Corticosteroid Use Before Stereotactic Brain Biopsy for Suspected Lymphoma

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BACKGROUND AND OBJECTIVES: Primary central nervous system lymphoma (PCNSL) is a rare, aggressive lymphoma requiring histopathological confirmation for diagnosis. Stereotactic brain biopsy (SBB) is the gold standard for definitive diagnosis, but preoperative corticosteroid therapy (CST), commonly administered to manage symptoms, may induce cytoreduction and obscure diagnostic features. Previous studies offer conflicting evidence on whether CST compromises diagnostic yield. This study assesses the impact of preoperative CST on the diagnostic yield of SBB in PCNSL, evaluating steroid timing, dose, duration, and associated postoperative complications.

METHODS: We retrospectively reviewed 725 patients who underwent SBB between 2014 and 2025 to identify 104 patients with pathologically confirmed PCNSL. Patients were categorized based on CST exposure and stratified by timing, cumulative dose, and duration of therapy. Clinical, radiological, and pathological variables were analyzed. Statistical tests included χ^2, Welch *t*-test, and Fisher exact test.

RESULTS: The overall diagnostic yield was 92.3%. Among patients who received CST (n = 43), the diagnostic yield was 95.3% (95% CI: 84.2%-99.4%), compared with 90.2% (95% CI: 79.8%-96.3%) in the non-CST group ($P = .46$). Yield remained high across intervals between CST administration and biopsy (<48 hours: 96.4%; 48-72 hours: 91.7%; >72 hours: 100%; $P = .658$), cumulative dose (≤20 mg: 95.5% [95% CI: 78.2%-99.2%], 21-40 mg: 100% [95% CI: 70.1%-100%], >40 mg: 100% [95% CI: 70.1%-100%]; $P = 1.0$), and duration (≤5 days: 94.6% [95% CI: 81.8%-99.3%] vs >5 days: 100% [95% CI: 91.8%-100%]; $P = 1.0$). Postoperative complications occurred in 4.8% of cases, with no significant difference by CST status.

CONCLUSION: Preoperative corticosteroids do not significantly reduce diagnostic accuracy in PCNSL in our cohort. These findings support the safe use of CST for symptom control in suspected PCNSL with expeditious biopsy. Optimizing biopsy timing, technique, and coordination among disciplines remains essential to ensure diagnostic success.

KEY WORDS: Biopsy, Corticosteroids, Diagnostic yield, Dexamethasone, Lymphoma, Neuro-oncology, Stereotactic

Primary central nervous lymphoma (PCNSL) is a rare and aggressive subtype of non-Hodgkin lymphoma.[1-8] It accounts for approximately 4% of all central nervous system tumors, carries a poorer prognosis than many other aggressive systemic lymphomas, and has shown a rising incidence in both immunocompromised and immunocompetent patients.[1-8] Affected patients typically present with neurological symptoms that necessitate a diagnostic workup that includes neuroimaging and cerebrospinal fluid (CSF) cytology.[7-13] However, these modalities may not reliably distinguish PCNSL from other neoplastic or infectious processes. As such, stereotactic brain biopsy (SBB) remains the gold standard for definitive histopathological diagnosis.[7-12] SBB enables the identification of malignant B cells typically exhibiting perivascular distribution, high proliferation rates, and/or immunoreactivity for pan B-cell markers.[14,15] In addition, biopsy facilitates immunohistochemical and genetic markers that may guide targeted oncological treatment.[14,15]

Timely diagnosis and early intervention are critical in PCNSL.[16,17] Symptomatic patients often receive preoperative corticosteroid therapy (CST) to reduce vasogenic edema and mass effect[18-21];

ABBREVIATIONS: CST, corticosteroid therapy; **PCNSL,** primary central nervous system lymphoma; **SBB,** stereotactic brain biopsy.

however, PCNSL cells are highly sensitive to corticosteroids, which may induce apoptosis and transient tumor regression, thereby complicating histological diagnosis.[6,9,10,16-18,20,22-26] Although existing studies report conflicting results on the impact of prebiopsy CST use on the diagnostic yield of SBB, they are limited by small sample sizes, reflecting the rarity of PCNSL.[3,8] While some advocate for withholding steroids before biopsy, others support their use for symptom control.[26] Moreover, data remain limited on how factors such as cumulative steroids dose, tapering pattern and timing of CST discontinuation before SBB influence diagnostic success.

