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Extraneural Metastasis in Oligodendroglioma: A Comprehensive Review and Outcome Analysis of 90 Cases

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ABSTRACT

Extraneural metastasis in oligodendroglioma is a rare and poorly characterized event. We report a case whereby liver and bone metastasis occurred 19 years after diagnosis. Next generation sequencing on brain and liver oligodendroglioma showed several common mutations and a few unique ones to each organ. We identified a total of 90 similar cases and analyzed them through univariate and multivariate regression analysis to assess the impact of relevant clinical variables on overall survival, post-metastasis survival, and extraneural metastasis latency. These analyses highlighted the role of 1p19q codeletion in prolonging overall survival and delaying extraneural metastasis onset but interestingly without impacting post-metastasis survival. Aggressive treatment before metastasis was associated with shorter post-metastasis survival. Other clinical factors showed limited impact. To our knowledge, this is the only reported case with next-generation sequencing data on the primary, recurrent, and metastatic oligodendroglioma tumors, providing rare molecular insights into disease evolution. Our study also represents the largest collection of oligodendroglioma extraneural metastasis cases to date, and the only one to include detailed analysis of clinical variables and their impact on survival and metastasis.

1 | Introduction

Oligodendroglioma extraneural metastasis (ENM) is a rare and incompletely characterized phenomenon. While ENM in gliomas is increasingly recognized, molecular profiling of metastatic lesions is not routinely performed. Most information on this topic comes from isolated case reports and few case series, providing fragmented insights into the molecular and clinical features of this phenomenon [1]. To address this gap, we present a case in which serial molecular profiling was performed of both the primary tumor and the metastatic lesions. Additionally we identified 90 oligodendroglioma ENM cases through a comprehensive literature review. We provide a detailed overview of the basic features of oligodendroglioma ENM, along with a regression analysis of the clinical and molecular factors influencing patient outcomes.

2 | Materials and Methods

Next-generation sequencing (NGS) was performed on extracted DNA using the Oncomine Comprehensive Assay v3 (Thermo Fisher Scientific Inc.) according to the manufacturer's protocol.

A comprehensive literature search was conducted through PubMed using the query terms “oligodendroglioma” and

Abbreviations: CT, Computed tomography; ENM, extraneural metastasis; FISH, fluorescence in situ hybridization; IDH1, isocitrate dehydrogenase 1; MRI, magnetic resonance imaging; NGS, Next-generation sequencing; NOS, not otherwise specified; OS, overall survival; PMS, post-metastasis survival; RT, radiation therapy; VAF, variant allele frequency; WHO, World Health Organization.

"metastasis." Also, a search was performed using the Elicit platform with the prompt: "Identify all oligodendroglioma extraneural metastasis cases" to find additional reports. We included cases with sufficient information on metastatic organ sites and clinical variables, including patient demographics, tumor characteristics, treatment history, and survival outcomes.

Three endpoints were analyzed:

1. Overall survival (OS): Time from initial diagnosis to death or last follow-up.
2. Post-metastasis survival (PMS): Time from extraneural metastasis to death or last contact.
3. ENM latency: Time from initial diagnosis to first ENM (no censoring, as all patients ultimately developed ENM).

Statistical analyses and graphic generation were performed using GraphPad Prism (version 10.5.0). Univariate Kaplan–Meier analyses with log-rank testing were performed to visually compare survival distributions and assess the individual impact of each variable. Cox proportional hazards regression was used for OS and PMS, accounting for censored observations. ENM latency was analyzed using multiple and simple linear regression models, as Cox modeling was not used due to lack of censoring.

3 | Results

3.1 | Case Presentation

Forty-five-year-old male presented with focal seizures in 2005 and a brain MRI revealed non-enhancing lesions in the left frontal and temporal lobes. Biopsy in 2005 and resection in 2006 confirmed WHO Grade 3 oligodendroglioma with isocitrate dehydrogenase 1 (IDH1) mutation and 1p19q codeletion and he received radiation therapy (RT) to both lesions. Over 14 years, he experienced slow deterioration, and radiographic progression in 2020 prompted resection, revealing the same grade tumor. Four years later, further progression led to resection again showing WHO grade 3 oligodendroglioma. Figure 1a shows 3 MRI pictures at relevant time points of the disease course.

