A FIVE-YEAR HISTOPATHOLOGICAL REVIEW OF CNS TUMOURS IN A TERTIARY CENTRE WITH EMPHASIS ON DIAGNOSTIC ASPECTS OF UNCOMMON TUMOURS

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ABSTRACT

BACKGROUND

Tumours of central nervous system (CNS) are of varied histogenesis and show divergent lines of differentiation and morphological features. These tumours show specific predilection for age and sex groups, more commonly than of tumours of other systems. Though tumours of glial tissue are more common, other tumours of neural, ependymal and meningeal origin are not uncommon. Metastatic disease is the common encounter in elderly. Tumour diagnosis is not always straight forward as many non-neoplastic lesions and reactive proliferations mimic tumours. Immunohistochemistry may help in problematic cases and thus can be used as an adjuvant tool in the diagnosis of such cases in addition to the routine histopathological staining methods. An accurate histological diagnosis is of extreme importance in these sites as exact diagnosis helps in proper management and favourable clinical outcome.

MATERIAL & METHODS

This study is on a retrospective and prospective basis in our institution from January 2011 to January, 2016. Our institute is a tertiary care center attached to a medical college catering to the needs of a rural based population. During this period, a total of 717 central nervous system tumour specimens were received and diagnosed based on examination of Haematoxylin and Eosin stained sections of formalin fixed and paraffin embedded specimens. Immunohistochemical markers (IHC) were applied in selective cases for an accurate diagnosis and a number of rare cases were diagnosed based on morphology and IHC marker studies.

RESULTS

Age and sex incidence and anatomic distribution of various tumours were studied. In adults, meningiomas occurred most frequently in the present study followed by nerve sheath tumours, astrocytomas, metastatic deposits, glioblastomas and pituitary adenomas. Embryonal tumours occurred frequently in children. Other rare tumours identified are amyloidogenic pituitary adenoma, central neurocytoma, glioneuronal tumour with neuropil-like islands, ganglioglioma, large cell medulloblastoma, rhabdoid and secretory meningioma, immature teratoma and gliosarcoma. Grading of the tumours was done according to the revised World Health Organization criteria.

CONCLUSION

In this study, we discuss the process of establishing accurately the diagnosis of central nervous system tumours including spinal tumours, with emphasis on rare tumours encountered and how IHC helped in the diagnosis.

KEYWORDS

Central Nervous System, Tumours, Intracranial, Spinal cord, Immunohistochemistry.

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INTRODUCTION: Diagnosis of a CNS tumour might not always be a straightforward process since many non-neoplastic diseases can present as space-occupying lesions, mimicking neoplastic process. CNS tumours by themselves are a heterogeneous group as they differ in histogenesis, and show a spectrum of morphological features. Thorough clinical data, imaging findings and per operative findings offer the valuable clues for the diagnostic approach. Histopathological examination; however, confirms the diagnosis. Whenever the pathologist is faced with a diagnostic dilemma, IHC markers help in exact diagnosis and subtyping of tumours.

This study is taken up to note the incidence of CNS tumours received in our institution along with reviewing the

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morphology of some rare tumours and how IHC aided in the final diagnosis.

MATERIALS AND METHODS: The present study was carried out in our department from January 2011 to January 2016. Neurosurgical specimens received from the department of neurosurgery comprised the material. Pertinent clinical data including details of imaging and preoperative findings were recorded. Only neoplastic lesions were included in our study and non-neoplastic lesions were excluded.

The biopsy specimens were fixed in buffered formalin routinely processed and sections cut from paraffin blocks. Histological examination was done on Haematoxylin and Eosin (H&E) stained sections to diagnose and classify various CNS tumours according to the World Health Organization (WHO) Classification of Central Nervous System tumours. A total number of 717 CNS tumours were recorded.

Immunohistochemistry was performed when there was differential diagnosis. Using three-micron thick sections on poly - L- lysine coated slides, antigen retrieval was done using microwave in citrate buffer at pH 6. Selected markers from a panel including Glial Fibrillary Acidic Protein (GFAP), Epithelial Membrane Antigen (EMA), S100, Pancytokeratin (Pan CK), Vimentin, Synaptophysin, CD99, Leukocyte Common Antigen (LCA), CD 20, NSE and Chromogranin were used for antigen detection by standard avidin biotin kit. Ki67 was used to grade meningiomas.

