

Choroid plexus tumors: A clinico-pathological and neuro-radiological study of 23 cases

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ABSTRACT

Background: Choroid plexus tumors are intraventricular tumors derived from choroid plexus epithelium.

Aim: To study the choroid plexus tumors with reference to their clinical, radiological, and pathological features.

Materials and Methods: The study was performed by the retrospectively reviewing the clinical, radiological, and pathological records of patients of choroid plexus tumors.

Results: A total of 23 cases (11 males, 13 females) of choroid plexus tumor were diagnosed from 1997 to 2009. Fourteen patients were below 15 years of age. Raised intracranial pressure was the main presenting feature in all the cases. Tumor was located in lateral ventricle ($n = 14$; in 3 cases tumor was going into third ventricle), fourth ventricle ($n = 7$), and cerebellopontine angle ($n = 2$). Total tumor excision was achieved in 21 cases. The histopathology was suggestive of choroid plexus papilloma ($n = 19$), atypical choroid plexus papilloma ($n = 1$), and choroid plexus carcinoma ($n = 2$). Clear cell areas were noted in three cases. Other histopathological features observed were foci of calcification ($n = 5$), Psammoma bodies ($n = 2$), hemorrhage ($n = 5$), hyalinization ($n = 2$), and oncocytic changes ($n = 1$).

Conclusions: Choroid plexus tumors are intraventricular tumors arising from choroid plexus epithelium. The predominant clinical presentation is raised intracranial pressure. Surgery is the mainstay of treatment; histopathologically, they include choroid plexus papilloma, atypical choroid plexus papilloma, and choroid plexus carcinoma.

Key words: Choroid plexus tumor, intraventricular, papilloma

Introduction

Choroid plexus tumors are rare intraventricular papillary neoplasms derived from choroid plexus epithelium, which account for approximately 2% to 4% of intracranial tumors in children and 0.5% in adults.^[1] Choroid plexus papillomas (CPPs) are more common than choroid plexus carcinomas (CPCs) in a ratio of a least 5:1; approximately 80% of CPCs arise in children, constituting 20% to 40% of all choroid plexus tumors in this

age group.^[1] The average annual incidence is approximately 0.3 per 1,000,000 population inhabitants.^[2] Approximately 80% of the lateral ventricle tumors arise in patients younger than 20 years, whereas fourth ventricle tumors are evenly distributed in all age groups.^[3] Congenital tumors have been observed in utero using ultrasound technique.^[4]

Presenting symptoms such as headache, diplopia, and ataxia are usually caused by hydrocephalus, regardless of tumor location. Direct mechanical obstruction of cerebro spinal fluid (CSF) flow, arachnoid granulation blockage from hemorrhage, and overproduction of CSF can contribute to hydrocephalus.^[5-7] Computed tomography (CT) and magnetic resonance imaging (MRI) show iso- or hyperdense, T1 isotense, and T2 hyperintense masses that appear enhanced with contrast, within the ventricles, generally associated with hydrocephalus.^[8] Macroscopic findings of CPPs include circumscribed cauliflower like masses that may adhere to the ventricular wall, but are usually well delineated from the brain tissue, and cysts and hemorrhages may occur. CPCs are usually invasive tumors that may appear solid, hemorrhagic, and necrotic.^[1] The benign papillary tumor is composed of delicate

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fibrovascular connective tissue fronds covered by a single layer of uniform cuboidal to columnar epithelial cells with round or oval, basally situated, monomorphic nuclei. Conspicuous mitotic activity, brain invasion, and necrosis are absent.^[1]

In contrast to benign CPPs (World Health Organization (WHO) grade I), CPCs (WHO grade III) are characterized by frank signs of malignancy, that is, frequent mitoses, nuclear pleomorphism, increased cellular density, blurring of the papillary growth pattern, and necrosis.^[1,9] Histological grading is recognized as an important prognostic factor in choroid plexus tumors and also affects the decision toward adjuvant radiotherapy and chemotherapy.^[10-13] The distinction between CPP versus CPC is not always clear-cut, as some tumors show only one or few histological features of malignancy, for example, pleomorphism, increased mitotic activity, and invasion.^[14-24] Those tumors have been termed atypical CPP, but clear diagnostic criteria have not been established. In this study, the authors have analyzed clinical, pathological, and neuroradiological features of 23 cases of choroid plexus tumors.