Postoperative complications prompted an abdominal Computed tomography (CT) scan revealing liver, lymph node, and osseous lesions. Figure 1b shows liver and osseous ENM lesions at diagnosis in April 2024 and at progression in August 2024.

Biopsy of the liver lesion confirmed oligodendroglioma (IDH mutant, 1p19q codeletion) (Figure 1c). Re-irradiation and temozolomide chemotherapy were initiated with good intracranial control but systemic ENM progression occurred, and he died shortly after, 19 years after the initial diagnosis.

Next generation sequencing (NGS) data from the 2020 and 2024 brain tumor resections and the liver metastasis biopsy are summarized in Table 1 by site, year, and variant allele frequency (VAF) or copy number (CN). *IDH1* mutation and 1p19q codeletion were identified in all three tumor samples. *CDKN2A/2B* deletions were unique to the central nervous system (CNS) tumors from 2020 and 2024, while *TERT*, *PIK3CA*, and *STK* mutations were shared between the 2024 CNS tumor and the liver metastasis. *PTEN* and *NOTCH* mutations were found exclusively in the liver metastasis. PTEN, a phosphatase involved in cell proliferation, and NOTCH1, a transmembrane receptor regulating developmental and cell–cell interactions, are recurrently altered in multiple malignancies including gliomas. A cBioPortal query of the GLASS diffuse glioma cohort of 693 samples showed PTEN mutations in 34% of cases (nearly half missense, though none matching the variant in our case) and NOTCH1 alterations in 15% (about one-quarter representing copy number gains). The 2020 CNS tumor harbored several unique mutations whereas no unique mutations were identified in the 2024 CNS tumor.

3.2 | Oligodendroglioma ENM Literature Review

Oligodendroglioma has an incidence rate of 0.29 per 100,000, accounting for 0.8% of all CNS tumors with a median age of diagnosis of 45 years and median OS of 207 months according to the 2024 Central Brain Tumor Registry of the United States [2]. The 2021 WHO classification of CNS tumors redefined oligodendroglioma as a molecularly defined entity characterized by IDH mutation and 1p/19q codeletion [3]. Therefore, the inclusion of histologically defined oligodendrogliomas provides a reasonable approximation of the current molecular definition. A comprehensive PubMed and Elicit review identified 90 published ENM cases, which were included in the final analysis if sufficient clinical data were available [1, 4–65]. These historical cases, together with our current patient, were analyzed to evaluate the impact of selected clinical factors on OS, PMS, and ENM latency.

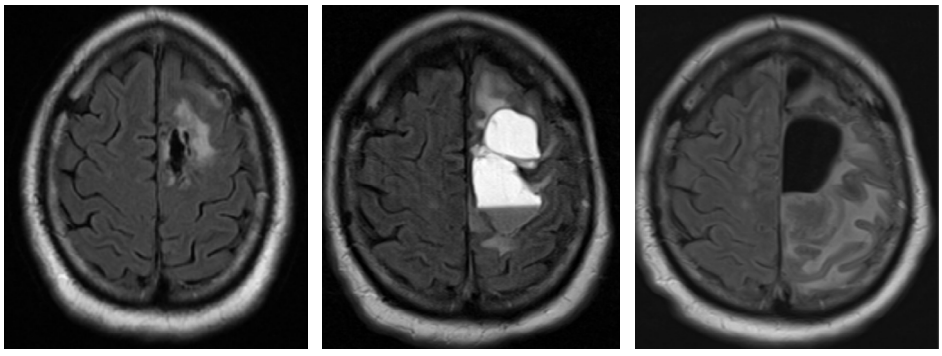
3.3 | Oligodendroglioma ENM Characteristics

Summary of the baseline features and outcomes of the 90 oligodendroglioma ENM cases identified is shown in Table 2. The most common metastatic site was bone, accounting for 45% of cases, followed by lymph nodes (18%) and soft tissue (16%). Lung and liver ENMs were seen in < 10% of the time.

3.4 | Oligodendroglioma ENM OS and PMS Analysis

The following clinical and molecular factors were chosen for the analysis due to their known or hypothesized impact on survival outcomes:

1. 1p19q status: codeleted vs. not otherwise specified (NOS), given the well-documented positive prognostic impact of 1p19q codeletion in diffuse gliomas [64].
2. Pre-ENM treatment modality: aggressive (defined as multimodal therapy with surgery, radiation and chemotherapy) vs. nonaggressive (surgery with or without radiation or chemotherapy but not all three modalities) [65].
3. Oligodendroglioma and ENM diagnoses age: Dichotomized at 40 years, reflecting its recognized prognostic significance in low-grade gliomas [66].