RESULTS: Age, sex incidence and anatomic distribution of the 717 tumours were studied. In our study, the majority of tumours were Meningiomas (27.1%) followed by Schwannomas (17.2%) and Astrocytomas (12.6%). Pituitary adenomas and Glioblastomas occurred with equal incidence (8.4%) followed by Metastatic (7.2%) and Embryonal lesions (4%). The other tumours were grouped under a miscellaneous category (15.1%) and that included the rare tumours. (Table 1) Grading of the tumours was done according to the revised 2007 WHO criteria.

Brain Tumours	No. of Tumours	Frequency in Percentage
Astrocytomas	90	12.6
Meningiomas	194	27.1
Tumours of cranial and spinal nerves	123	17.2
Metastatic tumours	52	7.2
Pituitary adenomas	60	8.4
Glioblastomas	60	8.4
Embryonal tumours	30	4.0
Miscellaneous Tumours	108	15.1
Total number of brain tumours	717	100

Table 1: Frequency of CNS Tumours

Astrocytomas were seen more in the 3rd decade whereas meningiomas and Schwannomas were seen mostly in the 4th decade. Understandably, metastatic lesions occurred in the 5th and 6th decade and embryonal tumours occurred frequently in children. (Table 2)

Histological Group	Age Group in years								
	1- 10	11-20	21-30	31-40	41-50	51-60	61-70	71-90	Total
Astrocytomas	5	16	18	27	15	6	1	2	90
Meningiomas	0	5	17	37	57	45	25	8	194
Tumours of cranial and spinal nerves	0	4	15	23	32	23	14	12	123
Metastatic tumours	1	0	1	8	13	14	14	1	52
Pituitary tumours	0	0	8	15	21	9	7	0	60
Glioblastomas	1	1	2	8	18	15	10	5	60
Embryonal tumours	12	8	2	5	1	1	1	0	30
Miscellaneous	19	13	14	14	30	12	2	4	108
Total	**38**	**47**	**77**	**137**	**187**	**125**	**74**	**32**	**717**

Table 2: Age Incidence of Various CNS Tumours

All tumours in our study except meningiomas and metastatic tumours were more common in men. Meningiomas were almost twice as common in women as men. (Table 3)

Histological Group	Males	Females	Total
Astrocytomas	53	37	90
Meningiomas	64	130	194
Tumours of cranial and spinal nerves	58	65	123
Metastatic tumours	24	28	52
Pituitary tumours	33	27	60
Glioblastomas	40	20	60
Embryonal tumours	20	10	30
Miscellaneous tumours	59	49	108
Total	**349**	**368**	**717**

Table 3: Sex Incidence of CNS Tumours

Our study showed that frontal location was the commonest site for tumours of CNS followed by cerebellopontine angle, suprasellar area and spine. (Table 4)

Sl. No.	Location	No. of cases	Percentage
1.	Frontal	133	18.5
2.	Parietal	65	9.1
3.	Frontoparietal	39	5.4
4.	Temporoparietal	18	2.5
5.	Temporal	25	3.5
6.	Parietooccipital	14	1.9
7.	Cerebellar	59	8.2
8.	Cerebellopontine angle	99	13.8
9.	Sellar, Suprasellar	99	13.8
10.	Intraventricular	19	2.7
11.	Spine	95	13.3
12.	Miscellaneous	52	7.3
	Total	**717**	**100**

Table 4: Location of CNS Tumours

Immunohistochemistry was done only for selected cases (Table 5).

