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Third Ventricular Chordoid Glioma: A Distinct Clinicopathologic Entity

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Abstract. We have encountered a series of 8 third ventricular neoplasms with a distinctive chordoid appearance that appear to represent a clinicopathologic entity. The tumors occurred in 7 females and 1 male, ranging in age from 31 to 70 years. In all cases, imaging studies showed a large well-circumscribed third ventricular mass; a cystic component was noted in 2. The tumors consisted of cords and clusters of cohesive, oval-to-polygonal epithelioid cells with abundant eosinophilic cytoplasm, relatively uniform round-to-oval nuclei, and inconspicuous nucleoli. Mitotic activity was absent. The stroma consisted of scant, coarse fibrillar processes, as well as prominent, slightly basophilic, extracellular mucin resembling that in chordomas. Throughout the tumor, and surrounding its well-defined borders, were infiltrates of mature lymphocytes and plasma cells. Russell bodies were prominent in the latter. Adjacent brain tissue showed reactive changes with gliosis and numerous Rosenthal fibers.

Immunohistochemically, tumor cells were strongly reactive for GFAP and vimentin, but negative or only weakly staining for EMA. The MIB-1 labeling index was approximately 1%. Ultrastructural examination of 4 cases revealed focal microvilli, scattered "intermediate" junctions, and focal basal lamina formation. Neither desmosomes nor cilia were seen. Total resections were achieved in 2 cases; only subtotal removals were achieved in 6. Subsequent tumor enlargement was noted in 3 of the 6 patients with incomplete resection, and of these, two died at post-operative intervals of 8 months and 3 years. The other patient survives 4 years post-operatively with stable residual disease. Of the 2 patients with total resection, 1 was lost to follow-up; the other, during a brief follow-up period, did well without evidence of recurrence.

Key Words: Brain neoplasms; Glioma; Suprasellar region; Third ventricle.

INTRODUCTION

Primary brain tumors occupying the third ventricle in adults form a heterogeneous group of uncommon lesions (1–6). Most often, they arise from the linings of the ventricular walls, or extend into the ventricle from their source in the pineal, suprasellar, or hypothalamic regions. Frequently encountered are colloid cysts, choroid plexus tumors, ependymoma, pilocytic astrocytomas, germinoma, and pineal parenchymal tumors. We have encountered a series of 8 neoplasms involving the third ventricle of adults. The lesions exhibited unique radiographic, histologic, immunohistochemical, and ultrastructural features. All appeared to arise in the hypothalamic/suprasellar/third ventricular region. Histologically composed of clusters of epithelioid cells, slightly basophilic and vacuolated extracellular mucin, as well as a lymphoplasmacytic infiltrate, the tumors had a low grade, "chordoid" appearance resembling that of chordoma and chordoid meningioma. Although immunohistochemical and ultrastructural studies indicate that these unique tumors are glial in nature, they find no place in current histopathologic classification schemes (1, 2).

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MATERIALS AND METHODS

All 8 cases reported herein were received in consultative services at Johns Hopkins Hospital or Mayo Clinic between the years 1988 and 1997. Consisting of 7 surgical and 1 autopsy specimens, they were formalin fixed and routinely processed. In addition to hematoxylin and eosin (H&E) stained slides, stains for mucins including mucicarmine and periodic acid Schiff (PAS) with and without diastase digestion were selectively applied. Mitoses were counted by examining 10 high power fields (HPF) 3 times for each tumor, and determining an average number per 10 HPF. For immunohistochemical studies, paraffin-embedded specimens were sectioned at 4 microns, deparaffinized, and subjected to antigen retrieval by either limited protein digestion or steaming (20 minutes at 80°C). Slides were then incubated at room temperature with antibodies directed towards glial fibrillary acidic protein (GFAP) (polyclonal, 1:6,000; Dako Co., Carpenteria, CA), epithelial membrane antigen (EMA) (monoclonal, 1:2,000; Dako Co.), S-100 protein (polyclonal, 1:6,000; Dako Co.), vimentin (monoclonal, 1:50; ICN ImmunoBiologicals, Lisle, IL), cytokeratin (AE1/AE3, 1:2,000; Boehringer Mannheim, FRG), and MIB-1 (monoclonal, 1:150; Immunotech, Inc., Westbrook, ME). Antibodies were detected using the avidin-biotin complex (ABC) method, diaminobenzadine serving as the chromogen. For quantitating the percentage of nuclei staining with MIB-1 antibody to the nuclear antigen Ki67, at least 500 tumor cells were manually counted 3 times in each case, and an average percentage of positively staining nuclei was calculated. Tissues permanently fixed and routinely processed for electron microscopy were available in 4 cases. Data regarding clinical presentation, radiological and operative findings, adjuvant therapy, and follow-up (tumor recurrence, present clinical status) were obtained when possible.

