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ORIGINAL ARTICLE

SOX10 immunohistochemical stain in differential diagnosis of posterior fossa tumors, a single centered study.

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ABSTRACT… Objective: To evaluate the diagnostic utility of SOX10 immunohistochemical staining in distinguishing posterior fossa tumors, particularly ependymomas and non-ependymomas. **Study Design:** Cross-sectional study. **Setting:** Department of Histopathology, Shaukat Khanam Memorial Cancer Hospital and Research Center, Lahore. **Period:** March 2025 to August 2025. **Methods:** This cross sectional study included 65 posterior fossa tumors. 33 ependymomas and 32 non-ependymomas: pilocytic astrocytoma, medulloblastoma, meningioma, diffuse midline glioma,andhemangioblastoma. Immunohistochemistry was performed using SOX10, OLIG2, GFAP, synaptophysin, SSTR2 and inhibin. Ependymomas were molecularly subclassified into PF-EPN-A and PF-EPN-B. Statistical significance was assessed using chi-square and Fisher's exact tests ($p < 0.05$). **Results:** PF-EPN-A was the most common ependymoma subtype (81.8%), with older median age than PF-EPN-B (5.0 vs. 2.5 years; $p = 0.031$). SOX10 was negative in all ependymomas (0/33) but highly expressed in pilocyticastrocytomas (85%) and the single diffuse midline glioma (100%), with significantly higher positivity in non-ependymomas (56.3%; $p < 0.001$). Synaptophysin was uniformly positive in medulloblastomas (100%). **Conclusion:** SOX10 is a sensitive and specific marker for distinguishing pilocytic astrocytoma from ependymoma. Its routine use in diagnostic panels enhances accuracy, reduces interpretive uncertainty and supports improved clinical decision-making in pediatric neuro-oncology.

Key words: Ependymoma, Immunohistochemistry, Posterior Fossa Tumor, Pilocytic Astrocytoma, Pediatric Neuro-Oncology, SOX10.

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INTRODUCTION

SOX10 (SRY-related HMG-box gene 10) is a transcription factor that plays an important role in the development and differentiation of neural crest cells, oligodendrocytes and Schwann cells.[1] It is essential for myelination and maintenance of glial lineage identity. SOX10 expression is typically seen in oligodendrocytes, Schwann cells, melanocytes and their neoplastic counterparts.

Posterior fossa tumors represent a heterogeneous group of neoplasms that arise in the infratentorial compartment of the brain. These tumors represent a significant proportion of central nervous system (CNS) tumors, particularly in children. These tumors account for approximately 50–60% of all childhood brain tumors and around 20–30% of adult intracranial neoplasms.[2] In such cases, immunohistochemistry (IHC) emerges as an importantfactor that makes tumor classification and differentiation accurate.[3]

Among the emerging immunohistochemical markers, SOX10 has gained increasing attention for its diagnostic utility in CNS tumors, especiallly glial and neural crest origin.[4] In case of posterior fossa tumors, SOX10 represents significant diagnostic potential, particularly in differentiating ependymomas and astrocytic tumors from embryonal tumors.[5]

Diffuse midline glioma (DMG), H3 K27me3 altered, represents a highly aggressive and infiltrative glioma that predominantly affects children and young adults. These tumors are commonly found in midline structures including the pons, thalamus and spinal cord.[6]

Histologically, diffuse midline gliomas show infiltrative growth patterns with moderate to high cellularity, nuclear atypia and variable mitotic activity.