Materials and Methods

All evaluable cases of choroid plexus tumors of the central nervous system from January 1997 to December 2009 were retrieved from the database files of the department of pathology. The clinical, surgical, and pathological data of the patients was retrieved from hospital information system, database files from Department of Neurosurgery and records of the Department of Pathology. The clinical data included patient age and gender, clinical presentation, physical examination, investigations (hematological, radiological), operative details, and clinical diagnosis. The pathological data included tumor location, gross appearance, tumor size (cm), and growth pattern, cellular atypia, mitosis and necrosis. A total of 19 cases of central CPP, 2 cases of atypical CPP, and 2 cases of CPC were diagnosed during this period. The formalin-fixed paraffin embedded tissue blocks and tissue sections were retrieved and reviewed. Additional sections of 3-5 μ were cut and stained with hematoxylin and eosin, as and when required. Neuropathological re-evaluation of choroid plexus tumors involved assessment of histological features proposed as atypical by the current WHO classification. The presence or absence of high cellularity, solid growth, necrosis, and nuclear pleomorphism was assessed. Mitotic activity was assessed by counting mitoses in 10 randomly selected high-power fields (HPF; area of view 0.23 mm²). In line with previous studies, increased mitotic activity was defined as the presence of more than one mitosis/10 HPF. Choroid plexus tumors displaying frank signs of malignancy, defined here as the presence of at least 4 of the previously mentioned atypical histological features, were classified as choroid plexus carcinoma. Based on light microscopic examination, representative sections were selected for immunohistochemistry for cytokeratin and

GFAP. All antibodies were procured from Dako Corporation, Denmark. Ki-67 proliferation index was calculated by counting 1000 cells under high power.

Results

The clinical, neuroradiological features, and operative findings of the 23 cases are summarized in Table 1.

During a period of 13 years, we identified 23 cases of choroid plexus tumors in the Department of Pathology at our institute. There were 11 males and 12 females. The female to male ratio was 1.09:1. The mean age of the patients in this study was 17.4 years, with a range of 6 months to 47 years. Fourteen patients were below 15 years of age. Symptoms of raised intracranial pressure were seen in all cases. In addition, one patient presented with cerebellar symptoms and one patient with gait ataxia. In all the cases, CT scan revealed isodense lobulated lesion, with homogenous contrast enhancement and MRI scan revealed isotense signal intensity on T1 weighted images, hyperintense signal intensity on T2 weighted images, and homogenous enhancement with contrast administration [Figure 1a]. In three cases, there were cystic component in the tumor. Tumor was located in lateral ventricle ($n = 14$; in 3 cases tumor was going into third ventricle), fourth ventricle ($n = 7$), and cerebellopontine angle ($n = 2$). Varying degree of hydrocephalus (HCP) was noted in all the cases and two cases required ventriculo-peritoneal shunt, and one case required subdural-peritoneal shunt. None of the cases underwent pre-operative embolization. Total tumor excision [Figure 1c] was achieved in 21 cases and in remaining 2 cases subtotal tumor excision was done. All patients improved in their symptoms following surgery except one who died in the postoperative period due to refractory ventricular fibrillation caused by hypokalemia. Two cases which had CPC were asymptomatic till 1.5 months follow-up, but long term outcome is not known as they were lost to follow-up after the initial follow-up of 1.5 months.

Histopathological examination

Grossly, the resection specimen consisted of portions of soft, friable, papilliferous pink-tan tissue [Figure 1b]. Microscopically, the architecture of CPPs resembled that of normal choroid plexus. The neoplastic villi were lined by simple epithelium, and the core consists of loose connective tissue stroma and a central blood vessel. The epithelial lining cells were uniform in size and shape, and the nuclei were round-to-oval and basally positioned. Apical cytoplasm was abundant, and cilia could be noted for some cells. No cellular atypia or mitotic figures were noted in 16 cases and were labelled as CPP [Figure 2a]. In two cases, tumor showed focal sheet pattern, with nuclear hyperchromasia, and occasional mitosis [Figure 2b]. These were labelled as atypical choroid plexus papilloma. In another two cases, tumor revealed a tumor displaying cellular pleomorphism, nuclear atypia, mitotic figures, and necrosis.

