DETAILS OF RESEACH CONTRIBUTIONS

My involvement in Neuro-oncology, encompasses both clinical and fundamental research. As a principal investigator, I've undertaken numerous extramural projects, resulting in the publication of nearly 230 papers. My central focus revolves around devising comprehensive strategies for **developing diagnostic and prognostic biomarkers**, along with **guidelines** for their seamless integration into clinical practice, particularly in regions with limited resources. The ultimate objective is to establish markers facilitating early detection and predictive insights.An exceptional facet of my work centers on **pediatric CNS tumors.** This entails identifying distinctive genetic characteristics in pediatric glioblastomas, challenging conventional oligodendroglioma classification norms, and uncovering the aggressive genetic landscapes inherent in pediatric meningiomas. Furthermore, my groundbreaking research on pleomorphic xanthoastrocytoma (PXA) and diffuse midline gliomas holds the potential for personalized treatments and innovative immunotherapy pathways. The key highlights of my research can be summarized as follows:

1 PEDIATRIC NEUROONCOLOGY

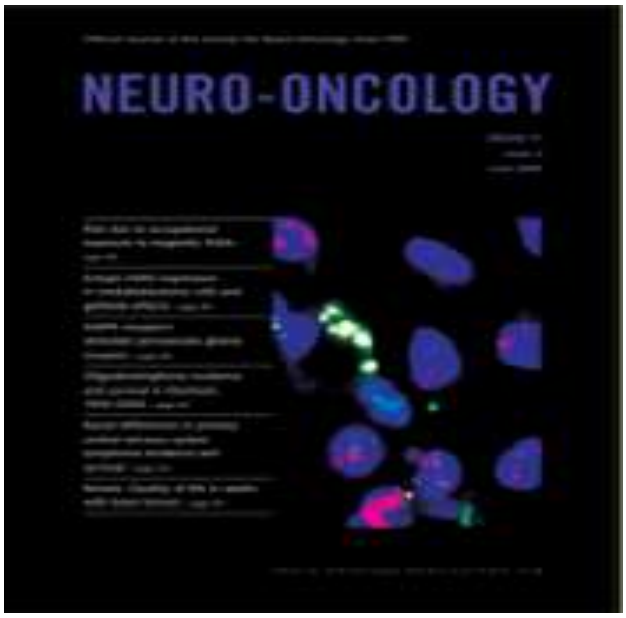
1.1 Pediatric Diffuse high Grade Gliomas

1.1.1 Pediatric glioblastomas: A histopathological and molecular genetic study

Glioblastoma multiforme (GBM), a highly malignant brain tumor, is relatively rare in children, constituting only 5%–10% of childhood brain tumors. Unlike the extensive research on adult GBMs, there have been fewer studies investigating the molecular properties of pediatric GBMs. We examined 30 cases of childhood GBMs diagnosed between 2002 and 2007.Histopathological features of these pediatric GBMs resembled those of adult GBMs, showing cellular characteristics like pleomorphism, mitotic activity, and necrosis. The study revealed that 63% of the cases exhibited p53 protein expression, a well-known marker for TP53 gene alterations. EGFR protein expression was noted in 23% of cases, although actual amplification of the EGFR gene was rare (5.5%). Similarly, deletion of the PTEN gene was also infrequent (5.5%). Loss of expression of the CDK inhibitors p16 and p27 was observed in 68% and 54% of cases, respectively.

Overall, the study suggested that pediatric GBMs exhibit genetic differences compared to their adult counterparts. While EGFR amplification and PTEN deletion are uncommon in pediatric primary GBMs, p53 alterations are more frequent in comparison to primary adult GBMs. Additionally, the frequency of loss of p16 and p27 expression in pediatric GBMs resembled that seen in adults. These findings underline the need for unique therapeutic strategies tailored to pediatric malignant gliomas, distinct from those used in adults. Since targeted therapies for adult GBMs are being developed, understanding the molecular differences between adult and pediatric cases is crucial for guiding effective treatment approaches for children with GBMs.

Building upon the insights garnered from our study and other relevant research, the World Health Organization (WHO) has acknowledged pediatric gliomas as a distinct entity. This recognition has led to the introduction of a separate classification for these tumors.



- **The microphotograph from our paper was published as cover image of Jounal**
- **Highlights of the study were published in nature (Clinical practice neurology)**

1.1.2 Clinico-pathological and molecular characterization of diffuse midline gliomas: is there a prognostic significance?

Recognized by the World Health Organization (WHO) as a distinct subgroup of central nervous system tumors, DMGs pose significant clinical challenges due to their rarity and aggressive nature. In recognizing the gap in understanding this entity within the Indian context, we undertook this research endeavor to illuminate the prevalence, clinical characteristics, and prognostic implications of diffuse midline gliomas(DMGs)in both pediatric and adult patients. The research underscores the necessity of molecular markers, particularly H3K27M immunohistochemistry with computer-derived scoring, in accurate diagnosis. Notably, this study reveals that the poor prognosis associated with DMGs remains consistent across age groups, locations, and radiological features, emphasizing the urgent need for innovative therapeutic strategies. The findings provide clinicians with a deeper understanding of the complex nature of these tumors and stress the importance of exploring novel treatment avenues. As the study calls for collaborative multicenter efforts, its clinical implications extend to shaping future research collaborations aimed at uncovering potential therapeutic targets and refining patient care approaches for DMGs, ultimately offering hope to individuals facing this challenging diagnosis.

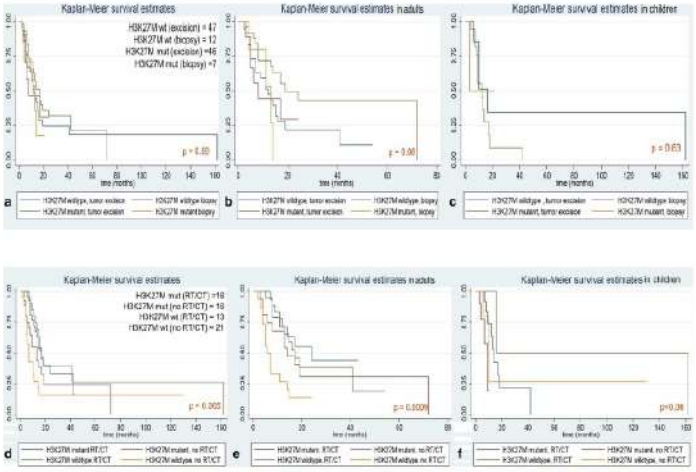


Fig. 3 Kaplan-Meier survival graphs correlating the H3K27M mutation with the surgical approach in **a** total study population, **b** adults, and **c** pediatric population. **d** Kaplan-Meier survival graph correlating survival with use of adjuvant therapy in H3K27M mutant and wild-type cases in total study population, **e** adults, and **f** pediatric population