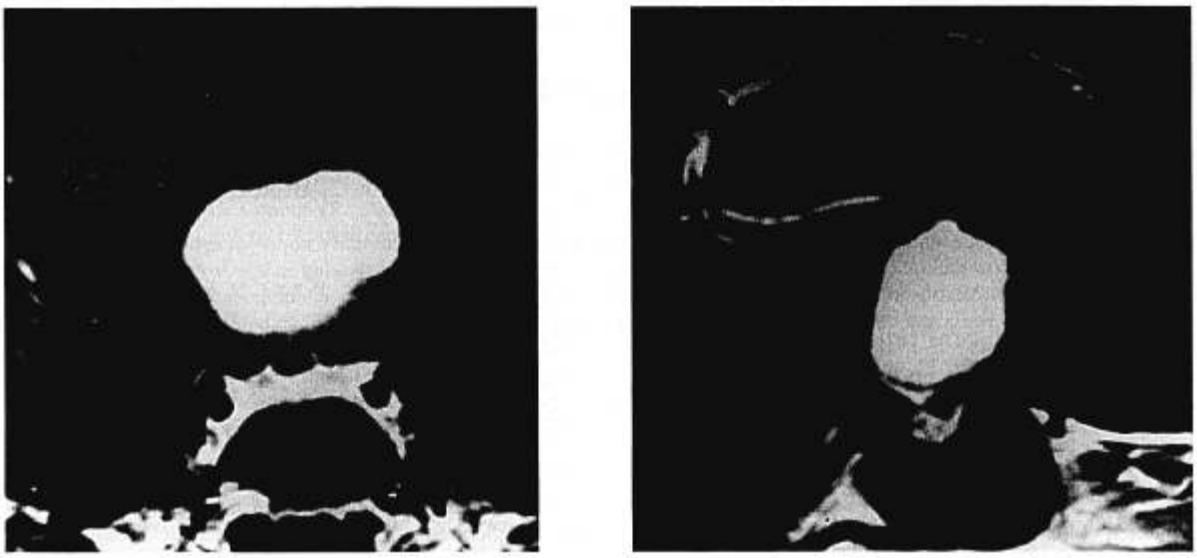

Fig. 1. Sagittal (left) and coronal (right) magnetic resonance images of a third ventricular "chordoid glioma" (Case 5). The tumor has a large, solid component within the third ventricle that is uniformly gadolinium enhancing, and is contiguous with the suprasellar/hypothalamic region. A cavum septum pellucidum is also present.

CASE REPORTS

Case 1

A 50-year-old female developed neurological symptoms including increased lethargy, urinary incontinence, and ataxia. A computed tomographic (CT) scan revealed a third ventricular mass and hydrocephalus. At surgery, the well-circumscribed lesion filled the third ventricle, but the precise site of tumor attachment to brain structures was not apparent. The mass was totally resected.

Case 2

A 70-year-old male developed progressive ataxia over a period of 2 years. Obstructive hydrocephalus was apparent on magnetic resonance imaging (MRI), which also showed a suprasellar mass with a solid, homogeneously enhancing component involving the third ventricle, as well as a multicystic component that extended laterally into the right temporal lobe and inferiorly into the sella turcica. The lesion was subtotally resected. No enlargement of the residual tumor was noted after 1 year.