Table 1: Summary of the clinical, radiological and operative details of choroid plexus tumors

Age(years)/ Sex	Clinical presentation	Extent of excision	Location	Neuro-radiological features (CT and MRI)	Operative findings	Histopathological diagnosis
2.5/F	Vomiting with persistent cry, deterioration of vision	Total	Right lateral ventricle	CT scan: Isodense, 4x4x4 cm with homogenous contrast enhancement with HCP	Tumor was well defined, reddish, irregular surface, pinkish, moderately vascular	Choroid plexus papilloma with atypia
5/M	Headache, vomiting, one episode of tonic posturing	Total	Left lateral ventricle	CT scan: Isodense to hyper dense lesion in the atrium and body of the ventricle, enhancing on contrast. HCP present	Greyish, soft, partly suckable, vascular, with plane of cleavage from the ventricular wall	Choroid plexus papilloma
27/F	Headache, gait ataxia	Total	4 th ventricle	CT: Isodense, contrast enhancement MRI: Iso on T1 hyper on T2, enhancing on contrast coming out of 4 th ventricle	Soft, suckable, vascular, friable tumor with no attachment on floor of 4 th ventricle	Choroid plexus papilloma
15/M	Increasing head circumference and downward gaze palsy	Total	Right lateral ventricle	CT: Isodense lesion with diffuse contrast enhancement MRI: iso on T1 hyper on T2	Pink, frond like, vascular friable mass	Choroid plexus apilloma
1.5/F	Difficulty in walking and feature of raised ICP, progressive enlargement of head	Total	Left lateral ventricle	CT: Hyperdense enhancing mass in the left trigone, temporal, occipital horn and body of lateral ventricle MRI: iso on T1 and heterogenous on T2, lobulated brilliantly enhancing mass	Lobulated mass, very vascular, soft to firm, well defined plane of cleavage	Choroid plexus apilloma
34/F	Raised ICP, progressive diminution of vision	Total	Fourth ventricle	CT: Hyperdense mass with contrast enhancement MRI: Enhancing mass, going into the cistern magna	Very vascular, firm, cauliflower like mass	Choroid plexus papilloma
45/F	Headache, cranial nerve paresis	Subtotal	Right cerebellopontine (CP) angle	CT: Hyperdense lesion, contrast enhancement with HCP MRI: Iso on T1 hyper on T2, enhancing on contrast	Cystic mass in temporal horn, pinkish, soft and vascular	Choroid plexus papilloma
25/F	On and off holocranial headache, progressive loss of vision	Total	Fourth ventricle	CT: 2.5x3.1 cm size isodense mass with enhancement MRI: Iso on T1 and hyper on T2 enhancing on contrast with HCP	Soft suckable frond like appearance arising from choroid plexus	Choroid plexus papilloma
10 month /M	Progressive enlarging head size	Total	Left lateral ventricle also going into Third ventricle	CT: 2.5x3.6 cm size isodense mass with contrast enhancement MRI: Frond like enhancing mass in third and left lateral ventricle with calcified foci and HCP	Reddish gray frond like friable highly vascular	Choroid plexus papilloma
20/M	Headache, decrease vision	Total	Fourth ventricle	CT: 4x4 cm, hypodense with dense area of calcification with HCP MRI: Enhancing mass in fourth ventricle	Pinkish grey vascular mass attached to 4 th ventricular floor	Choroid plexus papilloma
13/F	Headache, vomiting	Total	Fourth ventricle	CT: Lobulated hyperdense mass with specks of calcification with HCP MRI: ILobulated enhancing mass in fourth ventricle	Pink, soft, vascular fronds tumour	Choroid plexus papilloma
9/M	Altered sensorium, failure to take feeds, abnormal enlargement of head	Total	Right lateral ventricle	CT: Hyperdense lesion in the right ventricle taking up contrast MRI: Enhancing mass right lateral ventricle with calcified foci and HCP	Fronds of vascular tissue attached to choroid plexus	Choroid plexus papilloma
10 month /M	Vomiting, dullness with decreased feeding, increased head size	Total	3 rd and right lateral ventricle	CT: lobulated hyperdense enhancing mass MRI: T1 iso, T2 hyper with enhancement with contrast with HCP	Pink lobulated mass	Choroid plexus papilloma
6 months /M	Frequent upward rolling of eyes with enlargement of head	Total	Right lateral ventricle	MRI: Iso to hyper on T1 hyper on T2 lesion, with enhancement with contrast, with HCP	Tumor reddish grey highly vascular	Choroid plexus papilloma
7/M	Headache	Total	Left lateral ventricle	MRI: T1 iso- and T2 hyper lesion in trigone with contrast enhancement with HCP	Fragile, suckable	Choroid plexus carcinoma
25/F	Holocranial headache, Total off and on vomiting and progressive vision loss for 15 days, generalized seizure for 5 months	Total	Left lateral ventricle	MRI: Iso on T1 hyper on T2 homogenously enhancing on contrast lesion in the left lateral ventricle trigone with entrapment of the occipital horn with HCP	Tumor was greyish red, firm, moderately vascular, suckable at places	Choroid plexus papilloma with atypia

