DOI: 10.21088/ijprp.2278.148X.15226.1

Indian Journal of Pathology: Research and Practice
Volume 15 Number 2, May - August 2026

ORIGINAL ARTICLE

Clinico-Pathological and Immunohistochemical Study of Primary CNS Tumors in a Tertiary Care Centre

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HOW TO CITE THIS ARTICLE:

Kausar Tasnim, Shagufta Qadri, Maheshwari Veena. Clinico-Pathological and Immunohistochemical Study of Primary CNS Tumors in a Tertiary Care Centre. Ind Jr of Path: Res and Practice 2026; 15(2) 101-110.

ABSTRACT

Background: Primary CNS tumors show wide clinical, histomorphological, and immunohistochemical variation. Distinguishing these lesions from each other and from reactive process can be challenging due to overlapping cell morphology. So, the diagnosis and categorization of primary CNS tumors, is essential for planning the management and to determine the prognosis.

Objectives: To evaluate the clinico-radiological features in diagnosing CNS tumors; to study the occurrence of CNS tumors in relation to age, sex, and anatomical sites, and the histopathological features of various CNS tumors and to evaluate the role of immunohistochemistry in diagnosing as well as classifying various CNS tumors according to CNS5.

Materials and Methods: This study included a total of 242 case' biopsy of primary CNS tumors received in the Department of Pathology over 5 years from 2020 to 2024. All specimens were examined grossly, processed routinely, stained with H&E, and evaluated using a panel of IHC markers (IDH1, ATRX, p53, Ki-67, GFAP, Synaptophysin, EMA, PR, etc.).

Results: Out of 242, the most common age group was 15-39 years with male: female ratio of 1.3:1 with most common site- cerebrum. Majority of the cases were non-malignant (61%), while 39% cases were malignant. The most frequent tumors were gliomas, followed by meningiomas, schwannoma, pituitary adenomas, and medulloblastomas. IHC significantly assisted in differentiating astrocytic tumors, subtyping meningiomas, and confirming embryonal tumors. Ki-67 index correlated with the tumor grade.

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➢ **Received:** 20-12-2025 ➢ **Accepted:** 25-01-2026

Conclusion: A combined clinico-pathological and IHC approach markedly improves diagnostic accuracy of primary CNS tumors.

KEYWORDS

• Gliomas • Immunohistochemistry • Medulloblastomas • Meningiomas • Pituitary adenomas • Schwannoma

INTRODUCTION

Central nervous system (CNS) tumors constitute less than 2% of all neoplasms but remain a major cause of cancer-related morbidity and mortality.[1] Globally they account for about 1.6% of cancers, with India reporting a slightly higher incidence of 2.4%.[2] Their clinical manifestations depend on location, size, and growth rate.[3,4] In adults, gliomas, meningiomas, nerve sheath tumors, and pituitary lesions form over 85% of CNS tumors, with glioblastoma being the most prevalent.[3]. Malignant CNS tumors (e.g., Gliomas, embryonal tumours, and germ cell tumours occur more frequently in children and adolescents than benign tumours (e.g., pituitary tumours).[5]

In India, improved neuroimaging and the establishment of hospital-based brain tumor registries have strengthened epidemiological data.[6] Diagnosis of CNS tumors relies on radiology, histopathology, and immunohistochemistry (IHC).[3] The WHO CNS 5 classification integrates histology with molecular profiling for precise diagnosis and prognostication.[7] However, many centers continue to depend mainly on histopathology and essential IHC markers due to limited molecular facilities. The purpose of this study is to evaluate primary CNS tumors through clinico-radiological, histopathological, and immunohistochemical correlation, thereby reinforcing the diagnostic value of markers such as IDH1, ATRX, p53, Ki-67, EMA, GFAP, BRAF, Synaptophysin, SSTR2A, and others in CNS tumor classification.

MATERIALS AND METHODS

This study comprised a 3-year retrospective and a 2-year prospective analysis of primary CNS tumors conducted from January 2020 to December 2024 in the Department of Pathology, Jawaharlal Nehru Medical College, AMU, Aligarh, evaluating a total of 242 cases. Patients of all age groups who underwent excisional surgery in the Department of Neurosurgery, JNMCH, AMU, for primary CNS tumors or tumor-associated symptoms were included and the exclusion criteria were patients not giving consent, having incomplete clinical data, or inadequate biopsy material. Patients with Bone tumors, metastasis, hematolymphoid and mesenchymal malignancies were also not included. Clinical profiles, which were obtained from neurosurgery department and histopathological examinations, along with immunohistochemical analyses, were utilized to establish definitive diagnoses. Most of IHCs used in this study were manufactured by PathnSitu Biotechnologies. The study focused on assessing the nature of the lesions (benign vs. malignant), anatomical site of involvement, and patient age and sex distribution. Tissue samples were obtained from fresh sections of paraffin-embedded blocks and stained with H&E, relevant special stains, and required immunostains. All cases were diagnosed and classified according to the WHO CNS Classification 2021. Ethical clearance from the institutional ethical committee of JNMCH, AMU, Aligarh was obtained for the present study.