Case 3

A 59-year-old female developed symptoms of obstructive hydrocephalus and was found on imaging studies to have a large, solid and cystic tumor within the third ventricle. The lesion appeared to arise in the suprasellar region. The tumor was subtotally resected. Despite postoperative radiation therapy, the patient died of a recurrent tumor 3 years thereafter. At autopsy, the recurrence occupied the entire third ventricle.

Case 4

A 47-year-old female developed symptoms of obstructive hydrocephalus. An MRI scan revealed a third ventricular, egg-shaped mass arising in the suprasellar region. At craniotomy, the mass was subtotally resected. Five months later, imaging studies demonstrated slight growth of a residual tumor within the sella turcica. The patient developed multiple medical complications and died 8 months after surgery of causes not directly related to the neoplasm.

Case 5

Over a period of 6 months, a 31-year-old female developed headaches, nausea and vomiting and experienced short periods of unconsciousness. An MRI scan revealed a homogeneously enhancing tumor in the third ventricle that appeared to arise in the hypothalamic/suprasellar region (Fig. 1). The lesion was completely excised. No recurrence was noted after 6 months.

Case 6

A 56-year-old female developed hypothyroidism as well as diabetes insipidus. Imaging studies revealed a mass arising in the suprasellar region and occupying the third ventricle. The tumor was subtotally resected. No MRI evidence of recurrence was noted after one year. Postoperative complications included memory deficits and hypothalamic dysfunction.

Case 7

Hypothyroidism was detected in a 31-year-old female complaining of gastrointestinal symptoms and weight

TABLE 1
Clinical Characteristics of Third Ventricular "Chordoid Gliomas"

Age and sex	Symptoms	Imaging findings	Treatment	Outcome
50, F	Lethargy, ataxia, urinary incontinence	Well-circumscribed mass in 3rd ventricle; obstructive hydrocephalus	Total resection	NA
70, M	Ataxia	Solid and cystic 3rd ventricular mass, uniformly enhancing	Subtotal resection	No recurrence, 1 year
59, F	Obstructive hydrocephalus	Solid and cystic 3rd ventricular mass arising in suprasellar region	Subtotal resection; radiation	Recurrence, d.o.d., 3 years
47, F	Obstructive hydrocephalus	Solid, 3rd ventricular mass arising in suprasellar region	Subtotal resection	Residual tumor, 5 months died of post-op complications, 8 months slight tumor growth
31, F	Headache, nausea, vomiting	Solid, uniformly enhancing 3rd ventricular mass arising in suprasellar region	Total resection	Diabetes insipidis; no recurrence, 6 months
56, F	Hypothyroidism, diabetes insipidus	Solid, 3rd ventricular mass arising in suprasellar region	Subtotal resection	No recurrence, 1 year
31, F	Hypothyroidism, weight loss	Enhancing 3rd ventricular mass arising in suprasellar-hypothalamic region	Subtotal resection; radiation	Recurrent tumor, 4 years; slow tumor growth
35, F	Amenorrhea, psychotic disorder	Solid, enhancing 3rd ventricular mass compressing hypothalamic structures	Subtotal resection	Post-operative death

Abbreviations: NA, not available; d.o.d., dead of disease.

loss. On the CT scan a 2.5 cm contrast-enhancing suprasellar mass extending into the third ventricle could be seen. The lesion was subtotally resected and the patient underwent post-operative radiation therapy. Four years later, she was clinically stable and asymptomatic except for a persistent visual field defect. Sequential imaging studies performed during this period revealed that the residual tumor had grown slightly to 2.8 cm, but that it had not enlarged during the last year.

Case 8

A 35-year-old female with a one-year history of amenorrhea developed a psychotic disorder and was treated for schizophrenia for a period of 6 months. Due to progressive symptoms, an MRI was performed, showing a large, solid, contrast-enhancing mass that filled the third ventricle and compressed underlying hypothalamic structures. The mass was subtotally resected and the patient showed initial recovery, but died in the post-operative period due to pulmonary embolus.

