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New ultrastructural changes of the ependyma in experimental hydrocephalus*

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Summary. New ultrastructural alterations of the ependymal cells in the altered container model of experimental feline hydrocephalus are described. These include half desmosomes and a basal lamina on the apical surface of the ependymal cells, punctate adhesion-like structures between intraventricular mononuclear cells and the apical surface of the ependymal cells and unusual distortion of the ependymal cells. The significance of these previously unreported morphological alterations is unknown.

Key words: Ultrastructure – Ependyma – Experimental hydrocephalus

While studying the ependyma in kaolin-induced feline hydrocephalus, we encountered previously unreported findings, which are the subject of this communication.

Materials and methods

Progressive hydrocephalus was induced in anesthetized, adult cats using kaolin and the altered container model [3]. Animals were selected for study 1–6 weeks after inducing hydrocephalus. Transcardiac in vivo perfusion of the brains was performed using 4% paraformaldehyde and 5% glutaraldehyde in phosphate buffer (pH = 7.3–7.4). The fixed brains were rapidly removed, immersed in 5% glutaraldehyde and then rinsed with 50% sucrose. The brains were sectioned in the coronal plane and ventricular size assessed [8]. Samples were taken from the ependyma of the body and the temporal horn of the lateral ventricle. These specimens were post fixed in buffered osmium tetroxide for 2 h, stained *en bloc* with a saturated aqueous solution of uranyl acetate, dehydrated in alcohol and embedded in Epon. Ultrathin sections were cut for electron microscopic observation and stained with uranyl acetate and lead citrate.

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Results

These studies revealed the stereotyped changes of the ependyma usually associated with hydrocephalus [7, 9] and other unexpected ultrastructural changes described below.

In specimens of ependyma from the temporal horn, stretching of the cytoplasm of the ependyma was associated with unusual distortion of the three-dimensional orientation of these cells (Fig. 1). Thinner, layered, sheet-like projections of the elongated cytoplasm covered adjacent ependymal cells. The perinuclear cytoplasm was narrowed and the remaining cytoplasm appeared to extend as a process over the lateral aspect of the adjacent ependymal cell and traverse its apical surface. These elongated processes contained many fibrillary structures. This elongation and deformation of the cytoplasm distorted the normal orientation of interependymal junctions.

Half desmosomes were found on the apical surface of some flattened ependymal cells. These structures varied in length and did not establish contact with adjacent cells or basal lamina. A definite basal lamina was found on the apical surface of some ependymal cells. The electron density of this basal lamina was patchy compared to normal basal lamina and was often discontinuous (Fig. 2). Half desmosomes were sometimes found adjacent to the basal lamina. Caviola were occasionally found beneath the basal lamina.

Macrophages or mononuclear cells were occasionally found within the ventricular system, often in close contact with the ependymal surface (Fig. 3). Sometimes, punctate adhesion structures joined these mononuclear cells to the ependymal cells. These adhesions between cells were incompletely formed and

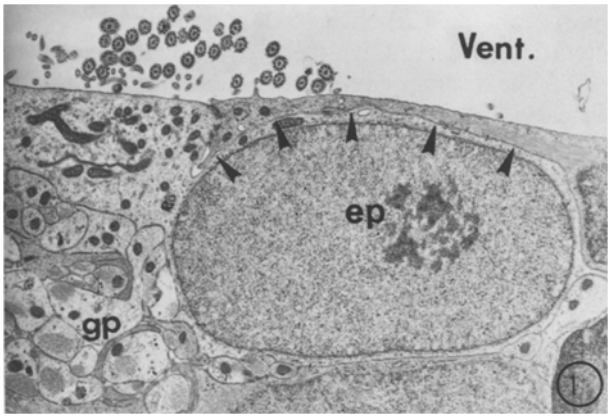

Fig. 1. Photomicrograph showing the distortion of the ependymal cells (*ep*) and the narrowing of the perinuclear cytoplasm. The process of this ependymal cell extends laterally to traverse the surface of neighboring ependymal cells (*arrows*). The glial cell processes (*gp*) below the ependymal cells have a circular appearance and bundles of glial fibrils. No obvious desmosomes are seen between the ependymal and glial cell processes. *Vent:* Ventrice; ×6,900

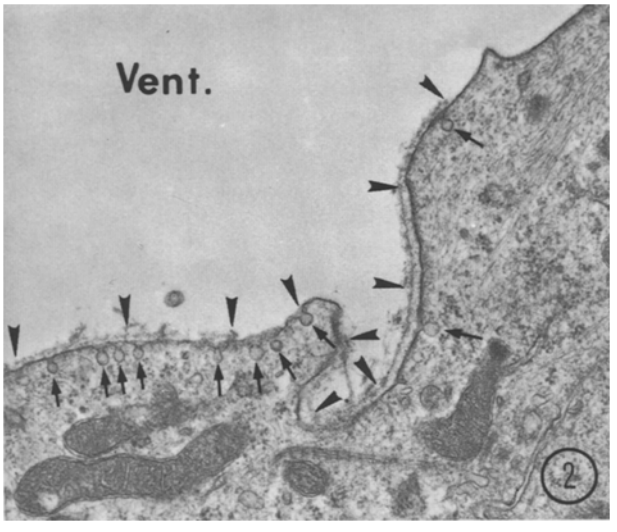

Fig. 2. Photomicrograph showing the basal lamina on the apical surface of the ependymal cells (*arrowheads*). The electron density of these structures are patchy and discontinuous. Caviola can be seen beneath the basal lamina in some areas (*arrows*). *Vent:* Ventricle; ×24,400

were usually thicker on the side of the mononuclear cells. Processes of the mononuclear cells sometimes overlapped the microvilli protruding from the surface of the ependymal cells. Half desmosome-like electron densities were found in the process of the mononuclear cells adjacent to the microvilli (Fig. 4).

