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Dural Lymphomas Unmasked: A Narrative Review of Extra-Axial Mimics and a Pragmatic Diagnostic Decision Algorithm

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Simple Summary

Primary dural lymphomas (PDL) are uncommon extranodal non-Hodgkin lymphomas arising from dura mater. Most of the published guidance on their management is extrapolated from systemic lymphomas involving the central nervous system. On imaging, dural lymphomas may resemble benign meningiomas, metastases, or inflammatory disorders that thicken the dura. Accordingly, most patients initially undergo aggressive surgical intervention instead of limited diagnostic procedures. This review summarizes current knowledge on PDL, describes the most common disease subtypes (indolent histologies versus aggressive disease confined to the dura or secondary involvement), and highlights the value of a multidisciplinary, integrated approach combining conventional imaging with advanced magnetic resonance features, alongside neurosurgical, and onco-hematological expertise. We propose a step-by-step diagnostic approach to support timely and limited biopsy, staging, and treatment selection, with the aim of reducing misdiagnosis, avoid unnecessary extensive surgery, and accelerate appropriate therapy.

Abstract

Dural-based lymphomas are rare extranodal non-Hodgkin lymphomas that span from indolent primary dural entities, most commonly extranodal marginal zone (mucosa-associated lymphoid tissue) lymphoma, to aggressive variants that are primary or represent secondary involvement from systemic lymphoma. They overlap radiologically with benign meningiomas, dural metastases, and immune-mediated pachymeningitis, creating a risk of anchoring bias, delayed diagnosis, and unnecessarily extensive resections when limited tissue sampling would be sufficient for diagnosis. We conducted a structured narrative review to synthesize the epidemiology, clinico-pathological classification, imaging phenotypes, and management principles of dural lymphomatous disease. This study will especially focus on primary dural lymphoma (PDL) with additional discussion of secondary dural involvement. Emphasis is placed on the clinical value of a multiparametric diagnostic approach that integrates computed tomography contrast-enhanced magnetic resonance imaging with functional techniques, and fluorodeoxyglucose positron emission

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tomography/computed tomography for systemic staging and for distinguishing truly localized primary dural lymphoma from secondary involvement. Potential diagnostic pitfalls related to somatostatin receptor–based tracer uptake will also be discussed. We introduced a pragmatic operational framework based on four clinico-biological clusters, translated into a step-by-step decision algorithm that prioritizes timely biopsy and comprehensive hematologic staging to guide surgical strategy. Given the absence of dedicated, multidisciplinary guidance for dural-based lymphomas, this algorithm is intended as a reproducible foundation for consensus recommendations and future multicenter validation.

Keywords: dura mater; meningeal neoplasms; primary dural lymphoma; central nervous system neoplasms; meningioma diagnosis; differential magnetic resonance imaging; lymphoma; non-hodgkin

1. Introduction

Dural-based lymphomas are rare but clinically relevant, ranging from (i) indolent primary dural lymphoma (PDL) —most commonly extranodal marginal zone lymphoma (EMZL)/mucosa-associated lymphoid tissue (MALT) lymphoma—to (ii) aggressive disease that may be primary dural (less often) or reflect dural involvement within secondary CNS lymphoma (SCNSL). These entities frequently share a "meningioma-like" radiological phenotype and therefore carry a non-negligible risk of delayed diagnosis and anchoring bias [1–3].

This distinction is not merely nosological but carries immediate management implications. When clinical and radiological red flags raise suspicion for a "meningioma-mimicking lesion" consistent with lymphoma, anchoring bias should be actively avoided and the diagnostic work-up promptly redirected towards histopathologic confirmation and appropriate staging.

Accurate classification (PDL EMZL/MALT, PDL non-EMZL/MALT including dural DLBCL, and secondary dural involvement/SCNSL) has direct consequences for the diagnostic work-up (extent of systemic staging and CSF assessment according to context), radiotherapy and surgical decision-making (biopsy vs resection), the need for CNS-directed chemotherapy (particularly high-dose methotrexate [HD-MTX]-based regimens in aggressive cases), and follow-up strategies [4,5].

The 5th edition of the WHO classifications for CNS tumors and hematolymphoid neoplasms, together with international EHA–ESMO guidance for primary and secondary central nervous system lymphoma (PCNSL/SCNSL), provides the conceptual framework within which dural lymphomas should be positioned across the spectrum of CNS lymphoma and SCNSL [4,6–8]. However, these documents devote limited space to the specificities of pachymeningeal disease. Consequently, diagnostic and therapeutic strategies for dural lesions are often extrapolated from the far more abundant evidence on parenchymal presentations. At present, there are no dedicated guidelines for the management of dural lymphomatous lesions.

The primary aim of this narrative review, intended for a multidisciplinary neuro-oncological audience, is to summarize available evidence on epidemiology, imaging patterns (conventional and advanced MRI; 18F-FDG PET and, in selected settings, somatostatin receptor [SSTR] tracers), histopathological correlates, and management strategies for dural lymphomas, with particular emphasis on PDL. We further propose a pragmatic, multidisciplinary operational framework comprising four clinico-biological clusters, along with a diagnostic pathway that integrates MRI and PET findings with hematologic staging, aimed at minimizing anchoring bias and avoiding unnecessary surgical resections.

2. Materials and Methods

Study Design and Objectives

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Owing to the rarity of dural lymphomas and the heterogeneity of reported data—frequently nested within PCNSL or SCNSL series—we conducted a structured narrative review and critical synthesis with the following objectives: (1) define an operational nosological framework distinguishing PDL from secondary dural involvement/SCNSL; and (2) propose an integrated decision pathway—including hematologic staging—supporting differential diagnosis, therapeutic stratification, and treatment planning [1,3,6]. Within this framework, we emphasize multimodal imaging analysis, leveraging morphological and metabolic patterns to guide pre-biopsy diagnostic orientation in "meningioma-like" presentations and to support differential diagnosis against major extra-axial mimics.

Data Sources and Search Strategy

We searched PubMed/MEDLINE, Embase, and Scopus from database inception to [LAST SEARCH DATE], using combinations of free-text terms and, for PubMed, MeSH terms and keyword equivalents. Boolean operators (AND, OR, NOT) and truncation (*) were used to refine results. Search strings included, among others: dural lymphoma, primary dural lymphoma, dural marginal zone, MALT lymphoma dura, pachymeningeal lymphoma, secondary CNS lymphoma, meningioma mimics, diffusion, perfusion, FDG PET, and somatostatin receptor PET. The search was designed to map the evidence base and identify recurring themes (imaging and early management) rather than to produce quantitative estimates or pooled effect sizes. An example PubMed string is provided in the Supplement (Table S1).

Study Selection and Inclusion/Exclusion Criteria

We included: (1) observational studies (single-center or multicenter case series) reporting histotype, imaging, treatment, and outcomes; (2) reviews, position papers, and guidelines relevant to PCNSL/SCNSL and diagnostic–therapeutic frameworks; and (3) neuroradiological studies focused on imaging patterns and differential diagnosis of dural masses and/or CNS lymphoma. We excluded studies in which the lymphoma was predominantly intra-axial (parenchymal) and lacked a primary or dominant dural component; conference abstracts without full data; isolated case reports without additional management or radiological value; and non-English papers (except highly clinically relevant exceptions, documented).