Sl. No.	IHC No	Clinical data	Differential diagnosis	IHC markers	Final diagnosis
1.	44/11	F 62 Frontal lobe	Glioblastoma Metastases	GFAP +	Glioblastoma multiforme
2.	47/11	M 30 Frontoparietal	Astrocytoma PNET	GFAP +	Diffuse fibrillary Astrocytoma WHO grade II
3.	2/12	F 35 posterior fossa	Glioblastoma Gliofibroma Gliosarcoma	GFAP + in glial GFAP – in spindle Area	Gliofibroma
4.	33/12	M 28 Basofrontal involving optic chiasm	PNET Neuroendocrine carcinoma	Pancytokeratin + Chromogranin +	Metastases from a neuroendocrine carcinoma
5.	38/12	F 58 Frontal intra axial tumour	Non-Hodgkin's lymphoma Astrocytoma	GFAP – CD45 + CD20 +	Non-Hodgkin's lymphoma B cell type
6.	50/12	M 12 4th ventricle	Ependymoma PNET	GFAP – CD99 +	PNET
7.	2/13	F16 Frontoparietal	Non-Hodgkin's lymphoma PNET	CD99 +	PNET
8.	14/13	M ch 5 post fossa	Ependymoma Medulloblastoma Neuroblastoma	GFAP + S100 –	Ependymoma
9.	25/13	F 64 Frontoparietal	Non-Hodgkin's lymphoma Metastases	CD45 + CD20 +	Non-Hodgkin's lymphoma B cell type
10.	43/14	F 41 Frontal	Non-Hodgkin's lymphoma Metastases	Pancytokeratin + CD45 -	Metastases
11.	51/14	F 46 Frontal	Glioblastoma Metastases	GFAP + EMA –	Glioblastoma multiforme
12.	58/14	F 41 CP angle	Clear cell Ependymoma Clear cell carcinoma metastases	EMA + GFAP –	Metastases
13.	70/14	F 11 Rt. Frontal	Oligoastrocytoma for grading	Ki 67 + less than 5%	Low grade Oligoastrocytoma
14.	51/15	F 26 Lt Temporal	Astrocytoma Metastases Non-Hodgkin's lymphoma	GFAP – Pancytokeratin – CD45 +	Non-Hodgkin's lymphoma

15.	52/15	M 45 Frontoparietal	Glioblastoma Metastases	GFAP – Pancytokeratin +	Metastases
16.	69/15	M 29 Rt. Parietal	Gliosarcoma Glioblastoma	GFAP + Vimentin +	Gliosarcoma
17.	147/15	M 12 Rt. Temporal	Ependymoma Glioblastoma	GFAP + EMA +	Ependymoma
18.	161/15	M 38 Ant cranial fossa	Olfactory Neuroblastoma Small cell carcinoma metastases	NSE – Chromogranin +	Olfactory Neuroblastoma
19.	167/15	M 38 Frontoparietal	Clear cell meningioma Metastases	EMA + Vimentin+ Pancytokeratin –	Clear cell meningioma
	33/16	M 21 Suprasellar	Astrocytoma Non-Hodgkin's lymphoma	Synaptophysin + GFAP – Pancytokeratin –	Neuroblastoma/PNET
21.	58/16	M63 Lt Temporal	Metastases Non-Hodgkin's lymphoma Anaplastic Astrocytoma	Pancytokeratin – CD 45 + GFAP -	High grade Non-Hodgkin's lymphoma
22.	62/16	M 4 Frontal	Meningioma Astrocytoma	GFAP +	Low grade astrocytoma

Table 5: Immunohistochemical Analysis of CNS Tumours

DISCUSSION: Tumours of the central nervous system are rare constituting less than 2% of all malignancies. In the present study, meningiomas were the commonest tumours (194 cases) constituting 27.1% of all CNS neoplasms. Datta[1] and Madabhushi[2] reported astrocytomas to be the commonest tumours in their respective studies. However, Das et al,[3] Suh et al[4] and Tamkeen et al[5] observed that meningiomas made up the largest subgroup of their studies.

Schwannomas were the second most common tumours constituting 17.2% of all CNS neoplasms which was not a common finding in other studies.

Diffuse astrocytoma represents 10–15% of all astrocytic brain tumours.[6] In our study, astrocytomas comprised 12.6% of all CNS tumours. Other studies in India and Pakistan reported an incidence much higher; up to 38%.[1,2,7]

60 cases (8.4%) of pituitary adenoma were reported, similar to other studies; where the incidence was ranged from 6 to 11%.[1,2,3,5,] However Javaria et al from Pakistan[8] reported a very low incidence of sellar tumours 2.6% and Kyon von Jung et al from Korea reported an incidence of 20%.[9]

Incidence of Glioblastomas constituted 8.4% of all CNS neoplasms in this study which is comparable to other studies in India, [1,2] Korea[9] and Singapore[3] where the incidence was from 5.2% to 15.5%. Javaria et al[7] reported a very high incidence of Glioblastomas in their study of 22.6% of all CNS tumours.