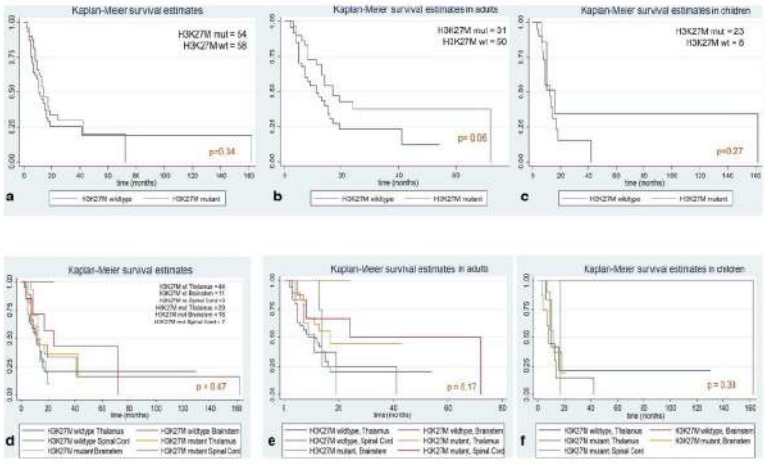
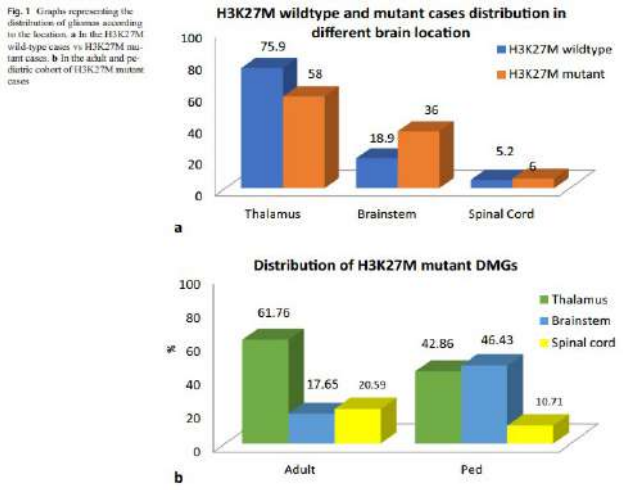


Fig. 2 Kaplan-Meier survival graph in relation to H3K27M mutation. **a** Overall survival in the total study population vs H3K27M mutation. **b** Overall survival in adults vs H3K27M mutation. **c** Overall survival in pediatric population vs H3K27M mutation. **d** Kaplan-Meier survival graph correlating overall survival in relation to tumor location and H3K27M mutation in total study population, **e** in adults. **f** in pediatric population



1.1.3 Analysis of PD-L1 expression and T cell infiltration in different molecular subgroups of diffuse midline gliomas.

The study analyzed a 126 diffuse midline gliomas (DMGs), both in adults and pediatric patients, focusing on their immune profile and genetic markers. The primary objective was to understand the potential of programmed death-ligand 1 (PD-L1) expression as a predictive biomarker for immunotherapy in DMGs.The analysis revealed four distinct molecular subgroups of DMGs based on genetic alterations, including the presence of H3K27M mutation, IDH1 mutation, ATRX mutation, and p53 protein expression. H3K27M mutation was more frequently observed in pediatric cases than in adults. Thalamus emerged as the most common site for DMGs, followed by the brain stem and spinal cord. Regarding PD-L1 expression, we found that approximately 31.7% of the DMGs exhibited PD-L1 expression, with varying levels of intensity. Interestingly, strong PD-L1 expression was predominantly observed in grade 4 tumors. Additionally, PD-L1 expression was more prevalent in elderly patients compared to pediatric and adult cases. The study also revealed a higher frequency of PD-L1 expression in H3K27M/IDH double-negative tumors and H3K27M wild-type/IDH1 mutant adult glioblastomas, indicating potential subgroups that might benefit from PD-L1 targeted therapy. In terms of immune infiltration, T cell presence was observed in the majority of cases, with CD3 and CD8 infiltration levels varying between pediatric and adult cases. While CD3 infiltration was similar in both age groups, CD8 expression was significantly greater in adults compared to children. Survival analysis revealed that PD-L1-positive cases tended to have poorer median overall survival, especially in adults, and high

CD3 and CD8 infiltration was associated with decreased survival in adult DMGs.. Multicenter studies with larger patient cohorts are recommended to further investigate the interplay between genetic alterations, immune profiles, and therapeutic responses in DMGs.

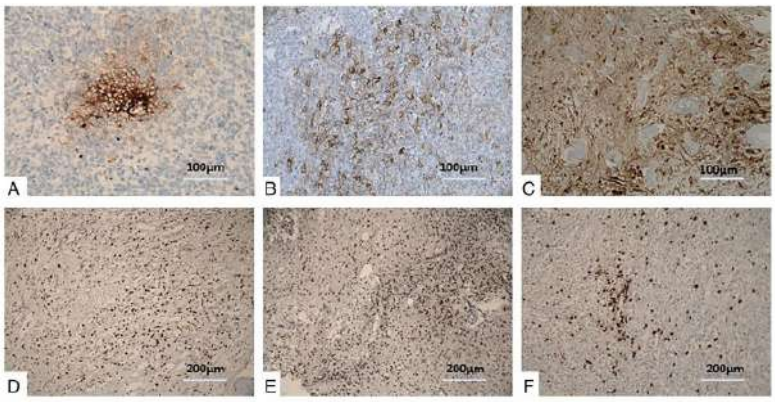


Fig. 2 Microphotographs of the tumor sections immunostained for PD-L1 (A-C), CD3 (D, E) and CD8 (F). PD-L1 immunoreactivity is localized in the cell membrane (A-C) and cytoplasm (C). CD3-immunoreactive T cells (D, E), diffusely infiltrating in the tumor, are also immunoreactive for CD8 (F).

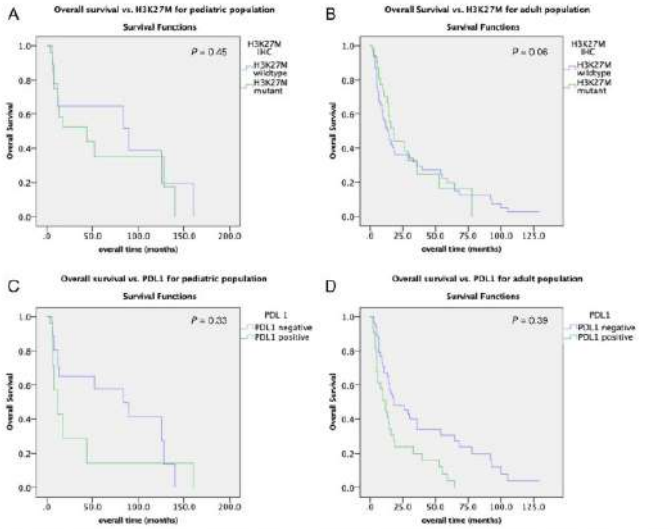


Fig. 3 Graphs by Kaplan-Meier survival analysis in pediatric population for H3K27M mutant versus wild-type (A), adult population for H3K27M mutant versus wild-type (B), pediatric population for PD-L1-positive versus negative status (C), adult population for PD-L1-positive versus negative status (D).