RESULTS

The diagnosis was primarily established by integrating the presenting clinical symptoms, radiological findings, histopathological examination & IHC evaluation (wherever applied). Out of 242 cases, Majority were non-malignant, 148 cases (61%), whereas 94 (39%) cases were malignant. Adolescent and young adult age group affected most (46.28%) as shown in table 2. Male population was more commonly affected (138/242 male cases) with male female ratio of 1.3:1 except in meningioma were female preponderance (42/64 female cases, 67%) with male: female ratio of 1:1.9 seen. The most common site of involvement was cerebrum in 96 cases

(39.7%) as shown in table 3. The most common presenting complaint was neurological deficit in 151 (62.4%) cases. Gliomas, glioneuronal, and neuronal tumors (71 cases, 75.6%) were the most common malignant tumors, while among non-malignant tumors, meningioma Grade I predominated (56 cases, 37.8%). Within gliomas, astrocytoma was the most frequent (30 cases, 31.9%), followed by glioblastoma (15 cases) as shown in figure 1.

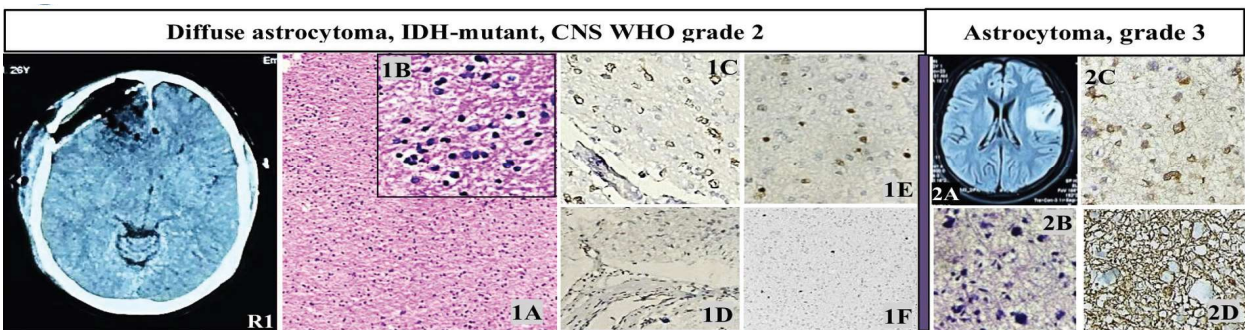

Figure 1: R1 MRI showed a hypodense, non-enhancing left frontal lesion with ill-defined margins and mild mass effect. Histomorphology (**Fig. 1A–1B**) showed increased cellularity with mild atypia of tumor cells in a dense fibrillary background (H&E10X & Inset 40X). IHC showed IDH mutation (**Fig. 1C**, 40X), ATRX loss (**Fig. 1D**,40X), p53 positivity (~10%) (**Fig. 1E**,10X), and a low Ki-67 index. (<5%) (**Fig. 1F**, 10X) in tumor cells.

Figure 2A: T2/FLAIR MRI shows a hyperintense lesion in the right frontoparietal white matter with irregular margins and perilesional edema, causing mild compression of adjacent sulci and ventricles, Histopathology reveals increased cellularity with nuclear atypia and mitotic activity (**Fig. 2B**, H&E, 40X), Tumor cells show IDH mutation (**Fig. 2C**, IHC, 40X) and diffuse cytoplasmic GFAP positivity (IHC, 40X) (**Fig. 2D**).

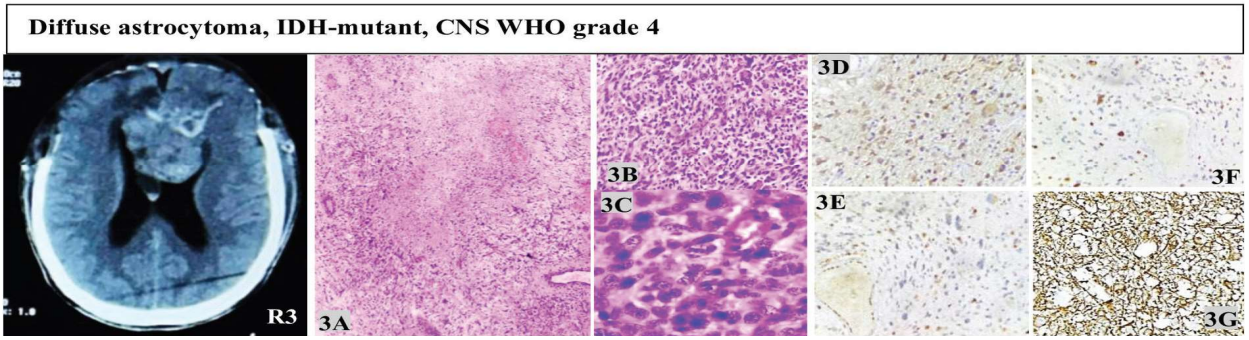

Figure 2: Fig. R3 MRI showed a heterogeneously enhancing fronto-parietal mass with central necrosis, vasogenic edema, and midline shift. Histomorphology (**Fig. 3A–3C**) showed diffusely infiltrating atypical cells with geographic necrosis, increased cellularity, microvascular proliferation, marked atypia, & mitoses. IHC showed IDH mutation (**Fig. 3D**), ATRX loss (**Fig. 3E**), p53 positivity in 30–40% of tumor cells (**Fig. 3F**) and diffuse GFAP positivity (**Fig. 3G**).