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Table 1: Summary of the clinical, radiological and operative details of choroid plexus tumors

Age(years)/Sex	Clinical presentation	Extent of excision	Location	Neuro-radiological features (CT and MRI)	Operative findings	Histopathological diagnosis
26months/F	Progressive increase in size of head, recurrent vomiting, with developmental delay	Total	Right lateral ventricle	MRI: T1 iso, T2 hyper with contrast enhancing lesion with HCP	Reddish, soft to firm frond like moderately vascular lesion	Choroid plexus papilloma
1/M	Progressive increase in size of head	Total	Left lateral ventricle	CT: Isodense, lobulated enhancing mass in trigonal area of left lateral ventricle MRI: Lobulated mass T1 iso, T2 hyper and homogenously enhancing with contrast with moderate HCP	Lobulated, vascular frond like mass	Choroid plexus papilloma
40/M	Raised ICP, right cerebellar symptoms	Total	Right CP angle	CT: Hyperdense with cystic component, mass with hydrocephalus MRI: Right CP angle extraxial lesion, iso to hypo in T1, and heterogenous hyperdense on T2 with HCP	Reddish, soft to firm frond like moderately vascular lesion	Choroid plexus papilloma
32/F	Holocranial headache, vomiting, diminution of vision, altered sensorium	Subtotal	Right CP angle	CT: Enhancing brain stem exophytic mass on right CP angle, MRI: Lobulated enhancing mass on right CP angle, coming out of foramen of Luschka	Grey white friable vascular frond like tumor	Choroid plexus papilloma
14/M	Headache and vomiting	Total	Right lateral ventricle and third ventricle	CT: Hyperdense mass, enhancing on contrast MRI: lobulated with both cystic and solid component Brightly enhancing on contrast	Tumor in right lateral ventricle and extending to into third ventricle. cyst has multiple septae	Choroid plexus papilloma
22/F	Headache and swaying while walking for, progressive diminution of vision	Total	Fourth ventricle	CT: Irregular frond like mass in fourth ventricle, mildly enhancing on contrast, with HCP	5×4 cm tumor reddish yellow fleshy	Choroid plexus papilloma
1.5/F	Vomiting, headache and visual deterioration	Total	Fourth ventricle	CT: Contrast enhancing lesion MRI: Small iso to hypo mass with contrast enhancement with HCP	Reddish pinkish lobulated mass	Choroid plexus carcinoma

The epithelial sheath overlying the papillae exhibited areas of stratification, and the stroma was edematous. The epithelial cells were columnar and tightly stacked in some areas, with high nucleus/cytoplasm ratios and hyperchromatic nuclei. They were labelled as CPC [Figure 2c]. Clears cell areas were identified in three cases [Figure 2d]. Foci of calcification were identified in five CPP, including Psammoma bodies in two cases and were located either in fibrovascular core and also lying in the stroma. Hemorrhage was identified in five tumors and hyalinization was identified in two tumors. Oncocytic changes in the tumor cells were identified in one tumor.

Discussion

CPPs are intraventricular papillary neoplasms derived from the choroid plexus epithelium, that account for 2% to 3% of intracranial tumors in children.^[9] Benign papillomas are reported to account for approximately four-fifth of the neoplasms and carcinomas one-fifth.^[1] On MRI scans, papillomas tend to appear as lobulated, homogeneous, enhancing masses, whereas carcinomas appear more heterogeneous because of areas of necrosis, calcification, or

hemorrhage. The advantage of using MRI in the diagnosis of choroid plexus neoplasms lies in the fact that the multiplanar view facilitates surgery as well as postoperative evaluation.

Clear cells are common in all choroid plexus tumors, benign, and malignant. As they are also very frequent in the fetal choroid plexus, they may suggest similarities between the neoplastic cells and immature related tissue. Psammoma bodies (PBs) are changes whose origin is still an enigma for the scientists, but their mechanism of formation is not clear.^[25,26] Following observation that laminated hyaline globules may be the precursor of concentric laminar calcification lacking clinical or histopathological relevance, they have been considered to be secondary to hypoxia or ischemia. They are found in normal meninges, choroid plexus.^[25,26] PBs are more frequent in choroid plexus of healthy older people, and during aging, they obtain larger dimensions more irregular contours, which is the result of their mutual merging.^[25,27] Amyloid deposition can also be incidentally observed in choroid plexus and choroid plexus tumors without clinical significance.^[28] Other unusual histological feature include mucinous degeneration, melanization, and tubular glandular structures.