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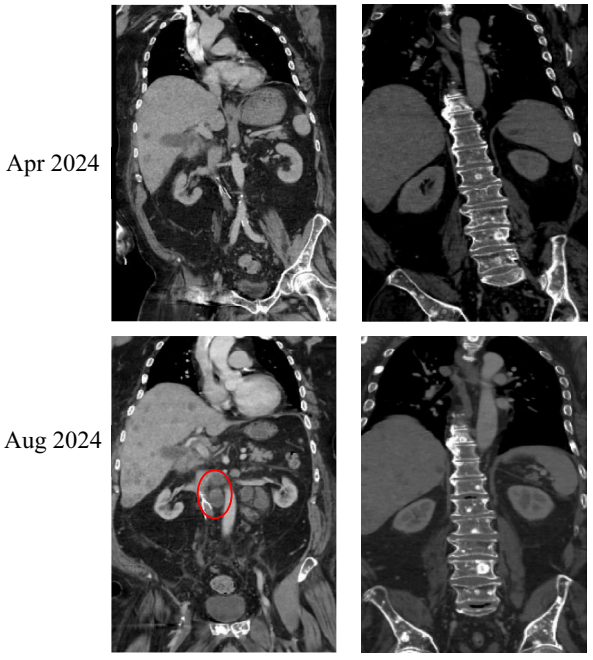

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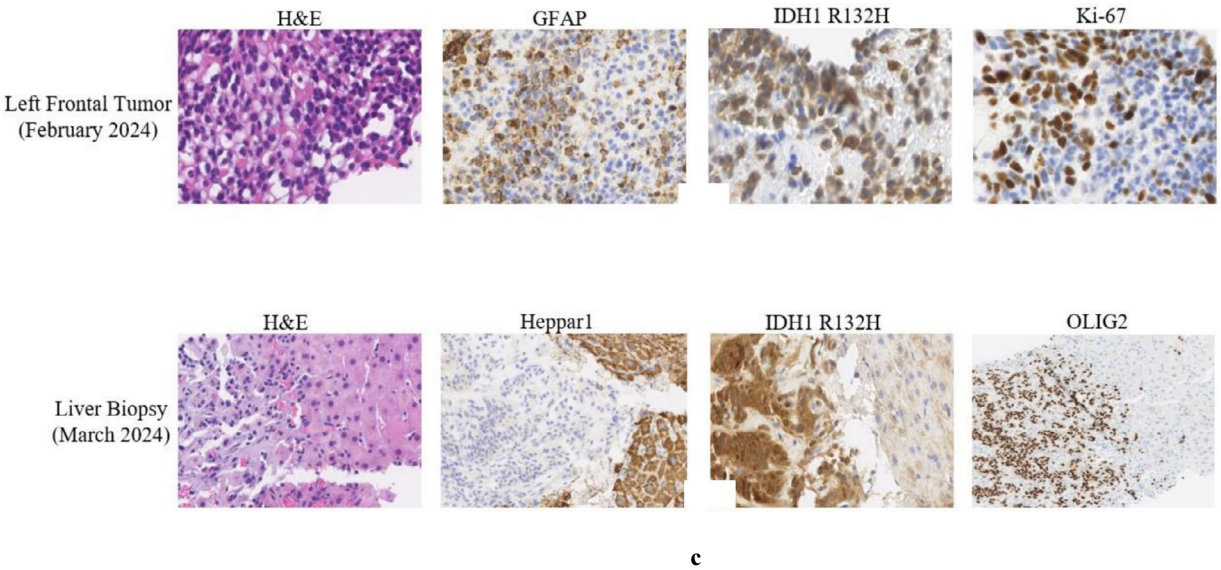

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FIGURE 1 | Legend on next page.

FIGURE 1 | (a) Axial MRI Brain FLAIR Images from diagnosis in December 2006, progression in June 2020, and CNS progression around systemic progression discovery in January 2024. (b) CT Chest/Abdomen/Pelvis coronal views showing increase in number of liver lesions, stable multiple osseous lesions and new retroperitoneal lymph node between April and August of 2025. (c) Immunohistochemical (IHC) staining of recurrent left frontal tumor and liver tissue samples using following markers for: General tissue structure (H&E), glial origin (GFAP), hepatocyte antigen (Heppar1), IDH1 R132H mutant isoform, proliferation index (Ki-67) and oligodendroglia (Olig2).