RESULTS

This series included 7 females and 1 male ranging in age from 31 to 70 years (mean, 47 years). Clinical information is listed in Table 1. In all cases, imaging studies showed a large, 2.5–3.5 cm, well-circumscribed third ventricular mass (Fig. 1). A cystic component was noted in 2 cases. Short of these cystic elements, tumors were uniformly contrast-enhancing, and appeared contiguous with hypothalamic or suprasellar structures. Given their size, however, the precise site of origin of the lesions could not be determined. A gross total resection was achieved in 2 of 8 cases; in one instance, follow-up revealed no recurrence at 6 months. The second patient was lost to follow-up. In the 6 other instances, only a subtotal resection was possible. Three of these incompletely resected tumors were noted to enlarge. Two of these patients died, one directly due to tumor recurrence after 3 years, and one at 8 months due to multiple postoperative complications. In the third patient, the residual mass grew slowly over a 4-year period following its subtotal resection. Of the other 3 patients with subtotal resections, one patient died in the post-operative period due to pulmonary embolus; no tumor recurrence has been noted in the other two, but their follow-up periods have been relatively short. Two patients (Cases 3 and 7) received adjuvant radiation therapy following subtotal tumor resection. In both cases, residual tumor enlarged during the years following radiation. One patient remained asymptomtatic after 4 years and the other died of tumor regrowth after 3 years. No patients underwent chemotherapy.

Histopathologic, immunohistochemical, and ultrastructural features of the tumors are summarized in Table 2.

TABLE 2
Histopathologic, Immunohistochemical, and Ultrastructural Features of Third Ventricular "Chordoid Gliomas"

Histopathologic features	Number of cases
Epithelioid cell clusters	8/8
Epithelial cords	8/8
Mucinous background	8/8
Lymphoplasmacytic infiltrate	7/8
Russell bodies	7/8
Rosenthal fibers in adjacent brain	6/6
Infiltrating component	0/8
0-1 mitoses/10 HPF	8/8
Nuclear pleomorphism	0/8
Immunohistochemistry	**Number of cases staining**
GFAP, strong diffuse	8/8
EMA, strong diffuse	0/8
EMA, focal weak	4/8
Cytokeratin	3/4
S-100 protein	4/8
Vimentin	8/8
Ultrastructural features	**Number of cases**
Cytoplasmic intermediate filaments	4/4
Abundant mitochondria	4/4
"Intermediate" junctions	4/4
Desmosomes	0/4
Basal lamina	3/4
Intercellular microvilli	4/4
Cilia	0/4
Interdigitating cell membranes	0/4

Abbreviations: HPF, high power field; GFAP, glial fibrillary acidic protein; EMA, epithelial membrane antigen.

All neoplasms consisted of clusters and cords of oval-to-polygonal epithelioid cells with abundant eosinophilic cytoplasm. Their stroma consisted of an abundant, slightly basophilic and vacuolated mucinous matrix. In selected tumors in which stains were applied, mucin demonstrated PAS staining with and without diastase digestion, and modest mucicarmine staining. Other regions of tumor stroma consisted of coarsely fibrillar processes, and a prominent lymphoplasmacytic infiltrate (Fig. 2). Russell bodies were a constant feature. No histological features of meningioma, e.g. whorls, psammoma bodies, or nuclear pseudoinclusions, were identified. Neither perivascular orientation of tumor cells nor pseudorosettes suggestive of ependymoma were seen. Tumor cell nuclei were moderate in size, round-to-oval, and relatively uniform. No anaplastic features were identified in any lesion. Mitotic figures were rare (<1 per 10 HPF) or entirely absent. The neoplasms appeared architecturally solid microscopically, and showed no appreciable infiltration of surrounding brain parenchyma. Rather, adjacent brain tissue featured reactive astrocytes, numerous Rosenthal fibers, and chronic inflammatory cells. Immunohistochemically, all tumors showed strong, diffuse reactivity for GFAP and vimentin. Four tumors showed no immunoreactivity for EMA, while 4 others featured focal weak reactivity. Immunoreactivity of tumor cells for S-100 protein was noted at least focally in 4 of the 8 tumors, whereas moderate diffuse reactivity for cytokeratin was present in 3 of 4 tumors tested. The MIB-1 labeling index was uniformly low in all cases, ranging from 0 to 1.5%. Ultrastructural examination of 4 cases revealed round to spindled cells with short cellular processes containing abundant cytoplasmic intermediate filaments and mitochondria (Fig. 3). Scattered "intermediate" junctions and focal intercellular projections resembling microvilli were identified in each of 4 cases examined. Focal basal lamina formation was evident in 3 instances. Neither cilia, well-formed desmosomes, nor interdigitation of cell membranes was seen in any case.

