

THE FUNCTIONAL STATE OF THE CHOROID PLEXUS EPITHELIUM IN THE BRAINS OF HUMANS DYING OF TUBERCULOUS MENINGITIS

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(Received August 24, 1956. Presented by Active Member Acad. Med. Sci. USSR I. V. Davidov)

Most investigators, in spite of the multiplicity of contradictory opinions, believe that spinal fluid is formed by the choroid plexus of the brain [3, 10, 12, 13]. Information shedding light on the functional state of these plexi in the course of various diseases in general and tuberculous meningitis in particular is quite scanty, although the pathological anatomy of the vascular plexi has been described rather fully [2, 7, 11, 14, 15].

With the aid of the double salt injection method of A. N. Gennadiev [4, 5], we were able to demonstrate on dogs the importance of the intercellular substance of the plexi in the brain for the transfer of electrolytes between the blood plasma and the spinal fluid; the cellular elements of the epithelial cover of the plexi surviving the moment of the organism's death and not taking the stain [1].

On this basis, it seems evident that we became interested in investigating the functional state of the epithelium covering the choroid plexi lining the lateral ventricles of the brain in individuals dying of tuberculous meningitis.

EXPERIMENTAL METHODS

Injections were undertaken in corpses of 22 patients dead of tuberculous meningitis and 8 individuals brought shortly after sudden violent death (control group).

The lateral ventricle of the brain was entered with a needle and 10-20 cc of a mixture composed of potassium iron blue solution (3.76%) made isotonic with blood plasma was introduced. Perfusion of the ventricles was accomplished by careful removal of the calvarium (making certain not to damage the dura mater), or by making a trephine opening in the parietal bone. Before injecting the fluid, an equivalent or somewhat lesser quantity of spinal fluid was removed. Several minutes after the above operation, 50-60 cc of a mixture of More's solution (double sulphate salt of iron and ammonia — 3.53%) made isotonic with blood plasma was injected into the homonymous carotid artery. The order of the injections of the mixtures and the time intervals between them were varied. As a result of the chemical reaction between the salts that had been introduced, insoluble Turnbull blue would precipitate in the tissues.

Following the second injection, the brain was removed and studied histologically. The choroid plexi of the lateral ventricles were fixed in 10% neutral formalin and then studied as total transilluminated preparations and histological sections stained with hematoxylin eosin and hematoxylin-picrofuchsin. All experiments were done from 4 to 12 hours after death.

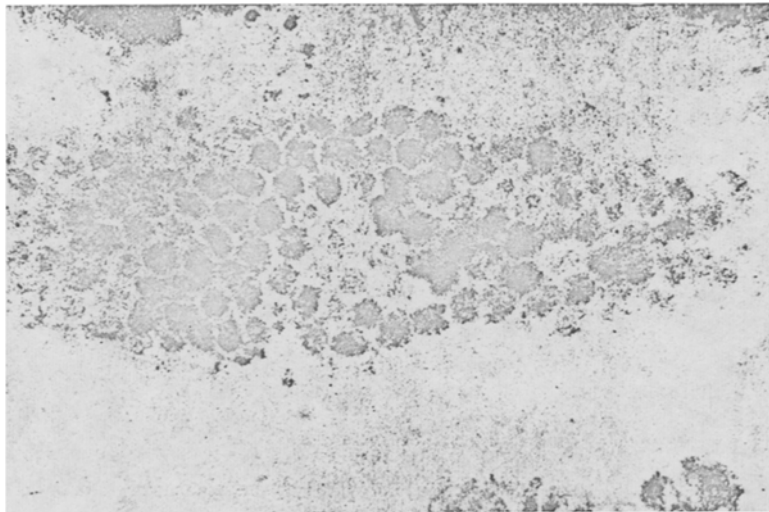
RESULTS

Microscopic studies of the villi of the vascular nets in the lateral ventricles taken from bodies of the control group within the first hours after death, in the total preparations, as well as in the histologic sections, showed the Turnbull blue to have precipitated on the surface of the cells and in the intercellular substance between the epithelium, in the connective tissue stroma, the endothelial lining of the capillaries as well as in the lumen of the vessels.

The results are analogous to those seen in the vessels of experimental animals under similar experimental conditions.

As the time interval after death was prolonged, there was seen a diffuse staining first of the surface and later of the protoplasm lying more deeply within the epithelial cells. As a rule, within 6 hours of death almost the entire epithelial plate would take the blue shading; the cellular elements taking more of the dye more evenly although some cells exhibited idiosyncracies, taking the dye more lightly.

Study of villi in patients dying of tuberculous meningitis and subjected to similar alternation of electrolytes, gave results differing in some particulars from those in the control group even when the time interval after death was the same for both.



Varying functional condition of the epithelial cells of the choroid plexus in the human brain during tuberculous meningitis. The following cells can be seen: those which do not take the Turnbull blue, those which have blue granules, and those which stain a diffuse blue. Intercellular spaces are widened. (Patient M., age 3 years, 4 hours after death; microphotograph, ocular 10x, objective 40x; hematoxylin-eosin).

In some instances, besides the usual picture of the villi in tuberculous meningitis, one could see increase in the size of the epithelial cells and of the spaces between them.

Turnbull blue was precipitated at various stages of the electrolyte transfer and lay in thick folds on the surface and between the epithelial cells. In one epithelial sheet there could be seen cells having a different response to the Turnbull blue. Some cellular elements proved to be intact: protoplasm stained evenly with eosin to a delicate rose, rounded nuclei, rich with chromatin. Among the unstained cells were cells having inclusions of blue granules within the protoplasm. Other cells held so much Turnbull blue as to stain the protoplasm blue, while some cells were colored diffusely (see Figure). As the time after death was lengthened, more cells were seen to take the stain diffusely.

D. N. Nasonov and V. Ya. Alexandrova [8, 9] have shown that intact cells will not take up the dye. Cells damaged by various agents and by death have increased absorptive capacities which permit the dye to penetrate. It follows, that the uncovering of intact cells in the epithelial cover of villi stricken by tuberculosis is proof of their vitality. Some cells are in a state of paranecrosis to which the blue granulation is proof. The epithelial cells which take a diffuse staining must have undergone far advanced alteration and death.

Tuberculous meningitis affects the epithelial cover of the vessel villi unevenly, as our observations show that the Turnbull blue is being absorbed variously, the cells being seen to be in different stages of vitality — this being reflected much more than is usually seen in the control studies.

The loosening of the epithelial cover, and the dystrophy reflected in the variable functional states of the cellular elements are probably the preconditions of the increased permeability for circulating substances carried in the

This state of the epithelial cover is reflected in the rise during tuberculous meningitis of the quantity of albumin present in the cerebrospinal fluid in the ventricles of the brain.

The material just presented serves as confirmation to I. V. Davidovsky who states that, "when other conditions are equal, the functional state of the organ and its tissues will determine its vulnerability" [6].

Apparently, one of the most important pathological physiologic properties of tissues is the variable functioning of the individual cells associated with the constant alternation between anabolic and catabolic processes; this determining very largely their variable susceptibility to being damaged. This last would be least in cells still immature.

SUMMARY

By studying the absorption of Turnbull blue in the corpses of 22 patients dead of tuberculous meningitis and 8 individuals brought shortly after sudden violent deaths and serving as controls, the author has shown that tuberculous meningitis destroys the cells of the epithelial cover of the choroid plexus quite unevenly: some cells necrotize and die while others are still quite viable. The metabolic variation of individual cells has an important influence in determining their susceptibility at the time of being subjected to toxic influences.

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