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Clinical–patient studies

Chordoid Meningioma, an uncommon variant of meningioma: A clinicopathologic study of 12 cases

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Key words: Chordoid meningioma, intracranial, intraventricular, mast cells, meningeal tumor

Abstract

Aims: The study has been undertaken to document the clinicopathological features of 12 cases of chordoid meningioma, operated at All India Institute of Medical Sciences during 1996 to June 2005.

Methods: Clinical information was retrieved from the records of our Neurosurgery Department. The cases were stained with H&E, Periodic Acid Schiff (PAS) with and without diastase, mucicarmine, giemsa, toluidine blue, alcian blue, reticulin and Masson trichrome. Immunohistochemistry for pancytokeratin, epithelial membrane antigen, vimentin, glial fibrillary acidic protein, MIB-1, Leucocyte common antigen (LCA), CD-3 and CD-20 was done in all cases.

Results: The age ranged from 12–67 years (mean 34.2 years) and three of them occurred in <18 years. Male to female ratio was 1:1.4. The duration of symptoms varied from 3.5 months to 5 years (mean 14.1 months). No systemic symptoms were noted. The location of tumor in eight cases was in the supratentorial and rest four in the infratentorial compartments. Interestingly, two cases were in intraventricular location, one in the lateral ventricle and other in the fourth ventricle. Microscopic examination showed lobulation with chordoid elements constituting >95% of the entire tumor area in 11 of the total 12 cases. In one case, chordoid pattern constituted about 30% of the total tumor area; the rest was predominant meningothelial (60%). Mild to severe lymphoplasmacytic cell infiltrate was present in all cases. The histochemical stains showed the pattern of acidic mucin and interestingly revealed the presence of mast cells both in connective tissue stroma and epithelial cell islands. The inflammatory infiltrate was B-cell predominant. MIB-1 labeling index was low (<2%) in all cases except two, which showed LI of 6% and 8%. Strong diffuse immunoreactivity for vimentin and focal positivity for epithelial membrane antigen was noted in all cases.

Conclusions: Chordoid meningiomas are predominantly tumors of young adults with predilection for supratentorial location. Intraventricular location, absence of systemic manifestations despite the presence of abundant B-lymphocytes, presence of mast cells and low MIB-1 LI are some of the interesting findings in the present series, which need documentation. Hence, larger number of cases with adequate follow-up data need to be studied further to establish the clinical significance of this variant.

Introduction

Meningiomas are common intracranial tumors and constitute between 13 and 20% of all primary intracranial tumors [1]. Predominantly, they are slow growing benign tumors attached to the duramater and composed of neoplastic meningothelial (arachnoidal) cells. They have a plethora of architectural patterns and wide range of histopathological appearances [1]. The 2000 WHO classification of tumors of central nervous system describes fifteen subtypes of meningiomas. Most of these share a common clinical course and are graded in the category of WHO grade I. Certain histological subtypes are more likely to recur and follow a more aggressive clinical outcome corresponding to WHO grades II and III. Apart from these histological subtypes, in the typical meningiomas there are certain morphological criteria of aggressive behavior [2] – viz. (i) A mitotic index of 4 or more per 10 hpf, (ii) The finding of 3 of 4 parameters (hypercellularity, sheeting growth, prominent nucleoli, presence of small cells) or (iii) Finding of brain invasion. The presence of either of the first two criteria are required for qualifying the tumor as WHO grade II [2] and presence of 20 mitoses or more per 10 hpf with or without obvious sarcoma-like areas is required for WHO grade III [3].

Chordoid meningioma was first described by Kepes et al. in 1988 as meningeal tumor in young patients associated with microcytic anaemia and/or dysgammaglobulinemiA [4]. Since then, there are only 73 published cases of chordoid meningiomas in the existing literature, most of which are in the form of isolated case reports [5–10] except for two series with a varied and contradicting clinicopathological features [4,11] because of which their characterization still remains to be complete.

To the best of our knowledge this is the second largest series in the existing literature, where we studied clinicopathological features of twelve cases of this uncommon tumor and reviewed the relevant literature.

Materials and methods

All cases, which were diagnosed as chordoid meningiomas, were retrieved from the files of Department of Pathology, All India Institute of Medical Sciences. All patients were operated and treated in this hospital during 1996 to June 2005. Five-micron thick sections were recut and routine Hematoxylin and Eosin stained sections of each case were reviewed and diagnosis was reconfirmed. Clinical information (patient's age and sex, presenting symptoms, neuroradiological data, operative findings and follow-up) was retrieved from the records of Neurosurgery Department of this hospital.