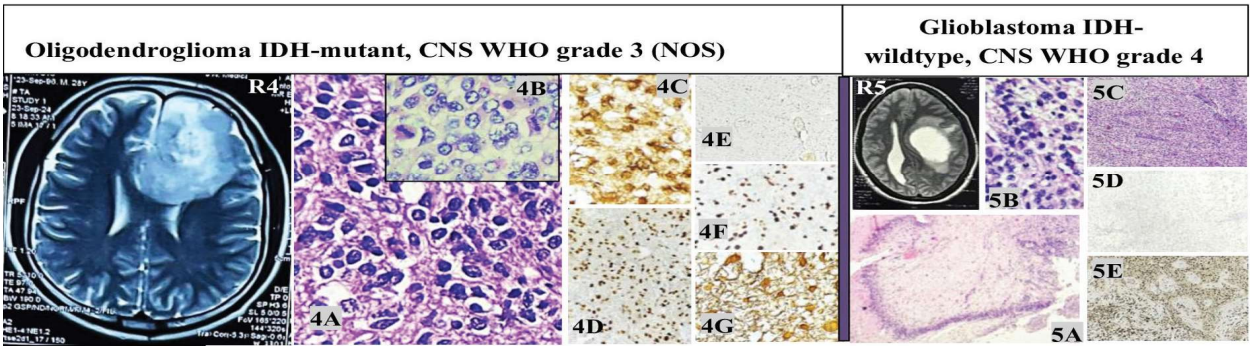

Figure 3: Fig. R4 MRI showed an ill-defined right frontal intra-axial mass with T2/FLAIR hyperintensity, cystic areas, vasogenic edema, and mild midline shift; histomorphology **(Fig. 4A&B,** H&E,10X) showed diffuse round cells with perinuclear halos and brisk mitoses (40X). IHC showed IDH-mutation (**Fig. 4C,** 40X), ATRX-retained (**Fig. 4D,** 40X), p53-negative (**Fig. 4E,** 10X), Ki-67 (30–40%, 10X) (**Fig. 4F,** 10X), and GFAP positivity (**Fig. 4G,** 40X) in tumor cells; **Figure R5** MRI showed an aggressive right frontoparietal mass with central necrosis, extensive edema, and marked midline

shift; histology (**Fig. 5A–5C**) shows palisading necrosis, atypia, and microvascular proliferation. IHC shows IDH-wild type (**Fig. 5D,** 10X) and ATRX retained (**Fig. 5E,** 40X).