Screening and Data Extraction

Two authors independently screened titles and abstracts and assessed full-text articles; disagreements were resolved by consensus. Extracted variables included: setting/denominator (PCNSL/SCNSL/systemic lymphoma cohorts), criteria for "dural-based disease," histotype (EMZL/MALT vs DLBCL vs others), location (convexity/falx/skull base/spinal), MRI patterns (DWI/ADC; perfusion when available), PET (FDG ± SSTR), treatment (surgery/radiotherapy/chemotherapy), and outcomes (response, local/systemic relapse, and overall or progression-free survival when reported).

Synthesis and Reporting

We organized the synthesis according to the framework: PDL (EMZL/MALT vs non-EMZL/MALT) versus secondary dural involvement (aggressive versus disseminated indolent lymphoma), focusing on practical consequences for diagnosis, staging, and treatment selection. A summary table of key clinical series is provided in Table 1. Given heterogeneous designs and denominators (PDL series vs cohorts derived from PCNSL/SCNSL), we did not perform a formal risk-of-bias assessment; instead, we prioritized operational definitions, reproducible imaging descriptors, and clinical and staging context.

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Table 1. Major published case series of dural lymphoma: A. Primary dural lymphoma / intracranial marginal zone lymphoma (meningioma-like) series; B. Secondary CNS lymphoma with dural/pachymeningeal involvement (SCNSL / secondary dural involvement).

Study (year)	Design / setting	N	Histology / cohort	Treatment / management	Key outcomes / notes
A. Primary dural lymphoma / intracranial marginal zone lymphoma (meningioma-like) series					
Tu et al. (2005) [34]	Clinicopathologic /genetic series: intracranial MZL mimicking meningioma.	15 (14/15 dural-based masses)	Marginal zone B-cell lymphoma; plasmacytoid differentiation common; trisomy 3 frequent.	Surgery/biopsy for diagnosis; RT and/or chemotherapy (as reported).	For cases with follow-up (10): no evidence of disease at 1-7.6 years.
Iwamoto (2006) [9]	PDL treatment/outcome series	8	Low-grade MZL in all cases	RT in all; chemotherapy in 3 (adjuvant/combined)	Complete response in all; no local failures; systemic relapse described in 3 (late extracranial disease)
Venkataraman (2011) [10]	Pathology-anchored series of dural MZL (IgG4 link explored)	32	MZL involving meningeal dura; IgG4-positive plasma cells in a subset	Treatment heterogeneous in reported cohort	For cases with follow-up available (17): 16/17 alive, disease-free (4-124 months; median ~19.5 months)
de la Fuente (2017) [11]	Multi-institution experience (MSKCC + Univ. Miami): dural marginal zone lymphoma	26	Predominantly MZL/EMZL (dural)	Biopsy/surgery common for diagnosis; focal RT frequently used; systemic therapy in selected	Evaluable (n=23): ORR 100%, CR 22/23; 3-year PFS 89%, 5-year PFS 76%, 5-year OS 92%; relapses reported (local and systemic)
Sunderland et al. (2020) [33]	International multicenter retrospective analysis: EMZL with CNS/dural involvement.	26	EMZL with histologically confirmed CNS/dural involvement; primary CNS MZL (n=15) vs secondary CNS MZL (n=11).	Heterogeneous; surgery/biopsy + RT and/or systemic therapy.	Dura most common site (50%); 2-year PFS 59%, OS 80%; secondary CNS MZL: 2-year PFS 54%, OS 58%.
Karschnia (2020) [1]	Bi-institutional neuro-oncology cohort (PDL within PCNSL denominator)	20	Mixed: DLBCL 10/20, MZL 6/20, follicular 2/20, other 2/20	Individualized: surgery/biopsy + adjuvant RT and/or systemic therapy (including CNS-directed regimens for aggressive histology)	Best response evaluable (n=16): response 88%, CR 69%; median follow-up 44 months; 3 deaths from progression
B. Secondary CNS lymphoma with dural/pachymeningeal involvement (SCNSL / secondary dural involvement)					
Hollender et al. (2000) [27]	Single-center retrospective cohort (systemic NHL with CNS involvement).	140	Systemic NHL with CNS involvement at diagnosis/relapse /progression.	Chemo +/- RT; HDT used in a small subset (as reported).	Median survival after CNS diagnosis 2.6 months; better prognosis when CNS involvement at initial diagnosis; paresis predicted survival.

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Study (year)	Design / setting	N	Histology / cohort	Treatment / management	Key outcomes / notes
Spectre et al. (2005) [36]	Retrospective single-center series (indolent B-cell lymphomas with CNS involvement).	7 patients.	Indolent B-cell lymphomas with CNS involvement; parenchymal (2), leptomeningeal (3), both (2); systemic disease in all (bone marrow in all but one).	Systemic + intra-CSF chemotherapy in 6/7; radiotherapy in 2.	CNS response in 5 patients; survival 1-9 years for treated patients (median 2 years); 3 died of CNS disease.
Jahnke et al. (2006) [28]	Single-center retrospective prognostic-factor study.	43	NHL with secondary CNS involvement.	IT chemotherapy 77%; systemic chemotherapy 58%; RT 37%.	Median survival 5 months; low LDH and response to CNS therapy associated with improved survival.
Ferreri et al. (2015) [29]	Multicenter phase II trial: HD-MTX/Ara-C -> R-HDS -> ASCT (+ IT liposomal Ara-C).	38	Aggressive B-cell lymphoma with secondary CNS involvement (diagnosis or relapse).	HD-MTX + cytarabine; rituximab; R-HDS; ASCT in eligible responders.	CR 63%; 2-year EFS 50% +/- 8%; 5-year OS 41% +/- 8% overall and 68% +/- 11% in transplanted; 4 treatment-related deaths.
El-Galaly et al. (2018) [25]	Large international retrospective cohort (registries/databases; immunochemotherapy era).	291 patients with SCNS involvement by DLBCL.	DLBCL with secondary CNS involvement during/after first-line; CNS sites categorized (parenchymal, leptomeningeal, both) but dural not separated in abstract.	Curative/intensive therapy in a subset (e.g., HD-MTX- or platinum-containing regimens); variable use of rituximab and HDC/ASCT in responders.	Median post-SCNS OS 3.9 months; 2-year OS 20% (95% CI 15-25). In patients <=60 with PS 0-1 treated with HD-MTX-based regimens for isolated parenchymal SCNS: 2-year OS 62% (95% CI 36-80).
Malikova et al. (2018) [32]	Retrospective imaging series: morphological MRI patterns in SCNSL.	21	SCNSL (histologically verified); steroids at MRI in 12/21.	Not a treatment study (diagnostic imaging focus).	Parenchymal lesions 86%; combined parenchymal + meningeal involvement 48%; diffusion restriction in 78% of parenchymal lesions.
Ferreri et al. (2021) [30]	MARIETTA trial (international, single-arm phase II): MATRix-RICE + ASCT.	79 enrolled; 75 assessable	DLBCL with secondary CNS involvement.	MATRix x3 -> RICE x3; carmustine/thiotepa-conditioned ASCT in responders.	1-year PFS 58%; 2-year PFS 46% overall and 83% in transplanted; 2-year OS 46% overall and 83% in transplanted.
Habringer et al. (2024) [31]	International prospective registry (SCNSL-R; NCT05114330).	279 enrolled; 243 analyzed	SCNSL of all entities (DLBCL 68%); synchronous 20% vs metachronous 80%.	Real-world induction regimens; HDT-ASCT consolidation in responders (PR or better) associated with OS benefit.	Median OS 17.2 months; longer OS in synchronous cohort (60.6 months) vs metachronous (11.4 months); HDT-ASCT in responders: adjusted HR 0.47 for OS.