DISCUSSION

The morphological features of these third ventricular neoplasms are unlike those of previously classified central nervous system (CNS) lesions, but remarkably uniform within this series of cases. Their solid, well circumscribed nature was evident both by neuroimaging procedures and by microscopy, which demonstrated a sharply defined border between tumor and brain parenchyma. A lymphoplasmacytic infiltrate was often present at the tumor-brain interface. No significant infiltration of adjacent brain by tumor was noted, nor was there evidence of normal CNS parenchyma within the masses. Instead, surrounding brain tissue showed only longstanding reactive changes, including reactive astrocytosis and numerous Rosenthal fibers. The cytological features were

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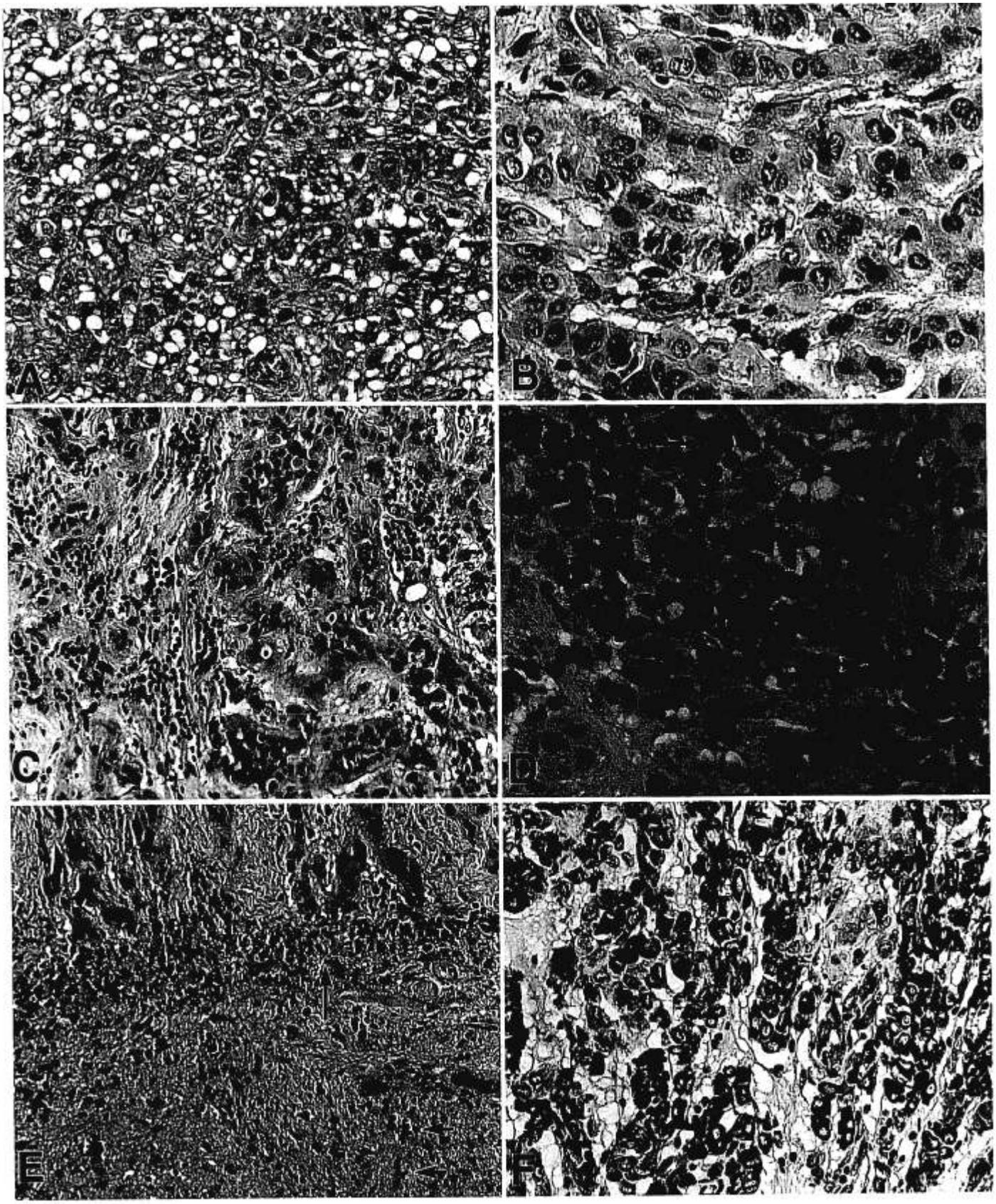
Fig. 2. Histopathological features of third ventricular "chordoid gliomas." Tumors consisted of cords and clusters of cohesive cells in a background of scant, coarse fibrillar processes, and a prominent, slightly basophilic and vacuolated extracellular mucinous material resembling that of chordomas (A). In some regions, linear arrays of tumor cells with abundant eosinophilic cytoplasm were noted. Nuclei were uniform round-to-oval and contained inconspicuous nucleoli (B). Throughout the tumor were infiltrates of mature lymphocytes and plasma cells (C). In regions containing lymphoplasmacytic clusters, Russell bodies (arrows) were a consistent finding (D). All tumors had sharply defined borders (long arrows) with adjacent brain tissue, which showed reactive changes including gliosis and numerous Rosenthal fibers (E, short arrows). Tumor cells were strongly immunoreactive for GFAP (F).