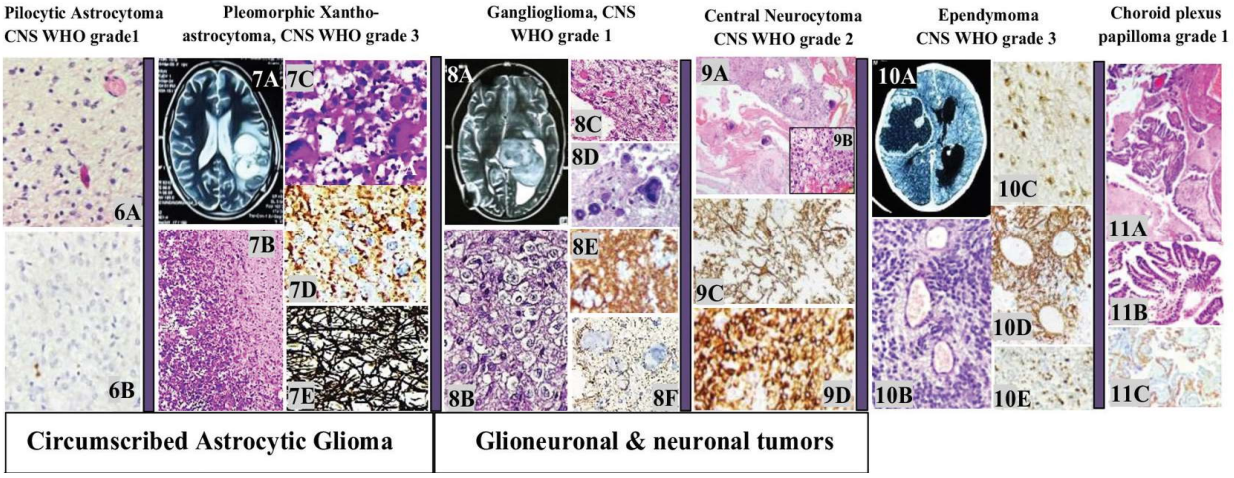

Figure 4: Figure 6A (H&E, 40X) showed moderate cellularity with piloid cells and Rosenthal fibers; **Fig. 6B** showed Ki-67 <2% (40X); **Fig. 7A** MRI showed a well-circumscribed cystic parietal lesion with an enhancing mural nodule. **Fig. 7B,** (H&E, 10X**)** showed peripheral invasion with pleomorphic cells with **(Fig. 7C** H&E, 40X**)** spindle/epithelioid cells, multinucleated astrocytes, and xanthomatous changes with BRAF V600E positivity **(Fig. 7D IHC-40X)** & rich reticulin network **(Fig. 7E,** 40X**)**; **Fig. 8A** MRI showed a large heterogeneous temporal solid-cystic mass with mass effect; **Fig. 8B** showed neuronal component (ganglion cells) within the tumor area (H&E, 40X). **Fig. 8C** showed glial component of tumor with eosinophilic granular bodies (H&E, 40X), and dystrophic calcification in ganglion cells (**Fig 8D,** H&E, 40X), IHC showed GFAP positivity in glial component (**Fig 8E**, IHC-40X) & synaptophysin positivity in neoplastic ganglion cells (**Fig. 8F**, IHC-40X); **Fig. 9A** Section shows uniform round cells, thickened and hyalinized blood vessels and psammomatous calcification (H&E, 10X) some cells showed perinuclear clearing and intratoumoral hemorrhage. (**Fig**. **9B** Inset H&E, 40X). **Fig. 9C** showed GFAP-positive reactive astrocytes (IHC, 40X) and synaptophysin-positive tumor cells **(Fig. 9D,** IHC 10X**)**; **Fig. 10A** MRI showed a well-defined lobulated parietal lobe lesion, heterogeneously T2-hyperintense with cystic areas, septations, mild edema, heterogeneous enhancement, and mass effect. **Fig. 10B** Histopathology showed pseudorosettes, true rosettes, with round-to-oval tumor nuclei (H&E, 40X) with Perinuclear dot/ ring-like EMA positivity (**Fig. 10C,** IHC-40X**)**, GFAP shows perivascular accentuation (**Fig. 10D,** IHC-40X**),** and the Ki-67 index >20% (**Fig. 10E**, IHC-40X) in tumor cells; **Figure 11A** Section shows papillary pattern composed of fibrovascular fronds (H&E, 10X) with single layer of cuboidal to columnar epithelial cells & round/ oval nuclei. (**Fig 11B** H&E, 40X) with GFAP positivity in reactive astrocytes or entrapped glial tissue. (**Fig 11C**, IHC-10X).

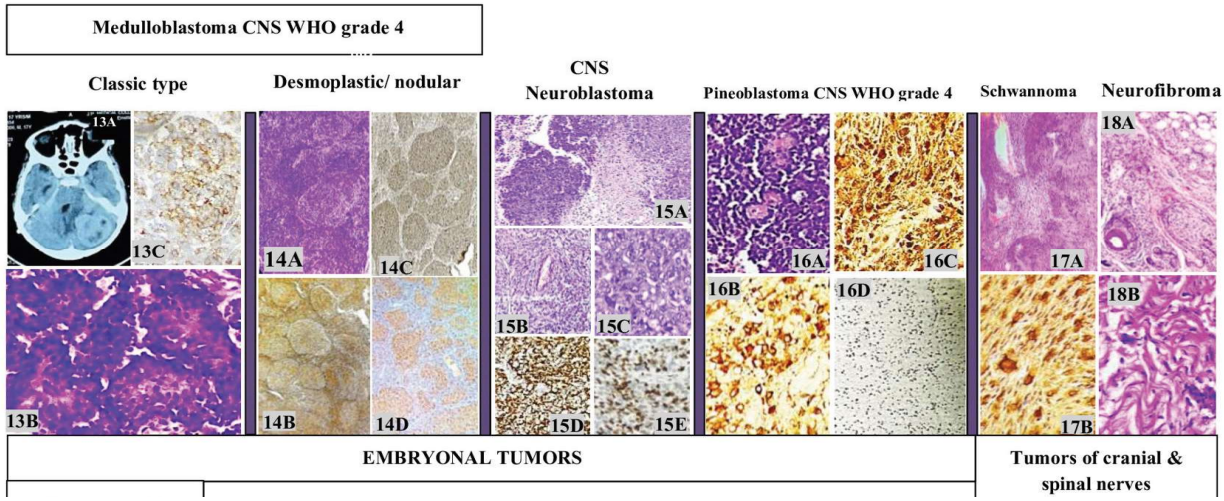

Figure 5: Fig. 13A MRI showed a hyperdense midline posterior fossa mass arising from the cerebellar vermis, compressing the 4th ventricle and causing obstructive hydrocephalus, with surrounding edema and heterogeneoenhancement; **Fig. 13B** showed small round blue cells with hyperchromatic nuclei and a high N:C ratio andHomer Wright rosettes with focal synaptophysin positivity **(Fig. 13C). Figure 14A** showed pale nodular areas with neuronal differentiation surrounded by densely packed hyperchromatic cells with reticulin-positive internodular regions **(Fig. 14B,** IHC-10X**)**, NSE +ve pale nodules **(Fig. 14C,** IHC-10X**)** and synaptophysin-positive nodules and negative internodular areas **(Fig.**