Surgical specimens were fixed in 10% neutral buffered formalin, routinely processed and paraffin embedded. Special stains – Periodic Acid Schiff (PAS) with and without diastase, mucicarmine, giemsa, toluidine blue, alcian blue, reticulin and Masson trichrome were done in all cases. In addition, immunohistochemical staining was done by LSAB technique using monoclonal antibodies to pancytokeratin (dil 1: 100), epithelial membrane antigen (dil 1: 50), vimentin (dil 1: 50), glial fibrillary acidic protein (dil 1: 5000), MIB-1 (dil 1: 200), CD-3 (dil 1:200) and CD-20 (dil 1: 100). All antibodies were obtained from M/S Dako Patts – Denmark. Antigen retrieval was done wherever required.

Results

During this period of 9.5 years (1996–June 2005), 1185 cases of meningiomas were diagnosed in our department, of which twelve were of chordoid meningiomas. Thus, chordoid meningiomas constituted 1.01% of all meningiomas.

Clinical features (Table 1)

The age ranged from 12 to 67 years with mean age of 32.4 years. Three (25%) of them were in the pediatric population (less than or equal to 18 years). There was slight female preponderance with male to female ratio of 1:1.4. The tumors occurred in different locations and the supratentorial location was the commoner with frontoparietal convexities being the most preferred location. (See Table 1) Two of them were in intraventricular location, one each in lateral and fourth ventricle. The duration of symptoms varied from 3 months to 5 years with a mean period of 14.1 months. The most common presenting symptoms were headache and vomiting and were present in all patients (100%). Other lesser common presenting symptoms were visual abnormalities in 5 cases (41.6%), seizures in 4 cases (33.3%) and paresis in two cases (16.6%). However, none of the patients had any systemic manifestations or hematological abnormalities.

Radiology

The radiological data with CT scans were available in seven cases and MR was available in two cases. CT scan showed isodense mass lesions, which were homogenous to heterogenously enhancing on contrast injection. On magnetic resonance imaging (MRI), the tumor was hypointense on T1 WI and iso to hyperintense on T2 WI. The tumor on post-gadollinum injection showed dense homogenous enhancement and were hypervascular on angiography. There was no peritumoral edema except in two cases, which showed marked peritumoral edema. One of the tumor was located in the fourth ventricle (Figure 1a and b). Both tumors showed presence of flow voids.

Pathological examination

On microscopic examination the predominant pattern was lobular arrangement with myxoid stroma in all tumors (Figure 2a). However, in one case the tumor showed sheet-like arrangement (Figure 2b). Within the

Table 1. Clinicopathological features of cases of chordoid meningioma

Case No.	Age/Sex	Duration of symptoms	Clinical features	Location	MIB-1 labeling index (%)	Follow up (Months)
1	17/M	3 months	Visual loss, progressive	Cerebellum (midline)	<1	24
2	12/M	1 year	GTCS, headache, vomiting, unsteadiness in gait	Cerebellum	<1	18
3	35/F	3 years	Focal seizures. Headache and vomiting	Right Parietal	<1	20
4	18/F	5 months	Visual loss, Headache and vomiting	Left Parietal	<1	16
5	26/F	5 years	Vomiting, headache & focal seizures	Left Frontal	2	12
6	32/F	Not available	Visual loss, Headache and vomiting	Right temporo-parietal	8	12
7	20/F	6 months	Headache, vomiting	4rth Ventricle	2	18
8	42/M	5 months	Visual loss, headache, pain in left half of face	Midbrain	1	12
9	42/F	Not available	Visual loss	Lateral ventricle	1	10
10	48/M	4 months	Headache, right hemiparesis	Left frontal	6	6
11	30/M	6 months	Headache, seizures and decreased vision	Left frontal	2	4
12	67/F	4 months	Headache, decreased vision, dysphasia and memory loss	Sphenoid wing	2	3