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Study (year)	Design / setting	N	Histology / cohort	Treatment / management	Key outcomes / notes

Abbreviations: PDL=primary dural lymphoma; MZL=marginal zone lymphoma; EMZL=extranodal marginal zone lymphoma; DLBCL=diffuse large B-cell lymphoma; SCNSL=secondary CNS lymphoma; CNS=central nervous system; RT=radiotherapy; HD-MTX=high-dose methotrexate; Ara-C=cytarabine; IT=intrathecal; ASCT=autologous stem-cell transplantation; HDT-ASCT=high-dose therapy with ASCT; CR=complete response; PR=partial response; EFS=event-free survival; PFS=progression-free survival; OS=overall survival; R-HDS=rituximab plus high-dose sequential chemoimmunotherapy; MATRix=rituximab, methotrexate, cytarabine, thiotepa; RICE=rituximab, ifosfamide, carboplatin, etoposide.

3. Operational Definition and Classification

Dural involvement in CNS lymphoma lies between primary CNS lymphoma and the SCNSL spectrum, yet its rarity and exclusion from prospective studies often lead to its integration within broader categories (PCNSL *sensu stricto* or generic SCNSL), with potential for improper treatment. A clear operational definition of PDL versus secondary dural involvement is therefore critical for any diagnostic–therapeutic algorithm [3,4,6,7].

We use "primary dural lymphoma (PDL)" to denote a lymphomatous neoplasm arising from the pachymeninges in the absence of detectable systemic disease on standard hematologic staging (e.g., whole-body PET/CT and/or CT chest–abdomen–pelvis; ± bone marrow biopsy depending on context). Available series suggest that the most frequently reported histotype is dural EMZL/MALT, biologically akin to MALT lymphomas at other sites, typically "meningioma-like" on imaging, and generally associated with favorable outcomes with locoregional approaches [1,2,9–11].

In neuro-oncological cohorts drawn from PCNSL denominators, the histologic distribution may include a comparatively higher proportion of dural DLBCL, plausibly reflecting selection effects and referral bias [1].

Less commonly, PDL presents with non-EMZL/MALT histotypes, most notably intracranial or spinal dural DLBCL (PD-DLBCL) or follicular lymphoma and other variants. In these cases, although the lesion is dural-based, clinical behavior and treatment intensity resemble more closely aggressive CNS lymphoma [1,11].

Variations in terminology and case composition reflect differences in WHO classification frameworks. In brief, WHO CNS5 includes "MALT lymphoma of the dura" among primary CNS lymphomas and promotes an integrated, layered diagnostic approach based on morphology, immunophenotype, molecular/genetic data, and clinico-radiological context [6]. Conversely, WHO-HAEM5 considers dural EMZL/MALT a site-associated variant of MALT lymphomas, emphasizing its biological affinity with extra-CNS extranodal MALT [7]. Practically, these perspectives support a cautious, integrative approach combining neuro-oncology and hematology: (1) complete systemic staging per hematology standards, not only to exclude secondary CNS involvement, but also to identify synchronous or occult extranodal sites; and (2) prolonged follow-up, given the possibility—albeit uncommon—of late extracranial relapse [9].

"Secondary dural involvement" refers to pachymeningeal disease occurring in the context of known or concurrently diagnosed systemic lymphoma, i.e., within SCNSL. Here, the dura is one possible compartment of disease alongside brain parenchyma, leptomeninges, cranial nerves, and/or spinal roots; by definition, an extra-CNS component is detectable on systemic staging [3,5]. Most commonly, it reflects systemic DLBCL with CNS involvement at presentation or relapse, although scenarios in indolent and other aggressive lymphomas have been reported [3]. Clinical management follows SCNSL principles and is associated with an overall poorer prognosis compared with most PDLs [3–5].

In the remainder of this review, we use "dural lymphoma" as an umbrella term encompassing primary and secondary dural disease, structured into four operational subgroups: (1A) PDL, EMZL/MALT ("classic" indolent form); (1B) PDL, non-EMZL/MALT (predominantly PD-DLBCL; less often follicular lymphoma or other variants); (2A) dural involvement in SCNSL from aggressive lymphomas (predominantly DLBCL); and (2B) dural involvement in disseminated indolent

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lymphomas. Although simplified, this grid provides a shared multidisciplinary language and facilitates integrated interpretation of presentation, imaging, and therapeutic choices [1,3,11]. To facilitate an operational, bias-resistant interpretation of "meningioma-like" pachymeningeal lesions, Table 2 summarizes the typical clinico-radiological profiles of the four dural lymphoma clusters, highlighting key discriminators, early diagnostic priorities (biopsy and hematologic staging), and initial management implications.

Table 2. Typical clinico-radiological profiles across the four operational clusters of dural lymphoma. The table summarizes, for each cluster, the usual clinical setting, presenting phenotype, key discriminator(s) used in day-to-day differential diagnosis, initial management priorities, and typical outcome patterns and follow-up implications. The schema is intended as a pragmatic aid to multidisciplinary decision-making and does not replace tissue diagnosis. PDL = primary dural lymphoma; EMZL = extranodal marginal zone lymphoma; MALT = mucosa-associated lymphoid tissue; DLBCL = diffuse large B-cell lymphoma; SCNSL = secondary central nervous system lymphoma; CNS = central nervous system; HD-MTX = high-dose methotrexate.

Cluster	Setting	Typical profile	Key discriminator	Initial management	Prognosis / follow-up
1A	Indolent PDL (extranodal marginal zone / MALT type)	Middle-aged (often female), immunocompetent; subacute mass-effect symptoms. Dural mass along convexity/falx/tentorium or skull base; frequently interpreted as meningioma.	Disease confined to dura at diagnosis; systemic staging negative (operational definition of PDL).	Tissue diagnosis (biopsy or limited resection if decompression needed); focal radiotherapy as indicated by location and symptom burden.	Excellent local control; prolonged follow-up to detect rare late extracranial relapse.
1B	PDL, non-EMZL/MALT (mainly primary dural diffuse large B-cell lymphoma; rare other histotypes)	Meningioma-like phenotype with rapid progression (weeks) and neurological deterioration. Skull base/cavernous sinus neuropathies; spinal dural/epidural compression may occur.	Aggressive clinical course with negative systemic staging; higher likelihood of requiring central nervous system-directed systemic therapy.	Central nervous system-directed systemic therapy (high-dose methotrexate-based regimens) ± focal radiotherapy; surgery mainly for diagnosis and/or urgent decompression.	Less favorable and more heterogeneous than Cluster 1A; outcome driven by biology and treatment intensity.
2A	Dural involvement in secondary central nervous system lymphoma from aggressive systemic lymphomas (predominantly diffuse large B-cell lymphoma)	Dural disease is usually not isolated; multi-compartment central nervous system involvement (parenchymal, leptomeningeal, cranial nerve/root). Often early in high-risk systemic subsets.	Systemic lymphoma present; systemic staging positive by definition (secondary involvement).	Intensive central nervous system-directed regimens (high-dose methotrexate and cytarabine backbones); consider intrathecal therapy and autologous transplant in selected patients.	Overall worse outcomes than PDL; prognosis driven by systemic risk profile, central nervous system burden, and feasibility of intensive therapy.
2B	Dural involvement in disseminated	Rare; single or multiple meningioma-like dural masses or diffuse	Imaging overlaps with Cluster 1A;	Integrate neuroimaging with systemic	Variable; depends on underlying

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	indolent systemic lymphomas	pachymeningeal thickening (including en plaque patterns). Mixed leptomeningeal/parenchymal patterns may occur.	practical discriminator is evidence of systemic indolent lymphoma on staging.	staging; tailor therapy to histotype, systemic burden, and pattern of central nervous system involvement.	indolent lymphoma biology and extent/pattern of central nervous system involvement.