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However, morphological features are often confused with other astrocytic tumors particularly pilocytic astrocytoma and high-grade gliomas.[7] SOX10 immunostaining can help in confirming glial differentiation and distinguishing these tumors from embryonal and metastatic neoplasms.[8] Ependymoma is one of the most common posterior fossa tumors in children. It arises from ependymal cells lining the ventricular system. The fourth ventricle is the most frequent site of origin.[9]

Differential diagnosis often includes pilocytic astrocytoma, medulloblastoma, diffuse gliomas and choroid plexus tumors. Immunohistochemically, ependymomas typically express GFAP, epithelial membrane antigen (EMA) in a dot-like patternand S100 protein.[10] Histologically, pilocytic astrocytoma is characterized by a biphasic pattern that consist of compact areas with Rosenthal fibers and loose microcystic regions with eosinophilic granular bodies.[11]

This resultsin confusion with diffuse gliomas and ependymomas.[12] Immunohistochemical markers such as GFAP, Olig2 and SOX10 are helpful in establishing astrocytic differentiation. SOX10 positivity in pilocytic astrocytoma supports its glial origin and aids in distinguishing it from embryonal tumors and vascular neoplasms.[13] The differential diagnosis of medulloblastoma includes other small round blue cell tumors such as atypical teratoid/ rhabdoid tumor, lymphoma and high-grade gliomas. Medulloblastomas typically express synaptophysin and neuron-specific enolase, while being negative for GFAP and SOX10. The absence of SOX10 staining serves as an important discriminator from glial tumors and ependymomas.[14]

METHODS

Cross sectional study was conducted in the Department of Histopathology at Shaukatkhanam memorial cancer Hospital and research center, Lahore in duration of 6 months (March 2025 to August 2025). Approval was obtained from the Institutional Review Board (EX-17-01-25-02 dated March 06, 2025) of Shaukat Khanam Memorial Cancer Hospital and Research Center, Lahore. A total of 65 histologically confirmed posterior fossa tumor cases were included in the study. Inclusion Criteria: All surgically resected posterior fossa tumors with adequate tissue available for histopathological and immunohistochemical evaluation. Histologically confirmed cases of Ependymoma, Pilocytic astrocytoma, Medulloblastoma, Diffuse midline glioma, Meningioma and Hemangio-blastoma. Exclusion Criteria: Inadequate tissue samples, poorly preserved specimens precluding reliable immunohistochemical interpretation, recurrent tumors and post-treatment specimens and cases lacking essential clinical information. Clinical and demographic data was taken from hospital medical records and pathology reports. Age distribution ranged from 1 to 50 years with most cases included children. The median age was 5.0 years for ependymomas and 7.0 years for non-ependymoma tumors. All specimens were fixed in 10% neutral buffered formalin, processed routinely and embedded in paraffin wax. Sections of 3–4 μm thickness were prepared and stained with hematoxylin and eosin (H&E) for histopathological evaluation. Tumors were classified and graded. Histological parameters including tumor cellularity, nuclear atypia, mitotic activity, necrosis, microvascular proliferation, presence of rosettes, pseudorosettes, biphasic architecture and stromal vascularity were noted. Immunostaining was performed on 3–4 μm thick FFPE sections using an automated immunostainer. Sections were then incubated with primary antibodies according to dilutions and incubation times. This was followed by visualization with 3,3′-diaminobenzidine (DAB) chromogen. Hematoxylin was used for nuclear counterstaining. Appropriate positive and negative controls were included with each staining run to ensure quality assurance.

Panel of Antibodies

Immunohistochemistry (IHC) was performed using the following panel of antibodies:

Marker	Purpose
SOX10	Glial / neural crest differentiation
OLIG2	Oligodendroglial lineage
GFAP	Astroglial differentiation
Synaptophysin	Neuronal / neuroectodermal differentiation
SSTR2	Meningioma marker
Inhibin	Hemangioblastoma marker

Immunohistochemical expression was evaluated independently by two experienced histopathologists. Nuclear staining for SOX10 and OLIG2, cytoplasmic staining for GFAP and synaptophysin, membranous staining for SSTR2 and cytoplasmic staining for inhibin were recorded. Staining intensity and distribution were graded as:

- **Negative:** <5% tumor cells
- **Focal positive:** 5–25%
- **Moderate positive:** 26–50%
- **Diffuse positive:** >50%

Statistical analysis was performed using SPSS version 2. Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR) while categorical variables were expressed as frequencies and percentages. Comparisons between categorical variables were conducted using the Chi-square test or Fisher's exact test. Continuous variables were compared using the Student's t-test or Mann–Whitney U test, depending on data distribution. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 65 posterior fossa tumors were analyzed, comprising 33 ependymomas and 32 non-ependymoma neoplasms.

