Leptomeningeal Enhancement in Multiple Sclerosis and Other Neurological Diseases: A Systematic Review and Meta-Analysis

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Meta-data

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Summary statement

Our systematic review and meta-analysis synthesize leptomeningeal enhancement proportions across a comprehensive panel of neurological diseases, including multiple sclerosis, and assesses its prognostic value in multiple sclerosis.

Summary data

•Leptomeningeal enhancement (LME) is a nonspecific imaging feature present across many neurological disorders, including neoplasm, infection, and primary neuroinflammation.

•The presence of LME is associated with worse clinical and imaging outcomes in multiple sclerosis, justifying its ascertainment in clinical practice.

•Neuroinflammatory animal models can be used to further investigate the pathophysiology of LME, including its pathological tissue signature and/or its association with cortical pathology.

Glossary

CIS, clinically isolated syndrome; CNS, central nervous system; EAE, experimental autoimmune encephalomyelitis; EDSS, Expanded Disability Status Scale; FLAIR, fluid-attenuated inversion recovery; Gd, gadolinium; LME, Leptomeningeal enhancement; MRI, magnetic resonance imaging; MS, multiple sclerosis (RR, relapsing-remitting); NMOSD, neuromyelitis optica spectrum disorder.

Abstract

Background: The lack of systematic evidence on leptomeningeal enhancement (LME) on MRI in neurological diseases, including multiple sclerosis (MS), hampers its interpretation in clinical routine and research settings.

Purpose: To perform a systematic review and meta-analysis of MRI LME in MS and other neurological diseases.

Materials and Methods: In a comprehensive literature search in Medline, Scopus, and Embase, out of 2292 publications, 459 records assessing LME in neurological diseases were eligible for qualitative synthesis. Of these, 135 were included in a random-effects model meta-analysis with subgroup analyses for MS.

Results: Of eligible publications, 161 investigated LME in neoplastic neurological (n=2392), 91 in neuroinfectious (n=1890), and 75 in primary neuroinflammatory diseases (n=4038). The LME-proportions for these disease classes were 0.47 [95%-CI: 0.37–0.57], 0.59 [95%-CI: 0.47–0.69], and 0.26 [95%-CI: 0.20–0.35], respectively. In a subgroup analysis comprising 1605 MS cases, LME proportion was 0.30 [95%-CI 0.21–0.42] with lower proportions in relapsing-remitting (0.19 [95%-CI 0.13–0.27]) compared to progressive MS (0.39 [95%-CI 0.30–0.49], p=0.002) and higher proportions in studies imaging at 7T (0.79 [95%-CI 0.64–0.89]) compared to lower field strengths (0.21 [95%-CI 0.15–0.29], p<0.001). LME in MS was associated with longer disease duration (mean difference 2.2 years [95%-CI 0.2–4.2], p=0.03), higher Expanded Disability Status Scale (mean difference 0.6 points [95%-CI 0.2–1.0], p=0.006), higher T1 (mean difference 1.6ml [95%-CI 0.1–3.0], p=0.04) and T2 lesion load (mean difference 5.9ml [95%-CI 3.2–8.6], p<0.001), and lower cortical volume (mean difference -21.3ml [95%-CI -34.7–-7.9], p=0.002).

Conclusions: Our study provides high-grade evidence for the substantial presence of LME in MS and a comprehensive panel of other neurological diseases. Our data could facilitate differential diagnosis of

LME in clinical settings. Additionally, our meta-analysis corroborates that LME is associated with key clinical and imaging features of MS.

