Subependymoma of the Lateral Ventricle
—Case Report—

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Abstract

A 56-year-old male presented with mild gait disturbance and short-term memory disturbance. Computed tomographic scans revealed an isodense mass with a large cyst in the left lateral ventricle, extending to the right. The tumor was removed totally via the left frontal transcortical approach. Light microscope examination found clusters of isomorphic cells separated by a dense fibrillar matrix. No ependymal rosettes or blepharoplasts were found. Some cluster cells had positive immunoperoxidase staining for glial fibrillary acidic protein and S-100 protein. Electron microscope observation found tumor cells with gap junctions and zonula adherens resembling the junctional complexes of normal ependymal cells, many microvilli and cilia, and long processes containing abundant glial fibrils. Such “transitional cells” may be important in establishing the origin of subependymoma.

Key words: subependymoma, electron microscopy, transitional cells

Introduction

Subependymoma is a rare, slow-growing noninvasive tumor of the central nervous system. The usual site is the fourth ventricle, and occasionally the lateral ventricles. Most reported subependymomas are small, asymptomatic tumors found incidentally at autopsy, often in young males. Tumor cytology includes astrocytes, ependymal cells, and mixed cells. However, the cytogenesis has yet to be established. We report an unusual case of subependymoma of the lateral ventricle, associated with colonic carcinoma. The ultrastructure and characterization of this tumor are discussed.

Case Report

A 56-year-old male presented with a 1-year history of mild gait disturbance. Four months prior to admission, he had developed ileus and received colectomy for adenocarcinoma of the colon. Three months prior to admission, his short-term memory had started to deteriorate. He had developed occasional headaches and progressive difficulty in walking with frequent falls.

Neurological examination on admission revealed recent memory disturbance, gait disturbance without muscle weakness, and bilateral papilledema. Precontrast computed tomographic scans demonstrated enlarged lateral ventricles and an isodense mass associated with a large cyst in the left lateral ventricle, extending to the right. The mass was homogeneously enhanced on the postcontrast scans. Magnetic resonance (MR) images showed a slightly low-intensity solid mass on the T1-weighted SE image and a high-intensity solid mass with an iso-intense cystic area on the proton-density image (Fig. 1). Cerebral angiograms demonstrated an avascular mass.

The intraventricular tumor was exposed via the left frontal transcortical approach. It resembled an extracerebral tumor except for the attachment to the medial wall near Monro's foramen. The tumor was totally removed. Postoperative course was uneventful. One year after surgery, he continues to do well.

Tumor tissue obtained at surgery was fixed in 6% formalin, embedded in paraffin, and stained with HE. Light microscopy showed the tumor cells characterized by clusters of isomorphic cells in a dense fibrillar matrix. The nuclei were oval and regular, and the cytoplasm was poorly defined. Focal
microcavitation was often present adjacent to cell clusters. No ependymal rosettes or mitotic figures were found (Fig. 2). Phosphotungstic acid-hematoxylin staining demonstrated no blepharoplasts in the cytoplasm. Some cluster cells demonstrated positive immunoperoxidase staining for glial fibrillary acidic protein, especially in the perikarya, and for S-100 protein.

For electron microscopy, tissue was immediately fixed in 2% glutaraldehyde and phosphate buffer (pH 7.4) and stood overnight. Specimens were postfixed in osmium tetroxide for 60 minutes, dehydrated by an ethanol and propylene oxide series, and embedded in Epon 812. Sections were cut with a diamond knife, stained with uranyl acetate and lead citrate, and examined with a H-7000 electron microscope (Hitachi, Katsuta, Ibaraki). Tumor cells were mostly clustered in the fibrillar matrix. The cytoplasm was irregular with gap junctions and zonula adherens resembling the junctional complexes in normal ependymal cells. Microvilli projected into the intracellular luminal formations. Cilia were abundant (Fig. 3A). Some tumor cells demonstrated irregular nuclei containing condensed chromosomes. Many cell processes with abundant glial fibrils and scattered free ribosomes were present near tumor cells. Rosenthal fibers were found in some intermingled

**Fig. 1** upper: Axial proton-density MR images (SE, 2000/40 msec), revealing a high-intensity solid mass with an isointense cystic area. lower: Coronal and sagittal T2-weighted MR images (SE, 500/30 msec), clearly demonstrating the solid mass in the left lateral ventricle.

**Fig. 2** Photomicrographs of the tumor. A: The fibrillar matrix and clusters of isomorphic cells are seen. HE stain, ×160. B: At high magnification, cell nuclei are oval and regular with scarce cytoplasm. Some compact cells are present (arrows). HE stain, ×320.

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glial filaments (Fig. 3B). A few tumor cells contained both astrocyte and ependymal cell features (Fig. 3C).

**Discussion**

Subependymoma is a rare tumor of uncertain cytogenesis, first reported in 1945. Several hypotheses about the origin have been proposed. Subependymal cells migrate and differentiate into neuronal and glial components, and are therefore multipotential. Scheinker considered that subependymoma developed from subependymal astrocytes, and should be classified as astrocytoma. Duffell et al. supported this theory by finding no ultrastructural differences between ordinary astrocytoma and subependymoma. Azzarelli et al. described “transitional tumor cells” with features morphologically intermediate between ependymal cells and astrocytes. Friede et al. called such cells “ependymoglia.”

However, the clinical features of subependymoma are not consistent with a mixed ependymoma and astrocytoma. We therefore think the ultrastructure of these cells is important. In this case, we observed “transitional cells” with gap junctions and zonula adherens resembling the junctional complexes of normal ependymal cells, many microvilli and cilia projecting into the lumen, and long processes containing abundant glial fibrils.

A unique feature of this case was the association with colonic carcinoma. Subependymoma has been associated with astrocytoma, choroid plexus papilloma, craniohypophygioma, and hemangioblastoma. Almost all associated intracranial tumors were adjacent to the subependymal cell plate. This association with an extracranial tumor was unusual, and probably incidental. The high incidence of subependymoma associated with other tumors is probably because of long survival times.

Most subependymomas are asymptomatic, but may grow sufficiently to cause symptoms. Scheithauer reported that 43 of 95 subependymoma cases had nonspecific clinical manifestations, either from hydrocephalus or compression of adjacent structures. In this case, the tumor was large enough to compress frontal lobe and fornix, and cause disturbance of gait and recent memory. The symptoms depend on the size and location of the subependymoma. Asymptomatic brain tumors, such
as colloid cyst of the third ventricle, are well known to cause unexpected death by acute obstruction of cerebrospinal fluid drainage, resulting in increased intracranial pressure.\textsuperscript{4,7,9}

We found insufficient specific features to distinguish subependymoma from ependymoma or astrocytoma preoperatively, although it should have distinct biological behavior and morphological features. Subependymoma should be suspected in patients presenting with an intraventricular mass.

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References


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