Among ependymomas, PF-EPN-A was the predominant subtype (27/33, 81.8%), followed by PF-EPN-B (6/33, 18.2%).

Immuno-histochemical analysis showed OLIG2 positivity in pilocytic-astrocytomas (100%) and in the single case of diffuse midline glioma (100%), with complete absence in medullo-blastoma, meningioma, and hemangio-blastoma ($p < 0.001$).

Pilocyticastrocytomas showed strong OLIG2 (100%) and SOX10 (85%) positivity ($p < 0.001$), while medulloblastomas demonstrated uniform synaptophysin expression (100%). Meningiomas and hemangioblastomas displayed selective SSTR2 and inhibin positivity.

Age distribution analysis demonstrated a pediatric predominance, with 90.8% of all posterior fossa tumors occurring in patients <18 years (median age 6.0 years).

SOX10 demonstrated high positivity in pilocyticastrocytomas (85%) and the single diffuse midline glioma case (100%), resulting in a significantly higher expression rate among non-ependymoma tumors overall (56.3%; $p < 0.001$). All medulloblastomas, meningiomas and hemangioblastomas remained SOX10-negative.

TABLE-I

Overall tumor distribution and demographics

Diagnostic Category	Subtype	Cases (n)	% of Total	Age Range (years)	Median Age (years)	Male	Female
Ependymoma	PF-EPN-A	27	41.5%	1–17	5.0	18	9
	PF-EPN-B	6	9.2%	1–5	2.5	3	3
	TOTAL	**33**	**50.8%**	**1–17**	**5.0**	**21**	**12**
Non-Ependymoma	Pilocytic Astrocytoma	20	30.8%	2–50	7.0	11	9
	Medulloblastoma	7	10.8%	2–36	4.0	5	2
	Meningioma	2	3.1%	15–17	16.0	1	1
	Diffuse Midline Glioma	1	1.5%	39	39.0	1	0
	Hemangioblastoma	2	3.1%	17–35	26.0	0	2
	TOTAL	**32**	**49.2%**	**2–50**	**7.0**	**18**	**14**
G. TOTAL	—	**65**	**100.0%**	**1–50**	**6.0**	**39**	**26**

TABLE-II

Immuno-histochemical Profile Comparison

Marker	Pilocytic Astrocytoma (n=20)	Medulloblastoma (n=7)	Meningioma (n=2)	DGM (n=1)	Hemangioblastoma (n=2)	P-Value*
OLIG2	20 (100%)	_	_	1 (100%)	_	<0.001
SOX10	17 (85.0%)	0 (0%)	0 (0%)	1 (100%)	0 (0%)	<0.001
Synaptophysin	--	7/7 (100%)	_	_	_	--
SSTR2	--	--	2/2 (100%)	_	_	--
Inhibin	--	--	_	_	2/2 (100%)	--

TABLE-III

Diagnostic immunophenotypic signatures

Tumor Type	Characteristic IHC Profile	Diagnostic Utility	Key Discriminator vs. Ependymoma
Ependymoma	GFAP+ / OLIG2– / SOX10–	Gold standard signature	Reference category
Pilocytic Astrocytoma	OLIG2+ / SOX10+ (85%) / GFAP+	High sensitivity for glial tumors	OLIG2+ (100% vs. 0%; p<0.001)
Medulloblastoma	Synaptophysin+ (100%) / OLIG2– / SOX10–	Neuroblastic differentiation	Synaptophysin+ (100% vs. not expressed)
Meningioma	SSTR2+ (100%) / OLIG2– / SOX10–	Membranous SSTR2 positivity	SSTR2+ (limited data)
Hemangioblastoma	Inhibin+ (100%) / OLIG2– / SOX10–	Stromal cell marker	Inhibin+ (limited data)