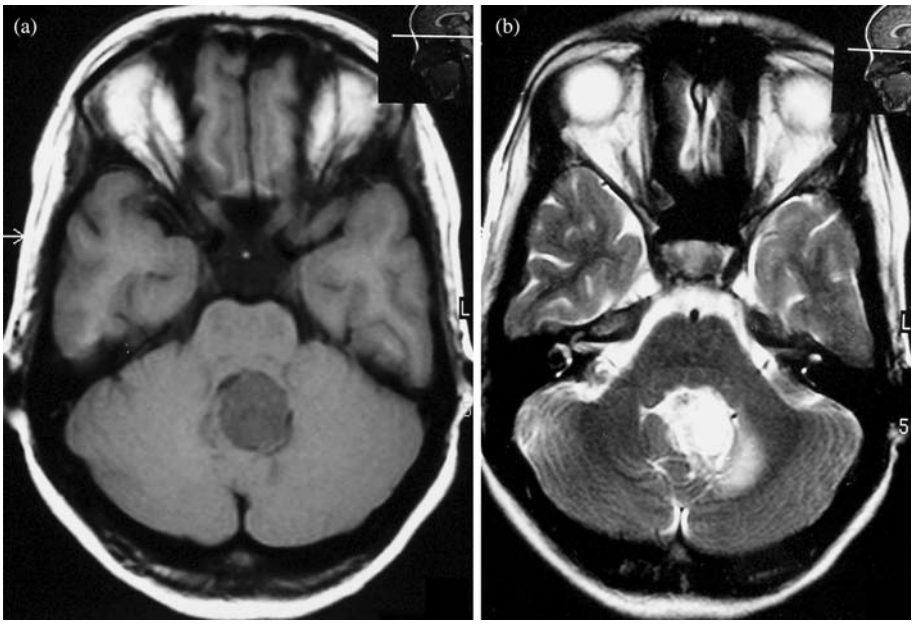

Figure 1. (a) MRI T1 WI of case no. 7 shows hypointense mass in the fourth ventricle. (b) The tumor is hyperintense on T2 WI.

myxoid stroma, the tumor cells displayed the classical cord-like arrangement (Figure 2c). The tumor cells in most of the cases were oval to polygonal in appearance without any spindling. Intranuclear cytoplasmic inclusions were seen in 4 cases and cytoplasmic vacuolation in 2 cases (pseudo-physalliferous cell) (Figure. 2d). Psammoma bodies and tumor giant cells were seen in one case each respectively. Nine cases showed 100% chordoid elements. In two cases, a small component (about 5%) of meningothelial and transitional forms of meningioma respectively was seen in addition to the chordoid element. In the other case, chordoid pattern was a minor component and constituted about 30% of the total tumor area; the rest of which was composed predominantly of meningothelial component. The histopathological hallmark of all the cases was the presence of lymphoplasmacytic cell infiltrate both within and at the periphery of the tumor (Figure 2e). The infiltrate was varying from mild to severe. The myxoid stroma stained red with mucicarmine, pink in Periodic acid Schiff and was resistant to diastase and bright blue with alcian blue staining (Figure 3a). On reticulin stain, the lobules of the tumors were reticulin poor with condensation of reticulin at the periphery. No collagenous stroma was seen in any of the cases on Masson trichrome stain. Interestingly, mast cells were seen both within the myxoid stroma and the epithelial cell islands on toluidine blue and giemsa stains in all cases (Figure 3b). They were sparsely populated, granulated, single in arrangement and more frequently seen at the interface regions.

Tumor cells were strongly immunoreactive to vimentin in all cases (Figure 4a). Epithelial membrane antigen was focally positive in all except in one case (Figure 4b), in which it showed diffuse positivity. All the tumors were negative for cytokeratin and GFAP. The MIB-1 labeling index was low and was <2% in all cases except two which showed 6% and 8% respectively (Figure 4c). The lymphocytic infiltrate was predominantly of B-cell (CD20 positive) immunophenotype (Figure 4d). T-cells (CD3 positive) were sparse and scant (Figure 4e).

Follow-up

Gross total resection was done in eleven cases and near total resection in one. The operative impression was benign meningioma in 7 cases, malignant meningioma in one, hemangioblastoma in one and in 3 cases no operative diagnosis was offered. The postoperative follow-up of the cases was varied from 3 months to 24 months (mean follow-up period: 12.9 months) and none of them had any clinical recurrence or metastasis.