14D, IHC-10X**), Fig. 15A** showed sheets of poorly differentiated cells infiltrating adjacent CNS tissue (H&E, 4X). The tumor showed embryonal cells with a high N:C ratio and Homer Wright rosettes (**Fig. 15 B&C; H&E, 10X, 40X),** IHC showed synaptophysin positivity (**Fig. 15D)**, and Ki-67 index > 50% (**Fig. 15E). Fig. 16A** showed a highly cellular small-round cell tumor with a high N:C ratio and rosettes **(**H&E, 10X). Tumor cells were chromogranin-positive **(Fig.16B,** 10X), S100 positivity **(Fig. 16C)** and Ki-67 index of ~35% **(Fig. 16D). Figure 17A** showed Antoni A areas with Verocay bodies in adjacent Antoni B area (H&E, 10X). Tumor cells- S100 positive **(Fig. 17B; IHC, 40X). Figure 18 A&B** showed bland spindle cells with thin, wavy nuclei in a loose myxoid stroma with collagen **(H&E, 10X, 40X).**

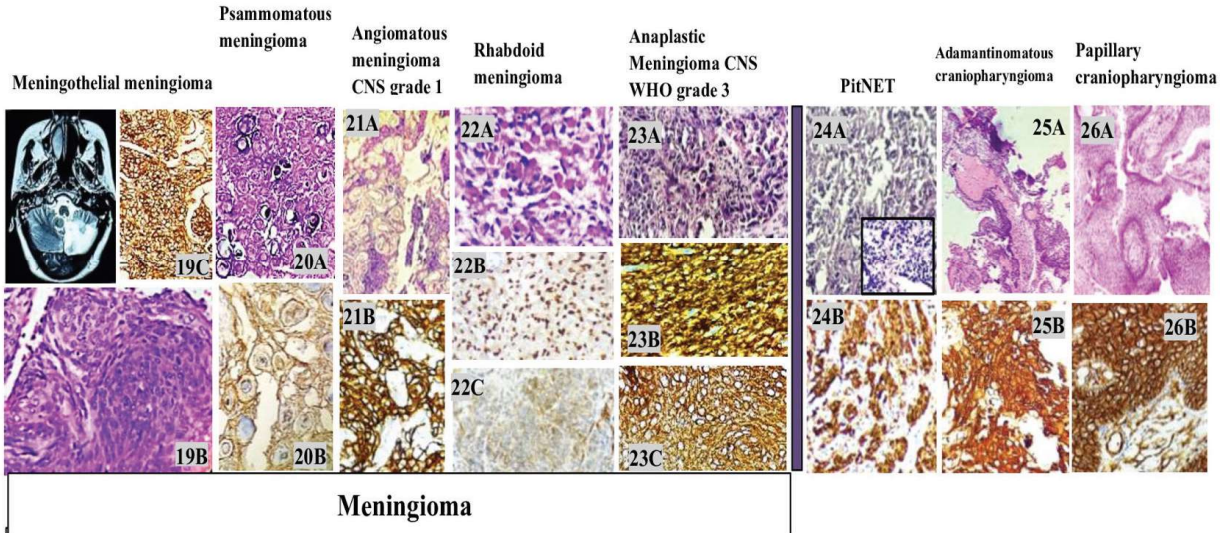

Figure 6: Fig. 19A showed a well-defined extra-axial CPA mass with dural attachment, compressing the cerebellum and brainstem, appearing iso- to hyperintense on T2 MRI. The tumor had syncytial meningothelial cells **(Fig. 19B,** 40X**)** with SSTR2A membranous and cytoplasmic positivity **(Fig. 19C, IHC,** 10X**); Fig. 20A** showed Meningioma replaced by psammomatous calcifications **(H&E,** 10X**)**, with EMA-positive cells seen between psammoma bodies **(Fig. 20, IHC,** 40X**); Fig. 21A** showed hyalinized small vessels with intervening meningioma cells and microcystic/metaplastic areas, and SSTR2A showed membranous and cytoplasmic positivity in tumor cells **(Fig. 21B,** IHC, 40X**); Fig. 22A** (H&E, 40X) showed plump cells with eccentric nuclei & eosinophilic paranuclear inclusions with PR showed nuclear positivity **(Fig. 22B,** IHC, 40X**),** and SSTR2A showed membranous & cytoplasmic positivity in tumor cells **(Fig. 22C,** IHC, 40X**); Fig. 23A** showed overtly malignant anaplasia resembling carcinoma or melanoma with ↑ mitotic activity (H&E, 40X) with SSTR2A positivity in tumor cells (**Fig. 23B,** IHC-40X) and Vimentin- positivity in tumor cells (IHC-10X) **Fig. 24A** showed monomorphic cells in sheets with bland nuclei & fine chromatin (H&E, 10X & H&E, 40X- inset) & **Fig. 24B** showed cytoplasmic synaptophysin positivity in tumor cells (IHC, 40X). **Fig. 25A** showed cords and trabeculae of cells with wet keratin, ghost cells, calcification, & basal palisading at the tumor–brain interface (H&E, 10X), while β-catenin showed nuclear positivity (**Fig. 25C**, IHC, 40X). **Fig. 26A** showed papillary structures lined by well-differentiated non-keratinizing squamous epithelium over fibrovascular cores with mixed inflammatory cells (H&E, 10X), & β-catenin showed cytoplasmic & membranous positivity (**Fig. 26C**, IHC, 40X).