4. Clinical Presentation, Epidemiology, and Therapeutic Overview

Although dural involvement is uncommon within the broader spectrum of CNS lymphomas, it carries significant clinical relevance due to the high risk of misclassification as a "meningioma-like" lesion and its resulting therapeutic implications. Misattribution to a benign extra-axial pathology may delay correct diagnosis and prompt inappropriate treatment strategies, in a setting where management differs substantially between indolent, predominantly locoregional disease and aggressive forms, whether primary or secondary.

Epidemiologic estimates are heterogeneous and strongly dependent on the denominator and referral patterns. One review suggested that PDL accounts for <1% of all CNS lymphomas; conversely, in PCNSL-based cohorts the proportion may appear higher, reaching 6.3% in a bi-institutional series (20/316 PCNSL) [1,2].

1A. PDL, EMZL/MALT

Historical series identify dural EMZL/MALT as the prototypical PDL presentation. In the series by Venkataraman et al. (32 cases), there was a female predominance (27F/5M) and a median age of approximately 50 years [10]. Patients most commonly present with mass-effect symptoms from an extra-axial tumor (headache, seizures, focal deficits), with lesions on the convexity/falx/tentorium or skull base frequently interpreted preoperatively as meningiomas [9,11]

In terms of natural history, disease is often confined to the dura at diagnosis, with excellent responses after locoregional treatment (surgery and/or focal radiotherapy) [9,11]. Nonetheless, late extracranial relapse after complete local response has been described; this may reflect occult disease not detected at baseline and/or subsequent dissemination rather than "metastasis" in a strict sense [9].

Histologic confirmation is crucial because it directly informs prognosis and management: dural EMZL/MALT, typically indolent and frequently localized, is associated with more favorable outcomes than high-grade primary CNS lymphomas. Dural EMZL/MALT typically shows positivity for B-cell markers and BCL2, negativity for CD10 and BCL6, usually lacking MUM1 expression, with low Ki-67 expression [10]. A potentially helpful feature is the presence of clonal IgG4-positive plasma cells, documented by light-chain restriction (predominant κ or λ), a pattern described in marginal zone lymphoma involving the CNS. Importantly, IgG4 positivity per se does not equate to IgG4-related disease (IgG4-RD); some EMZL/MALT lymphomas may arise in an IgG4-RD background, and an etiopathogenetic relationship has been hypothesized [12].

In patients with EMZL/MALT lymphoma associated with hepatitis C virus (HCV) infection, initial antiviral therapy is recommended and may improve outcomes and, in select cases, induce lymphoma regression. Screening includes anti-HCV antibodies and, if positive, HCV-RNA PCR to confirm active infection and quantify viral load [13]. Direct-acting antivirals (DAAs) have transformed HCV treatment, achieving very high rates of sustained virologic response across most genotypes [14].

Beyond radiotherapy, systemic approaches—including HD-MTX–based and non–HD-MTX regimens—have been reported [15]. However, combined chemo-radiotherapy carries a substantial risk of neurotoxicity and is therefore generally unnecessary and not routinely recommended in this indolent, often localized setting [16].

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1B. PDL, Non-EMZL/MALT (DLBCL and Other Histotypes)

Non-EMZL/MALT PDL is uncommon in traditional dural pathology series, yet it may be relatively overrepresented in cohorts from tertiary neuro-oncology centers. In the series by Karschnia et al., DLBCL comprised 10 of 20 PDL cases, whereas MZL accounted for 6 of 20 [1]. Taken together, these data suggest that "MZL predominance" can coexist with a clinically meaningful DLBCL subset—an apparent discrepancy likely driven by differing denominators, referral bias, and center-specific case mix.

In immunocompetent patients, the immunophenotype typical of PCNSL-DLBCL reflects mature B-cell origin with post-germinal-center activation and a predominantly non-GCB (activated B-cell-like) profile: CD20+, CD79a+, PAX5+, usually CD10−, variably BCL6+, strong MUM1/IRF4+, high Ki-67 (>80–90%), and typically negative for CD5/CD138 in classic immunocompetent cases; an ABC/non-GCB phenotype is prevalent [17].

Clinically, PD-DLBCL shares a dural-based extra-axial localization and "meningioma-like" appearance with EMZL but tends to exhibit faster growth and neurological deterioration over weeks. Skull-base/cavernous sinus involvement with cranial neuropathies and spinal dural/epidural masses causing compressive myelopathy or radiculopathy have been reported [2,12].

Biologically and clinically, these forms often require systemic CNS-directed therapy analogous to aggressive PCNSL [1,4,8]. Combinations of HD-MTX with other cytotoxic agents capable of crossing the blood–brain barrier are recommended (ideally evaluated in prospective trials). Regimens cited include MATRix, R-MBVP, rituximab–HD-MTX–carmustine–etoposide–prednisone, R-MPV, and R-MT. High-dose chemotherapy followed by autologous hematopoietic stem cell transplantation (HDC-ASCT) is recommended as consolidation in eligible patients with responsive or stable disease after adequate induction therapy [4].

Beyond DLBCL, other PDL histotypes have been described (e.g., dural follicular lymphoma, mantle cell lymphoma with dural infiltration, and very rare dural presentations of other lymphoproliferative neoplasms), largely as case reports or small series. For these variants, treatment is necessarily extrapolated from experience with dural EMZL, PCNSL, and systemic lymphomas of the corresponding histotype [18–22]. Overall, compared with dural EMZL/MALT, these variants tend to exhibit more aggressive biology and less favorable outcomes.

2A. Dural Involvement in SCNSL from Aggressive Lymphomas

Within SCNSL, dural disease typically occurs as part of a multicompartment pattern involving brain parenchyma, leptomeninges, and/or neuroaxial structures such as cranial nerves and spinal roots; isolated dural disease is uncommon. In unselected cohorts of systemic DLBCL, CNS involvement is overall infrequent and limited to a minority of patients; however, risk is heterogeneous and increases substantially in the presence of unfavorable clinical and/or biological features, including high-risk extranodal sites, high disease burden, and aggressive molecular profiles (e.g., lymphomas arising in immune-privileged sites, the MCD molecular class per LymphGen, and double-hit/double-expressor phenotypes), reaching approximately 10–15% in high risk patients [3,23,24].

SCNSL events typically arise early in the disease course—often within the first year—suggesting that a subset of patients may already harbor subclinical CNS involvement at baseline. These events are associated with a poor prognosis despite intensive treatment strategies [3,25]. Evidence informing prognostication, contemporary intensive strategies (including transplant-based regimens), neuroradiological MRI phenotypes, and extranodal marginal zone lymphoma cohorts with CNS/dural involvement is derived from retrospective series, prospective trials, and real-world datasets [26–34].

2B. Dural Involvement in Disseminated Indolent Lymphomas

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CNS involvement in indolent lymphomas is rare. In a large cohort the reported frequency was 2.8% (33/1163) [35]. In series focusing on indolent lymphomas with CNS involvement, the dural compartment may be under-represented or not consistently separated radiologically. Therefore, practical classification often relies on integrating neuroimaging with systemic staging [36]. Imaging may show solitary or multiple dural "meningioma-like" masses or diffuse pachymeningeal thickening, noting that mixed patterns with leptomeningeal/parenchymal components are not uncommon [36].