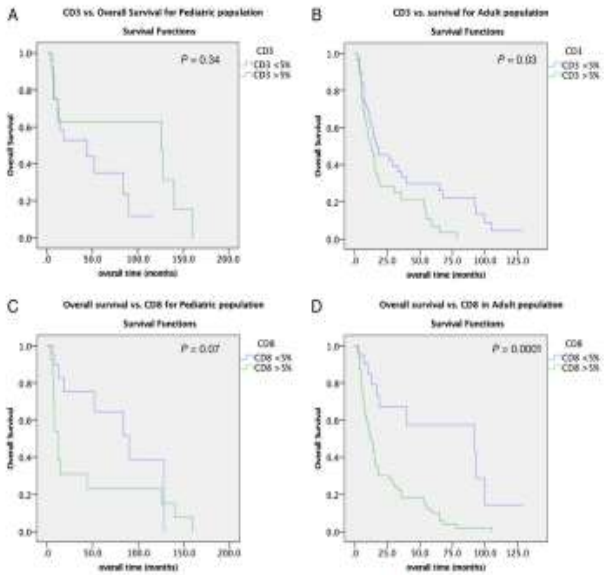


Fig. 4 Graphs by Kaplan-Meier survival analysis of overall survival (OS) in pediatric population CD3-identified T lymphocyte low versus high infiltration (A), adult population for CD3-identified T lymphocyte low versus high infiltration (B), pediatric population for CD8-identified cytotoxic T lymphocyte low versus high infiltration (C), and adult population for CD8-identified cytotoxic T lymphocyte low versus high infiltration (D).

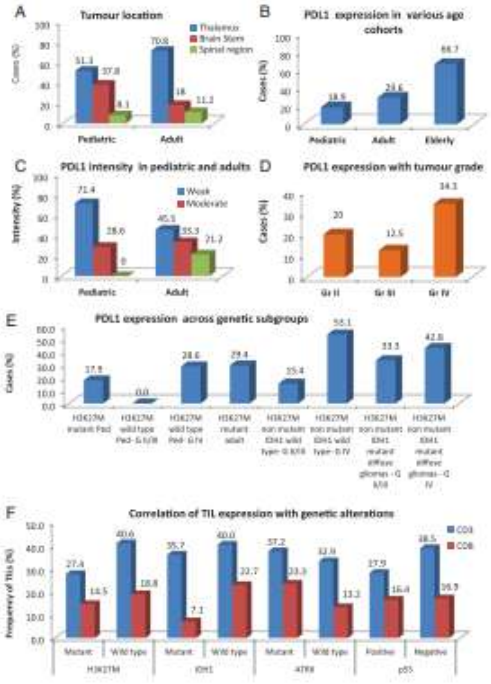


Fig. 1 Quantitative analyses in diffuse midline gliomas, such as tumor location (A) and PD-L1 expression status (B) and staining intensity (C) in different ages, PD-L1 expression in different age groups (C) and tumor grades (D), PL-D1 expression across genetic subgroups (E), correlation of TIL expression with genetic alterations (F).

1.2 Paediatric oligodendrogliomas

Molecular profile of oligodendrogliomas in young patients

Tumours with ologodendroglioma(OGs) like histology are extremely rare in childrenThis study aimed to comprehend the distinct genetic patterns and clinical implications of OGs in pediatric and young adult patients, as their molecular basis had been poorly understood compared to adult OGs. While previous research highlighted specific genetic features such as 1p/19q deletions, MGMT methylation, and IDH1 mutation in adult OGs with diagnostic, prognostic, and therapeutic significance, it remained unclear if these markers applied to younger patients. Analyzing 14 cases of OGs in individuals aged 25 years or younger, divided into pediatric (≤18 years) and young adult (19–25 years) subgroups, the study unveiled key insights. Notably, 1p/19q deletions were absent in pediatric cases, while 57% of young adults exhibited this deletion and 14% showed isolated 1p loss. Neither subgroup displayed IDH1 mutations or TP53 mutations. MGMT gene promoter methylation was prominent in both groups (71% each). These findings underscored the distinct molecular profile of OGs in young patients compared to adult OGs, with variations in 1p/19q deletions and IDH1 mutations. However, shared features included MGMT promoter methylation and the absence of TP53 mutation, indicating that molecular characteristics influencing prognosis and clinical management in adult OGs might not translate directly to pediatric cases. The study advocated for further extensive research to uncover additional relevant genetic alterations in young patients with OGs, ultimately enhancing treatment strategies and prognostic evaluations.WHO 2021 now reognized s that ogs do not occur in pediatric age gropup and all such tumours should be investigated throufgngs or methylation analysis for proper characterization.

In 2021, the World Health Organization officially acknowledged that oligodendrogliomas (OGs) are not typically found in the pediatric age group, suggesting that any such tumors occurring in this age range should undergo thorough investigation

using techniques like next-generation sequencing (NGS) or methylation analysisfor achieving accurate characterization of these tumors.

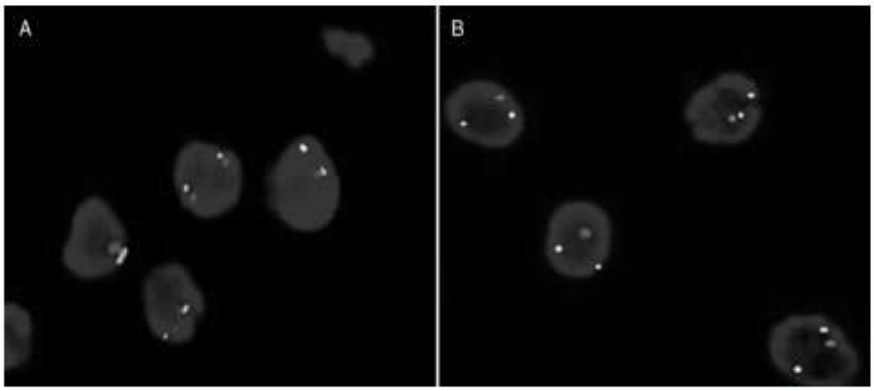


Fig. 1. Hetrozygosity status of 1p chromosome, as determined by FISH). (A) A case showing 2 red (test: 1p36) and 2 green (control: 1q 25) signals, implying no 1p deletion. (B) A case showing 1 red (test) and 2 green (control) signals, implying 1p deletion.