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Introduction

Abnormal meningeal contrast enhancement may take two distinct forms: pachymeningeal enhancement, referring to dural-arachnoidal enhancement, which follows the contour of the inner table of the skull and includes intradural veins and sinuses; and leptomeningeal enhancement (LME), which follows the pia-arachnoid abutting the cortical surface and extending into the sulci. LME is often caused by neoplastic or infectious processes. However, LME is also gaining increased attention as a putative imaging biomarker of meningeal inflammation in neuroinflammatory diseases, including MS and neurosarcoidosis (**Figure 1**).(1)

Because LME can be present in a wide variety of neurological diseases,(2) differential diagnostic considerations are paramount for proper patient workup. However, there is a lack of systematic evidence on LME proportions in MS and other neurological diseases. Furthermore, a wide variety of methodological approaches to imaging LME has been published,(3, 4) impeding the implementation of an appropriate imaging protocol for sensitive LME detection. With respect to MS, several studies have presented conflicting findings regarding the association of LME with clinical and imaging parameters,(5) such that high-level evidence would benefit clinicians and researchers.

Based on these shortcomings, we set out to systematically summarize the available evidence on LME in neurological diseases with a focus on MS. This study had the following goals: (1) synthesize data on LME proportions in neurological diseases, including potentially distinct LME features such as phenotype or temporal evolution; (2) qualitatively and quantitatively summarize the potential association of LME with clinical and imaging features in MS; (3) propose an appropriate imaging protocol to detect LME in clinical and research practice; (4) summarize the data on pathological correlates of LME in neuroinflammation; (5) summarize the available evidence on LME in animal models of neuroinflammation.

Materials and Methods

We registered the study protocol in the International prospective register of systematic reviews (PROSPERO, CRD42021235026, https://www.crd.york.ac.uk/PROSPERO/) and used the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) Guidelines for reporting.(6)

Search strategy

We searched for original studies published in full up to February 2, 2021, in PubMed, Scopus, and Ovid EMBASE. See **Table S1** for the search string in each of these databases.

Inclusion and exclusion criteria

We included publications on human or animal data that reported on any outcome related to leptomeningeal inflammation on magnetic resonance imaging (MRI) in any neurological diseases. Case reports were also included in the systematic review. Exclusion criteria: conference abstracts, non-English articles, and publications that reiterated previously reported quantitative data. Reviews were excluded but retained as potential sources of additional records.

Study selection and data extraction

Titles and abstracts of studies were screened for their relevance in the web-based application Rayyan by two reviewers (CT and BVI), followed by full-text screening.(7) Subsequently, the following data were extracted: title, authors, publication year, study design, neurological disease, and number of subjects per group. For studies with ≥10 subjects, MRI sequences/field strength, LME location (spinal cord, convexities, basal, cerebellar, brainstem), main study findings (in narrative manner), pattern of LME (for example, nodular or linear), temporal dynamics of LME, and proportions of LME in experimental and control groups were also extracted.

Quality assessment

The quality of each study with ≥10 included subjects was assessed against predefined criteria by two reviewers (CT and BVI) using the Newcastle-Ottawa scale for evaluating risk of bias in nonrandomized studies.(8) Discrepancies were resolved by discussion.

Data synthesis and analysis

Only diseases/disease classes with ≥2 publications describing ≥10 adult subjects each were included in the meta-analysis, and only summary-level data were used. As primary outcome, log-transformed proportions of LME were used. A random-effects model was fitted to the data. The amount of heterogeneity ($\tau 2$), was estimated using the DerSimonian-Laird estimator.(9) In addition, the Q-test for heterogeneity (10) and the I^2 statistic (11) are reported.

For subgroup analyses of clinical and imaging outcomes in MS, the analysis was carried out using the log-transformed proportions or mean difference as the outcome measure. Subgroup analyses were computed for MS to assess the proportion of LME in clinical MS phenotypes when ≥3 studies were available. Subgroup analyses in MS for clinical and imaging outcomes were computed when ≥3 studies reported at least mean, variance, and n for LME+ and LME- groups on respective outcomes.

A two-tailed P value <0.05 was considered statistically significant.

Publication bias

The rank correlation test and the regression test, using the standard error of the observed outcomes as predictor, were used to check for funnel plot asymmetry. The analysis was carried out using R (version 3.6.1) with the *meta* and *metafor* packages (version 2.4.0).(12)

Results

1. Eligible publications

In total, 2292 original publications were retrieved from our comprehensive database search and an additional 10 publications from reference lists of reviews on related topics. After abstract and title screening, 1089 studies were eligible for full-text search. After screening the full text of these studies, 458 articles (35% of deduplicated references) were included for qualitative synthesis and 135 articles (10%) for quantitative synthesis (**Figure S1**).