Metastatic tumours were 7.2% in our study, which was low when compared to other studies where the incidence was from 10 to 12%.[1,2,3,8] In comparison, Javaria et al[8] reported a low incidence of only 4.9% of secondary deposits in their study.

The peak incidence of CNS tumours was seen between 41-50 years. Astrocytomas were commonly seen in the age range of 31-40 years whereas meningiomas, glioblastomas, pituitary adenomas and Schwannomas were seen mostly between 41-50 years. As expected metastatic tumours occurred between 51 -70 years and embryonal tumours occurred in children. These statistics were comparable to most studies in literature.

Males were affected more than females in most of the CNS tumours including Glioblastomas and Embryonal tumours; meningiomas being an exception with higher frequency in females.[10] Meningiomas were twice as common in females as men as observed in other studies.[3,4,6,7]

Intracranial tumours constituted 86.7% and spinal 13.3% similar to that observed by other studies.[2] Frontal lobe was the commonest site (20.7%) of involvement in the intracranial location. This is in agreement to the findings of Jalali and Datta[11], Tamkeen et al[5] and Javaria et al.[8]

Immunohistochemistry was done for cases where there was a differential diagnosis. Over a period of 5 years, 22 cases were subjected to immunohistochemistry that guided us in arriving at final diagnosis (Table 5). That IHC plays a crucial supplementary role in resolving diagnostic dilemmas in the routine practice of neurosurgical pathology has also been proved in our study and in other studies like that of Madabhushi et al.[2]

Several rare and interesting tumours were reported in our series. The first was that of a 54-year-old male who presented with headache of one year duration, recent onset of diplopia and visual field disturbances whose hormonal analysis revealed extremely elevated levels of serum prolactin; 4700 ng/mL (normal 4.04–15.2 ng/mL). MRI Brain images revealed heterogenous hypointense with central hyperintense irregular lobulated mass lesion in sellar and suprasellar area measuring 4.7 x 3.8 x 3.9 cm. The patient underwent trans-sphenoidal adenomectomy and histology revealed only few scattered monotonous numbers of round to polygonal tumour cells with round to oval nuclei and variable amounts of eosinophilic cytoplasm. The cells were intervened by vascularised fibrous stroma. Mitoses were

absent. The striking feature within the tumour was the presence of masses of spherical, laminated eosinophilic material that form coral-like structures which stained positively with Congo red and showed apple green birefringence under polarised light (Figure 1) and reported as amyloid deposits in pituitary adenoma as accumulation of coral-like spheres.

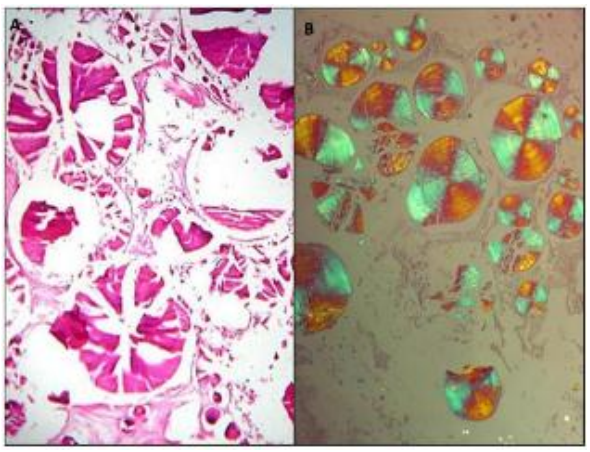

Fig. 1A & 1B

Fig. 1A: Amyloidogenic pituitary adenoma with masses of spherical, laminated amyloid (H and E x 100).
B: Congo red stain of tumour showing apple green birefringence under polarised light (Congo red x 100).