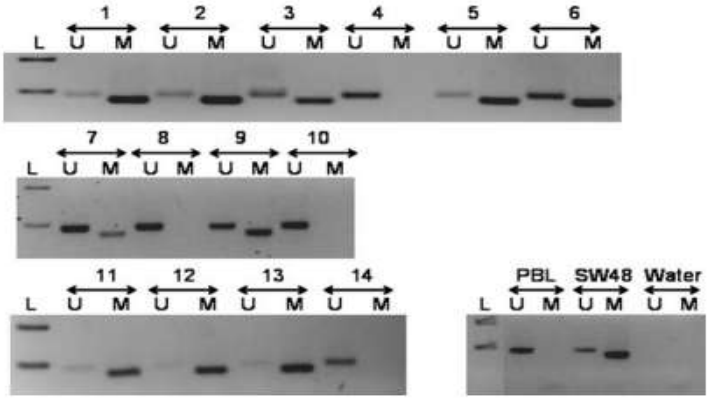
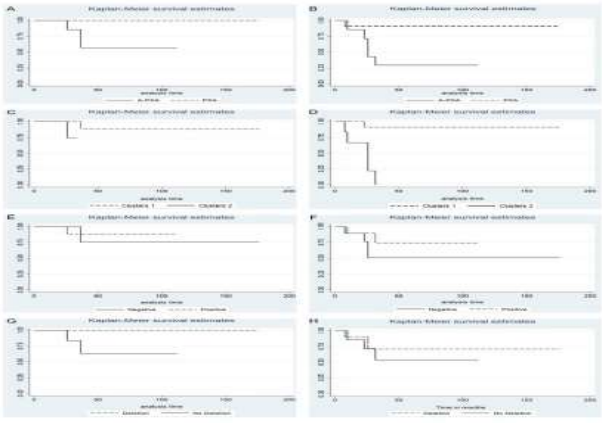


Fig. 2. Methylation status of the MGMT promoter, as determined by methylation-specific PCR assay. DNA from normal peripheral blood lymphocytes (PBL) was used as a control for the unmethylated MGMT promoter (U), enzymatically methylated lymphocytic DNA (SW48) served as a positive control for the methylated MGMT promoter (M), and water was used as a negative control for the PCR. A 100-bp marker ladder (L) was loaded to estimate molecular size. M: PCR product amplified by methylation-specific primers; U: PCR product amplified by unmethylated-specific primers; L: ladder; SW48: methylated control DNA; PBL: unmethylated control DNA. Samples 1, 2, 3, 5, 6, 7, 9, 11, 12, and 13 were methylated with presence of band in methylated "M" lane. Band in "U" lane is because of lymphocytes and normal tissue present with tumor tissue. Cases 4, 8, 10, and 14 were unmethylated with no band in "M" lane. In control samples, PBL had a band only in "U" lane, whereas SW48 had a band only in "M" lane.

1.3 Pleomorphic Xanthoastrocytoma

1.3.1 Gene expression based profiling of pleomorphic xanthoastrocytoma highlights two prognostic subgroups

In this pioneering study, the we conducted a comprehensive analysis of pleomorphic xanthoastrocytomas (PXAs), a rare type of brain tumor, to uncover their distinct molecular characteristics. Through genome-wide expression profiling, we identified two distinct molecular clusters within PXAs, revealing an unexpected level of molecular heterogeneity even within the same histological grade. This finding challenges traditional diagnostic and prognostic approaches solely based on histology. Furthermore, the study pinpointed specific dysregulated genes, including CDK14 and MTFP1, which could serve as potential prognostic markers and therapeutic targets. Importantly, the gene expression-based prediction of recurrence demonstrated superior accuracy compared to traditional histological grading, BRAF mutation status, or CDKN2A deletion status. **This research not only enhances our understanding of PXAs' underlying biology but also opens up new avenues for more accurate risk stratification and personalized treatment strategies, potentially revolutionizing the way these tumors are diagnosed and managed in the clinical setting.**



Kaplan Meier survival estimates showing that patients with histological diagnosis of APXA had a relatively poorer OS (A) and significantly poorer PFS (B) as compared to PXA (P=0.0804, P=0.0194). The cases in cluster 2 had a poor OS (C) and significantly worse PFS (D) as compared to cluster 1 (P=0.0973, P=0.003). The difference in OS (E) and PFS (F) among *BRAF* mutant and wild type cases was not significant (P=0.8995, P=0.3022). There was no significant difference in OS (G) and PFS (H) in cases with *CDKN2A* deletion versus no deletion (P=0.0977, P=0.4999).

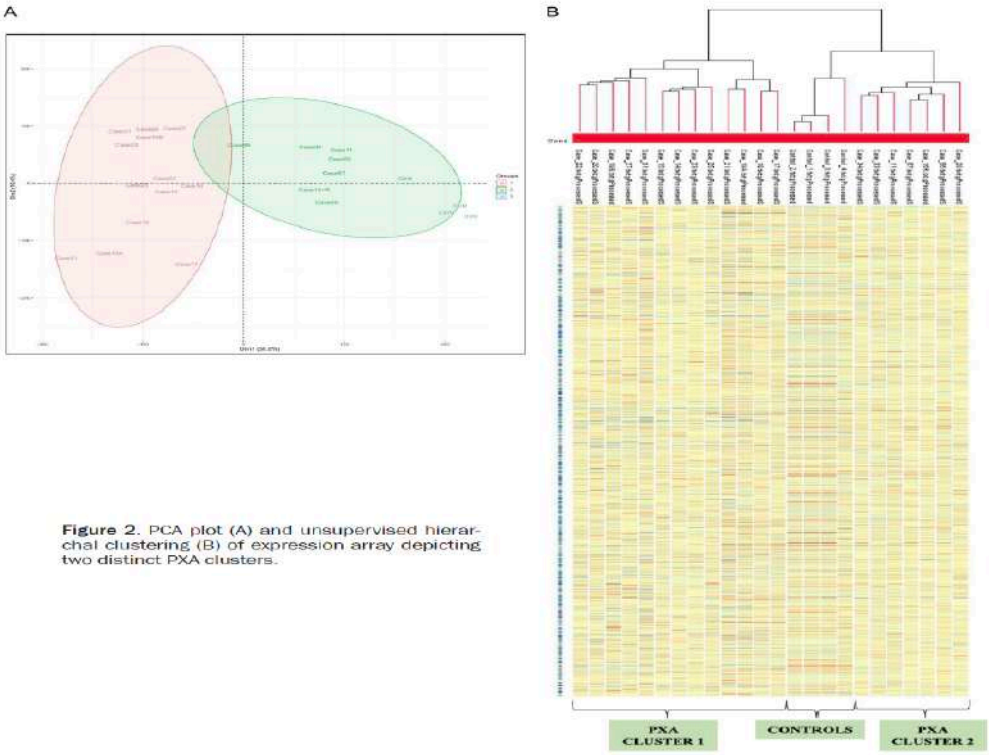


Figure 2. PCA plot (A) and unsupervised hierarchal clustering (B) of expression array depicting two distinct PXA clusters.

