely spread pathology remains byways of learning. Therefore, the study of the morphological peculiarities of the cerebral arteries in the patients with hemorrhagic and ischemic stroke on the MS background became the aim of our research.

Materials and methods

We conducted the morphological study of the cerebral vessels in 30 cases sectional materials in patients with hemorrhagic stroke and in 32 patients with the ischemic stroke in combination with such manifestations of the metabolic syndrome as hypertension, diabetes mellitus of the II type and dislipidemia. The comparison group was formed by 30 cases, where the patients’ death was not connected with the vascular disease of cerebrum.

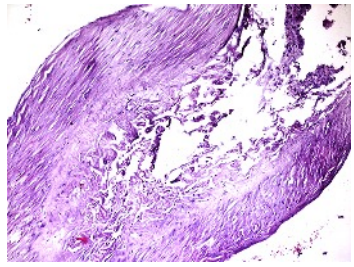
We fixed the materials in the 10% neutral formalin solution, dehydrated them in the series of alcohol of the increasing concentration, embedded them in paraffin; to colorize paraffin sections of 5-7mcm thickness we used hematoxylin and eosin, trichromium after Mason (detection of collagen fiber), fuxelin after Hart (identification of elastic fiber) picrofuxin after Van Gizen to detect the connective tissue and sudan III to detect lipids. The study was carried out using the microscope MBR-3 model by different magnification power (ocular 10, objective 10-20-40).

Results and discussion

Patients of the research groups were from 40 to 60 years old. The average age of the died patients with the hemorrhagic stroke was52,6 +2,8 years, with the ischemic stroke – 54,8+3,6 years. We examined internal carotid arteries, middle cerebral arteries and cerebral floor arteries, which main function is blood supply, and microcirculatory vessels, which provide the exchange processes in cerebrum substance.

Under the hemorrhagic stroke, we noticed considerable thickening of vessels due to the fibrosis and proliferation of all wall's layers (see Figure 1).

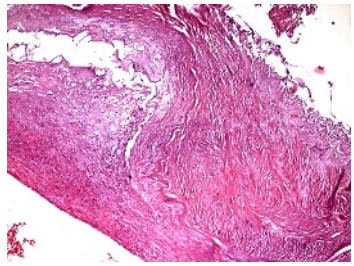
Figure 1: Considerable thickening of the artery wall with narrowing of the lumen. Hematoxylin&Eosin [x200]



Source: Author

In the majority of large vessels, we found atherosclerotic plaques, which were represented by cellular, fibrous and lipid component – foam cells with detritus (see Figure 2).

Figure 2: Fibrous-lipid plaque of artery. Hematoxylin&Eosin [x200]



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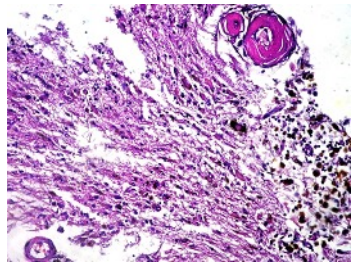
In most vessels of the smaller size, we discovered hyalinosis of wall with thickening and lumen narrowing, sometimes considerable. Usually we found out accumulation of hemosiderofag (Figure 3).

Figure 3: Hyalinosis of the small vessels, accumulation of the hemosiderofags. Hematoxylin&Eosin [x200]



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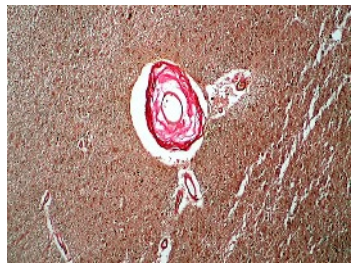
In the small vessels, we also noticed thickening of the vessels' walls with lumen narrowing due to the proliferation of the collagen fibers in the wall (see Figure 4).

Figure 4. Artery wall thickening due to the proliferation of the collagen fibers. Trichrome after Mason's staining [x200]



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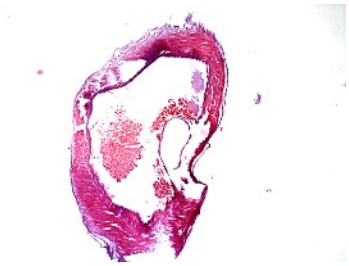
In some vessels, we discovered destructive changes in the middle layers of arteries, sometimes media necroses (see Figure 5).

Figure 5: Destructive changes, necrosis in the middle layer of artery. Hematoxylin&Eosin [x200]



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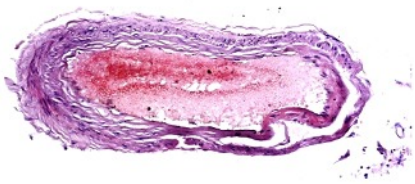
Destructive changes in the middle layers of arteries under arterial hypertension can develop when plasmoraghia presents, because of insudacia of plasma components in the vessel wall that causes its necrosis. The reason for the isolated necrosis of the middle coat of arteries can be also prolonged vasospasm, which causes vessel wall hypoxia with ion exchange disorders in myocytes (Kistenev et al, 2007). Spasm can develop in muscular type arteries, such as the arteries of cerebral surface and intracerebral arteries with the diameter of 150-500mcm. The middle coat of these arteries, which is represented by 5 layers of smooth muscle cells, well-marked inner elastic membrane as well as great number of nerve adrenergic fiber, are exactly that wall structures, which can cause artery's spasm under hypertensive crisis (Ceska, 2007).