Given these gaps, we present our findings from one of the largest retrospective cohorts to date, examining the relationship between preoperative CST and the diagnostic yield of SBB in patients with PCNSL. We evaluate both timing and cumulative dosage of corticosteroids and assess their association with biopsy success and postoperative outcomes.

METHODS

Study Design and Population

We conducted a retrospective cohort study of patients with suspected cranial PCNSL who underwent SBB at our institution between January 2014 and January 2025. Institutional review board and local ethics committee approval were obtained; patient consent was waived due to the retrospective design.

Our analysis was limited to patients who underwent SBB for lesions with a high preoperative suspicion of PCNSL based on clinical and radiographic criteria. A database query using CPT (Current Procedural Terminology) code 61750 identified eligible cases. Comprehensive chart reviews assessed biopsy outcomes—diagnostic (e.g., lymphoma, glioma, and metastasis) or nondiagnostic. For nondiagnostic cases, follow-up data, including repeat or open biopsies and clinical progression, were used to determine final diagnoses.

We excluded patients who had non-SBB procedures (e.g., open biopsies or resections), biopsies conducted for therapeutic rather than diagnostic purposes, incomplete clinical data, absence of corticosteroid exposure documentation, unconfirmed PCNSL diagnoses, or repeat/open biopsies unrelated to an initial nondiagnostic result.

Data Collection

Collected clinical variables included age at biopsy, sex, number of SBBs, date of initial biopsy and follow-up reports, comorbidities (HIV, transplant history, hypertension, diabetes mellitus, and dyslipidemia), medication history including immunosuppressants and steroid type(s), route, dose, frequency, and timing relative to biopsy. Radiographic imaging reports, specifically MRI with gadolinium contrast, were reviewed to assess enhancement patterns, lesion location and number, and associated features (e.g., edema, mass effect, hemorrhage, and leptomeningeal involvement) using a standardized template.

Steroid Exposure

Steroid exposure was defined as documented inpatient and outpatient administration of CST before biopsy. Patients were classified as CST-naive (no steroids before biopsy) or CST-exposed (received steroids prebiopsy). For CST-exposed patients, the interval between the last steroid dose and biopsy was recorded and grouped as follows: ≤48 hours (0-2 days), 48-72 hours (>1 to ≤3 days), and >72 hours. Duration of CST use before biopsy was also recorded and categorized as ≤5 or >5 days. To assess cumulative CST exposure, prebiopsy dexamethasone-equivalent doses were extracted and grouped into 3 categories: ≤20 mg, >20-40 mg, and >40 mg.

Location Classification

Biopsy targets were classified based on anatomical location. Deep lesions included those located in the thalamus, basal ganglia, corpus callosum, hypothalamus, or splenium. Superficial lesions encompassed cortical, subcortical, cerebellar, insular, parietal, temporal, frontal, or occipital regions.

Outcomes

The primary outcome was diagnostic yield, defined as the ability of SBB to provide a conclusive histopathological diagnosis, and was calculated as the percentage of patients with PCNSL who received a definitive diagnosis on initial SBB. Secondary outcomes included SBB-related complications: hemorrhage, seizure, infection, or neurological deficits.

Statistical Analysis

Descriptive statistics were computed using RStudio. The Fisher exact test and Welch *t*-test were used for categorical and continuous variables, respectively. Diagnostic yield across steroid timing categories was assessed using the χ^2 tests. Multivariate logistic regression was not performed due to the limited number of nondiagnostic cases. A *P*-value <.05 was considered significant.

RESULTS

Figure 1 presents the chart review results. Of 776 patients initially identified, 51 who underwent non-SBB procedures were excluded. Of the remaining 725 SBB cases, 621 patients with non-PCNSL diagnoses were excluded, leaving 104 patients with confirmed PCNSL. The diagnostic yield for PCNSL was 92.3% (96 diagnostic, 8 nondiagnostic biopsies). For non-PCNSL patients, diagnostic yield was 95.0% (590 diagnostic, 31 nondiagnostic). Gliomas (66%) were the most common non-PCNSL diagnosis, followed by metastases (7%), abscesses (4%), demyelinating lesions (3%), normal brain (1%), and others (19%) (Figure 2).

Patient Characteristics

Table 1 summarizes patient data. The median age at diagnosis was 67.5 ± 12.0 years; 51% were female. All patients had histologically confirmed large B-cell lymphoma. Deep lesions were present in 12.5%. Comorbidities included HIV (1.9%), extracranial lymphoma (28.8%), and transplant history (5.8%). Common medical conditions included hypertension (67.3%), dyslipidemia (49.0%), and diabetes (29.8%). No significant differences were observed between CST-exposed and CST-naive groups.