1.3.2 Long Non-coding RNA and mRNA Co-expression Network Reveals Novel Players in Pleomorphic Xanthoastrocytoma

In this study, the gene expression profiles of pleomorphic xanthoastrocytoma (PXA) patients were analyzed to uncover potential biomarkers and therapeutic targets. Using microarray data, differentially expressed mRNAs and long non-coding RNAs (lncRNAs) in PXA were identified. Pathway enrichment and protein-protein interaction analyses highlighted key pathways like MAPK, PI3K/AKT, and EGFR signaling that might contribute to PXA pathogenesis. Notably, several hub genes emerged as potential drivers, some of which are known oncogenes. Survival analysis linked the expression of specific lncRNAs and mRNAs to patient prognosis, suggesting their potential as prognostic markers. These findings provide valuable insights into the molecular mechanisms underlying PXA, suggesting novel therapeutic avenues and prognostic indicators that could impact clinical management and patient outcome

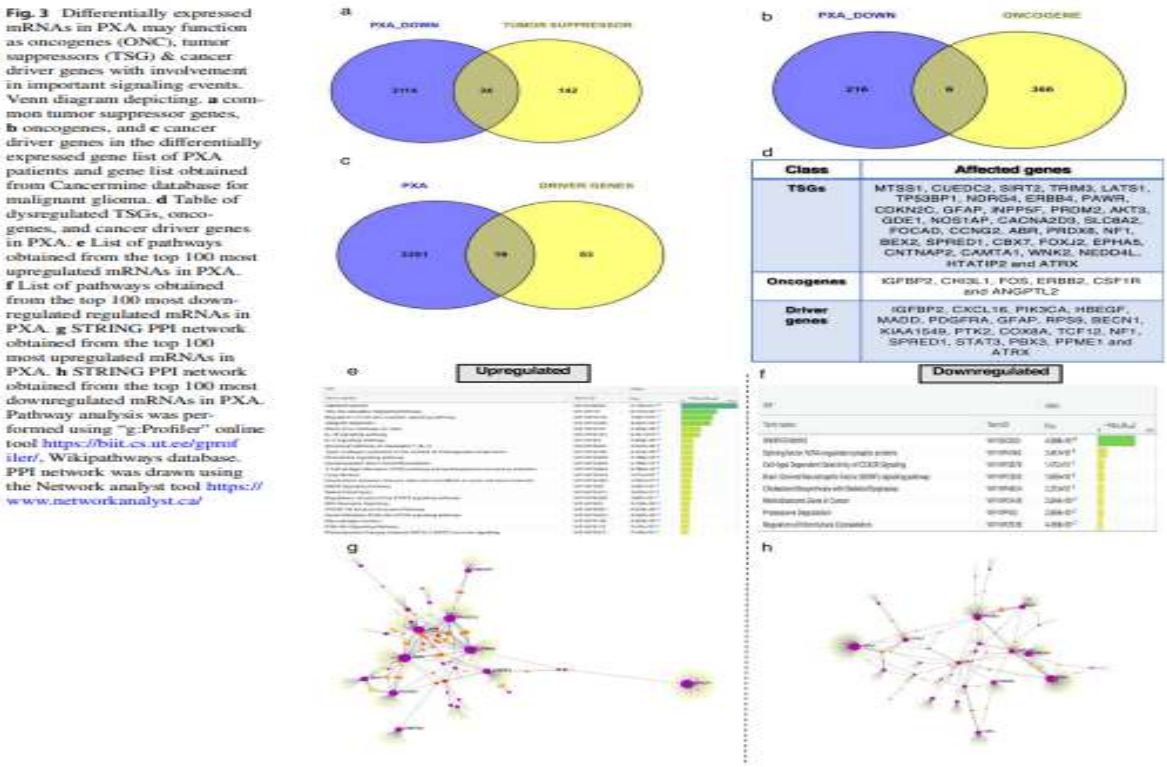


Fig. 3 Differentially expressed mRNAs in PXA may function as oncogenes (ONC), tumor suppressors (TSG) & cancer driver genes with involvement in important signaling events. Venn diagram depicting. **a** common tumor suppressor genes, **b** oncogenes, and **c** cancer driver genes in the differentially expressed gene list of PXA patients and gene list obtained from Cancermine database for malignant glioma. **d** Table of dysregulated TSGs, oncogenes, and cancer driver genes in PXA. **e** List of pathways obtained from the top 100 most upregulated mRNAs in PXA. **f** List of pathways obtained from the top 100 most downregulated regulated mRNAs in PXA. **g** STRING PPI network obtained from the top 100 most upregulated mRNAs in PXA. **h** STRING PPI network obtained from the top 100 most downregulated mRNAs in PXA. Pathway analysis was performed using "g:Profiler" online tool https://biit.cs.ut.ee/gprofiler/, Wikipathways database. PPI network was drawn using the Network analyst tool https://www.networkanalyst.ca/

1.4 Paediatric Meningiomas

In this study, the clinical, morphological, and molecular characteristics of pediatric meningiomas were analyzed. A total of 40 cases were studied, revealing distinct features compared to their adult counterparts. The majority of pediatric meningiomas were located in the cerebral convexity and intraventricular regions, with a higher prevalence of higher-grade tumors. Chromosome 22q deletion and Merlin loss were common across all grades. Although the frequency of combined 1p/14q deletion increased with tumor grade, this difference was not statistically significant. Notably, none of the cases showed SMO, AKT, KLF4, TRAF7 mutations, or TERT promoter mutations observed in adult meningiomas. GAB and stathmin co-expression was prevalent and independent of tumor grade. **The study highlights the unique molecular profile of pediatric meningiomas and emphasizes their differences from adult cases**.