Discussion

Meningiomas, which were earlier, reported as 'myxoid' and 'vacuolated' meningiomas in 1977 and 1979 [12,13], probably belong to this category. However, it was the reported case of Connors et al. in 1980 [14] which brought the attention to this variant. At that time, there was no consensus in the nomenclature with the University of California preferred a histological diagnosis of chordoma and the Armed Forces Institute of Pathology favoured an unusual variant of angioblastic meningioma of the hemangioblastic type. Subsequently after 5 years in 1982, Diamond et al. [15] reviewed this same case and favoured the diagnosis of a lymphoproliferative disorder, either angiomatous lymphoid hamartoma or plasma cell granuloma.

The term 'chordoid meningioma' was first introduced by Kepes et al. in 1988 [4] and described it as a distinct entity occurring in pediatric population associated with systemic manifestations and/or hematological abnormalities. In the present existing literature, apart from two large series (Kepes et al. [4] and Couce et al. [11], the majority of the documented cases are subjects of case reports [5–10]. Brief review of comparison of

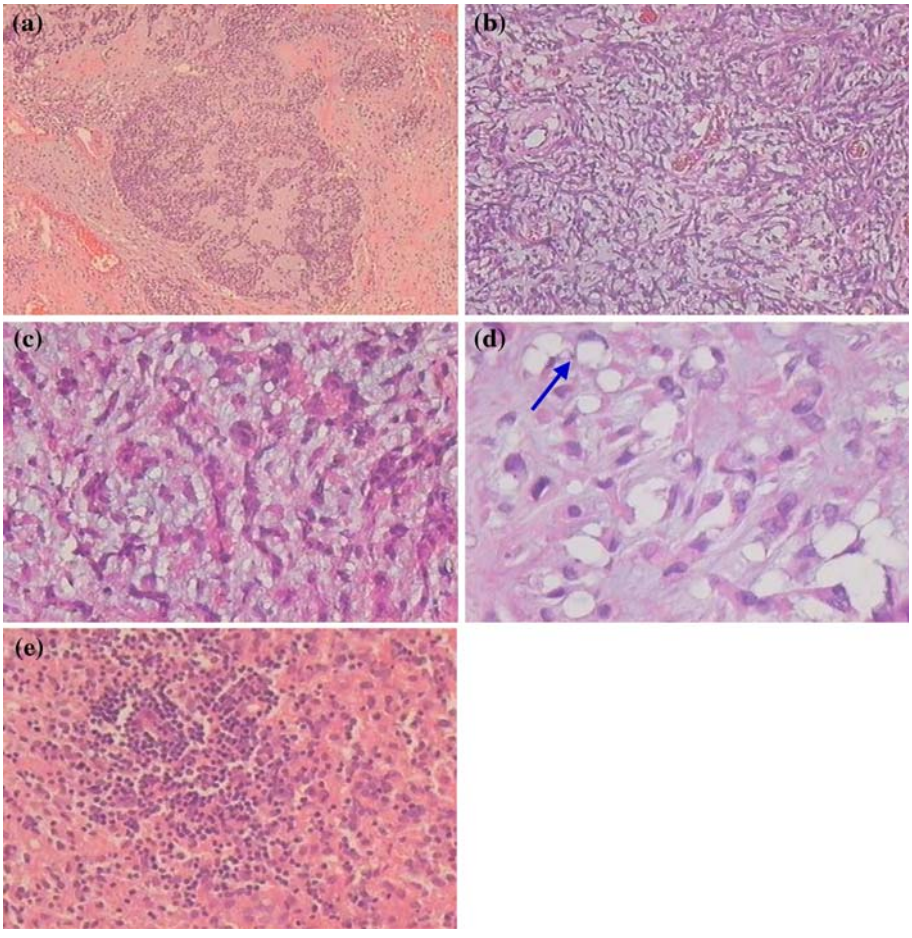

Figure 2. (a) (H&E ×0). Photomicrograph showing the lobular arrangement admixed with myxoid stroma. (b) (H&E ×100). Photomicrograph showing the sheeting pattern of the tumor. (c) (H&E ×200). Photomicrograph showing the cord-like arrangement of the tumor cells within the myxoid stroma. (d) (H&E ×400) Photomicrograph showing cytoplasmic vacuolation of the tumor cells and assuming the appearance of physalliferous cells (pseudo-physalliferous cell). (e) (H&E ×200) Photomicrograph showing the conspicuous presence of lymphoplasma cell infiltrate within the tumor.

clinicopathological features observed in different series is shown in Table 2.