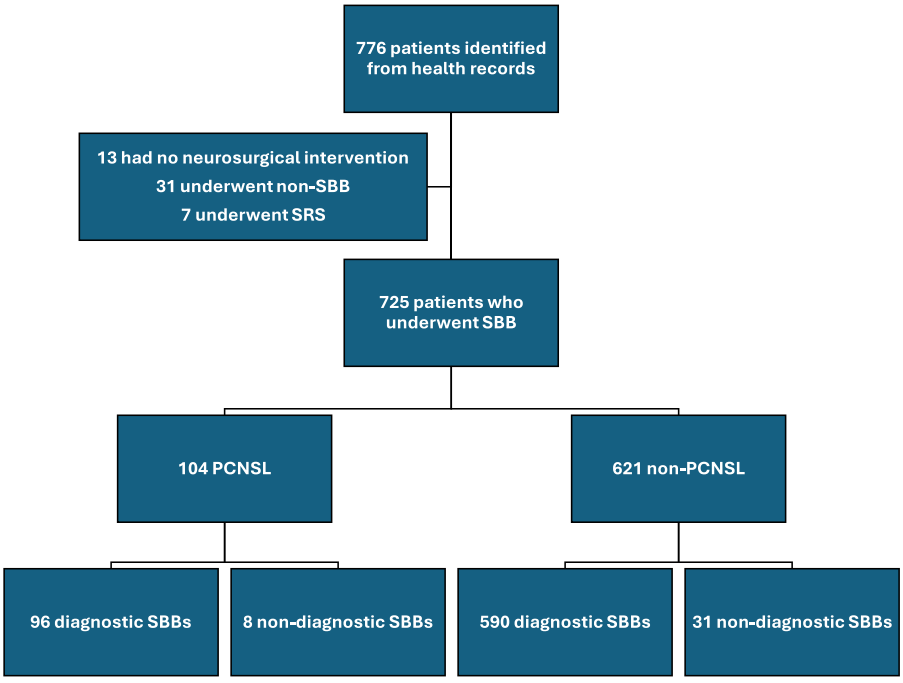

FIGURE 1. *Patient selection and diagnostic yield of SBB in PCNSL and non-PCNSL lesions. PCNSL, primary central nervous system lymphoma; SBB, stereotactic brain biopsies; SRS, stereotactic radiosurgery.*

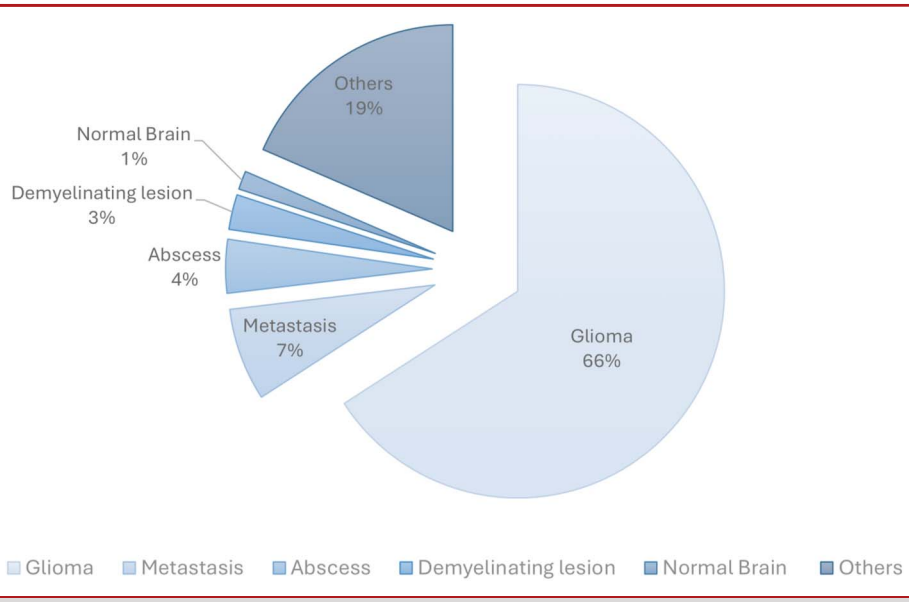

FIGURE 2. *Distribution of histopathological diagnoses from non–primary central nervous system lymphoma lesions (n = 590).*