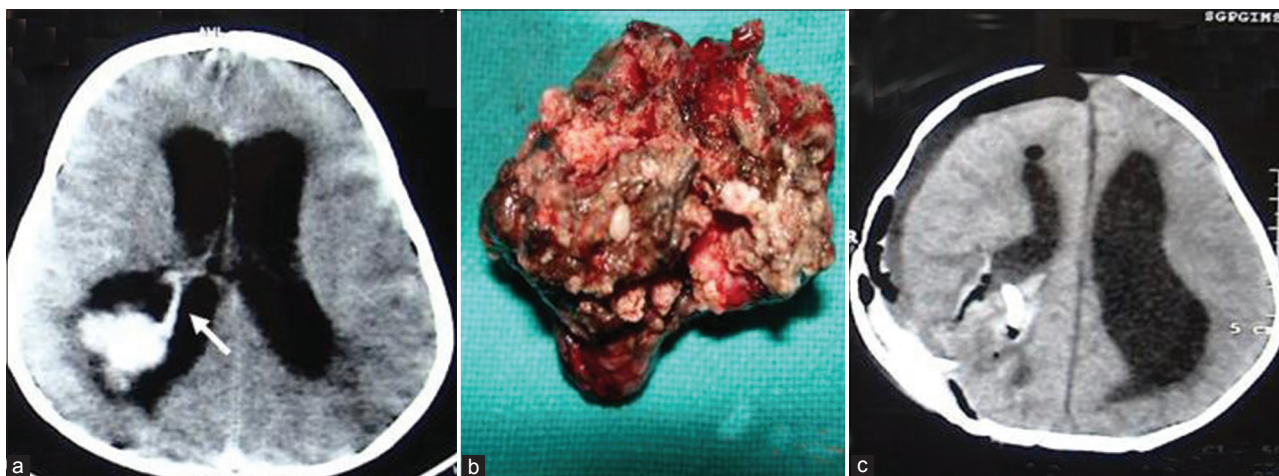


Figure 1: Contrast enhanced CT picture of choroid plexus papilloma in the left lateral ventricle. (a) The arrow is showing the vascular pedicle attached with the choroid plexus is also seen, (b) Gross specimen of choroid plexus papilloma, (c) Post operative CT scan showing complete excision of the tumor

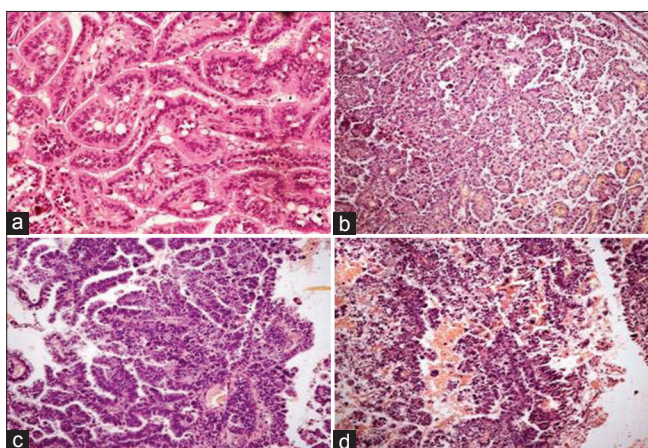


Figure 2: Photomicrograph of choroid plexus papilloma: (a) Tumor displaying papillae lined by single layer of cubo-columnar cells having isomorphic nuclei and moderate amount of cytoplasm, (b) Mild to moderately anisomorphic nuclei and scarce mitosis, (c) Pleomorphic nuclei, coarsely clumped chromatin, irregular nuclear membrane and moderate amount of cytoplasm with increased mitotic activity, (d) Clear cell change in choroid plexus carcinoma

Primary CPP may also arise in cerebellopontine angle from embryonic remnant of choroid plexus anlage. CPP of cerebellopontine angle may present as direct extension from primary intra fourth ventricular CPP or from choroid tuft projecting from the foramen of Luschka, referred to as Bochdalek's flower basket.