These spheroid type of amyloid deposits are rare and are almost exclusive to prolactinomas.[12,13]

Two uncommon cases of neuronal tumours were reported in our study. Histology of a Cerebellopontine angle tumour and a parietal lobe tumour from male patients aged 32 years and 51 years respectively showed monomorphic cells in nests and sheets with intervening fibrillar matrix. Vacuolated cytoplasm and round nuclei with speckled chromatin were seen (Figure 2 A). The differential diagnoses in both cases was between oligodendroglioma and periventricular neurocytoma. IHC showed positivity of fibrillary matrix for synaptophysin which is diagnostic of periventricular neurocytoma. GFAP positivity was observed in trapped neuronal cells. Neurocytoma corresponds to WHO grade II tumour, carries good prognosis and complete excision is sufficient in most cases.[14]

Histology of three lesions of frontal, frontoparietal and cerebellopontine angle location showed clusters of enlarged and multinucleated neurons admixed with small lymphocytes. A diagnosis of ganglioglioma of WHO Grade I was given (Figure 2 B). Ganglion cells like astrocytes are differentiated from neoplastic neurons by positive expression for GFAP. Neoplastic neurons are distinguished from native neurons by architectural disarray, abnormal clustering, pleomorphism, multinucleation, and diffuse cytoplasmic positivity for chromogranin.[15]

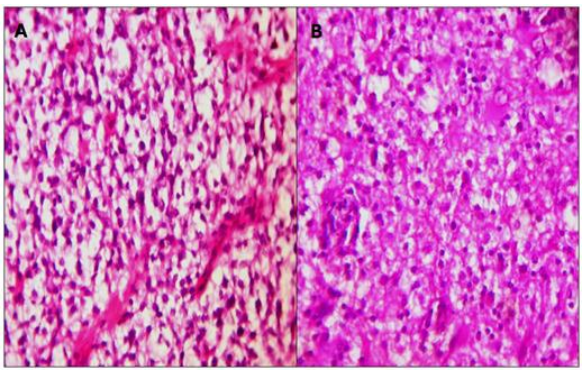

Fig. 2A & 2B

Fig. 2A: Neurocytoma with monomorphic cells in nests and sheets, intervening fibrillar matrix and cells with vacuolated cytoplasm, round nuclei and speckled chromatin (H and E x 400).
B: Ganglioglioma with clusters of enlarged and multinucleated neurons admixed with small lymphocytes and ganglion cells like astrocytes (H and E x 400).

A rare case of a glioneuronal tumour with neuropil-like islands was reported in our institute. The tumour was in the intramedullary location of the cervical region and had round to oval islands of neuropil-like matrix. These islands were surrounded by oligodendrocyte like cells and larger anaplastic glial cells. The tumour also showed more mitotic activity and microvascular proliferation but no necrosis. (Figure 3 A) These uncommon tumours are WHO grade II or III and seem to behave in a manner comparable to neoplasms of diffuse astrocytic type when matched for WHO grade of their glial components.[16]

Teratomas of the parasellar regions are rare and constitute only 1% of the parasellar and sellar tumours. We signed out a case of immature teratoma in the sellar, parasellar area in a female child aged 4 years. The immature neuroepithelial and mesenchymal component comprised a minor component of the tumour and was labelled low grade. (Figure 3 B).

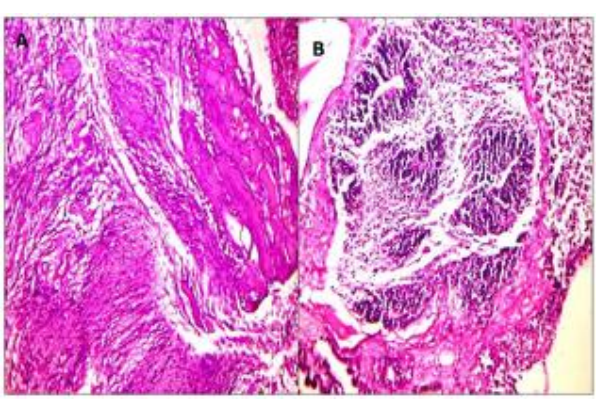

Fig. 3A & 3B

Fig. 3A: Glioneuronal tumour with islands of neuropil-like matrix surrounded by oligodendrocyte like cells and larger anaplastic glial cells (H and E x100).
B: Immature teratoma with neuroepithelial tissue (H and E x400).