TABLE 1. Demographic and Clinical Features of PCNSL Patient Cohort

Variable	All (n = 104)	No preoperative CST (n = 61)	With preoperative CST (n = 43)	*P*-value
Age at biopsy (mean ± SD)	67.51 ± 12.04	62.2 ± 20.8	67.8 ± 16.6	.13
Sex (female)	51%	47.5%	58.1%	.32
Diagnostic yield (%)	92.3%	90.2%	95.3%	.46
Histology	Large B-cell lymphoma (100%)	Large B-cell lymphoma (100%)	Large B-cell lymphoma (100%)	—
Deep location (%)	12.5%	16.4%	7.0%	.23
Postoperative complications (%)	4.8%	4.91%	4.65%	.84
HIV positive (%)	1.93%	3.3%	0%	.51
Extracranial lymphoma	28.84%	29.51%	27.91%	—
Transplant history (%)	5.77%	8.2%	2.3%	.40
Hypertension (%)	67.3%	65.6%	69.8%	.44
Diabetes mellitus (%)	29.8%	29.5%	30.2%	.64
Dyslipidemia (%)	49.03%	49.2%	48.8%	.66
Mean cumulative steroid dose (mg)		0	33.2 ± 38.43[a]	—
Mean duration of steroid before biopsy (d)		—	3.27 ± 2.14	—
Days since last dose (mean)		—	1.3	—

CST, corticosteroid therapy; PCNSL, primary central nervous system lymphoma.
[a]2 patients who received steroids had methylprednisolone and were not included in the cumulative dose calculation.

Radiographic Characteristics

MRI was available in 96.1% (100/104) of cases. Homogeneous enhancement was most common (65%), followed by heterogeneous (35%). Lesions were frequently periventricular (78%), with other sites including basal ganglia (45%), corpus callosum (32%), thalamus (18%), and cerebellum (15%). Associated features included vasogenic edema (89%), multifocality (73%), mass effect/midline shift (61%), hemorrhage (12%), and leptomeningeal/dural involvement (9%).

Postoperative Complications

Complications occurred in 4.8% of patients, including 4 hemorrhages and 1 ocular abnormality. Complication rates were similar between CST-exposed (4.7%, 95% CI: 0.6%-15.8%) and CST-naive patients (4.9%, 95% CI: 1.0%-13.7%) (P = .84; Table 1).

CST Among Treated Patients

Of the 104 patients included in the final cohort, 43 (41.3%) received preoperative CST, while 61 (58.7%) did not (Table 1). Dexamethasone was the predominant agent (98%). Dosing schedules varied, with the most common being every 6 hours (Q6, n = 21), followed by Q12 (n = 7), Q8 (n = 4), Q24 (n = 1), and Q4 (n = 1). Seven patients received a single dose. Among CST-exposed patients, the mean duration of prebiopsy steroid use was 3.27 ± 2.14 days, and the mean interval since the last dose was 1.3 days. Regarding cumulative prebiopsy dexamethasone dosage: 22 patients (54%) received ≤20 mg, 9 (22%) received 21-40 mg, and 10 (24%) received >40 mg.

Diagnostic Yield and the Role of Corticosteroid Timing and Duration Before Biopsy

Diagnostic yield did not differ significantly by corticosteroid exposure or timing. Among CST-naïve patients, the diagnostic yield was 90.2% (55/61; 95% CI: 79.8%-96.3%), compared with 95.3% (41/43; 95% CI: 84.2%-99.4%) in CST-exposed patients (P = .46; Table 1). Diagnostic yield remained consistently high across all cumulative dexamethasone dose categories: 95.5% for ≤20 mg (95% CI: 78.2%-99.2%), 100% for 21-40 mg (95% CI: 70.1%-100%), and 100% for >40 mg (95% CI: 70.1%-100%) (P = 1.0; Table 2). Similarly, diagnostic success did not differ by duration of corticosteroid exposure: 94.6% for ≤5 days (95% CI: 81.8%-99.3%) vs 100% for >5 days (95% CI: 91.8%-100%) (P = 1.0; Table 3A). Time from last steroid dose to biopsy was not associated with diagnostic failure: yield was 96.4% for ≤48 hours, 91.7% for 48-72 hours, and 100% for >72 hours (P = .66; Table 3B).

TABLE 2. Diagnostic Yield by Cumulative Dexamethasone Dose

Cumulative dexamethasone dose (mg)	Total patients[a]	Diagnostic	Nondiagnostic	Diagnostic yield (%)
≤20 mg	22	21	1	95.5
21-40 mg	9	9	0	100
>40 mg	10	10	0	100

[a]Excluded 2 patients who received methylprednisolone.