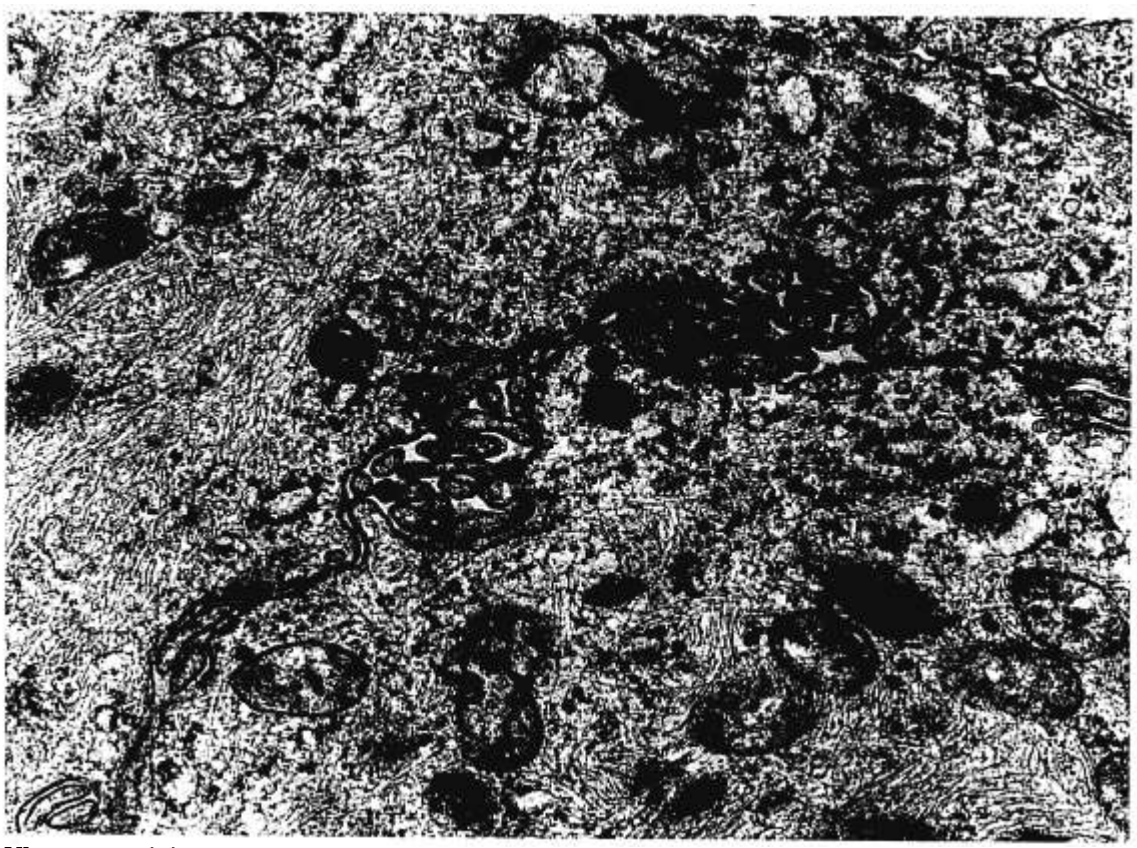


Fig. 3. Ultrastructural features of third ventricular "chordoid gliomas." Typically, the cytoplasm contained abundant intermediate filaments and numerous interspersed mitochodria (lower right). Focal microvilli were identified in each of the cases, but cilia were not seen.

those of a low-grade neoplasm. Neoplastic cells possessed abundant eosinophilic cytoplasm (a low nuclear to cytoplasmic ratio), the nuclei being round-to-oval without significant pleomorphism or atypia. Mitoses were either exceedingly rare or absent. The MIB-1 proliferation index was extremely low.

In the sense that these tumors are solid, histologically low grade, and lack a significant infiltrating component, they bear a similarity to pilocytic astrocytomas or ependymomas. Unlike the former, however, they lacked piloid cells, microcystic formation, and intratumoral Rosenthal fibers or granular bodies. Similarly, was there was no perivascular orientation of tumor cells to suggest ependymoma. Instead, the neoplastic cells were epithelioid in appearance and arranged in clusters that suggested a meningothelial, choroid plexus or notochordal derivation. Although features of classic meningioma such as cellular whorls, psammoma bodies, and nuclear vacuoles were lacking, similarities to the so-called "chordoid" meningiomas were noted, specifically clustering of epithelioid cells and the presence of a lymphoplasmacytic infiltrate (7). However, the "chordoid glioma" described herein differs in many respects from the lymphoplasmacyte-rich lesion described