TABLE 1 | Side-by-side NGS data.

CNS 2020	CNS 2024 (VAF/CN)	Liver ENM 2024 (VAF/CN)
IDH1-R132H	IDH1-R132H (30.1%)	IDH1-R132H (15.9%)
1p19q codel	1p19q codel [1]	1p19q codel (N/A)
BRCA2 G1771D	TERT 124C>T (33.7%)	TERT 124C>T (35.9%)
ARID1A loss	PIK3CA-E110del (24.2%)	PIK3CA-E110del (17.1%)
AXL loss	STK11 gain (3.2)	STK11 gain (3.3)
CDKN2A/2B loss	CDKN2A (0.63)/2B (0.51) loss	PTEN-L57S (22.1%)
NOTCH2 loss		NOTCH1 gain (3.1)
RAD51B loss		
ERCC2 loss	TMB 3.4 Mutions/Megabase	TMB 2.5 Mutions/Megabase
EIF3E-RSPO2 fusion	Microsatellite-Stable	Microsatellite-Stable

Note: Shared mutations are underlined and shaded in: Gray (for mutations common in all 3 tumors), yellow (for mutations common between CNS 2024 tumor and liver ENM), or orange (for mutations common between CNS 2020 and CNS 2024 tumors).

TABLE 2 | Baseline features and outcomes summary of 90 oligodendroglioma ENM cases.

Gender	35 F vs. 46 M
Median age at oligodendroglioma diagnosis	42 years (1–72)
Median age at ENM diagnosis	45 years (1–74)
Median post oligodendroglioma diagnosis OS	48 months (4–215)
Median PMS	8 months (1–48)
Median time of ENM occurrence	36 months (2–324)

4. Gender: male versus female.
5. Concurrent CNS recurrence: present versus absent.
6. Metastatic burden: 1 ENM versus > 1 ENM

Univariate analyses using Kaplan–Meier survival estimates identified 1p19q codeletion, metastatic burden, and aggressive treatment as the only statistically impactful variables for either OS or PMS.

1. 1p19q codeletion: Patients with codeleted tumors demonstrated significantly longer OS compared to those with unknown 1p19q status. In contrast, there was no significant difference in PMS between the groups (Figure 2a).
2. Metastatic burden: Patients with a single site of metastasis had significantly longer OS compared to those with > 1 metastatic sites. However, the number of ENM sites did not significantly impact PMS (Figure 2b).
3. Aggressive treatment: Lack of aggressive treatment was associated with improved PMS. However, aggressive treatment did not significantly impact OS (Figure 2c).

None of the other variables statistically influenced OS or PMS.

To better characterize the impact of these clinical variables on OS and PMS, we performed multivariable Cox regression analysis. Although 90 cases were identified, complete data on all relevant clinical variables were available for only 58 patients in the OS analysis and 54 patients in the PMS analysis. Our results showed that 1p19q codeletion was the only variable significantly associated with improved OS, but it did not impact PMS. Aggressive treatment was associated with worsened PMS, and age < 40 years at the time of ENM diagnosis was associated with improved PMS, though neither affected OS. All other variables had no impact on either OS or PMS (Table 3).

3.5 | Oligodendroglioma ENM Latency Analysis

ENM Latency was analyzed by dividing the 85 cases with known ENM into quartiles: Q1 (2–18 months), Q2 (19–36 months), Q3 (37–84 months), and Q4 (85–324 months). The distribution across quartiles was relatively balanced, with 25%–27% of cases in each of the first two quartiles and slightly fewer in the upper quartiles.

Because latency to ENM was known for all cases, no censored data was present, precluding the use of Cox regression. Therefore, linear regression models were used to evaluate associations between clinical variables and ENM latency.