Of the 8 patients with nondiagnostic initial SBBs (Table 4), 2 received CST before biopsy. One received a single 14 mg IV dose of dexamethasone, discontinued 2 days before biopsy; the other received high-dose IV methylprednisolone (32 mg every 8 hours for 4 days; total 324 mg) without taper. The remaining 6 patients were steroid naïve. Initial pathology revealed findings such as gliosis, reactive astrocytosis, chronic inflammation, or atypical B-cell infiltrates, all interpreted as nondiagnostic. Ultimately, all 8 patients were confirmed to have PCNSL through repeat SBB (n = 6), CSF cytology (n = 1), or postmortem examination (n = 1). Notably, 2 patients (Patients 1 and 3) received low-dose dexamethasone while awaiting second biopsy: one also received rituximab, which was later discontinued, and another was on tacrolimus at the time of initial biopsy. No postoperative complications were reported in this subgroup, supporting the safety and potential diagnostic value of repeat biopsy in selected cases.

DISCUSSION

The optimal approach to preoperative CST in patients undergoing SBB for suspected PCNSL weighs neurological deficit against the possibility of nondiagnostic biopsy. This study contributes to this discussion by analyzing corticosteroid use including dose, timing, frequency, and discontinuation, in a large, homogeneous cohort with pathologically confirmed PCNSL who underwent SBB. These findings are interpreted in the context of existing literature to offer important clinical insights.

Diagnostic Yield and Comparison with Prior Literature

Our diagnostic yield of 92.3% aligns with prior reports (88%-96%) for SBB in PCNSL.[24-26] This level of accuracy also aligns with our findings from non-PCNSL cases (95%) and broader results for SBB in other intracranial lesions with reported yields ranging from 90% to 99.3%.[27-29] SBB remains the preferred modality due to its minimally invasive nature and ability to access deep lesions.[30]

Previous studies reported lower yields ranging from 26% to 48%, attributed to variation in surgical technique, imaging guidance, and pathological interpretation.[31,32] Preoperative CST is frequently cited as a key factor, as it can induce tumor regression and obscure histological features.[33] Multiple studies have raised concerns about corticosteroid use before biopsy. For instance, Scheichel et al[34] (2021) reported a of 22.2% failure rate in CST-exposed vs 7.7% in steroid-naïve patients, with pathological evidence indicating a marked reduction in atypical lymphoid cells in the corticosteroid-treated group. A meta-analysis of 463 patients reported nondiagnostic rates of 19% with CST vs 5.7%

TABLE 3. Diagnostic Yield by Duration of CST (A) and Timing of Last Steroid Dose (B)

A.

CST duration	Total patients	Diagnostic	Nondiagnostic	Diagnostic yield (%)
≤5 d	37	35	2	94.6
>5 d	6	6	0	100

B.

Timing of last dose	Total patients	Diagnostic	Nondiagnostic	Diagnostic yield (%)
<48 h	28	27	1	96.4
48-72 h	12	11	1	91.7
>72 h	3	3	0	100

CST, corticosteroid therapy.

TABLE 4. Detailed Report of all 8 PCNSL Patients Who Initially Had Nondiagnostic SBB

Patient	Sex	Age at biopsy (y)	Biopsy location	Steroid exposure before initial biopsy	Initial biopsy surgery	Initial pathology report	Steroid exposure after initial biopsy	Follow-up report type	Follow-up pathology report	Postoperative complications	Immunosuppressants before biopsy
Patient 1	F	62	Cerebellar	No steroids were given	SBB	Atypical B-cell infiltrate	Pt discharged on dexamethasone 2 mg Q12h PO until second biopsy	Postmortem	B-cell lymphoma of brain lesion	None	Tacrolimus
Patient 2	M	88	Frontal	Methylprednisolone; 32 mg Q8H; IV; total dose of 324 mg over 4 d; No tapering before biopsy	SBB	Brain tissue with numerous macrophages and chronic inflammation	No steroids were given	CSF analysis 2 d later	Atypical cells highly suggestive of PCNSL	None	No
Patient 3	F	61	Frontal	Dexamethasone; 14 mg once; IV; stopped 2 d before biopsy	SBB	Gliosis	Pt discharged on dexamethasone 4 mg Q8h PO until second biopsy	SBB 2 wk later	CD-20 positive large B-cell lymphoma	None	No
Patient 4	F	64	Temporal	No steroids were given	SBB	Reactive astrocytosis	No steroids were given	SBB 1 mo later	CD-20 positive large B-cell lymphoma	None	No
Patient 5	M	43	Frontal	No steroids were given	SBB	Brain parenchyma with gliosis and scattered T-cells	No steroids were given Discontinued rituximab until second biopsy	SBB 5 wk later	CD-20 positive large B-cell lymphoma	None	Rituximab
Patient 6	F	69	Splenium	No steroids were given	SBB	Atypical B-cell rich inflammatory demyelinating lesion	No steroids were given	SBB 6 mo later	Primary CNS lymphoma, large B-cell type	None	No
Patient 7	M	40	Thalamus	No steroids were given	SBB	Inflammatory demyelinating lesion	No steroids were given	SBB 10 mo later	CD-20 positive large B-cell lymphoma	None	No
Patient 8	F	59	Parietal	No steroids were given	SBB	White matter with mild gliosis	No steroids were given	SBB 2 wk later	CD-20 positive large B-cell lymphoma	None	No