In the present series, the chordoid meningioma included in the study comprised 1.01% of all meningiomas as compared to 0.5% in the series by Couce et al. [11]. Some of the features are at variance with large series reported by Kepes et al. [4] and Couce et al. [11]. The age of our patients ranged from 12 to 67 years (mean 32.4 years) with 25% (3 cases) of them in the pediatric age group. Though the age range is wide but most of the cases were in younger age group, this in contrast to the reported mean age of 11.4 and 47.4 years in Kepes [4] and Couce series [11] respectively. In the present study, there is a slight female preponderance (with male to female ratio of 1:1.4) which is similar to that reported in the literature, a pattern commonly observed in typical meningiomas. But atypical and anaplastic meningiomas show conspicuous predominance in males [16]. A significant 33.3 % (4 cases) were in the Infratentorial compartment and interestingly 2 cases (16.7%) were intraventricular in location, an unusual rare site of occurrence with only one documented case of intraventricular location reported to date [17]. However, unlike this variant, meningiomas are generally known to occur within the ventricles [18] and are assumed to arise from the meningothelial inclusion bodies normally present in the arachnoid of the tela choroidea [19]. Most of the reported Chordoid meningiomas are intracranial in location except a few rare stray reports of occurrence in the extracranial sites like cervical spine [20] and lungs [21].

Systemic manifestations like anemia and dysgammaglobulinemia have been reported to be associated with the tumor [4] and these symptoms cured with the excision of these tumors. None of our patients like those in the series by Couce et al. [11] and some of the other single isolated case reports, showed any systemic manifestations. None of our cases showed any recurrence, while Couce et al. [11] reported a recurrence rate of 42% and Kepes et al. [4] reported a recurrence rate of about 28.6%. The available clinical follow-up period of the present study is not adequate for any valid affirmative inference and the short follow-up may explain the lack of recurrences in the present study (Table 2). Similarly, most of the other isolated case reports also do not have adequate clinical follow-up information and thus cannot be commented anything about recurrences.

The histopathological spectrum of tumor patterns observed in the study was same as that of the existing literature with lobular pattern being the predominant

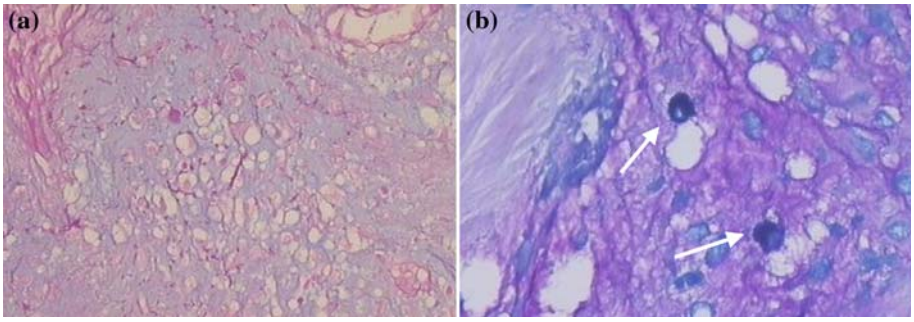

Figure 3. (a) (alcian blue ×100) Photomicrograph showing bright blue stroma on alcian blue, which confirms the acidic nature of the connective tissue mucin. (b) (giemsa ×400). Photomicrograph showing the presence of granulated forms of mast cells within the tumor. These are arranged singly and are not associated with any vessels.

pattern and consistent presence of lymphoplasmacytic infiltrate. In the study, the predominant population of the lymphocytes being the B-cells as observed by Kepes et al. [4]. However, Couce et al did not observed the consistent presence of lymphoplasmacytic cell infiltrate and found only in 59.5% of the cases [11]. Some of the isolated case reports also did not find any intratumoral infiltrate [7] while few others reported a predominance of T-cell population. The reasons for such variable and inconsistent observations are not fully understood at the moment, but it probably infers the heterogeneous nature of these lesions. Though the presence of T-cells seen in the series by Couce et al. [11], can be explained as a part of tumor immune reaction and can also be seen rarely in other subtypes of menigiomas [22]. However, the significance of the presence of B-cell infiltrates in the present study is not clear at the moment and lack of associated systemic manifestations negates this being a part of lymphoproliferative disorder. Because of the nature of the lymphocytic infiltrate the tumors in the study appears to be different from those of the Couce et al. Mucicarmine, alcian blue and PAS with diastase