Discussion

Unexpected findings in this study were the presence of the half desmosome and the basal lamina on the

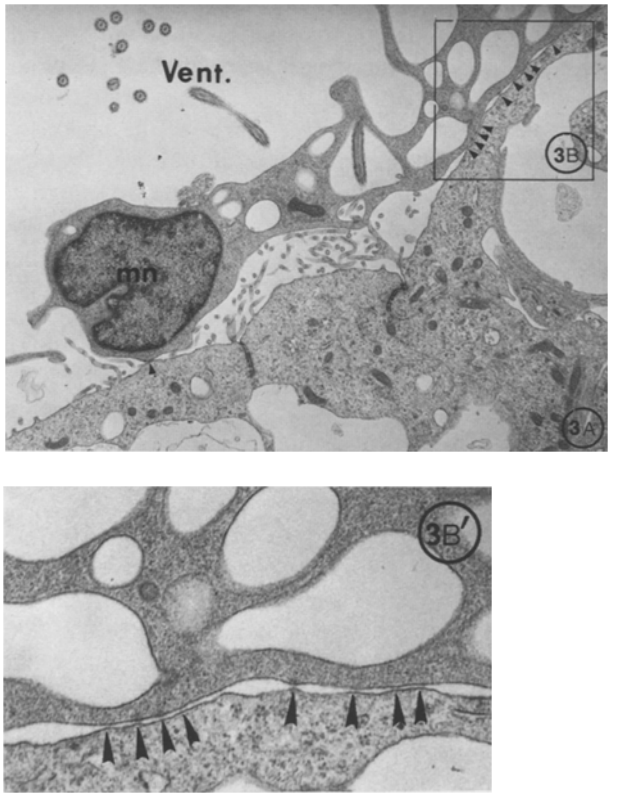

Fig. 3A, B and B′. Photomicrograph showing the close contact between an intraventricular mononuclear cell (*mn*) and the ependymal apical surface. Punctate adhesions can be seen between these two cells (*arrowhead*). *Vent:* Ventricle; **A, B** ×4,400; **B′** ×12,300

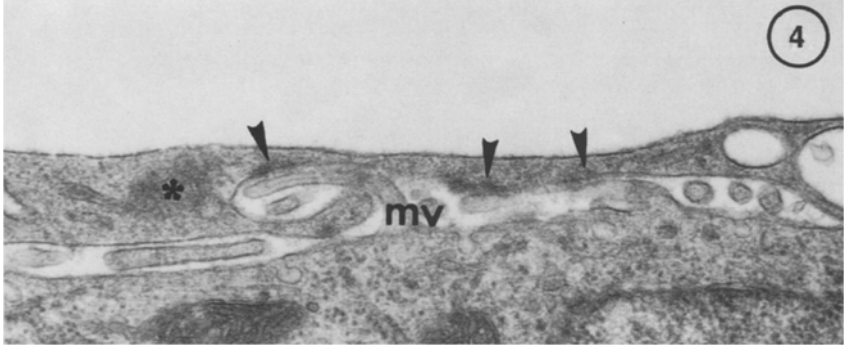

Fig. 4. The process of this mono nuclear cell overlaps the microvilli (*mv*) on the ependymal surface. Half desmosome-like electron density is seen in the mononuclear processes overlying some microvilli (*arrowhead*). *Vent:* Ventricle, ×32,400

apical surface of ependymal cells as previously reported from this laboratory [6]. Half desmosomes are usually found at the intercellular junctions of the ependymal cells. The basal lamina is usually found in regions near the perivascular space of the brain and lamina terminalis of the spinal cord [2]. Both the half desmosome and the basal lamina can be found on the pial surface of normal brain [1].

One possible reason for the appearance of the basal lamina on the ependymal surface is the biological influence created by the presence of the mesenchymal cells. Kusaka et al [4] showed that glial cells, such as subpial astrocytes, can form a basal lamina in the presence of the mesenchymal cells in tissue culture preparations. Although separation of the ependymal cells by the enlargement of the ventricles could distort the architecture sufficiently to displace the separated desmosomes to the apical surface of the ependyma, we found neither separation of the apical interependymal junctions, nor the half desmosomes on this surface. Therefore, we believe those structures were not simply created by the anatomic distortion of the ependymal and might be a biological response to the hydrocephalic process.

The elongated, unipolar processes of the ependymal cell were another unusual finding often observed in this model of feline hydrocephalus. Vacuolated distension of the extracellular space between the lateral aspects of adjacent ependymal cells has been noted by others [5, 9], but not in association with the thinner-layered sheet-like processes of the ependyma described above. Since these abnormalities have only been identified in this study, which used the altered container model of feline hydrocephalus, it is unclear whether these abnormalities are restricted to this model.

This report documents several new ultrastructural changes in the morphology of the ependyma covering the periventricular white matter in experimental feline hydrocephalus. Although some of the morphological alterations observed by others in this region were also found, the findings enumerated above appear unique. We cannot determine if these changes are caused by the progressive and extreme degree of ventricular enlargement associated with this model of hydrocephalus or influence of mesenchymal cells. The significance of these morphological alterations and their reversibility cannot be determined at present.

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