by Kepes et al. Chordoid glioma occurred in adults and arose in the suprasellar/hypothalamic region, whereas Kepes's chordoid meningiomas occurred in adolescents (ages 8–19 years), had an association with systemic Castleman's syndrome, and were dura-based, involving the tentorium or cerebral convexities. In contrast, the chordoid gliomas in the present study were not dura-based and showed no association with Castleman's syndrome or any other hematopoietic disorder. Consistent with the association of chordoid meningiomas with Castleman's syndrome is the fact that the lymphoplasmacytic component is far more prominent than that in chordoid gliomas, where stromal lymphocytes and plasma cells, although readily apparent, did not represent a significant portion of the tumor mass. Lastly, reactive lymphoid follicles with germinal centers, a feature typical of chordoid meningiomas, were not encountered in the present series.

The distinction of chordoid glioma from choroid plexus tumors poses no real problem (1, 2). In low-grade form, the latter are papillary lesions, uniformly S-100 protein immunoreactive, and only focally GFAP-positive at best. Ultrastructurally, they are distinctly epithelial, featuring microvilli, cilia and complex, well-formed junctions.

Chordoma-like lesions termed "giant ecchordosis physaliphora" rarely form symptomatic, intradural masses with compression of overlying brain parenchyma (8). Such tumors feature physaliphorous cells, lack an inflammatory component, are strongly cytokeratin and EMA immunoreactive, and lack GFAP staining.