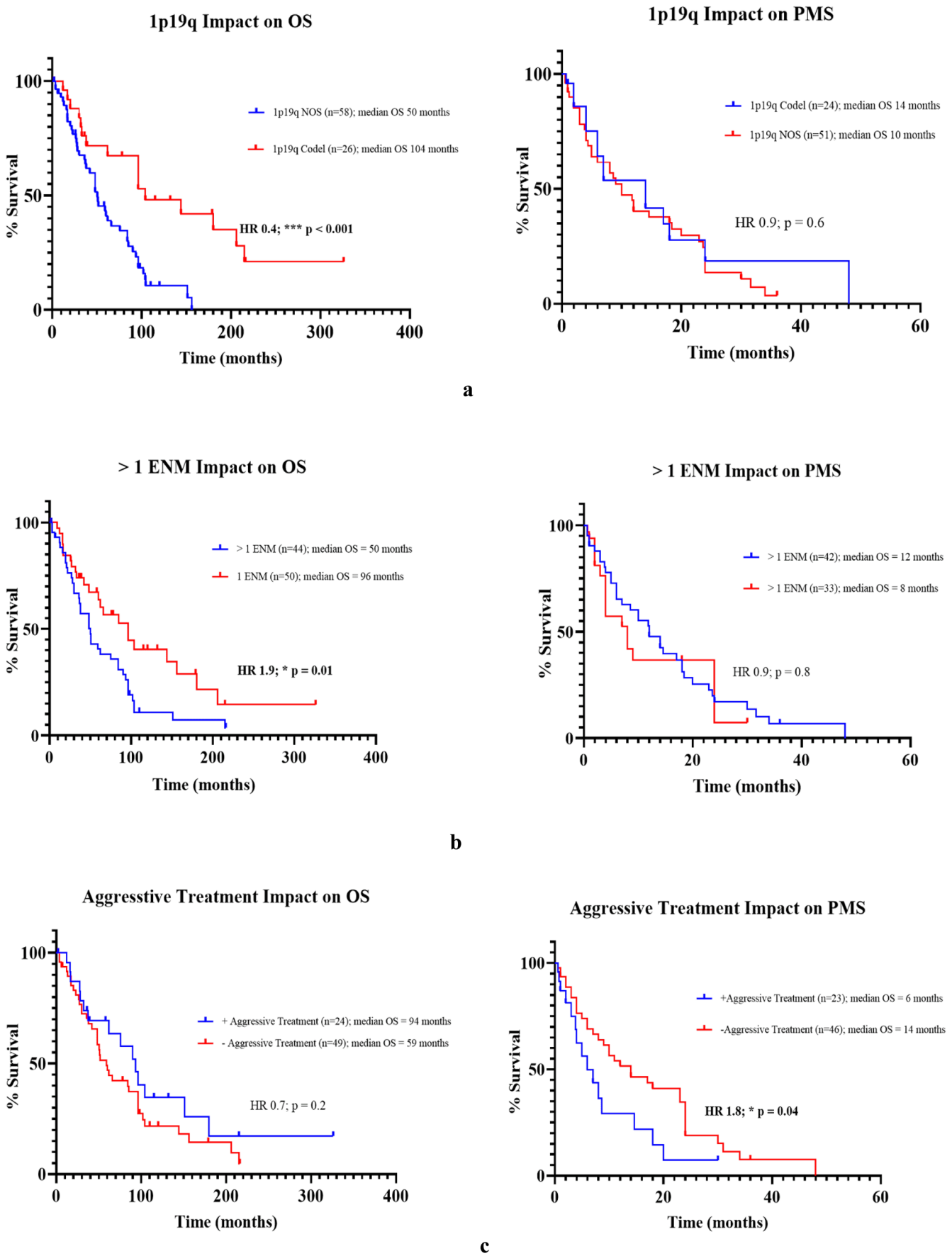

FIGURE 2 | Kaplan Meier survival curve of the impact of 1p19q codeletion (a) > 1 ENM site (b), and aggressive therapy (c) on OS and PMS.

TABLE 3 | OS ($n = 58$) and PMS ($n = 54$) Cox regression analysis.

Variable	OS HR; *p*	PMS HR; *p*
1p19q codeletion	0.5; 0.049*	0.6; 0.13
Aggressive treatment	1.0; 0.96	4.1; 0.0006***
ENM AGE < 40 years	1.9; 0.52	0.03; 0.03*
Diagnosis age < 40 yearsrs	0.4; 0.37	10.8; 0.11
Male gender	1.1; 0.82	1.3; 0.53
CNS recurrence	1.2; 0.57	2; 0.07
> 1 ENM Site	1.5; 0.27	0.6; 0.22

*$p < 0.05$; **$p < 0.005$; ***$p < 0.001$.