PCNSL, primary central nervous system lymphoma; PO, per os; SBB, stereotactic brain biopsies.

without, nearly a 3-fold increase in risk.[35] Collectively, these data have shaped a prevailing clinical recommendation to withhold corticosteroids, when clinically feasible, in patients undergoing biopsy for suspected PCNSL.[19] Realistically, many patients with suspected PCNSL receive CST before their diagnosis often due to neurological compromise. These scenarios are complex, and a variety of measures to taper steroids or withhold them as long as feasible before biopsy have been proposed.[25,36-38] The mechanism by which corticosteroids induce cytoreduction in PCNSL, primarily through glucocorticoid receptor–mediated proapoptotic signaling and subsequent tumor regression, is illustrated in Figure 3.

In our cohort, diagnostic yield remained high regardless of CST duration (≤5 vs >5 days), timing (≤48, 48-72, >72 hours), or cumulative dose (≤20, 21-40, >40 mg). None of these differences reached statistical significance.

These results stand in contrast to earlier work by Manoj et al[39] (2014), who reported a decline in diagnostic yield with corticosteroid exposure exceeding 7 days, suggesting that prolonged CST may obscure key histological features. In our cohort, even short-interval or moderate-dose corticosteroid use did not significantly affect diagnostic success. Our findings are consistent with more recent literature in smaller cohorts. Tosefsky et al[35] (2024) found no significant relationship between the interval of steroid cessation and biopsy success. Bullis et al demonstrated that all 18 patients who continued corticosteroids up to the time of biopsy achieved successful diagnoses, and Binnahil et al (2016)