TABLE-IV

Age distribution across diagnostic categories

Diagnostic Category	n	Median Age (years)	Pediatric (<18 yrs)	Adult (≥18 yrs)	P-Value*
Ependymoma (Total)	33	5.0	33 (100%)	0 (0%)	—
PF-EPN-A	27	5.0	27 (100%)	0 (0%)	0.031
PF-EPN-B	6	2.5	6 (100%)	0 (0%)	—
Non-Ependymoma (Total)	32	7.0	26 (81.3%)	6 (18.8%)	—
Pilocytic Astrocytoma	20	7.0	17 (85.0%)	3 (15.0%)	—
Medulloblastoma	7	4.0	6 (85.7%)	1 (14.3%)	—
Other rare tumors	5	26.0	2 (40.0%)	3 (60.0%)	—
GRAND TOTAL	65	6.0	59 (90.8%)	6 (9.2%)	—

TABLE-V

SOX10 Expression Pattern across All Tumor Types

Tumor Type	Total Cases	SOX10 Positive	SOX10 Negative	Positivity Rate	p-value vs. Ependymoma
Ependymoma (PF-EPN-A)	27	0	27	0.0%	__
Ependymoma (PF-EPN-B)	6	0	6	0.0%	1.000
All Ependymomas	33	0	33	0.0%	—
Pilocytic Astrocytoma	20	17	3	85.0%	<0.001
Medulloblastoma	7	0	7	0.0%	1.000
Meningioma	2	0	2	0.0%	1.000
Diffuse Midline Glioma	1	1	0	100.0%	0.317
Hemangioblastoma	2	0	2	0.0%	1.000
Non-Ependymomas	32	18	14	56.3%	<0.001

IMMUNOHISTOCHEMICAL STAINING

FIGURE-1

Ependymoma and negative SOX10Immunostain in ependymoma
(a) Hematoxylin and eosin (H&E) stainedsection of posterior fossa ependymoma demonstrating a highly cellular tumor with uniform round to oval nuclei and characteristic perivascular pseudorosette formation.
(b) Immunohistochemical (IHC) stain of SOX10showing complete absence of nuclear immunoreactivity in tumor cells.
(c)H&E section highlighting the typical architectural features of ependymoma including perivascular pseudorosettes and a fibrillary background.
(d) Corresponding SOX10 stain again showing diffuse negativity.

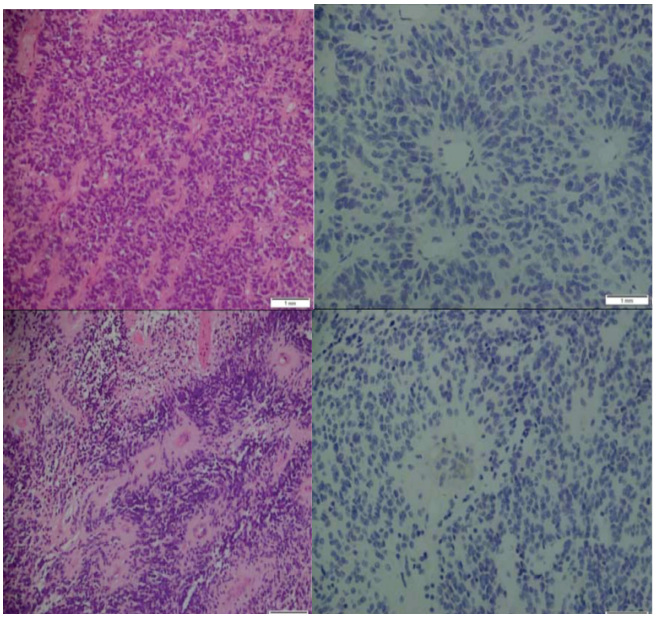


FIGURE-2

Pilocytic astrocytoma and Positive SOX10 immunostain in pilocytic astrocytoma
(a,c) H&E section of posterior fossa pilocytic astrocytoma demonstrating a moderately cellular tumor composed of bipolar astrocytic cells within a loose microcystic background. (b,d) Strong positive SOX10 immunohistochemical stain.