TABLE 4 | Simple (univariate) and multiple (multivariate) linear regression analysis on ENM latency.

Variable	Univariate estimate; *p*	Multivariate estimate; *p*
1p19q codeletion	+48.7; 0.0003***	+37.8; 0.03*
Male gender	−33.5; 0.02*	−32.4; 0.06
Diagnosis age < 40 years	+11.7; 0.4	+7.6; 0.7
Aggressive treatment	+6.2; 0.7	+7.6; 0.7
CNS recurrence	−17.9; 0.3	−14.1; 0.4
> 1 ENM site	−15.3; 0.3	−22.8; 0.2

*$p < 0.05$; **$p < 0.005$; ***$p < 0.001$.

Individual impact of each variable on ENM onset was studied via simple linear regression analyses. The analysis showed that 1p19q codeletion was associated with a 49-month delay in ENM onset. Additionally, male patients had a trend toward a 34-month reduction in latency, though this model explained only a small proportion of the variance ($p = 0.02$; $R^2 = 0.07$). None of the other variables (diagnosis age < 40 years, aggressive treatment, CNS recurrence, > 1 ENM site) impacted ENM onset. Multiple linear regression was then performed to assess the combined impact of these clinical variables. The regression model was statistically significant ($p = 0.018$), with an R^2 of 0.247, indicating that approximately 25% of the variance in ENM latency was explained by the included variables. Among these, 1p19q codeletion was the only factor independently associated with delayed ENM onset (38 months), consistent with the univariate findings. There was also a borderline trend for earlier ENM onset in male patients (32 months), though this was not statistically significant ($p = 0.06$). None of the other variables showed significant independent associations with ENM onset in either univariate or multivariable linear regression models (Table 4).

4 | Discussion

Oligodendroglioma ENM remains a rare and poorly understood phenomenon with growing recognition. Our case adds unique value by providing longitudinal molecular and clinical characterization over nearly two decades. This allowed us to highlight the stability of core alterations (IDH1 mutation, 1p19q codeletion) and the emergence of additional mutations at different disease stages (e.g., TERT, PIK3CA, STK shared by later CNS and metastatic lesions, PTEN and NOTCH1 restricted to the metastasis). Analysis of the GLASS diffuse glioma cohort on cBioPortal.org showed PTEN alterations in ~34% (none matching our case) and NOTCH1 mutations in 15% of the 693 samples available. These findings indicate that the alterations are common in gliomas and unlikely to represent liver- or metastasis-specific driver mutations. Comprehensive molecular analysis of ENM was rare among published cases. Only one prior report performed full NGS [4], two conducted targeted sequencing of select genes [6, 7], and two described unspecified molecular testing [16, 45]. Most relied solely on immunohistochemistry or fluorescence in situ hybridization (FISH). To our knowledge, this is the only report with comprehensive NGS performed on tissue from multiple intracranial resections and the ENM lesion, providing rare insights into the molecular heterogeneity of ENM in oligodendroglioma.

Our analysis was based on histologically defined oligodendrogliomas, as molecular classification was not routinely applied in earlier reports. However, histologic diagnosis correlates well with true 1p19q codeletion in approximately 61%–89% of cases [67], supporting the validity of including these cases when analyzing historical ENM data. This inclusive approach reflects real-world reporting patterns and underscores one of our key findings that although 1p19q codeletion is associated with improved OS, its favorable prognostic effect did not extend to PMS. This suggests that post-metastatic tumor biology overrides pre-metastatic molecular advantage.

Also, histologic grade was not included as an independent variable in our analysis given the shift in clinical practice toward molecularly guided treatment decisions. Our pooled analysis of 90 ENM cases is the largest to date and provides a detailed characterization of clinical features, metastatic patterns, and survival outcomes. We found that bone was the most frequent metastatic site (45%), followed by lymph nodes (18%) and soft tissue (16%). The median ENM latency occurrence was 3 years, yet the range extended over two decades, underscoring the unpredictable nature of extraneural spread in this disease. PMS was dismal, with a median of only 8 months despite prior multimodal therapy in many patients. These findings highlight the importance of long-term surveillance in oligodendroglioma patients.

Kaplan–Meier analyses offered comparative insights into survival trends, while Cox regression for OS and PMS offered a more accurate evaluation of prognostic factors. ENM latency was assessed using linear regression because the time to metastasis was known for all patients, eliminating the need for censoring-adjusted modeling.