Treatment is guided by histology and systemic disease burden. Published cases of indolent B-cell lymphomas with dural/CNS involvement report rituximab-based systemic approaches, including bendamustine–rituximab (BR) and R-CHOP [37,38]. BTK inhibitors (e.g., ibrutinib) have also been used in selected scenarios [39].

5. Imaging

Imaging is central to the diagnostic–therapeutic pathway in CNS neoplasms, enabling non-invasive characterization and longitudinal monitoring through morphological, functional, and metabolic parameters. In dural lymphomas—where appearances can be nonspecific and mimic meningioma, pachymeningitis, or dural metastases—integrating CT, multiparametric MRI, and PET/CT is pivotal to strengthen suspicion, guide targeted biopsy, and help distinguish primary from secondary disease within staging workflows [40,41]. Management implications of imaging–staging integration and criteria prompting tissue sampling are addressed in "Future directions" (Box 1–2; Figure 1).

Evidence on CNS lymphoma imaging supports a shift toward multiparametric assessment beyond conventional sequences, incorporating diffusion, perfusion, spectroscopy, and PET (including hybrid PET/CT or PET/MRI). This aligns with WHO integrated diagnostics and major international recommendations [41,42]. However, most quantitative DWI/ADC and perfusion data derive from parenchymal PCNSL; applying these metrics to dural-based lesions is partly conceptual and requires caution, given overlap with atypical/anaplastic meningiomas and other "meningioma mimics." Advanced parameters should therefore be interpreted alongside morphology, bone changes, growth pattern, and clinical context, rather than as stand-alone criteria [43,44].

A systematic approach is not merely technical quality control but a methodological requirement: reproducible MRI and PET/CT protocols share terminology among neuroradiology, neurosurgery, hematology, and oncology. A stable conceptual framework (here, the four clinico-biological clusters) are prerequisites for using imaging as a stratification tool rather than a static depiction of the lesion.

Diagnostic Modalities: CT, MRI, and PET/CT

In suspected dural lymphoma, the imaging work-up is often sequential: initial cranial CT, followed by contrast-enhanced MRI and, when indicated, PET/CT (or PET/MRI) to define systemic extent.

CT remains essential as a first-line choice of imaging. In CNS lymphomas, including dural forms, lesions may appear iso- to relatively hyperdense and show intense, relatively homogeneous enhancement after iodinated contrast [40]. For dural masses, CT is particularly valuable for bone assessment: lymphoma more often shows a normal calvarium or erosive/osteolytic changes, whereas frank hyperostosis is less typical than in meningioma. Bone lysis should broaden the differential diagnosis to include lymphoma and metastasis [44,45].

Contrast-enhanced MRI remains the modality of choice for dural evaluation. A standardized protocol should include at a minimum, pre- and post-contrast T1, T2, FLAIR, DWI with ADC maps, SWI/T2*, and, when feasible, perfusion spectroscopy and high-resolution 3D acquisitions may be added [41]. MRI enables: (1) confirmation of extra-axial origin and mapping of dural extent; (2) assessment of relationships with parenchyma, leptomeninges, venous sinuses, and bone; (3) functional characterization via diffusion and perfusion [40,42].

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18F-FDG PET/CT is particularly useful in hematology to identify extra-CNS sites and distinguish truly confined PDL from secondary disease with systemic involvement; it may also support targeted biopsy and response monitoring [41,46]. A separate consideration is SSTR PET (e.g., 68Ga-DOTATATE/DOTATOC), widely used in meningioma. When adjacent calvarial involvement is present, bone tracer uptake on SSTR PET may support the diagnosis of transosseous meningioma and help differentiate it from lymphoma-related osteolysis [47]. SSTR expression has been documented in some lymphomatous subtypes, creating a potential "meningioma-mimicking" pitfall if interpreted without multiparametric MRI and clinical context [48].

MRI Sequences: Key Features and Practical Interpretation

Moving from modalities to sequences, MRI interpretation of dural lymphoma relies on combinations of findings rather than isolated signs.

Post-gadolinium T1 sequences define enhancement patterns: dural lymphomas frequently show intense, relatively homogeneous enhancement of a dural-based extra-axial mass, often with a "dural tail," a nonspecific feature that contributes to confusion with meningioma. Some series report relatively extensive vasogenic edema, a less sharp cortical interface, and possible erosive bone changes [44,45].

T2 and FLAIR sequences, while less specific, help characterize edema and possible leptomeningeal involvement. Dural lymphomas are often iso- to moderately hyperintense on T2; cortico-subcortical edema may be more evident in biologically aggressive or secondary forms, and FLAIR facilitates detection of leptomeningeal extension beyond contiguity effects [40,41].

Diffusion (DWI) and ADC maps are among the most useful parameters to raise suspicion for lymphoma, but they are not pathognomonic in dural masses. Hypercellularity and a high nuclear-to-cytoplasmic ratio often produce diffusion restriction and low ADC values; however, restriction can also be seen in atypical/malignant meningiomas and other highly cellular dural tumors. Diffusion should therefore be interpreted alongside perfusion and morphology [40,43,44].

Only a limited number of studies have specifically investigated diffusion-weighted imaging in the assessment of dural lymphoma, and no validated ADC cut-off values are currently available to reliably differentiate dural lymphoma from its mimickers. Published case reports and small case series suggest that diffusion restriction is often homogeneous and may be more pronounced than in adjacent brain parenchyma; moreover, restriction appears more marked in DLBCL/SCNSL compared with EMZL/MALT [49–53]. A systematic evaluation of diffusion restriction patterns and quantitative ADC metrics in dural lymphoma represents, therefore, a promising area for future research.

MRI perfusion (DSC/DCE) may provide additional information. Typical meningiomas often show higher perfusion than many mimics, including some lymphomas, but exceptions exist and universally validated cut-offs are lacking. rCBV and related parameters should therefore be considered supportive markers within a multiparametric approach (morphology, bone involvement, DWI/ADC, and clinical context). Reported perfusion metrics vary across studies because of heterogeneity in acquisition and post-processing [44,55,56].

SWI/T2* can aid differential diagnosis: dural lymphomas typically show little or no intralesional susceptibility, consistent with limited hemorrhagic or calcified components, whereas susceptibility foci are more frequent in some dural metastases and in calcified meningiomas [42,44].

MR spectroscopy (MRS) contributes metabolic characterization, typically showing elevated choline with reduced N-acetylaspartate and possible lipid/lactate components in highly cellular lesions, including lymphomas. In meningiomas, an alanine peak is often described and may add supportive value [40]. Magnetic resonance spectroscopy of dural-based lesions remains technically challenging: susceptibility-related field inhomogeneities near bone and air-containing cavities can result in suboptimal shimming, and lipid signals from adjacent scalp and diploic marrow may contaminate spectra, limiting reliability and reproducibility [54].

Cluster-Based Analysis: Radiological Correlations

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The practical value of these techniques emerges when applied to the four clinico-biological clusters. The goal is not to assign histology from imaging alone, but to use radiological information to strengthen or weaken the likelihood of each cluster in an individual patient.

In cluster 1A (PDL, EMZL/MALT), the typical scenario is an immunocompetent middle-aged woman with one or more "meningioma-like" extra-axial masses. MRI often shows iso- to hypointense T1 and iso- to moderately hyperintense T2 signal, homogeneous enhancement with a dural tail, and usually normal or minimally altered adjacent bone. Diffusion restriction may be present but not necessarily marked; perfusion and spectroscopy, when available, may support a less aggressive profile than dural DLBCL while remaining within the lymphomatous spectrum. FDG PET/CT is negative outside the dural lesion, and negative systemic staging supports an indolent PDL classification.