We also discovered middle-sized intacerebral arteries with focal thinning and destructive changes of all wall's coats (see Figure 6). These changes can result in wall's rupture that is the reason for hemorrhage of the brain.

Figure 6: Focal thinning of artery wall. Hematoxylin&Eosin [x200]



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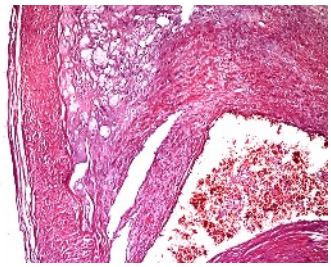
In cases of ischemic stroke while investigating cerebrum arteries of large and middle size, we found out the significant thickening of the wall with the considerable lumen narrowing and presence of the atherosclerotic plaques (see Figure 7).

Figure 7: Considerable thickening of the artery wall with lumen narrowing, and presence of the atherosclerotic plaques. Hematoxylin&Eosin [x200]



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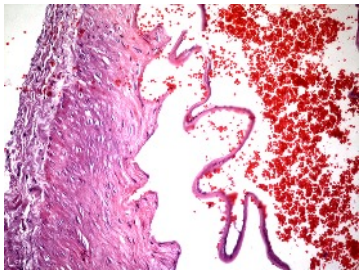
In artery wall, we noticed the morphological changes in the form of proliferation, desquamation of intima (see Figure 8).

Figure 8: Artery wall – desquamation of intima. Hematoxylin&Eosin [x200]



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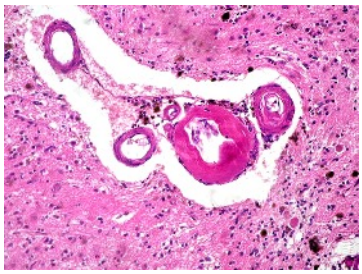
Damage of endothelium causes the penetration of blood plasma components into inner arteries coats, that stimulate proliferation of myocytes and connective tissue cells that results in the local thickening of inner coat (Hansen, Bray, 2008).

In the progress of atherosclerosis, the condition of metabolism in inner and middle artery coat is very important, considering the fact that they are nourished by way of plasma perfusion. Therefore, under hyperplasia of intima there exist conditions for plasma perfusion disorders through the vessel wall that creates the conditions for the development of lipidosis of intima and media of the cerebral vessels (Sharipova et al., 2009, Leng, 2013).

The occurrence of dislipoproteinemia of atherogenic character, lipoprotein flux into the inner coat of arteries, cellular rearrangement in the inner coat and capture by cells lipoproteins of diminished density, which come from blood, as well as formation of fiber tissue by mesenchimal cells are necessary factors for atherosclerosis development (Hansen, Bray, 2008, Kim et al, 2012). Some substances, e.g. cholesterol and lipoproteins, as well as changes in endothelium can stimulate atherogenesis; activated and damaged endothelium can take an active part in atherogenesis. Activation of endothelium is attended by the production of cellular growth factor (Sitia et al., 2010).

In the vessels of smaller diameter, we also noticed the formation of atherosclerotic plaques, thickening of the whole artery wall due to intima and media proliferation with the considerable narrowing of lumen. At the same time, we often noticed hyalinosis of the wall in the vessels of smaller diameter (Figure 9).

Figure 9: Hyalinosis of the cerebral artery. Hematoxylin&Eosin [x200]



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While examining vessels of the microcirculatory channel, we noticed in some cases growth in their number because of neoangiomatosis, which is connected with hypoxia of the cerebral tissue.

Later on, we are planning further study of the morphological and functional mechanisms of changes in vascular wall of cerebral arteries using immunohystochemical, ultrastructural methods for the correction of these changes under metabolic syndrome.

Conclusion

Under the hemorrhagic stroke on the background of MS we observed atherosclerotic affection in cerebral vessels in the form of plaques, hyalinosis of the vessels walls, and destructive and necrotic changes of the middle coat of the vessel wall, that are, from our point of view, the most important in morphogenesis of hemorrhagic stroke.

Atherosclerotic affection of the cerebral vessels with lipidosis and fiber plaques in vessels wall, disorganization of the vessel wall in form of intima proliferation, structural change of the internal elastic membrane, which results in vessels lumen narrowing and ischemia development, are the morphological basis for development of the ischemic stroke under the metabolic syndrome.

Diabetic microangiopathy with development of hyalinosis, sclerosis of vessels wall that damages the microcirculation process of metabolism in medullary substance that complicates clinical course, is the main factor of derangements in cerebral blood circulation.

Pathogenetic cure and prevention measures of the cerebral blood circulation damages in patients with metabolic syndrome should contain both antithrombotic and endotelioprotective therapy, and that kind of treatment, which normalize the manifestations of metabolic syndrome, hypotension and hypoglycemic means in particular.

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