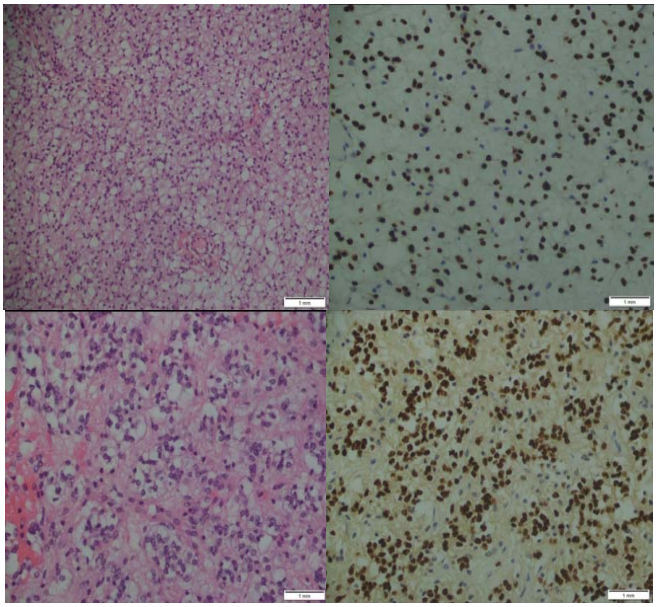


FIGURE-3

Medulloblastoma and Negative SOX 10Immunostainin medulloblastoma
(a) H&E stain shows histomorphology of medulloblastoma. The tissue demonstrates the typical features of medulloblastoma including uniform cell population with occasional mitotic figures.
(b) IHC section demonstrates complete negativity for SOX10 in medulloblastoma. The absence of brown nuclear staining confirms that medulloblastomas consistently lack SOX10 expression.

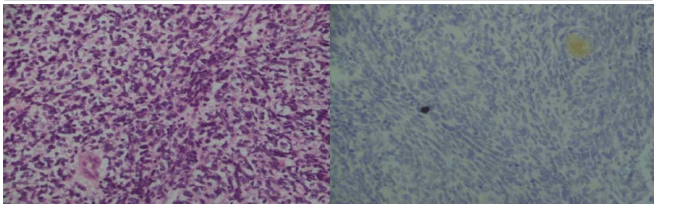


FIGURE-4

Diffuse Midline Glioma and positive SOX10 in DMG
(a)(H&E) stain showing the characteristic histomorphology of diffuse midline glioma including moderate cellularity with occasional mitotic figures and a fibrillary background. (b) IHC section demonstrates strong nuclear positivity for SOX10 (indicated by brown staining) throughout the tumor cells. This pattern of diffuse nuclear staining is characteristic of SOX10 expression in glial tumors.

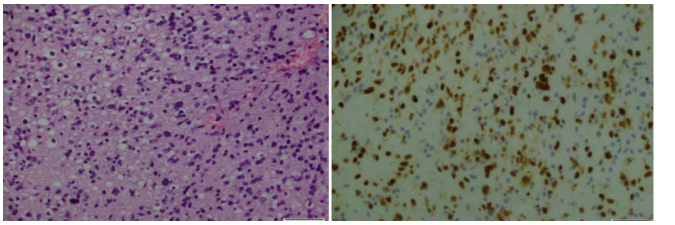


FIGURE-5

Hemangioblastoma with negative SOX10 immunostain in hemangioblastoma
(a) H&E stain shows the characteristic histomorphology of hemangioblastoma. The image shows a mixture of vascular channels (appearing as white spaces) surrounded by stromal cells (purple-stained cells).
(b) Immunohistochemical (IHC) stain of SOX10showing complete absence of nuclear immunoreactivity in tumor cells.