In cluster 1B (PDL, non-EMZL/MALT; especially PD-DLBCL), the initial appearance may be similar but with stronger aggressiveness signals: faster growth, more extensive vasogenic edema, a less sharp parenchymal interface, and more marked diffusion restriction with low ADC values. Bone erosion/osteolysis or more invasive involvement of the cortical plane may occur compared with typical meningioma [44,45]. FDG uptake may be high at the dural lesion while systemic staging remains negative; this combination favors cluster 1B.

Cluster 2A (dural involvement in SCNSL from aggressive lymphoma) is suggested primarily by the fact that the dura is rarely the sole compartment involved. Imaging may show a multicompartment pattern (parenchymal lesions, leptomeningeal involvement, diffuse or nodular dural thickening), and PET/CT may reveal systemic sites. Here, MRI characterizes CNS compartments, whereas integrated staging—often via PET/CT—defines systemic extent and biology [41].

Finally, cluster 2B (dural involvement in disseminated indolent lymphoma) can be radiologically similar to PDL MALT, with solitary or multiple "meningioma-like" dural masses, sometimes en plaque. In this setting, the discriminator is typically systemic context and staging: brain imaging alone may not reliably distinguish 1A from 2B, and classification depends largely on evidence of systemic disease [36,41].

Radiological Differential Diagnosis and Reasoning Schemes

In routine neuroradiological practice, many dural-based lesions are initially interpreted as meningiomas. However, a subset of "meningioma-like" masses represents mimics (metastatic, inflammatory, or lymphomatous) with substantially different management implications. To reduce anchoring bias, we propose a set of red flags (Box 1) and a decision pathway that prompts early tissue diagnosis and staging when appropriate.

Radiological differential diagnosis requires a multiparametric approach integrating extra-axial features, bone changes, enhancement patterns, and functional sequences (DWI/ADC and perfusion), acknowledging that no single sign is definitive and that advanced parameters are typically supportive within dural lesion assessment [43,44,57]. In selected contexts, a peripheral rim-like enhancement pattern may provide supportive value in distinguishing meningiomas from malignant dural tumors [58].

Dural metastases are an important source of diagnostic confusion: they can appear as solitary or multiple nodules, en plaque thickening, or diffuse "carpet-like" enhancement. Bone lysis (more than hyperostosis), coexisting parenchymal metastases, and PET/CT findings suggesting an extracranial primary would support this diagnosis; perfusion may help, but overlaps exist and vary by histotype [44,55,56].

Immune-mediated pachymeningitis (including IgG4-related forms) and neurosarcoidosis represent major inflammatory considerations in the differential diagnosis. MRI often shows diffuse or multifocal dural thickening with intense enhancement, sometimes symmetric; marked diffusion restriction is less typical, and perfusion tends not to show profiles strongly suggestive of a hypercellular neoplasm. Clinical context remains central: association with systemic manifestations

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(orbit, paranasal sinuses, salivary glands, lung parenchyma) and steroid responsiveness can be informative; nevertheless, targeted biopsy is often decisive [59]. In IgG4-related forms, serum IgG4 and inflammatory markers may support suspicion; on tissue, distinguishing a polyclonal from clonal plasma cell infiltrate through κ/λ light-chain restriction is critical and may indicate an underlying lymphoproliferative process (e.g., IgG4+ MZL).

Future Directions and a Pragmatic Management Proposal in the Absence of Dedicated Guidelines

In a field lacking specific recommendations, particularly for PDL, developing and validating shared approaches to the initial management of "meningioma-like" dural-based lesions is a priority. The goal is twofold: to reduce anchoring bias and standardizing clinically decisive choices (tissue diagnosis, timing and extent of staging, extent of surgery, and peri-diagnostic corticosteroid use). In practice, phenotypic overlap between meningioma and mimics (metastatic, inflammatory, or lymphomatous) and the inherently probabilistic nature of advanced imaging markers contribute to meaningful inter-operator variability, potentially affecting time to diagnosis, staging appropriateness, and selection of the initial surgical procedure. Figure 1 shows a reproducible decision pathway integrating key clinico-radiological nodes and corresponding actions (targeted biopsy, systemic staging, role of surgery, corticosteroid management) into a pragmatic sequence shared across disciplines. As a future adjunct, CSF-based liquid biopsy may complement this pathway—particularly when tissue sampling is high-risk—by enabling detection of lymphoma-associated alterations in CSF ctDNA/cfDNA (e.g., MYD88 L265P and CD79B Y196), with CSF generally offering higher sensitivity than plasma in CNS settings [60]. Exploratory CSF biomarker approaches (including microRNA/exosome and cytokine/chemokine profiles) remain investigational and are not yet standardized for routine practice, requiring interpretation within the full clinical and radiological context [61,62].

Radiomics-based pipelines, coupled with machine-learning classifiers, may enable a more objective differentiation of dural lymphoma from meningioma and other dural mimickers by leveraging high-dimensional shape and texture features extracted from routine MRI (and, where available, functional sequences such as DWI/perfusion) and/or FDG PET. In primary CNS lymphoma, preliminary radiomics/ML studies have shown encouraging performance for non-invasive diagnostic classification, but translation to the dural compartment will require harmonized acquisition/segmentation workflows and rigorous external validation, ideally aligned with standardized feature definitions (e.g., IBSI) [63–65].

Within this framework, a structured set of clinico-radiological red flags (Box 1) and early management principles (Box 2) is needed to ensure pathway reproducibility across neuroradiology, neurosurgery, and hemato-oncology. Box 1 is intended to identify contexts in which a mimic—particularly a lymphomatous mimic—is sufficiently plausible to justify reprioritization from standard dural mass management to an accelerated pathway centered on tissue diagnosis and staging. Box 2 translates this suspicion into operational decisions, clarifying when to prefer a low-morbidity "tissue-first" approach, when to reserve surgery for selected indications (decompression/debulking), and how to optimize the biopsy–staging–therapy sequence (including pragmatic considerations on corticosteroid timing). Figure 1 integrates Box 1 and Box 2 into a threshold-based algorithm (low/intermediate/high suspicion) that makes explicit when to deviate from conventional pathways and how to prioritize diagnostic and staging steps.

Box 1. Red flags suggesting a "meningioma-like" dural lesion that may represent a lymphomatous, metastatic, or inflammatory mimic, prompting an accelerated tissue-first diagnostic pathway.

Box 1. Red flags for meningioma-like dural lesions: when to suspect a lymphomatous mimic and adopt a biopsy-first pathway
Note. Pragmatic checklist synthesized from the dural-lymphoma and dural-mimic imaging literature (not a formal guideline).
A. Clinical red flags (context and time)

 doi:10.20944/preprints202602.0174.v1

Major: rapid symptom progression (days-weeks) or neurological deterioration atypical for meningioma.
Major: hematologic history (known/recent lymphoma) and/or clinically significant immunosuppression.
Major: skull-base phenotype (multiple cranial neuropathies) or suspicion of multicompartment disease.
Minor: clinicoradiologic incongruence or disproportionate symptoms.
B. Morphologic/structural red flags (CT/MRI, including bone)
Major: atypical bone pattern: erosion/osteolysis or permeative change (rather than frank hyperostosis).
Major: infiltrative behavior: blurred cortical interface or diffuse/multifocal pachymeningeal extension not fitting classic meningioma.
Minor: disproportionate vasogenic edema relative to lesion size/implantation.
Minor: en plaque or multifocal appearance that does not integrate with the clinical-radiologic context.
C. Functional/metabolic red flags (multiparametric MRI and PET)
Major: marked diffusion restriction (DWI/ADC) suggestive of hypercellularity in a non-convincing meningioma setting.
Major: positive systemic staging (FDG-PET/CT or equivalent) suggesting secondary involvement/SCNSL.
Major: receptor-imaging pitfall: SSTR-PET uptake compatible with meningioma but discordant with MRI and/or clinical context.
Minor: perfusion/MRS parameters not aligned with typical meningioma (supportive, not decisive).
Practical decision rule
Biopsy-first is recommended when >=1 Major red flag is present, or when >=2 Minor red flags coexist, in the absence of a primary decompressive indication (critical mass effect, uncontrolled refractory seizures, or deterioration requiring debulking).
Preferred goal: rapid, minimally invasive tissue diagnosis (stereotactic biopsy or diagnostic mini-craniotomy) to align early staging and therapy with disease biology and to reduce surgical overtreatment.
Supporting sources (all cited in the main text): Karschnia et al., 2020; Lyndon et al., 2019; Maireche et al., 2023; Juntikka et al., 2021.