Table 1: Distribution of 1° CNS tumors according to the histopathological and immunohistochemical diagnosis

Diagnostic Category	Cases	(%)
Adult Type Diffuse LGG (IHC not applied)	4	4.7
Adult Type Diffuse HGG (IHC not applied)	10	12
Astrocytoma	30	35.7
Oligodendroglioma	4	4.7
Glioblastoma	15	18.0
Paediatric Type Diffuse HGG	3	3.6
Circumscribed Astrocytic Glioma	6	7.1
Ependymoma	6	7.1
Glioneuronal & Neuronal Tumors	6	7.1
Total: Glioma, glioneuronal & neuronal tumours	84	100
Choroid Plexus Papilloma	2	0.8
Medulloblastoma	13	5.4
CNS neuroblastoma	1	0.4
Pineoblastoma	1	0.4
Schwannoma	36	14.9
Neurofibroma	2	0.8
Meningioma	64	26.4
PitNET	29	12
Craniopharyngioma	10	4.1
Total	242	100

In MRI-large irregularly marginated lesion with T1-hypointense and T2 FLAIR mismatch

seen in astrocytoma IDH mutant whereas rim enhancement around central necrosis with peritumoral edema was seen in astrocytoma grade 4 as well as in glioblastoma. Per operatively they were found easily suckable. Immunohistochemical markers including IDH, ATRX, p53, Ki-67, and GFAP were used for differentiating astrocytoma, oligodendroglioma, and glioblastoma.

Among circumscribed astrocytic gliomas, 5 cases of pilocytic astrocytoma presented with neurological deficit and 1 case of PXA presented with multiple episodes of seizures. MRI showing well circumscribed masses which were identified histomorphologically and using GFAP, S100, BRAF, CD34 and reticulin stain.

Six cases of Glioneuronal and neuronal tumors were found in this study-3 cases of ganglioglioma with mixed solid and cystic component on MRI, and 3 cases of Central neurocytoma which were confirmed by synaptophysin positivity, NSE positivity, EMA negativity, Ki-67 < 1% and IDH 1 negativity.

6 cases of ependymoma with imaging showing well-defined solid-cystic lesions with heterogeneous T2 signals and contrast enhancement. Histology revealed round to oval cells forming perivascular pseudorosettes. IHC showed EMA paranuclear dot-like positivity (except in 1 case of myxopapillary ependymoma), GFAP positivity with perivascular accentuation, and variable Ki-67.

Table 2: Age wise distribution of different histo-pathological diagnosis

Histopathological Diagnosis	**0-14 Years (paediatric age)**		**15-39 Years (adolescent and young adult age)**		**40-50 Years (Older adults)**		**51-70 Years (Older adults)**	
	Cases	**(%)**	**Cases**	**(%)**	**Cases**	**(%)**	**Cases**	**(%)**
Adult Type Diffuse LGG	-	-	4	3.6%	-	-	-	-
Adult Type Diffuse HGG	-	-	5	4.5%	2	3.7%	2	4.0%
Astrocytoma	-	-	19	17.0%	3	5.6%	9	18.0%
Oligodendroglioma	-	-	2	1.8%	2	3.7%	-	-
Glioblastoma	-	-	5	4.5%	6	11.1%	4	8.0%
Paediatric Type Diffuse HGG	3	11.5%	-	-	-	-	-	-
Circumscribed Astrocytic Glioma	3	11.5%	3	2.7%	-	-	-	-
Ependymoma	1	3.8%	2	1.8%	2	3.7%	1	2.0%
Glioneuronal & Neuronal Tumors	1	3.8%	4	3.6%	1	1.9%	-	-
Total: Glioma, glioneuronal & neuronal tumours	8	30.8%	44	39.5%	16	30%	16	30%
Choroid Plexus Papilloma	-	-	2	1.8%	-	-	-	-
Medulloblastoma	8	30.8%	5	4.5%		-	-	-
CNS neuroblastoma	-	-	1	0.9%	-	-	-	-
Pineoblastoma	1	3.8%	-	-	-	-	-	-
Schwannoma	-	-	20	17.9%	7	13.0%	9	18.0%
Neurofibroma	-	-	2	1.8%	-	-	-	-
Meningioma	2	7.7%	21	18.8%	22	40.7%	19	38.0%
PitNET	-	-	14	12.5%	9	16.7%	6	12.0%
Craniopharyngioma	7	26.9%	3	2.7%	-	-	-	-
Total	26	100%	112	100%	54	100%	50	100%