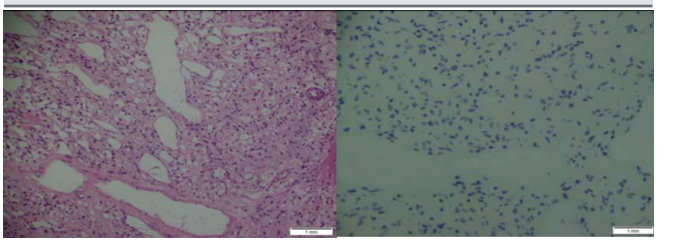
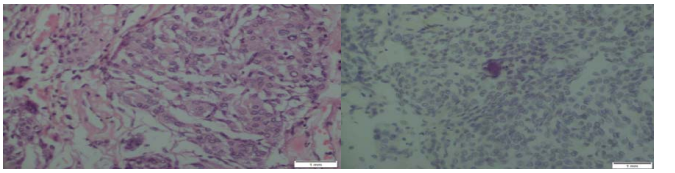


FIGURE-6

Meningioma and Negative SOX10 immunostain in meningioma
(a) H&E stain showing the histomorphology of meningioma. This shows whorled patterns of uniform tumor cells with oval nuclei and moderate cytoplasm along with psammoma bodies.
(b) IHC section demonstrating complete negativity for SOX10 in meningioma. The absence of brown nuclear staining confirms that meningiomas consistently lack SOX10 expression.



DISCUSSION

Posterior fossa tumors represent a diagnostically challenging group of central nervous system (CNS) neoplasms. Accurate diagnosis is critical, as therapeutic strategies, prognostic stratification and clinical outcomes differ significantly among tumor subtypes. The present study systematically evaluated the immunohistochemical utility of SOX10 and OLIG2, alongside conventional markers, in the differential diagnosis of posterior fossa tumors with particular emphasis on differentiating ependymoma from pilocytic astrocytoma and medulloblastoma.[15]

In our study of 65 posterior fossa tumors, ependymomas constituted slightly over half of the cases (50.8%), reflecting the well-established predominance of this tumor entity in the pediatric posterior fossa. The non-ependymoma group was mainly composed of pilocyticastrocytomas (62.5%) and medulloblastomas (21.9%), consistent with previously reported epidemiological distributions.[16]

A major diagnostic problem in posterior fossa tumors arises from the morphological overlap between ependymoma and pilocytic astrocytoma. While classical histological features such as perivascular pseudorosettes favor ependymoma, their absence in small biopsies often leads to diagnostic uncertainty. Our findings demonstrate that SOX10 and OLIG2 are highly reliable markers for resolving this diagnostic challenge.[17]

In our study, OLIG2 demonstrated 100% positivity in pilocyticastrocytomas and the single diffuse midline glioma while showing complete negativity in all ependymomas. This finding further strengthens the role of OLIG2 as a glial lineage marker. This is particularly useful in differentiating astrocytic tumors from ependymal neoplasms.[18] Previous studies have consistently reported OLIG2 expression in astrocytic and oligodendroglial tumors.[19] Ligon et al. first described OLIG2 as a lineage-specific transcription factor in gliomas. They also noted high SOX10 sensitivity in astrocytic neoplasms showing its value as a glial lineage marker.[20]

Another finding in our study was the uniform GFAP positivity in all ependymomas. However, GFAP expression is not specific and is also seen in astrocytic tumors. Similar observations have been reported in multiple studies where GFAP demonstrated high sensitivity but poor specificity for ependymal differentiation.[21]

SOX10 was completely negative in all medulloblastomas in our study. This finding further supports its role in excluding embryonal tumors from the differential diagnosis of glial neoplasms. Kleinschmeidt et al. similarly reported consistent SOX10 negativity in medulloblastomas, reinforcing its specificity for glial lineage tumors.[22]

CONCLUSION

This single-center study shows that SOX10 immuno-histochemical staining is a highly reliable and powerful diagnostic marker in the differential diagnosis of posterior fossa tumors. SOX10 is a sensitive and specific marker for distinguishing pilocytic astrocytoma from ependymoma. Its routine use in diagnostic panels enhances accuracy, reduces interpretive uncertainty and supports improved clinical decision-making in pediatric neuro-oncology.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Ayesha Iftikhar:** Manuscript writing, data collection.
2	**Sajid Mushtaq:** Conception of idea.
3	**Asif Loya:** Proof reading.
4	**Usman Hassan:** Data collection.
5	**Hina Maqbool:** Data analysis.
6	**Shaarif Bashir:** Data entry.