Box 2. Pragmatic early management principles for suspected dural lymphoma, prioritizing timely biopsy and comprehensive hematologic staging before defining surgical strategy.

Box 2. Guideline-consistent early management when CNS lymphoma is suspected: applied to meningioma-like dural lesions
Interpretation. Dedicated international guidelines for PDL are limited. The following principles are derived from PCNSL/SCNSL recommendations and apply when lymphoma is a credible differential diagnosis.
1) Prioritize tissue diagnosis (“biopsy-first”)
Obtain targeted tissue sampling (stereotactic biopsy; alternatively diagnostic mini-craniotomy when stereotaxy is impractical).
CSF studies can be supportive when positive but have limited sensitivity and should not replace tissue diagnosis when an accessible lesion exists.
2) Reserve resection/debulking for selected emergencies
Routine gross-total resection is not a standard initial strategy for suspected CNS lymphoma.
Consider decompression/debulking only when required for life-threatening mass effect/intracranial hypertension or when needed to obtain safe diagnostic tissue in a critical anatomical setting.
3) If SCNSL is suspected, stage systemically and choose the safest biopsy site
Perform prompt systemic staging (FDG-PET/CT or equivalent total-body imaging) plus standard hematologic work-up.
With concurrent systemic disease, diagnosis may be established from a systemic-site biopsy when CNS imaging is consistent after expert review; for isolated CNS relapse or atypical imaging, CNS biopsy is often required.
4) Corticosteroids: avoid pre-biopsy if clinically feasible
When suspicion is high and the patient is stable, avoid or defer corticosteroids before biopsy due to potential reduction in diagnostic yield.
If steroids are required (edema/intracranial hypertension/neurological deterioration), do not delay biopsy and document dose and duration.
Practical one-line rule (for meningioma-like dural lesions)
When red flags suggest a lymphomatous mimic and there is no primary decompressive indication, proceed with rapid minimally invasive tissue diagnosis and complete staging before planning extensive “meningioma-like” resection.

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Key sources (canonical publication years): Hoang-Xuan et al., 2023; Ferreri et al., 2024; Bobillo et al., 2023; Cwynarski et al., 2023.

Beyond immediate clinical value, explicit criteria (Box 1–2) and algorithmic implementation (Figure 1) enable more robust evidence generation by harmonizing definitions and thresholds, building more comparable multicenter cohorts, and measuring practice-relevant endpoints: time to tissue diagnosis, rate of unnecessary extensive resections, completeness and appropriateness of staging, impact of pre-treatment steroids on diagnostic yield, and pathway safety. Without such minimum standards, the literature—dominated by retrospective series and reviews—remains intrinsically heterogeneous and difficult to translate into operational recommendations.

A realistic and methodologically coherent next step would be to develop multidisciplinary consensus recommendations (ideally via structured Delphi/expert panel processes with scientific society endorsement) formalizing: (i) a minimum set of red flags (Box 1), (ii) early management principles (Box 2), (iii) imaging requirements and criteria for PET/staging, and (iv) pathway quality indicators. These recommendations would provide a shared basis for multicenter validation of the proposed algorithm (Figure 1) and prospective studies, thereby laying the groundwork for future dedicated guidelines.

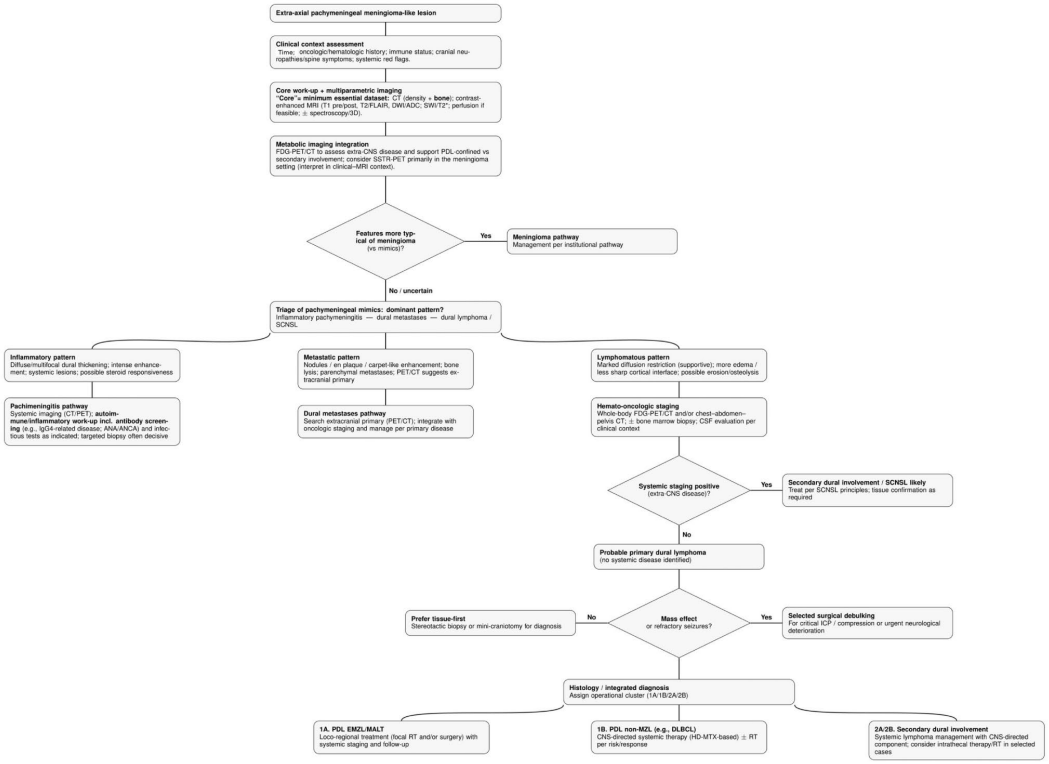

Figure 1. The algorithm applies to a "meningioma-like" extra-axial pachymeningeal lesion and proceeds through four main steps.