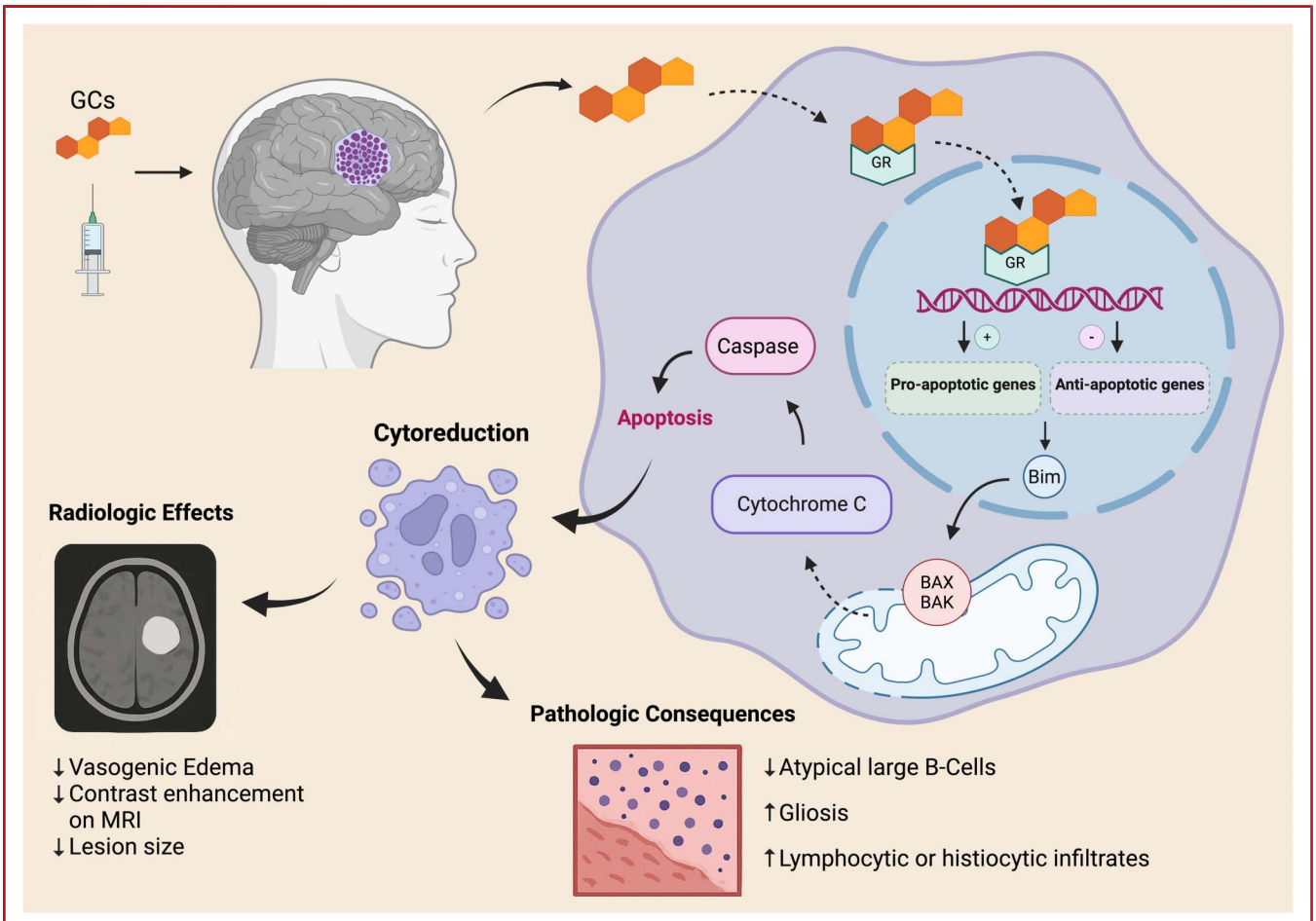

FIGURE 3. *Mechanism of corticosteroid-induced regression in PCNSL. GCs induce apoptosis in lymphoma cells primarily through GR-mediated transcriptional reprogramming of Bcl-2 family proteins. On binding cytoplasmic GR, the receptor translocates to the nucleus and binds transcription factors, upregulating proapoptotic genes while suppressing antiapoptotic genes. This imbalance activates Bax/Bak, triggering mitochondrial outer membrane permeabilization and cytochrome c release. Cytochrome c binds to other factors to form the apoptosome complex, activating caspases which dismantle the cell through DNA fragmentation and structural degradation, ultimately leading to cytoreduction of the tumor. Radiologically, GCs reduce vasogenic edema, contrast enhancement on MRI, and overall lesion size. Pathological consequences include diminished numbers of atypical large B cells, increased gliosis, and increased lymphocytic or histiocytic infiltrates. BAK, BCL2-antagonist/killer; BAX, Bcl-2-associated X; GCs, Corticosteroids; GR, glucocorticoid receptor; PCNSL, primary central nervous system lymphoma. Created in BioRender. Ghoche, M. (2025) https://BioRender.com/ffbzezd.*

found that 15 of 16 corticosteroid-treated patients were successfully diagnosed on first biopsy attempt.[40,41]

Unlike earlier studies that reported adverse effects often associated with prolonged or uninterrupted CST, our cohort largely received short-course steroids (mean duration 3.27 days), with biopsy typically occurring within 1-2 days of the last dose. This shorter exposure period may have minimized histological regression.

The 8 patients with initially nondiagnostic SBBs (Table 4) represent an instructive subgroup. Notably, only 2 of these received corticosteroids before the initial biopsy, while 6 were steroid-naïve, supporting our finding that steroid exposure alone is not the dominant factor in diagnostic failure. Pathology in these cases often showed gliosis, reactive astrocytosis, or nonspecific inflammation, which can obscure lymphoma. In some, factors such as rituximab exposure (e.g., Patient 5), deep or eloquent lesion locations (e.g., thalamus, splenium), or small sampling volumes may have contributed. All 8 were ultimately diagnosed through repeat biopsy, CSF cytology, or autopsy. No postoperative complications occurred in this group, supporting repeat biopsy when needed.