Table 3: Distribution of 1° CNS tumors according to radiological site

Radiological Site	Cases	(%)
Cerebrum	96	39.67
Frontal	26	10.74
Parietal	16	6.61
Temporal	7	2.89
Occipital	2	0.83
Fronto-Parietal	20	8.26
Temporo-Parietal	13	5.37
Parieto- Occipital	6	2.48
Insular	6	2.48
Brainstem & Thalamus	5	2.07
Pineal region	2	0.83
Sellar-Suprasellar Region	42	17.36
Cerebellum	4	1.65
Ventricle	10	4.13
Posterior Fossa	10	4.13
CP Angle	17	7.02
Olfactory Groove	3	1.24
Sphenoid Wing	4	1.65
Inter-Hemisphere Fissure	2	0.83
Para-Sagittal	2	0.83
Spine	42	17.36
Paraspinal	3	1.24
Total	**242**	**100**

2 Cases of choroid plexus papilloma CNS WHO grade 1, presented with headache and vomiting. MRI showed site of involvement-lateral ventricle and 3rd ventricle respectively. IHC showed Pan-CK positivity, weak EMA positivity and GFAP positivity in reactive astrocytes or entrapped glial tissue.

Of 15 embryonal tumors (all CNS WHO grade 4), 13 were medulloblastomas diagnosed with the aid of synaptophysin, NSE, chromogranin, S100, and Ki-67. One CNS neuroblastoma (20-year-old male) showed a frontal mass on CT imaging, with synaptophysin, S100, vimentin positivity and high Ki-67 (30–40%). One pineoblastoma in an 11-year-old female presented with headache, diminution of vision, diplopic seizure showed a pineal gland lesion on MRI.

In 38 cranial and paraspinal nerve tumors, 36 cases of schwannoma (confirmed by S100 and vimentin positivity) and 2 cases of neurofibroma were studied.

In 64 cases of meningioma, grade 1 tumors were most commonly seen (87.5%) in which meningothelial meningioma predominated (29 cases, 45.3%). SSTR2A, EMA, Vimentin, PR, and Ki-67 were used for confirmation and diagnosis.

29 cases of pituitary adenoma (PitNET) were studied and confirmed by synaptophysin & chromogranin. Lastly, Craniopharyngioma, 10 cases were studied and subtyping was done by using β-catenin. The adamantinomatous type showed nuclear positivity while papillary type showed membranous positivity.

DISCUSSION

The classification of CNS tumors has evolved markedly from 1979 to 2021 with adding immunohistochemical and molecular markers. Hence, this study documents the spectrum of CNS tumors according to the latest WHO 2021 classification. In our study, out of a total 242 cases, gliomas were the most common tumor type (78 cases, 31.8%), followed by meningiomas (64 cases, 26.4%), schwannoma-36 cases (14.9%) and pituitary neuro-endocrine tumors (PitNET) - 29 cases (12%). Other less frequent tumors included medulloblastoma, 13 cases (5.4%), craniopharyngioma, 10 cases (4.1%), and glioneuronal tumors, 6 cases (2.5%). This study was comparable with the studies conducted by **Meel** *et al.* **(2021)**[8] and **Price et al, (2024).**[9] The common age group affected was 15-39 years (AYA age group) that included 112 cases (46%), followed by 40–50 years (54 cases, 22%) and 51–70 years (50 cases, 21%) which was supported by **Meel** *et al.*, **(2021).**[8] and **Jaiswal et. al. (2016).**[6] In pediatric age group the most common 1° CNS tumors were glioma, glioneuronal & neuronal tumors (30.8%), medulloblastoma (30.7%), and craniopharyngioma (26.9%). This finding was agreed with studies by **Qadri** *et al.* **(2017)** [10] and **Meel** *et al.*, **(2021).**[8] Our study found CNS lesions to be more common in males, except meningiomas, which showed female predominance—consistent with findings by **Thambi** *et al.* **(2017)**[11] and **Jaiswal** *et al.* **(2016.**[6] The most common presenting complaint was neurological deficit (151 cases, 62.4%) followed by headache (133 cases, 55%), and seizure

(39 cases, 16.1%) which is corraborated with the study of **Ozawa et. al., (2019).**[12] In this study, the cerebrum was the most frequently involved radiological site (in 96 cases, 39.7%), followed by spinal (45 cases, 18.5%) and the sellar–suprasellar lesions (42 cases, 17.4%) which closely correlates with the findings of **Price** *et al.* **(2024).**[9]

Table 4: Comparison of diagnostic classification of primary CNS tumors between WHO CNS5 and WHO CNS4