1. Clinical context assessment and acquisition of core imaging: CT (density and, crucially, bone changes: hyperostosis vs erosion/osteolysis) plus contrast-enhanced MRI (pre-/post-contrast T1, T2, FLAIR, DWI/ADC, SWI/T2*; perfusion if feasible; ± spectroscopy/high-resolution 3D).
2. PET/CT integration: FDG PET/CT to identify extra-CNS sites and distinguish confined versus secondary disease; SSTR PET in the meningioma setting, acknowledging potential lymphoma-related pitfalls.
3. Initial branching: if findings are more typical of meningioma (e.g., hyperostosis; often higher perfusion in typical meningiomas; alanine on spectroscopy), follow the standard dural mass

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management pathway. Otherwise, triage major pachymeningeal mimics according to the dominant pattern:

- Inflammatory: diffuse/multifocal thickening with intense enhancement; possible systemic lesions; targeted biopsy is often decisive.
- Metastatic: nodules/en plaque/"carpet-like" disease; bone lysis; possible parenchymal metastases; PET/CT may suggest an extracranial primary.
- Lymphomatous: DWI/ADC and other parameters as supportive elements; more edema/less sharp cortical interface; possible erosion/osteolysis; attention to SSTR PET pitfalls.

4. Lymphomatous branch: stage before defining the surgical strategy. Perform hematologic staging (whole-body PET/CT and/or CT chest–abdomen–pelvis; ± bone marrow biopsy; CSF assessment as clinically indicated). If staging is positive, secondary involvement/SCNSL is likely (cluster 2A/2B); if negative, PDL is likely. In the absence of mass effect or medically refractory seizures, prioritize biopsy to avoid unnecessary craniotomy; reserve resection for clinically or surgically indicated cases. After tissue diagnosis, assign the operational cluster (1A/1B/2A/2B) and tailor therapy accordingly.

7. Limitations

This narrative review has methodological limitations. First, because it was not designed as a formal systematic review, it remains susceptible to selection bias despite a database search strategy and independent screening by two authors. In addition, language and publication biases may persist because we did not include grey literature, non-English studies, or contributions available only as abstracts.

The evidence base on dural and CNS lymphomas consists predominantly of retrospective studies, often with small cohorts and heterogeneous populations (PDL series versus cohorts derived from PCNSL/SCNSL), limiting comparability and generalizability—particularly for imaging findings and clinical outcomes. Finally, the lack of a formal risk-of-bias assessment and quantitative meta-analysis precludes robust effect-size estimation and an overall certainty rating of the available evidence.

8. Conclusions

Extra-axial dural lesions are frequently evaluated with insufficient radiological rigor, creating a risk of premature diagnostic impact that may inappropriately shape clinical management and expose patients to incorrect treatments. In cases with diagnostic uncertainty—particularly in the absence of significant mass effect or medically refractory seizures—direct referral to craniotomy rather than a biopsy procedure may entail substantially greater procedural risk than a minimally invasive diagnostic approach.

This issue is especially relevant when imaging raises suspicion for dural lymphoma: in such contexts, a biopsy-first pathway may prevent unnecessary resections and facilitate timely initiation of disease-directed therapy. Despite increasing recognition of pachymeningeal lymphomatous involvement, dedicated guidelines for dural lymphoma management are still lacking, and current practice often relies on evidence extrapolated from parenchymal primary CNS lymphoma.

Against this backdrop, the present narrative review aims to provide above all, a critical and as comprehensive as possible, synthesis of the radiological characteristics of extra-axial dural lesions, with particular attention to entities with overlapping phenotypes. The overarching goal is to support a more accurate and efficient diagnostic pathway and better-informed treatment decisions through a structured methodological approach and a practical neuroradiological guide.

Supplementary Materials: The following supporting information can be downloaded at the website of this paper posted on Preprints.org.

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Abbreviations

The following abbreviations are used in this manuscript:

18F-FDG = fluorine-18 fluorodeoxyglucose
3D = three-dimensional
68Ga-DOTATATE/DOTATOC = gallium-68 somatostatin receptor tracers
ABC = activated B-cell-like
ADC = apparent diffusion coefficient
Ara-C = cytarabine
ASCT = autologous stem cell transplantation
BCL6 = B-cell lymphoma 6
BR = bendamustine–rituximab
BTK = Bruton tyrosine kinase
CD = cluster of differentiation
CI = confidence interval
CNS = central nervous system
CR = complete response
CSF = cerebrospinal fluid
ctDNA = circulating tumor DNA
cfDNA = cell-free DNA
CT = computed tomography
DAAs = direct-acting antivirals
DCE = dynamic contrast-enhanced (MRI)
DLBCL = diffuse large B-cell lymphoma
DSC = dynamic susceptibility contrast (MRI)
DWI = diffusion-weighted imaging
EFS = event-free survival
EHA-ESMO = European Hematology Association–European Society for Medical Oncology
EMZL = extranodal marginal zone lymphoma
FDG = fluorodeoxyglucose
FDG-PET/CT = fluorodeoxyglucose positron emission tomography/computed tomography
FLAIR = fluid-attenuated inversion recovery
GCB = germinal center B-cell-like
HDC = high-dose chemotherapy
HDC-ASCT = high-dose chemotherapy followed by autologous stem cell transplantation
HD-MTX = high-dose methotrexate
HDS = high-dose sequential chemoimmunotherapy

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HDT = high-dose therapy
HDT-ASCT = high-dose therapy with autologous stem cell transplantation
HCV = hepatitis C virus
HCV-RNA = hepatitis C virus ribonucleic acid
HR = hazard ratio
IBSI = Image Biomarker Standardisation Initiative
IgG4 = immunoglobulin G4
IgG4-RD = IgG4-related disease
IRF4 = interferon regulatory factor 4
IT = intrathecal
Ki-67 = Ki-67 proliferation index
LDH = lactate dehydrogenase
MALT = mucosa-associated lymphoid tissue
MARIETTA = MATRix-RICE and autologous stem cell transplantation trial acronym
MATRix = rituximab, methotrexate, cytarabine, thiotepa (regimen)
MCD = molecular class MCD (LymphGen)
MeSH = Medical Subject Headings
ML = machine learning
MRI = magnetic resonance imaging
MRS = magnetic resonance spectroscopy
MSKCC = Memorial Sloan Kettering Cancer Center
MUM1 = multiple myeloma oncogene 1
MYD88 = myeloid differentiation primary response 88
MZL = marginal zone lymphoma
NHL = non-Hodgkin lymphoma
non-GCB = non–germinal center B-cell-like
ORR = objective response rate
OS = overall survival
PAX5 = paired box 5
PCR = polymerase chain reaction
PCNSL = primary central nervous system lymphoma
PD-DLBCL = primary dural diffuse large B-cell lymphoma
PDL = primary dural lymphoma
PET = positron emission tomography
PET/CT = positron emission tomography/computed tomography
PET/MRI = positron emission tomography/magnetic resonance imaging
PFS = progression-free survival
PR = partial response
rCBV = relative cerebral blood volume
R-CHOP = rituximab, cyclophosphamide, doxorubicin, vincristine, prednisone
R-HDS = rituximab plus high-dose sequential chemoimmunotherapy
RICE = rituximab, ifosfamide, carboplatin, etoposide
R-MBVP = rituximab, methotrexate, carmustine, etoposide, prednisone
R-MPV = rituximab, methotrexate, procarbazine, vincristine
R-MT = rituximab–methotrexate
RT = radiotherapy
SCNSL = secondary central nervous system lymphoma
SCNSL-R = Secondary CNS Lymphoma Registry
SSTR = somatostatin receptor
SSTR-PET = somatostatin receptor positron emission tomography
SWI = susceptibility-weighted imaging

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T1 = T1-weighted (MRI)
T2 = T2-weighted (MRI)
T2* = T2*-weighted (MRI)
WHO = World Health Organization
WHO CNS5 = WHO Classification of Tumors of the Central Nervous System, 5th edition
WHO-HAEM5 = WHO Classification of Hematolymphoid Tumors, 5th edition

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