Morphological changes in the brain in severe traumatic brain injury aggravated by alcohol intoxication

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ABSTRACT

All components of the human dura mater and its vascular system in traumatic brain injury were studied: all layers of the dura mater, vessels of the arterial and venous type, the nature of branching, their condition. Pathological processes in the form of hyalinosis and fibrosis of the layers of the vascular wall under the influence of alcohol cause bifurcation-hemodynamic insufficiency, causing protrusion of the vascular wall. In this case, we can talk about alcohol-dependent bifurcation-hemodynamic insufficiency.

Keywords: dura mater, alcohol intoxication, meningeal bleeding

Introduction. One of the main factors predisposing to the occurrence of meningeal bleeding of traumatic origin are various changes in the vascular bed of the dura mater (dura mater). It is known from a review of the literature that the pathology of the vascular wall as a cause of meningeal bleeding can be represented by various pathological conditions and diseases that are either local or widespread. These issues are most fully studied in the aspect of meningeal hemorrhages against the background of arterial hypertension and/or in the age aspect [1, 2, 3, 4, 5]. With traumatic meningeal bleeding, this issue is relevant, and this nosological unit against the background of alcohol intoxication remains a practically unexplored issue, which required a detailed study.

Purpose: to study the components of the DM and its vascular system in traumatic brain injury (TBI): all layers of the DM, vessels of the arterial and venous type, the nature of branching, their condition. Materials and research methods. The material for the study was autopsy and biopsy material (dura mater fragments) taken from patients with meningeal bleeding. Methods - histological, morphometry, statistical.

Results of the study and their discussion. When studying the places of branching of the arteries of the dura mater, local expansions of the vascular wall are found, which are always located precisely in the places of branching of the vessels of both arterial and venous types. Pathological processes in the form of hyalinosis and fibrosis of the layers of the vascular wall under the influence of alcohol cause bifurcation-hemodynamic insufficiency, causing protrusion of the vascular wall. In this case, we can talk about alcohol-dependent bifurcation-hemodynamic insufficiency (ABGN).

Pathological changes were detected in all layers of the vascular wall. Endothelial defects were the most constant phenomenon. A change in the shape and orientation of endotheliocytes, total and/or partial desquamation was revealed. Pathological processes in the form of

hyalinosis and fibrosis of the layers of the vascular wall under the influence of alcohol cause bifurcation-hemodynamic insufficiency, causing protrusion of the vascular wall. In this case, we can talk about alcohol-dependent bifurcation-hemodynamic insufficiency (ABGN).

Pathological changes were detected in all layers of the vascular wall. Endothelial defects were the most constant phenomenon. A change in the shape and orientation of endotheliocytes, total and/or partial desquamation was revealed. In the naked intima, in all cases, leukocyte infiltration, the appearance of individual phagocytes, formed parietal thrombosis, sludge phenomenon, total thrombosis and diapedesis of erythrocytes into the perivascular space were noted. In traumatic recurrent meningeal bleeding in patients with severe TBI and with an unaggravated alcohol history, pathomorphological changes were much more pronounced, with microscopy they showed clearly traced phenomena of occlusion of the microvasculature by aggregates and agglutinates of blood cells. This phenomenon can be regarded as "pre-thrombotic readiness" of the microvasculature. In the vast majority of cases (more than 73%), diapedetic perivascular hemorrhages were visualized against the background of the disorders described above. Massive hemorrhages in the DM were often combined with areas of microerosion formation and even zones of necrosis (37.1% of cases). The reason for the formation of microerosions and zones of necrotic changes during relapses of meningeal gemorrhagia in patients with severe TBI are pronounced microcirculatory disorders in the microvascular bed of the dura mater due to prolonged exposure to alcohol.

The formation of longitudinal folds of the vessel wall was revealed, protruding into its lumen and narrowing it by half, as a result of which it acquired a stellate appearance. This phenomenon is due to changes in the muscle layer and a sharp tortuosity of the internal elastic membrane (fibrillation, thickening, increased tortuosity). In transverse sections, the folding of the middle layer of the vascular wall has the form of oval bays, which determine the crimpiness of the inner contour of the vessel. Muscle cells in the folds had a disturbed orientation. The bundles of myocytes in the area of the bottom of the bays are sharply stretched and tightly adjacent to each other. In separate folds, dystrophic and necrotic changes are determined. In the muscular membrane there is diffuse edema - stratification of intermuscular spaces. In separate folds, in the muscle layer, the formation of collagen fibers is determined. The revealed structural disorders characterize the mosaic pattern of damage to the dura mater and its vascular system during meningeal bleeding of traumatic origin in patients with severe TBI and an unaggravated alcohol history. The morphological substrate for the development of relapses of meningeal bleeding of traumatic origin in patients with severe traumatic brain injury aggravated by an alcoholic history is: an increase in the density of the vascular network of the dura mater, its expansion and hemorrhagic impregnation due to significant changes in microvessels; the presence of zones with empty microvessels - zones of "ischemia"; the presence of veno-venular, arteriolo-venular and arteriolo-arterial anastomoses; pronounced tortuosity and a different diameter of the lumen of the anastomoses throughout. The vessels have a tortuous course; along their length, multiple areas of bulging of the walls are determined. Moreover, the bulging of the walls of the vessels of the microvasculature of the DM is characteristic of the vessels of the middle layer. In the lumen of most of the vascular system of the dura mater, erythrocyte aggregates are determined. Dilatation of the vessels of the postcapillary-venular link is noted. The tortuosity of the efferent microvessels in the inner layer of the DM sharply increases. Erythrocyte aggregates are determined, which fill the gaps of both the outlet and afferent microvessels. Local hemorrhages

are determined around individual capillaries, postcapillaries and venules.

In the middle layer of the DM, in the occipital and frontal regions, single main vessels with obliterated branches and a depleted capillary network, elongated into single chains, are determined. In the parietal and temporal regions, there are a large number of convoluted arteries and arterioles with a microvascular network, with a high density of capillaries. Local hemorrhages are determined around individual postcapillaries of venules. In the middle and inner layer of the dura mater, the number of arteriolovenular anastomoses increases, their outlet sections are expanded in places. By means of non-functioning capillaries, low-vascular zones are formed. There are signs of hemorrhagic and leukocyte infiltration in the paravasal connective tissue. Along with this, there is a restructuring of the vessels of the postcapillary-venular link. As part of the structural-functional unit, there are bifurcations and trifurcations of arterioles, the daughter branches depart at acute, obtuse and unfolded angles, the lumens of the venules are expanded. There are multiple arteriolo-venular anastomoses between the structural and functional units of the microvasculature. In the outer layer of the DM, the location of the venous component of structural and functional units loses its alternation with respect to the middle layer of the DM. The detected changes in the microvasculature of the DM (erythrocyte aggregation, slugging phenomenon, plasma separation) inevitably lead to the formation of micro- or macrothrombi in the vessels. Mixed thrombi were most often determined, while erythrocyte and hyaline thrombi were much less common. Fibrin thrombi were determined in the form of a thin mesh.

Thrombosis phenomena were noted mainly in small vessels, while sludge syndrome prevailed in larger vessels. Thrombotic changes in the microvascular bed of the DM in traumatic meningeal hemorrhages correlated with the nature and severity of TBI, as well as with the hemorheology and hemocoagulation disorders caused by it.

Conclusion. With traumatic meningeal bleeding in patients with a burdened alcohol history, the formation of fibrin or mixed thrombi quite often took place. Around such vessels in more than half of the observations, the phenomena of plasma impregnation were noted.

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