CST protocols were not standardized and were based on clinician judgment, reflecting real-world variability. This heterogeneity enabled stratified analysis by timing and dose to evaluate their effect on diagnostic yield. Although corticosteroid regimens varied widely in dose, frequency, and use of tapering, we did not observe a consistent pattern correlating specific CST regimens with biopsy failure. One of the most frequent scenarios encountered is a single large dose of CST, followed by initiation of scheduled dosing of CST given at a nontertiary site or before neurosurgical evaluation. This raises concern that diagnostic yield may be immediately affected. We hope that this manuscript can address this clinical scenario in that successful diagnostic outcomes were observed across both tapered and nontapered protocols, supporting the conclusion that regimen variability did not substantially affect yield in this cohort.

Given the potential for corticosteroids to induce transient lesion regression or reduced contrast enhancement, accurate biopsy targeting may be challenged. At our institution, stereotactic planning is based on contrast-enhanced MRI performed within 24-48 hours before biopsy, which typically is after the initiation of CST if neurological deficit requires treatment initiation, or if CST is started at an external care site. This standardized imaging-procedural coordination may partially explain the consistently high diagnostic yield observed in our study despite CST exposure.

These findings suggest that nondiagnostic biopsies result from multifactorial causes. Factors such as concurrent immunosuppressants, biopsy location, lesion heterogeneity, and procedural variables may be more influential than CST alone. For example, mistargeting has been identified as a primary contributor to nondiagnostic results in other series.[28,34]

Limitations

Our study has several strengths, including a large, well-characterized cohort of pathologically confirmed PCNSL patients diagnosed exclusively through SBB over 10 years. Our granular data set included clinical, radiographic, surgical, and histopathological details, enabling comprehensive CST analysis. We examined timing, dose, tapering, and outcomes beyond binary CST status.

Limitations include the retrospective design, which may introduce documentation and selection bias. Small subgroup sizes (e.g., >72 hours, >40 mg) limited statistical power. Few nondiagnostic events precluded multivariable modeling. Procedural variables such as number of biopsy passes or operator experience were not uniformly recorded, which may influence diagnostic success. Given the 10-year duration of the study, it is also possible that procedural refinements, such as improvements in stereotactic navigation or intraoperative targeting occurred over time, we acknowledge the potential influence of technological advancements on diagnostic performance. It is plausible that such improvements may have contributed in part to the consistently high diagnostic yield observed in both steroid-exposed and steroid-naïve patients, although this effect cannot be directly measured from our data.

CONCLUSION

Our findings suggest that preoperative CST does not significantly impair the diagnostic yield of SBB in patients with PCNSL. Diagnostic yield remained consistently high across a range of steroid doses and timing intervals. These findings suggest that short-course CST, often necessary for symptom control, may be safely administered before biopsy in select patients. Future prospective studies incorporating standardized corticosteroid protocols, serial imaging, and coordinated biopsy workflows may help clarify best practices and minimize clinical variability in the diagnostic management of suspected PCNSL.

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COMMENTS

This paper provides a valuable contribution to our understanding of the impact of preoperative corticosteroid therapy (CST) on the diagnostic yield of stereotactic brain biopsy (SBB) in patients with primary central nervous system lymphoma (PCNSL). The study's findings are reassuring, as they demonstrate that diagnostic outcomes remain high despite varying doses and durations of steroid exposure. This suggests that corticosteroids, while often necessary for symptom management, may not negatively impact the success of biopsy procedures as much as previously believed.

A particularly significant takeaway from the paper is that even in patients with extended or high-dose CST exposure, diagnostic success was largely unaffected. This challenges the conventional thinking that CST could induce lesion regression or reduce contrast enhancement to the point of rendering biopsies nondiagnostic. The study highlights that,

although CST is a key factor in the treatment of PCNSL, it may not be as detrimental to biopsy outcomes as once thought, opening up new possibilities for managing patients preoperatively.

The study also highlights the multifactorial nature of nodiagnostic biopsies. Factors such as lesion heterogeneity, biopsy location, and procedural issues like miss-targeting may be more critical than CST exposure alone. This insight is crucial, as it emphasizes that the technical aspects of the procedure and the inherent complexity of the lesions may contribute more significantly to biopsy failure than the use of steroids.

While the study provides strong evidence supporting the safe use of corticosteroids before biopsy, there are some areas for future investigation. The retrospective design and small subgroup sizes limit the ability to draw firm conclusions, and further research incorporating standardized protocols, more detailed procedural data, and larger cohorts would be beneficial. In addition, exploring the impact of technological advancements in stereotactic navigation and intraoperative techniques may help refine biopsy strategies further.

In conclusion, the study offers promising findings for clinicians managing PCNSL patients and underscores the need for continued exploration of best practices in biopsy procedures. With more prospective studies and detailed data, we can continue to improve the diagnostic approach to this complex and challenging condition.

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