Changes in Mast Cells of Vascular Plexuses of Human Cerebral Ventricles in Atherosclerosis of Precerebral Arteries

T. M. Babik

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Scanty mast cells with high saturation and weak degranulation located perivascularly and in the subepithelial zone of the villi were detected in vascular plexuses of human brain. Cerebral atherosclerosis was associated with pronounced changes in their morphofunctional organization: changed shape, decreased volume and index of saturation and degranulation, predominating in the villous part of the vascular plexus of the lateral ventricle.

Key Words: *mast cells; vascular plexuses; brain; atherosclerosis*

One of the pathogenetic mechanisms disordering the function of the blood-brain barrier (BBB) is ischemic and hypoxic damage to BBB cells [1] most frequently caused by cerebral atherosclerosis [3,6]. Mast cells (MC) are structural elements of BBB [10] and regulate its permeability via secretion of bioactive substances (histamine, serotonin, heparin, catecholamines, and some proteolytic enzymes) and their absorption from tissues [8,14]. In cerebral vascular plexuses MC mediators seem to modulate not only blood vessels, but also choroidal epithelium [9,13], thus stimulating the production of the cerebrospinal fluid. Studies of the morphofunctional organization of MC will help to clear out the role of MC in vascular plexuses of human cerebral ventricles in health and cerebrovascular diseases. This determined the aim of our study.

MATERIALS AND METHODS

Vascular plexuses of the lateral, third and fourth ventricles were studied in 24 human corpses of both sexes (aged 49-60 years). All had pronounced (involvement of more than 50% intima) pathomorphological signs of atherosclerosis of precerebral arteries (PCA; internal carotid, main, vertebral) with stenosis of more than $^1/_2$ of the vascular lumen; the deaths were not directly caused by this condition. The control group consisted of age-matched subjects without apparent signs of cardiovascular diseases, dead from accidental causes. Compartments of vascular plexuses are named in accordance with previous classification [4].

The material was fixed in 10% formalin and embedded in paraffin, serial sections (10 μ) were stained with 1% toluidine blue aqueous solution. Mast cells were counted per 100 villi for each compartment or part of the vascular plexuses. In order to calculate the saturation index, all MC were divided into 4 groups, depending on the number of granules and degree of metachromasia [7]: very dark cells, dark cells, clear cells, and very clear cells; for calculation of the degranulation index, the cells were classified as cells with intensive, moderate, and weak degranulation.

RESULTS

Mast cells are scanty in human cerebral ventricular vascular plexuses and can be even absent in some serial sections. The cells are predominantly located near the sinusoidal capillaries and in the villous subepithelial zone. Normally MC have round-oval shape; non-degranulating or weakly degranulating

Department of Human Anatomy, Chelyabinsk State Medical Academy. ***Address for correspondence:*** a_andy@mail.ru. T. M. Babik

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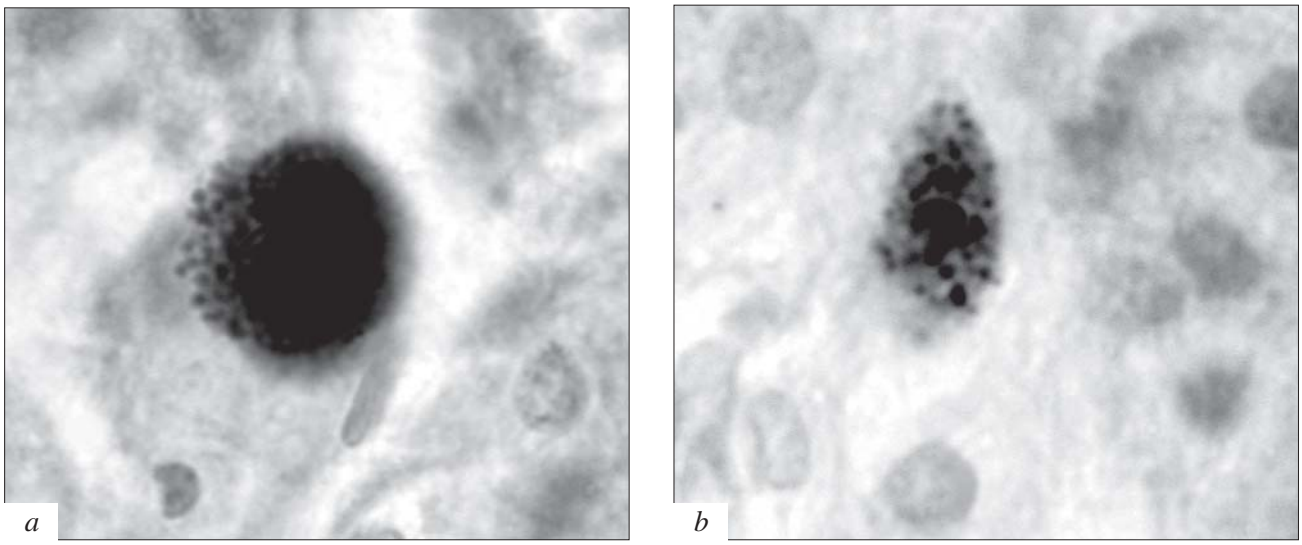

Fig. 1. Mast cells of the anterior compartment of the vascular plexus of human cerebral lateral ventricle. *a*) mast cell with high saturation and weak degranulation in the control group (man aged 55 years); *b*) non-degranulating mast cell with poor saturation in atherosclerosis of precerebral arteries (man aged 56 years). Toluidine blue staining, ×1000.