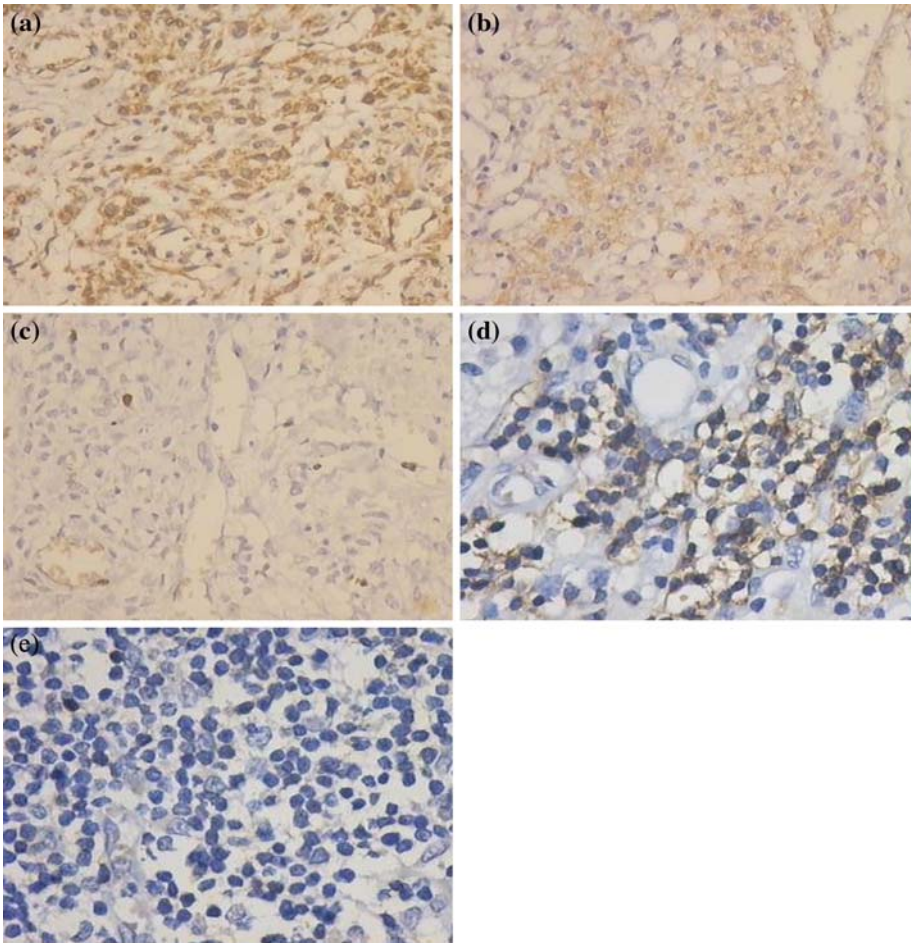

Figure 4. The tumor cells are immunoreactive for vimentin (a ×200) and epithelial membrane antigen (b ×200). MIB-1 shows an occasional labeled cell (c ×200). The lymphocytic infiltrate of the tumor is predominantly immunoreactive for B-cell marker CD20 (d ×200); while most of it is negative for T-cell marker CD3 (e ×200).

Table 2. Comparison of clinicopathological features observed in different series

	Couce et al.	Kepes et al.	Other single case reports	Present study
Total no of cases	42	7	18	12
Age range (in years)	12–77	8–19	15–62	12–67
Mean age	47.4	11.9	–	32.4
Children (less than or equal to 18 yrs)	2 (5.2%)	6(85.7%)	2 (11.1%)	25%
Sex ratio	1:1.1	1:1.3	0.87:1	1:1.4
Location				
Supratentorial	88%	71.4%	100%	66.7%
Infratentorial	12%	28.6%		33.3%
Intraventricular	None	None		16.6%
Systemic manifestation	None	7 (100%)	2 (11.1%)	None
Typical	88%	100%	100%	100%
Atypical	12%	None	None	0%
Chordoid elements				
> 50%	81%	–	–	91.6%
< 50%	19%			8.3%
Lymphoplasmacytic (LP) infiltrate	59.5%	100%	All except one	100%
Nature of LP infiltrate	T > > B	B > > T	–	B > > T
Mast cells	Not demonstrated	Not demonstrated	Not demonstrated	Present (100%)
MIB-1 LI	0.4–11.4% (5.2%)	–	–	1–2% (except 6 and 8% in 2 cases respectively)
Number of cases with available follow-up	33	7	–	12
Follow-up period	2 mon–16 yrs	6 mon–5 yrs	–	3 mon–2 yrs
Recurrences	42% (14/33)	28.6% (2/7)	Cannot be commented	None