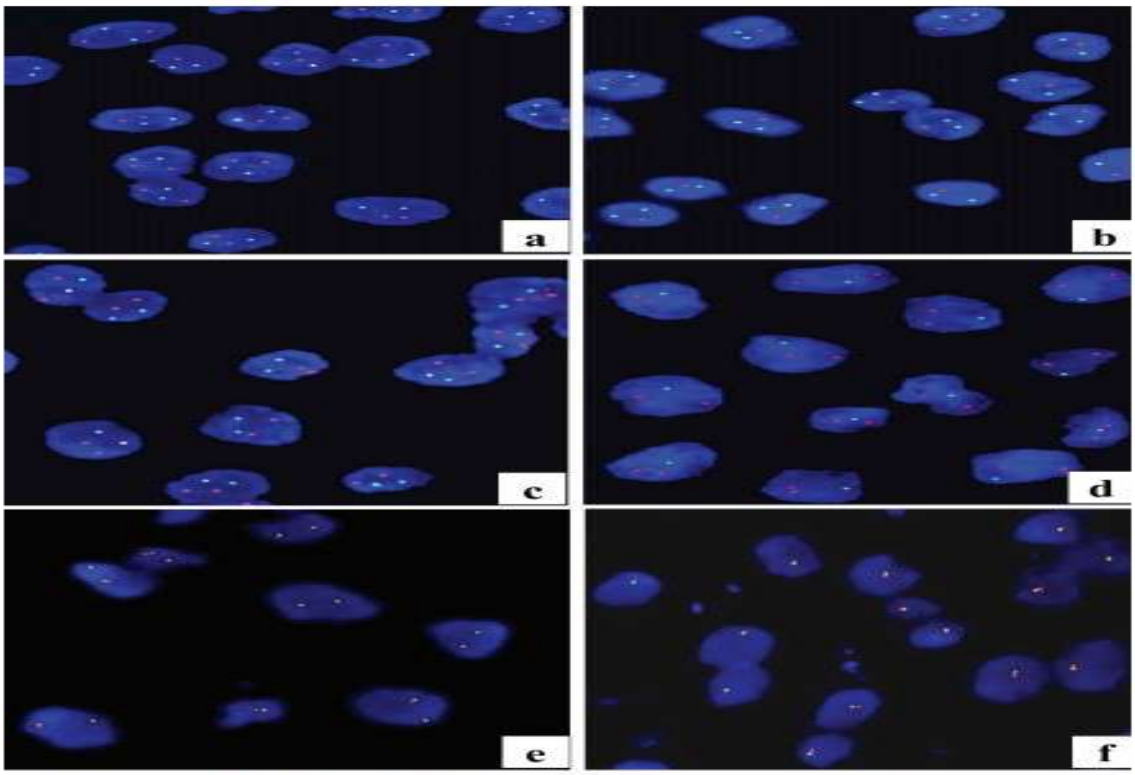


Fig. 1 (a–f).Photomicrograph of florescence *in situ* hybridization analysis for 1p (a, b), 14q (c, d) and 22q (e, f). (a) Case of transitional meningioma (WHO grade I) showing normal 1p status: nuclei show two red (test) and two green (control) signals (1000×). (b) Case of atypical meningioma (WHO Grade II) showing 1p deletion: Majority of nuclei show one red (test) and two green (control) signals (1000×). (c) Transitional meningioma (WHO Grade I) with normal 14q status: nuclei show two green (test)and two red (control) signals (1000×). (d) Atypical meningioma (WHO Grade II) showing 14q deletion: majority of nuclei show one green (test) and two red (control) signals (1000×). (e) Transitional meningioma (WHO Grade I) showing normal 22q status: nuclei show two red (test) and two green (control) merged signals (1000×). (f) Atypical meningioma (WHO Grade II) showing 22q deletion status: nuclei show one red (test) and one green (control) signal.

2MOLECULAR ANALYSIS OF CNS TUMORS TO DEVELOP COST-EFFICIENT TESTS SUITABLE FOR ROUTINE INTEGRATION INTO CLINICAL PRACTICE.

2.1 IDH Mutant and IDH wild type Astrocytomas

2.1.1 Role of CDKN2A deletion in grade 2/3 IDH-mutant astrocytomas: need for selective approach in resource-constrained settings.

The objective of this study was to determine the frequency of homozygous CDKN2A deletion in isocitrate dehydrogenase (IDH)–mutant diffuse astrocytomas (grade 2/3) and to identify specific clinicopathological indications where the CDKN2A fluorescence in situ hybridization (FISH) assay would be cost-effective in resource-constrained settings. The study analyzed IDH-mutant astrocytomas for various molecular markers and used FISH to assess CDKN2A deletion and 1p/19q codeletion. Survival outcomes were evaluated based on these molecular markers. The results showed that homozygous CDKN2A deletion was absent

in grade 2 and rare in primary grade 3 IDH-mutant astrocytomas. We proposed a selective approach for performing the CDKN2A FISH assay, focusing on cases with histomorphological features of anaplasia, p16 loss, or recurrent tumors, which achieved a sensitivity and negative predictive value of 100%. This approach would save on costs and workforce while maintaining accurate identification of cases with CDKN2A deletion, making it a more resource-efficient strategy in the clinical setting.

This study provides valuable insights into optimizing the use of the CDKN2A FISH assay for assessing CDKN2A deletion in IDH-mutant astrocytomas. By proposing a selective approach based on specific clinicopathological features, the authors offer a practical strategy to implement this molecular marker more efficiently and economically, particularly in settings with limited resources. This approach not only helps in accurate risk stratification of patients but also addresses the challenges of incorporating new molecular markers into routine clinical practice, ensuring better patient care and cost-effectiveness.

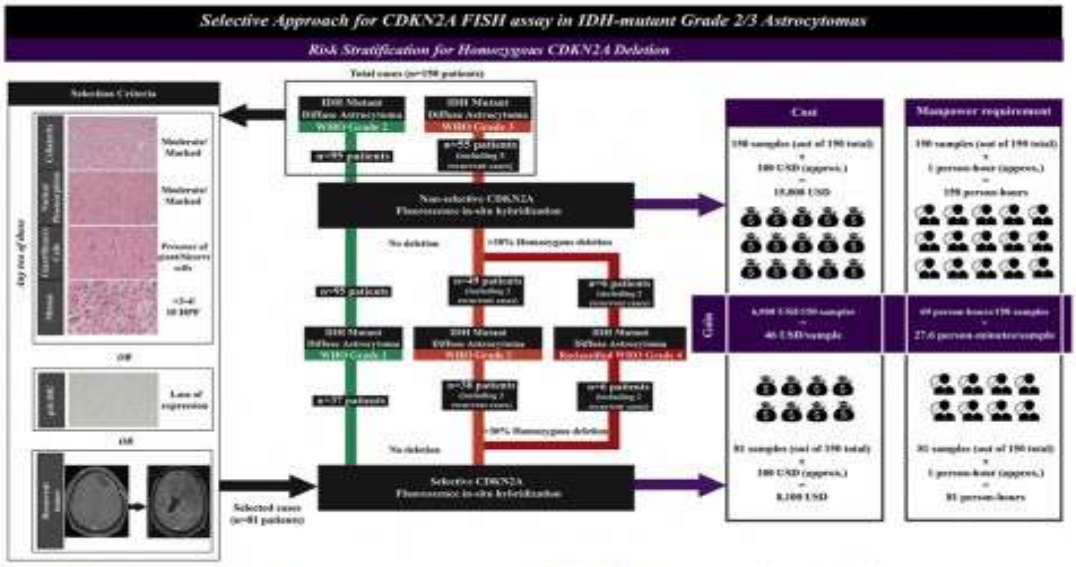


FIG. 6. Comparison of nonselective and selective approaches to *CDKN2A* FISH assay in terms of cost and workforce requirements.