Diagnosis with grade (WHO CNS5, 2021)	Previous name (WHO CNS4, (2016)	Present study (2025)	Price et. al. (2024)[23]
Diffuse astrocytoma			
Astrocytoma, IDH-mutant CNS WHO grade 2	Diffuse astrocytoma, IDH-mutant	12 (24.4%)	8.9 %
Astrocytoma, IDH-mutant CNS WHO grade 3	Anaplastic astrocytoma, IDH-mutant	6 (12.2%)	7.2%
Astrocytoma, IDH-mutant CNS WHO grade 4	Glioblastoma, IDH-mutant	12 (24.4%)	1.41%
Oligodendroglioma			
Oligodendroglioma IDH-mutant, CNS WHO grade 2	Oligodendroglioma IDH-mutant	2 (4.1%)	4.2%
Oligodendroglioma IDH-mutant, CNS WHO grade 3	Anaplastic oligodendroglioma IDH-mutant	2 (4.1%)	2.1%
Glioblastoma			
Glioblastoma, IDH-wildtype, CNS WHO grade 4	Glioblastoma, IDH-wildtype	15 (30.6%)	47.1%

In this study, IHC application of IDH1R132H, categorized the diffuse gliomas according to WHO CNS5 and was compared by study of **Price et. al., (2024).**[9] Astrocytoma, IDH-mutant Grade 4 (15.6% cases), reflecting the major WHO CNS5 change that reclassified many tumors previously labelled as glioblastoma, IDH-mutant. This shift also explains why our rate of glioblastoma, IDH-wildtype (19.5%) is much lower than theirs (Table 4). These molecular updates altered the incidence of different tumor types which was fruitful in prognostication as Grade 4 IDH-mutant astrocytomas usually have a better clinical outcome than IDH-wildtype glioblastomas.

Six cases of Glioneuronal and neuronal tumors were found in our study - 3 cases of ganglioglioma and 3 cases of Central neurocytoma. Their findings were compatible with **Jaiswal et. al., (2016)**.[6]

6 Ependymoma cases were identified: 3 supratentorial, and one each in the posterior fossa, spine, and a myxopapillary subtype. Our distribution differed **from Basu** *et al.* **(2024),**[13] who reported highest frequencies in the posterior fossa.

2 Cases of choroid plexus papilloma were found in this study & their finding were comparable with the study of **Anghileri et. Al., (2025).**[14]

13 cases of Medulloblastoma CNS WHO grade 4, observed in this study with classic (7 cases), desmoplastic/ nodular (3 cases), medulloblastoma with extensive nodularity- 2 Cases) and large cell/ anaplastic medulloblastoma (1 case). Their findings were agreed with **Kool** *et al.* **(2012).**[15] One case of CNS Neuroblastoma studied and its finding were comparable with **Bianchi** *et al.* **(2018).**[16] One case of pineoblastoma studied whose finding were agreed with **Jaju et. al., (2019).**[17]

In this study 64 meningiomas cases (26.4%) in which, a clear female predominance and peak incidence in the 40–50-year age group. The cohort included meningothelial (29 cases), psammomatous (3 cases), angiomatous (1case) – all Grade 1; chordoid (2 case) – Grade 2; and rhabdoid (2 cases), atypical (3 cases), and anaplastic (1case) – all Grade 3. These findings were aligned with the study of **Price** *et al.*, **2024**.[9]

In tumors of sellar and suprasellar region, this study included- pituitary adenoma (29 cases) and craniopharyngioma CNS WHO grade 1(10 cases) with Adamantinomatous craniopharyngioma (8 cases) and papillary craniopharyngioma (2 cases) whose findings were agreed with **Saeger** *et al.*, **(2007).**[18]

CONCLUSION

This study delineates the broad spectrum of neoplastic and non-neoplastic primary CNS lesions and outlines the relative frequency of various CNS malignancies encountered at a tertiary care center in North India. Neoplastic

lesions were observed to be more common than non-neoplastic entities, with the majority occurring in the adolescent and older age groups. An overall male predominance was noted, with meningioma being the sole exception, showing a female preponderance. Among neoplastic lesions, gliomas emerged as the most frequent tumor. Additionally, several uncommon and noteworthy cases, including pleomorphic xanthoastrocytoma CNS WHO grade 3, central neurocytoma CNS WHO grade 2 (NOS), myxopapillary ependymoma CNS WHO grade 2, choroid plexus papilloma CNS WHO grade 1, desmoplastic nodular medulloblastoma, medulloblastoma with extensive nodularity, Large cell /Anaplastic Medulloblastoma CNS WHO grade 4, CNS neuroblastoma CNS WHO grade 4 (NOS), pineoblastoma, CNS WHO grade 4 angiomatous meningioma CNS WHO grade 1, Chordoid meningioma CNS WHO grade 2, Rhabdoid meningioma CNS WHO grade 3 and Anaplastic meningioma CNS WHO grade 3 were identified in this series. The integration of clinical presentation, radiology, histopathology, and surrogate IHC markers significantly improves diagnosis and grading of primary CNS tumors.

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