Of the clinical and molecular factors analyzed, 1p19q codeletion emerged as the most consistently impactful. Metastatic burden, young age at ENM diagnosis, and aggressive multimodal therapy prior to ENM diagnosis also played meaningful roles in disease outcomes.

4.1 | 1p19q Codeletion

1p19q codeletion was significantly associated with improved OS in both univariate and multivariable analysis. It was also strongly associated with delayed ENM onset, extending latency by 49 months in simple regression models and by 38 months in multiple regression. However, its advantage did not extend to PMS reinforcing that its favorable prognostic role is lost once systemic spread occurs.

4.2 | Metastatic Burden

The number of metastatic sites showed potential impact on OS with patients having a single ENM demonstrating significantly longer OS in univariate analysis compared to those with multiple sites. However, this association did not persist in multivariable Cox regression, likely due to the confounding influence of other clinical variables considered in the multivariate model. Metastatic burden did not influence PMS or ENM latency, emphasizing its limited prognostic value once ENM has occurred.

4.3 | Age at ENM Diagnosis

Age <40 years at the time of ENM diagnosis was associated with significantly improved PMS in multivariable analysis without impacting OS or ENM latency. This finding suggests that younger patients may experience a more favorable post-metastatic disease course, although the lack of significance in univariate analysis indicates that other clinical variables may have influenced the outcome.

4.4 | Aggressive Treatment

Unexpectedly, aggressive multimodal therapy prior to ENM diagnosis was associated with worse PMS in both univariate and multivariable analysis. This paradoxical association likely reflects selection bias, where patients with more aggressive or treatment-refractory disease are more likely to receive intensive therapy. Aggressive treatment had no significant association with OS or ENM latency, suggesting its influence is largely restricted to the post-metastatic period.

4.5 | Male Gender

Male gender showed a trend toward reduced ENM latency, with men developing ENM approximately 34 months earlier than women in simple regression, though this effect was borderline in multivariable analysis and did not impact OS or PMS. Therefore, the biological significance of this variable remains questionable.

Notably, the latency to ENM demonstrated a relatively even distribution across quartiles, suggesting that extraneural spread can occur both early and late in the disease course without a strong temporal pattern.

Other clinical variables, including age at oligodendroglioma diagnosis and presence of concurrent CNS recurrence, did not demonstrate significant associations with OS, PMS, or ENM latency in either multivariable or univariate analyses.

These findings highlight that favorable molecular features, such as 1p19q codeletion, and a lower metastatic burden contribute to extended survival and delayed systemic spread. Cases categorized as NOS likely included more aggressive tumor variants, such as IDH-wildtype or astrocytic tumors, which may have contributed to their poorer outcomes compared to 1p19q codeleted oligodendrogliomas. However, once ENM develops, these advantages are lost. The improved PMS in younger patients at ENM diagnosis suggests potential differences in biological behavior or treatment response based on age, emphasizing the complex nature of extraneural disease, which may represent a distinct biological stage of oligodendroglioma with varied impact on patient outcomes. This emphasizes the need for novel systemic treatment approaches for patients with ENM, where traditional prognostic factors may no longer be relevant. Additionally, molecular profiling of metastatic lesions could identify new therapeutic targets and mechanisms of resistance, paving the way for more effective interventions in this challenging clinical scenario.

While this analysis provides important insights, its retrospective nature introduces inherent limitations, including potential selection bias and variability in treatment approaches. Furthermore, several ENM cases were discovered m, meaning the organ distribution and the kinetics of survival and latency may differ in real-life scenarios. Nevertheless, our work provides valuable data that can help guide future research and clinical management of oligodendroglioma ENM. Collaborative multi-institutional data sharing will be critical for advancing our understanding of this rare phenomenon.

Author Contributions

Ahmad Daher: conceptualization, data curation, formal analysis, investigation, methodology, supervision, writing – original draft, writing – review and editing. **Muhammad Yaman Jaber:** data curation, formal analysis, validation. **Stefania Maraka:** methodology, visualization, writing – review and editing. **Hiranmayi Vemaganti:** methodology, visualization, writing – review and editing. **Tibor Valyi-Nagy:** methodology, visualization, writing – review and editing.

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Ethics Statement

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

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