A case of Giant cell Glioblastoma was reported in a 55 year old male in the right temporal location that showed numerous multinucleated giant cells, some were extremely bizarre with prominent nucleoli. (Figure 4A) The tumour showed high mitotic activity with atypical mitoses and geographic necrosis. Giant cells are usually immunopositive for S-100 protein, Vimentin, class III ß-tubulin, p53 and EGFR, but their GFAP expression is highly variable.[17]

Six cases of Gliosarcomas were diagnosed in our study with an age range from 9 years to 47 years and the tumours were mostly located in the cerebral cortex of frontal and parietal lobes.

Gliosarcomas are WHO grade IV tumours and show a mixture of gliomatous and sarcomatous tissues with a striking biphasic growth pattern. This was also seen in our cases. (Figure 4B) The glial portion is astrocytic in nature and anaplastic, mostly showing the typical features of a glioblastoma. Epithelial differentiation, has carcinomatous features and sarcomatous component often demonstrates the typical pattern of fibrosarcoma or a malignant fibrous histiocytoma.[17]

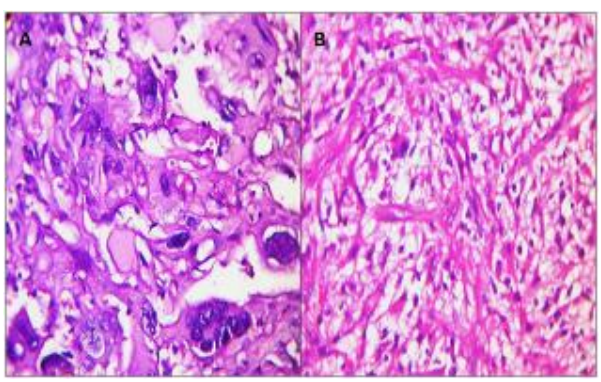

Fig. 4A & 4B

Fig. 4A: Giant cell glioblastoma with numerous multinucleated giant cells, some being extremely bizarre with prominent nucleoli. Atypical mitoses and geographic necrosis also seen (H and E x 400).
B: Gliosarcoma showing a biphasic pattern exhibiting a mixture of gliomatous and sarcomatous tissues (H and E x 400).

One rare case of secretory meningioma (WHO grade I) was reported with histological features of focal epithelial differentiation and intracellular lumina with eosinophilic secretions. (Figure 5A) These structures, known as pseudopsammoma bodies show immunoreactivity for Carcinoembryonic antigen (CEA) while the surrounding tumour cells are both CEA and cytokeratin positive.[18]

Another uncommon tumour, rhabdoid meningioma was identified in the supratentorial location in a male aged 29 years. The tumour showed sheets of large cells with eccentric nuclei, open chromatin and abundant eosinophilic cytoplasm. (Figure 5B) Rhabdoid meningiomas often undergo an aggressive clinical course and correspond to WHO grade III. A minority of meningiomas with rhabdoid features shows this only focally and lacks other histological features of malignancy.[18]

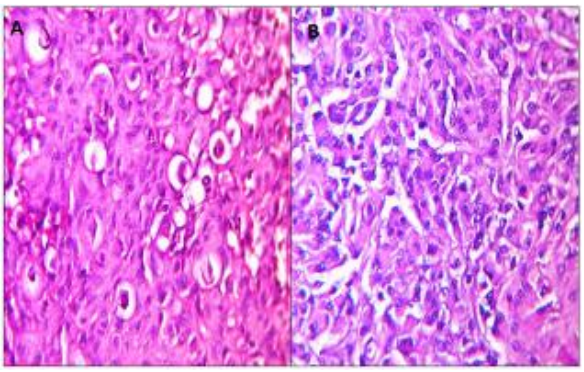

Fig. 5A & 5B

Fig. 5A: Secretory meningioma with focal epithelial differentiation and intracellular lumina with eosinophilic secretions (H and E x400).
B: Rhabdoid meningioma showing sheets of large cells with eccentric nuclei open chromatin and abundant eosinophilic cytoplasm. (H and E x400)

2 cases of meningeal hemangiopericytoma were reported in elderly males and in the parietal location. These tumours were highly vascular with slit like vascular channels and composed of closely packed round to oval monomorphic tumour cells with scant stroma. (Figure 6A) There was no necrosis or increased mitotic activity. Hemangiopericytoma corresponds histologically to WHO grade II tumours that are invariably solitary and attached to the cranial or spinal dura.[19]