(degranulation index 0.83-1.38) cells with high saturation with granules of bioactive substances predominate (saturation index 2.13-3.67; theoretically possible maximum 4.0; Fig. 1, *a*). In PCA atherosclerosis the count of MC decreased, the cells acquire elongated spindle-like shape, the degranulation index decreased by 51.79% and saturation index by 26.67%, on average (Fig. 1, *b*).

In the control group the maximum counts of MC in vascular plexuses were found in the villi of the anterior compartment, glomus, posterior compartment, and posterior pole of the lateral ventricle vascular plexus (Table 1); the concentration of bioactive substances in the MC cytoplasm in these compartments was maximum (Fig. 2). Degranulating MC predominated in the villi of the vascular plexus in the fourth ventricle (degranulation indexes in the median and lateral parts 1.29 and 1.38, respectively; Fig. 3). Mast cells in the vascular plexus of the third ventricle and smooth part of the vascular plexus of the lateral ventricle were characterized by minimum saturation with granules and weak degranulation. In cerebral atherosclerosis the count of MC decreased significantly mainly in regions with their maximum content; the decrease in saturation index was also most pronounced here: by 26.5% in the anterior compartment, by 35.15% in the glomus, by 35.71% in the posterior compart-

TABLE 1. Changes in the Content of MC in Vascular Plexuses of Human Cerebral Ventricles in PCA Atherosclerosis (*M*±*m*)

Parameter	Control group (*n*=24)	PCA atherosclerosis (*n*=24)
Lateral ventricle vascular plexuses		
anterior pole	1.17±0.17	0.79±0.10
anterior compartment	2.00±0.17	1.25±0.13^{+}
glomus	1.83±0.13	1.33±0.14*
posterior compartment	2.33±0.14	1.67±0.14^{+}
posterior pole	1.67±0.14	1.08±0.08^{+}
smooth part	0.33±0.12	0.21±0.10
Third ventricle vascular plexuses	0.42±0.15	0.17±0.11
Fourth ventricle vascular plexuses		
median part	0.25±0.13	0.13±0.09
lateral part	0.75±0.12	0.33±0.12*

Note. *$p<0.05$, $^{+}p<0.01$ compared to the control group.

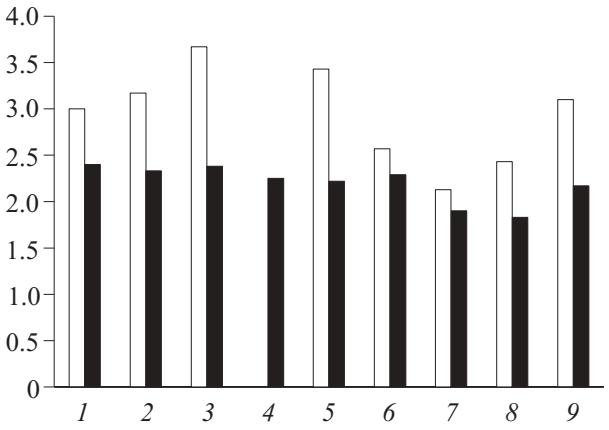

Fig. 2. Changes in mast cell saturation index in human cerebral ventricular vascular plexuses (VP) in atherosclerosis of precerebral arteries (PCA). Here and in Fig. 3: light bars: control group; dark bars: PCA atherosclerosis. *1*) anterior pole of lateral ventricle VP; *2*) anterior compartment of lateral ventricle VP; *3*) lateral ventricle VP glomus; *4*) posterior compartment of lateral ventricle VP; *5*) posterior pole of lateral ventricle VP; *6*) smooth part of lateral ventricle VP; *7*) third ventricle VP; *8*) median part of fourth ventricle VP; *9*) lateral part of fourth ventricle VP.

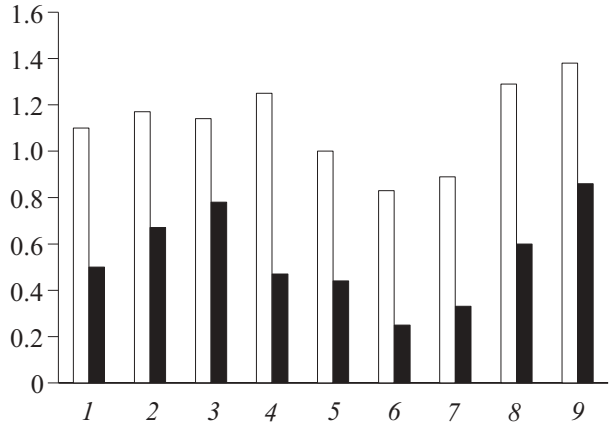

Fig. 3. Changes in mast cell degranulation index in human cerebral ventricular vascular plexuses in PCA atherosclerosis.

ment, and by 35.28% in the posterior pole. Mast cell degranulation index decreased more evenly in all compartments of vascular plexuses. Analysis of correlations revealed a strong correlation between MC count and their saturation with bioactive substances in the control group (r=0.868) and a decrease of correlation coefficient (r=0.632) in PCA atherosclerosis. The relationship between MC count and degranulation degree was weak in both groups (r=0.256 and r=0.224, respectively).

Low count of MC in human cerebral ventricular vascular plexuses in different age groups [5] with normally high saturation with bioactive substances and weak degranulation can indicate that the need in MC mediators emerges under conditions of increased functional load. In chronic cerebral hypoxia caused by PCA atherosclerosis the capillaries are reduced (their walls are thickened because of fibrosis and obliteration of the lumen) [2, 12], as a result of which MC count decreases. The synthesis of bioactive substances and degranulation are energy-consuming processes stimulating MC to oxygen consumption [11]. Under hypoxic conditions these processes are suppressed and hence, adaptation potential of the brain is impaired.

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