2.1.2 Molecular Characterization of IDH Wild-type Diffuse Astrocytomas: The Potential of cIMPACT-NOW Guidelines[6]

This study focuses on IDH wild-type (wt) grade 2/3 astrocytomas, a group of brain tumors with diverse molecular and clinical characteristics. The study employed a panel of molecular markers to reclassify these tumors and investigate their clinical outcomes in comparison to IDH-wt glioblastomas (GBMs. The analysis revealed that a subset of grade 2 tumors with specific molecular alterations exhibited a clinical course similar to GBMs. These tumors were

reclassified as molecular GBMs, emphasizing the importance of molecular testing in guiding treatment decisions. Additionally, it was found that grade 3 tumors lacking these molecular alterations behaved similarly to GBMs regardless of their molecular profile. The study highlights the significance of molecular characterization in accurately categorizing and managing these tumors.

The research contributes to the understanding of IDH-wt grade 2/3 astrocytomas, providing insights into their molecular heterogeneity and clinical behavior. By employing a panel of molecular markers, the study suggests a more accurate classification of these tumors, which can guide prognosis and treatment decisions. The findings highhlight the need for integrating molecular information into the diagnostic process to better predict patient outcomes and tailor treatment strategies. This approach has the potential to impact clinical practice by improving the management of IDH-wt grade 2/3 astrocytomas, ultimately leading to better patient care and outcomes.

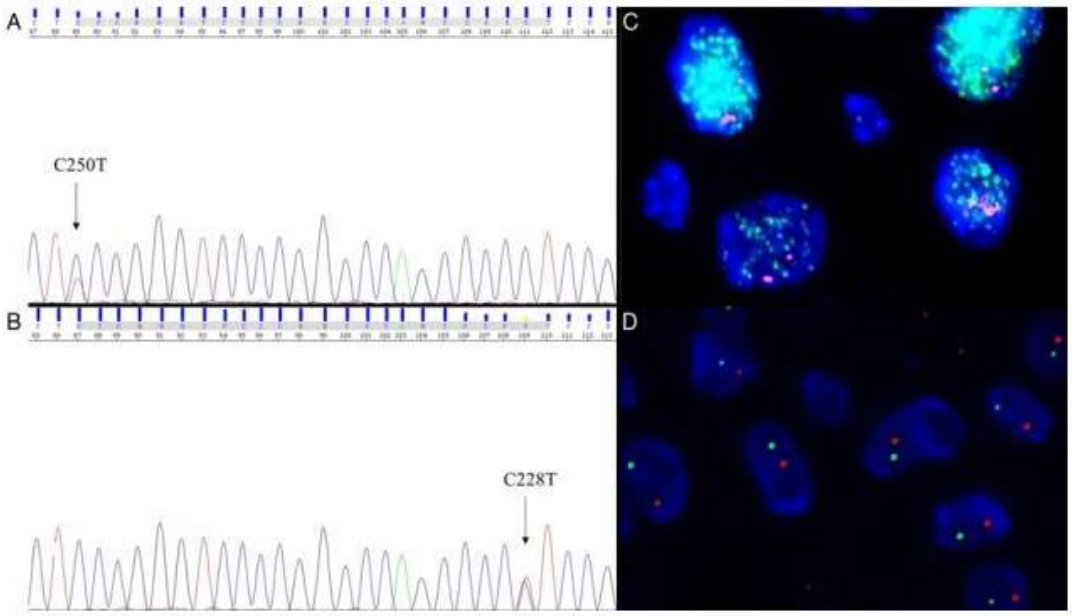


FIGURE 2. Electropherograms depicting *TERT* C228T (A) and *TERT* C250T (B) promoter region mutation in *IDH*-wt grade 2/3 diffuse astrocytic gliomas; dual-color fluorescence in situ hybridization for epidermal growth factor receptor (green signal); and centromere 7 (orange signal) (C) showing amplification in *IDH*-wt grade 3 diffuse astrocytic glioma (D) probe for PTEN (orange)/ CEP10 (green) showing monosomy 10 in a case of *IDH*-wt grade 2 diffuse astrocytic glioma. full color

2.2DNA methylation profiling of meningiomas highlights clinically distinct molecular subgroups.

The study aimed to investigate the molecular classification of meningiomas based on DNA methylation profiling and its clinical implications. DNA methylation profiling, copy number analysis, targeted sequencing, and H3K27me3 expression were performed on 35 meningiomas and 5 controls. Unsupervised hierarchical clustering identified four distinct

molecular subgroups: Malignant, Intermediate, Benign A, and Benign B. Within the same histological grade, molecular heterogeneity was observed. Specific mutations (NF2, AKT1, SMO, TRAF7, pTERT) were associated with different methylation subgroups. Chromosomal alterations were linked to different subgroups. Loss of H3K27me3 expression was observed in aggressive subgroups. Over 38,000 genes were dysregulated across subgroups. The DKFZ classifier achieved 71% accurate classification. Methylation profiling significantly impacted progression-free survival in WHO grade 1 and 2 meningiomas. The study highlighted the potential of DNA methylation profiling to enhance meningioma classification, prognostication, and treatment strategies.