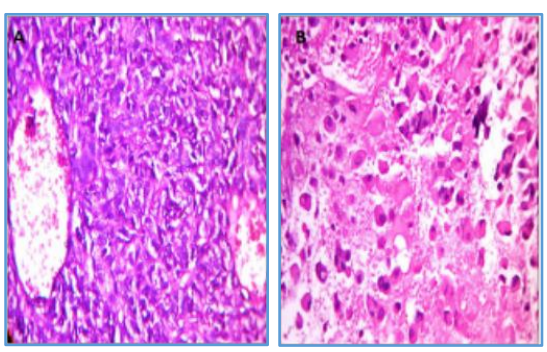

Fig. 6A & 6B

Fig. 6A: Meningeal hemangiopericytoma with scant stroma, slit like vascular channels and closely packed round to oval monomorphic tumour cells (H and E x100).
B: Atypical teratoid/rhabdoid tumour with nests and sheets of cells with classic rhabdoid features, smaller embryonic cells and spindle shaped cells. (H and E x400).

A rare case of a typical teratoid/rhabdoid tumour was diagnosed in a 12-year-old male child in the left parietal lobe. Microscopically the tumour was composed of nests and sheets of cells with classic rhabdoid features: eccentrically placed nuclei containing vesicular chromatin, prominent eosinophilic nucleoli and homogenous eosinophilic cytoplasmic inclusions. Smaller embryonic cells along with spindle shaped cells were seen. Extensive areas of geographic necrosis and haemorrhages were the prominent components of the tumour. Fig. 6B.

These tumours correspond to WHO grade IV and demonstrate a broad spectrum of immunohistochemical reactivity that is consistent with their histologic diversity.[20]

Two rare variants of medulloblastoma were reported. These were large cell medulloblastoma in a 13-year-old female child and desmoplastic medulloblastoma in a 4-year-old female child.

Large cell medulloblastoma showed monomorphic cells with large, round, vesicular nuclei, prominent nucleoli and variable amount of eosinophilic cytoplasm. Tumour exhibited high mitotic activity. (Figure 7 B)

Large cell medulloblastoma represents approximately 2–4% of medulloblastomas. Large cell and anaplastic medulloblastomas have considerable cytological overlap since large cell medulloblastoma frequently contains anaplastic regions, and in several studies, a combined large cell/anaplastic category has been proposed.[21]

Desmoplastic medulloblastoma was diagnosed by nodular zones ('pale islands') surrounded by densely packed, cells with hyperchromatic and moderately pleomorphic nuclei. (Figure 7 A)

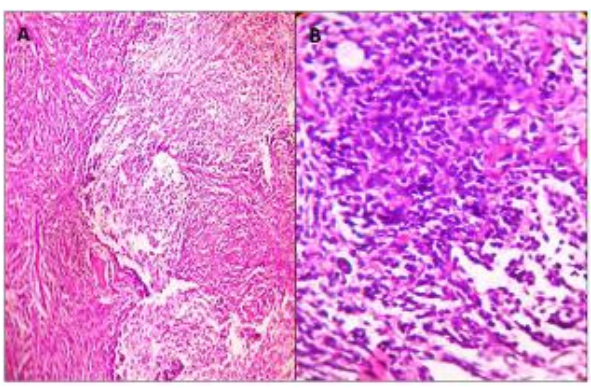

Fig. 7A & 7B

Fig. 7A: Desmoplastic medulloblastoma nodular pale zones surrounded by densely packed, cells with hyperchromatic and moderately pleomorphic nuclei. (H and E x100)
B: Large cell medulloblastoma showing monomorphic cells with large, round, vesicular nuclei, prominent nucleoli and eosinophilic cytoplasm. (H and E x100)

Medulloblastomas with extensive nodularity may resemble desmoplastic/nodular variant, but exhibit a more prominent lobular microarchitecture and elongated reticulin-free zones in fibrillary matrix.[22]

CONCLUSION: In this study, we discussed the incidence of the various histological patterns of tumours of the CNS including the spine, and emphasized the process of accurately establishing the diagnosis of central nervous system tumours including spinal tumours with the aid of morphology and IHC marker studies. 717 tumours were studied, the majority of tumours being meningiomas.