Recently, Wanschitz et al described a solid, third ventricular tumor in a 24-year-old woman that had similar, if not identical, histologic and immunohistochemical features as those described in the present study (9). In their case report, the authors concluded that the tumor represented a meningioma with a "peculiar expression of GFAP." Indeed, meningiomas are known to arise in the suprasellar region and to occasionally occupy the third ventricle (10–13), and aberrant immunoreactivity for GFAP has been reported in nonglial neoplasms (14, 15). However, several lines of evidence suggest that the neoplastic cells in our third ventricular chordoid tumors are glial. Although the majority of its cells were epithelioid in appearance, some exhibited a coarse cytoplasmic fibrillarity consistent with glial differentiation. Immunohistochemical studies provide the strongest evidence that the tumor cells are glial in nature. Immunoreactivity for GFAP was strong and diffuse in all 8 tumors, and was the most constant immunohistochemical finding. Although some meningiomas, including so-called "chordoid meningiomas," reportedly show GFAP immunoreactivity, the staining is typically weak and perhaps represents cross-reactivity with other intermediate filament antigens (14, 16). Certainly, staining is not of the degree noted in chordoid gliomas. Cytoplasmic EMA staining was weak and labeled only scattered cells in 4 cases, and was completely negative in 4 cases. While recognizing that a negative or only weakly positive result does not exclude an entity, i.e. meningioma in this case, none of the 8 cases demonstrated the strong EMA immunoreactivity expected of meningioma. Limited EMA staining of this sort may be seen in ependymoma (17), but the lesions had no other features suggesting ependymal derivation. Cytokeratin (AE1/AE3) immunoreactivity was noted in 3 of 4 tumors, but these findings are difficult to interpret, since astrocytomas, ependymomas, meningiomas and choroid plexus tumors may show immunoreactivity for cytokeratin (18–22). Thus, a glial derivation for these tumors is suggested by their strong, consistent staining for GFAP, and their relatively weak or negative EMA staining (18–22).

Although electron microscopy also supported the glial nature of chordoid glioma, it was not helpful in subclassifying them as astrocytic or ependymoma tumors (23–26). Abundant cytoplasmic intermediate filaments, moderate numbers of mitochondria, and occasional "intermediate junctions" were the most consistent, albeit nonspecific, findings. Microvilli were identified focally in all cases examined, suggesting the possibility of ependymoma, although cilia, and desmosomal junctions were not present, nor did other histological or ultrastructural features suggest the diagnosis. Significantly, there were no ultrastructural features suggestive of meningioma, such as complex interdigitation of cell membranes or well-formed desmosomes (27, 28).

Despite their low-grade histologic appearance, the location of chordoid gliomas in the third ventricle and their attachment to hypothalamic and suprasellar structures precluded their complete surgical removal in 6 of the 8 cases. Of these 6, 3 demonstrated new growth on neuroimaging studies; of these, 2 were associated with poor post-operative outcomes resulting in death. One of the 2 developed medical complications unrelated to tumor regrowth and died with evidence of residual tumor 8 months after operation. The second patient, despite receiving radiation therapy following a subtotal resection, was deemed unfit for reoperation of her enlarged tumor and died 3 years after the initial resection. In a third case of incomplete resection, the tumor regrew slowly despite post-operative radiation therapy; the patient remained clinically stable and another operation was not performed.

Many issues regarding these unique third ventricular neoplasms remain unresolved. First among these is the distinct female predominance. To address the possibility that these tumors may be hormonally responsive, we studied a subset for immunoreactivity to estrogen and progesterone receptors, but found no immunoreactivity in any of the 4 neoplasms tested. In addition, the specific location of these tumors within the third ventricle and their apparent origin from the suprasellar/hypothalamic compartment was a consistent, yet unexplained finding. This presumably reflects their cellular derivation, but a more complete explanation is not available.

Although the precise classification of these tumors can not be defined presently, and their biological behavior is not entirely clear, we believe that chordoid gliomas represent a unique form of low-grade glioma, one exhibiting characteristic clinical, radiographic, and histopathologic features. Only additional cases and longer clinical follow-up will permit a complete characterization of this distinctive third ventricular lesion.

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