The introduction of DNA methylation profiling for meningioma classification has provided a revolutionary shift in the diagnostic approach. By identifying distinct molecular subgroups and heterogeneity within the same grade of meningiomas, this methodology has transformed the traditional histology-based classification. This molecular classification has proven to be clinically relevant in predicting prognosis, treatment strategies, and disease course. **By accurately categorizing meningiomas into different subgroups based on their molecular signatures, healthcare professionals can better tailor patient management, deciding on more aggressive follow-up, appropriate adjuvant therapies, and personalized treatments. Additionally, this study highlights the importance of integrating molecular data into the diagnostic process, further emphasizing the role of molecular biomarkers in the understanding and management of brain tumors.**

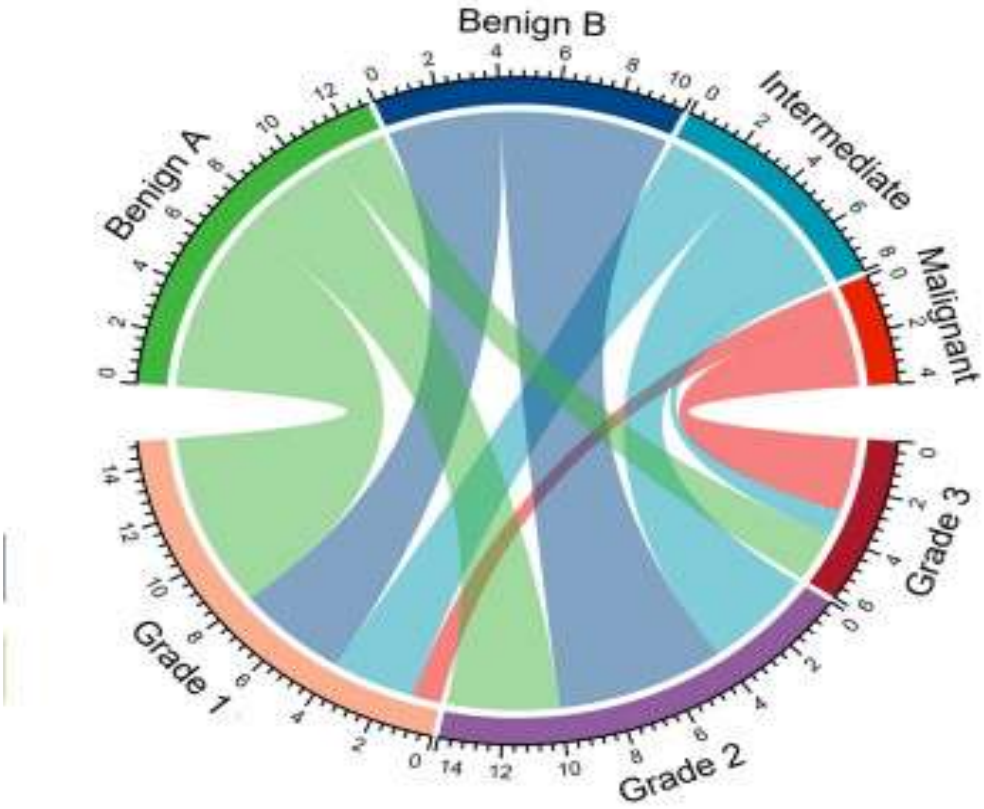


Fig. 3 Chord plot illustrating the distribution of conventional WHO grade meningiomas into the classifications provided by UHC

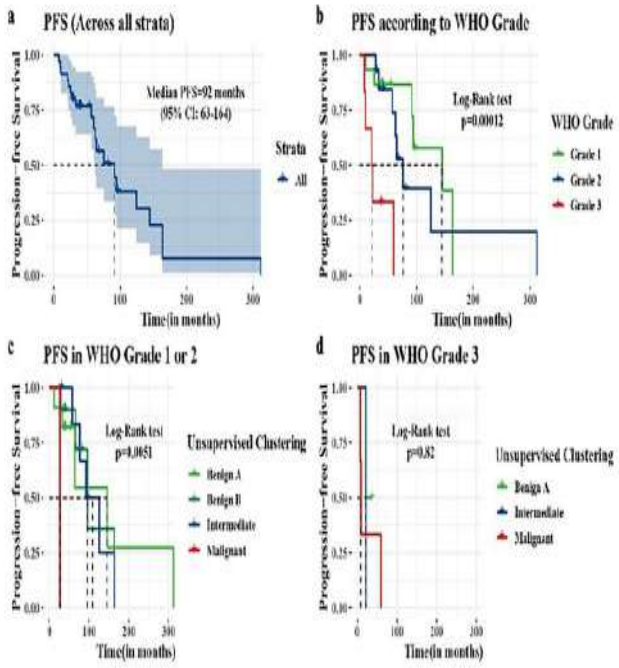


Fig. 4 Kaplan–Meier analysis showing **a** PFS across all strata, **b** PFS according to all WHO grades, **c** PFS in WHO grade 1 or 2 meningiomas and **d** PFS in WHO grade 3 meningiomas stratified according to methylation profiling subgroups

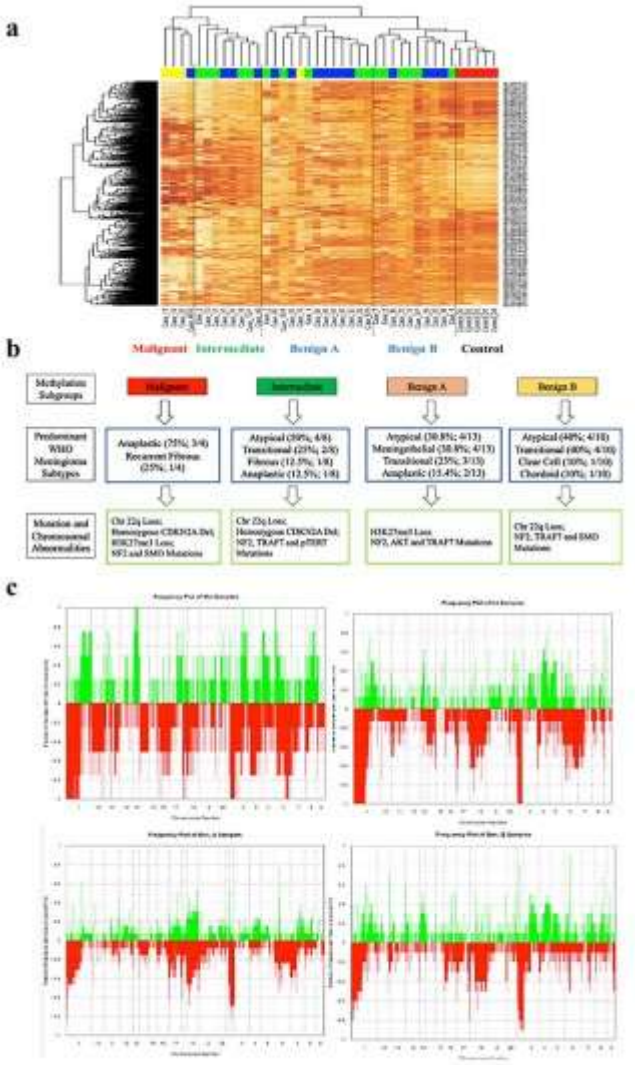


Fig. 2 Unsupervised hierarchal clustering reveals four methylation subgroups (**A**), Overview of histological and molecular features (**B**), and copy number profile of methylation subgroups (**C**)

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