A study by Jeibmann *et al.* examined a series of 164 choroid plexus tumors for the presence of atypical features and correlated these features with prognosis in an effort to create criteria for the diagnosis of atypical CPP.^[29] They showed that mitotic activity is the only feature that impacted the probability of recurrence in patients diagnosed with CPP. The group proposed that a mitotic count greater than or equal to 2 per 10 high-power fields could define a choroid plexus tumor as atypical, and the same study suggested that if at least

two of the following histological features are seen, including increased cellularity, nuclear pleomorphism, solid growth pattern, and/or necrosis, a diagnosis of atypical CPP would be warranted. Using these criteria, the study examined a series of 124 previously diagnosed CPPs, and 15% of these were atypical CPPs. Malignant evolution of a CPP which recurred as CPC has been reported in two patients, but documented transition from benign to malignant histology is extremely rare.

In comparison with the papilloma, the diagnosis of CPC is rendered in the setting of increased cell density, increased mitotic figures (usually greater than 5 per 10 high power fields), nuclear pleomorphism, and necrosis.^[1] Diffuse invasion of the adjacent brain parenchyma is often present. While distinct papillary configuration is a hallmark of low-grade CPP, in CPC the papillary features are blurred or can be lost in the sheets of epithelial tumor cells, and this ill-defined growth pattern can make diagnosis challenging.^[30] Proliferation indices such as Ki-67/MIB-1 can be useful in the diagnosis of CPC, with reported mean nuclear labelling rates of 1.9% for CPP and 13.8% in CPC. Choroid plexus carcinoma is classified as a WHO grade III tumor.^[1]

Differential diagnosis of an intraventricular papillary tumor includes several possibilities. Villous hypertrophy of the choroid plexus which is a benign proliferation causing enlargement of the choroid plexus in the ventricles is one such possibility. These lesions have normal choroid plexus microscopically, and can also be distinguished from a neoplastic lesion using proliferation markers.^[1] Papillary variant of ependymoma, is an additional consideration; however, characteristic cytological and morphological features on histological examination, including monomorphic cuboidal cells with a flattened surface, indistinct nucleoli, granular chromatin, ependymal rosettes, and perivascular pseudorosettes consisting of tapered cell processes oriented toward a central blood vessel can

help.^[9,31] Another tumor to consider as differential diagnosis is myxopapillary ependymoma, a tumor that is usually located at the spinal cord; it is formed by papillae within a myxoid matrix and PBs, surrounded by cuboidal epithelial cell, and has GFAP-and CK-positive reaction. Papillary meningioma, a rare high grade (WHO grade III) variant of meningioma, can arise in the choroid plexus, especially in patients in the pediatric age group. This tumor is described histologically as having a pseudopapillary or perivascular pattern in part of the tumor, which makes this meningioma a WHO grade III neoplasm.^[31,32] In infants, PNET (primitive neuroectodermal tumors) can occur in the same area. Apart from all these, atypical teratoid/rhabdoid tumor (AT/RT), due to its aggressive nature, deserves a special mention. Histologically, AT/RT usually has groups of rhabdoid cells with eccentric, pleomorphic nuclei, and abundant eosinophilic cytoplasm. These tumors are high grade, with increased mitoses and necrosis. Atypical teratoid/rhabdoid tumor can be somewhat challenging to distinguish from CPCs that are poorly differentiated with foci of epithelial differentiation. In immunohistochemistry, CPCs stained positively for INI1, whereas the majority of AT/RTs are negative. This immunostain could help in distinguishing between a poorly differentiated CPC and an AT/RT with extensive epithelial differentiation. Finally, metastatic neoplasms to the choroid plexus, especially in the adult population, are possible. The reported incidence of solitary metastases to the choroid plexus with no evidence of tumor in the brain parenchyma is rare, accounting for 0.14% of all cerebral metastases, the most common in adults being renal cell carcinoma. Metastases of papillary carcinomas to choroid plexus occur in 10% of the patients; hence, we should consider them as a differential diagnosis because they may have identical histological appearance. There are some markers that have been used to distinguish them, such as BerEP4 and CEA, which are positive in metastatic carcinomas and negative in CPPs.

Total excision by surgical extirpation is expected to be curative, with infrequent recurrences.^[2] However, a gross total resection sometimes may not be feasible because of the deep intraventricular location of lateral ventricular tumors and the proximity of third and fourth ventricular tumors to critical brain-stem structures.^[33]

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