Grading of the tumours was done according to the revised World Health Organization criteria.

Age and sex incidence were discussed along with several rare tumours received in our department.

Our study showed that frontal location was the commonest site for tumours of CNS followed in decreasing order by tumours in cerebellopontine angle, suprasellar area and spine.

Immunohistochemistry was done only for selected cases and that aided in establishing a definitive diagnosis.

REFERENCES

1. Jalali R, Datta D. Prospective analysis of incidence of central nervous system tumours presenting in a tertiary cancer hospital from India. J Neurooncol 2008;87(1):111-114.
2. Madabhushi V, Venkata RI, Garikaparthi S, et al. Role of immunohistochemistry in diagnosis of brain tumours: a single institutional experience. J NTR Univ Health Sci 2015;4(2):103-111.
3. Das A, Chapman CAT, Yap WM. Histological subtypes of symptomatic central nervous system tumours in Singapore. J Neurol Neurosurg Psychiatry 2000;68(3):372-374.
4. Soo YL, Koo H, Kim TS, et al. Tumours of the central nervous system in Korea: a multicentre study of 3221 cases. J Neurooncol 2002;56(3):251-259.
5. Masoodi T, Gupta RK, Singh JP, et al. Pattern of central nervous system neoplasms: a study of 106 cases. JK-Practitioner 2012;17(4):42-46
6. Von Deimling A, Burger PC, Nakazato Y, et al. Diffuse astrocytoma. In: Louis DN, Ohgaki H, Wiestler OD, et al, ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:25-29.
7. Ahsan J, Hashmi SN, Muhammad I, et al. Spectrum of central nervous system tumours–a single center histopathological review of 761 cases over 5 years. J Ayub Med Coll Abbottabad 2015;27(1):81-84.
8. Ahmed Z, Muzzafar S, Kayani N, et al. Histological pattern of central nervous system neoplasms. J Pak Med Assoc 2001;51(4):154-157.
9. Kyu-Won J, Ha J, Lee SH, et al. An updated nationwide epidemiology of primary brain tumours in republic of Korea. Brain Tumour Res Treat 2013;1(1):16-23.
10. Perry A, Louis DN, Scheithauer BW, et al. Meningiomas. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the nervous system. Lyon: IARC 2007:164-172.
11. Rosenblum MK. Central nervous system. In: Rosai J, ed. Rosai and Ackerman's surgical pathology. 10th edn. St Louis: Mosby 2011:2307-2410.
12. Molitch ME, Elton RL, Blackwell RE, et al. Bromocriptine as primary therapy for prolactin-secreting macroadenoma: results of a prospective multicenter study. J Clin Endocrinol Metab 1985;60(4):698-705.
13. Kubota T, Kuroda E, Yamashima T, et al. Amyloid formation in prolactinoma. Arch Pathol Lab Med 1986;110:72-75.

14. Figarella-Branger D, Söylemezoglu F, Burger PC. Central neurocytoma and extraventricular neurocytoma. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:106-109
15. Becker AJ, Wiestler OD, Figarella-Branger Det al. Ganglioglioma and gangliocytoma. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:103-105.
16. Kleihues P, Burger PC, Rosenblum MK, et al. Anaplastic astrocytoma. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:30-32.
17. Kleihues P, Burger PC, Aldape KD, et al. Glioblastoma. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:33-49.
18. Perry A, Louis DN, Scheithauer BW, et al. Meningeal tumours. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:164-172.
19. Paulus W, Scheithauer BW, Perry A. Mesenchymal, non-meningothelial tumours. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:173-180
20. Judkins AR, Eberhart CG, Wesseling P. Atypical teratoid/rhabdoid tumour. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:147-149
21. Giangaspero F, Eberhart CG, Haapasalo H, et al. Medulloblastoma. In: Louis DN, Ohgaki H, Wiestler OD, et al. ed. WHO classification of tumours of the central nervous system. Lyon: IARC 2007:132-146.
22. Giangaspero F, Perilongo G, Fondelli MP, et al. Medulloblastoma with extensive nodularity: a variant with favorable prognosis. J Neurosurg 1999;91(6):971-977.