confirmed the acidic nature of the mucin of the connective tissue type as also been observed by Couce et al. [11]. None of the other authors studied this aspect. The lobules of this tumor are poor in reticulin like other meningiomas. Interestingly for the first time, we demonstrated the presence of mast cells both in the connective tissue stroma and epithelial cell islands of these tumors. Its occurrence is well known in other myxoid containing tumors like synovial sarcoma [23], myxoid leiomyosarcoma [24], nerve sheath tumors [24], basal cell carcinoma [25], pleomorphic adenoma [26] and Warthins tumor [27] but it is described in very few primary brain tumors like hemangioblastoma [28], hemangiopericytoma, subependymal giant cell astrocytomas [29] and few meningothelial meningiomas [22,30]. Though mast cells are seen in very few cases of meningiomas, they are inconsistent and reported only in the syncytial types of meningothelial meningiomas [22,30]. The mast cells does store and release a number of biologically active mediators and have very diverse functions yet to be known completely. They are even considered as one of the host mediated effectors of tumor invasion [31]. The presence of mast cells can be either because of the influence of the nature of the connective tissue stroma, as a part of the inflammatory cell infiltrate or may be just bystander cells. These cells are found separate from the main inflammatory cell infiltrate, did not show any density correlation with severity of the inflammation and moreover the presence in the interface regions of the connective tissue stroma and epithelial cell islands. Because of all the fore mentioned features, it is highly unlikely that the mast cells in these tumors are part of the inflammation observed in these tumors. More importantly, the mast cells if at all regulate the lymphocytic infiltration; they do so for T-cells by production of IL-8 [25] but no association was observed with B-cell infiltrate. Because of their constant presence, this being just bystanders is also unlikely. The functional significance of consistent occurrence of these cells in these tumors is unclear and it is premature to formulate any hypothesis which is open to speculation. This aspect needs to be investigated, studied and explored for its role, if there is any. Additionally, more studies should be done on its closest morphologic mimics like chordomas and chordoid gliomas to see whether this feature of presence of mast cells in chordoid meningioma can play any contributory role in diagnoses of cases on frozen section.

The immunohistochemical profile of positivity for vimentin and epithelial membrane antigen (EMA), in conjunction with negativity for GFAP and Cytokeratin helps in differentiating other morphologically simulating tumors such as chordoma, chordoid glioma, epithelial hemangioblastoma and metastatic mucinous carcinoma. The identification of any component of typical meningioma, if present, is enough for establishing the diagnosis but immunohistochemistry is still a must for correct diagnosis and differentiating it from its closest morphologic mimics [32].

MIB-1 labeling index was more commonly low in the present study. However, Couce et al. [11] observed a wide range of MIB-1 LI (0.4–11.4%; mean 5.2%) in their series and also reported a high rate recurrence (42%), which made them to categorize this uncommon tumor as a grade II lesion. Though the size of the present study is small to be drawn into a comparison with the series by Couce et al., but the discordance in the observations raises a possible doubt about this being an entity with a true higher proliferative potential or is it a tumor with a relatively higher recurrence rates because

of the physiochemical properties of the connective tissue content.

To conclude, the chordoid meningiomas are an uncommon variant of meningioma and have a distinct clinicopathological profile, which is quite different from other subtypes of meningioma. From the knowledge of the existing literature and the present study it appears that this is a heterogeneous entity rather than a single distinct entity. The reasons for this may be due to lack of any WHO prescribed cut off value of chordoid elements for diagnosing this entity and because of scant literature on this uncommon tumor. Therefore, a multicenteric pooling of data using uniform criteria for diagnosis with long-term follow-up of the patients is likely to uncover the hidden facts of this uncommon tumor and shed light on the possibility of predicting the heterogenous biological behavior of these tumors. The present study is a contribution towards this direction and more pooling of data from other centers is also required. Thus, for proper characterization of these tumors more cases needs to be studied and common uniform diagnostic criteria have to be established.

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