2. General study characteristics

2.1. Included publications

Of the eligible publications, 144 investigated LME in neoplastic neurological diseases (2392 subjects including 183 children), 91 in infectious neurological diseases (1890 subjects including 48 children), and 76 in primary neuroinflammatory diseases (4038 subjects including 11 children). Additionally, 147 publications assessed LME in neurological diseases that did not belong to mentioned categories (1961 subjects including 762 children). We also included 5 publications in animal models of neurological diseases (mouse experimental autoimmune encephalomyelitis [EAE] model, bacterial meningitis in rats, bacterial CNS infection in a dog, subarachnoid diverticulum in a cat).

2.2. Risk of bias assessment

Most studies showed a low risk of bias for the selection domain (that is, whether patients and controls were defined according to acknowledged diagnostic criteria); see **Table S2 and S3**. Many studies did not report on adjusting their statistical analyses for subject age, sex, or other potential confounders (comparability domain), thus potentially inducing biases.

3. Leptomeningeal enhancement in neuroinflammatory diseases including multiple sclerosis

3.1. Primary neuroinflammatory diseases overall

3.1.1. Diseases

Studies reporting on LME in neuroinflammatory diseases were in neurosarcoidosis (20 publications), MS (17 publications), MOG-antibody diseases/encephalitis (8 publications), neuromyelitis optica spectrum disorder (NMOSD) (8 publications), primary angiitis of the CNS (6 publications), Susac syndrome (5 publications), NMDA-receptor encephalitis (4 publications), Behçet syndrome (1 publication), and GFAP astrocytopathy (1 publication).

3.1.2. LME pattern

21 studies did not report on the LME pattern, whereas the remaining studies reported on different LME patterns. The pattern of LME has most extensively been described in MS in 12 publications, mostly as either nodular and/or laminar/spread-and-fill.(13-19) Similar LME patterns have been described in Susac syndrome (20) and neurosarcoidosis.(21, 22) A spreading/laminar phenotype has been described in NMOSD,(23-25) NMDA-receptor encephalitis,(26) and GFAP astrocytopathy.(27)

3.1.3. MRI acquisition of LME

Most studies employed a postcontrast T2w-FLAIR sequence to visualize LME (19 publications) followed by a postcontrast T1w sequence (6 publications). 11 studies did not report the sequences used to detect LME.

3.1.4. Meta-analysis

A meta-analysis of LME in primary neuroinflammatory diseases, including a total of 2284 patients, showed an overall proportion of 0.26 [95%-CI: 0.20–0.35] with substantial heterogeneity across studies (I^2=90%, p<0.001) (**Figure 2**). NMOSD had the lowest proportion of LME with 0.06 [95%-CI 0.02–0.23], and neurosarcoidosis had the highest LME proportion with 0.41 [95%-CI 0.13–0.52].

3.2. Multiple sclerosis

3.2.1. LME proportion in MS and subgroups

Two studies from 2015 first described LME in MS.(2, 28) These and subsequent studies comprised 1605 MS patients, 303 non-MS controls, and 126 healthy controls (**Table 1**).

The overall proportion of LME in MS was 0.30 [95%-CI 0.21–0.42] (**Figure 3**). However, LME proportions in MS patients with a relapsing-remitting clinical phenotype (0.19 [95%-CI 0.13–0.27]; 7 publications) and CIS patients (0.06 [95%-CI 0.02–0.20]; 3 publications) were significantly lower compared to progressive MS patients (0.39 [95%-CI 0.30–0.49], p=0.002 and p=0.003, respectively; 6 publications).

3.2.2. MRI acquisition of LME

14 studies acquired MRI at 3T (or complementing 1.5T) and 3 studies at 7T.(13, 16, 18) In a meta subgroup-analysis with the B_0 magnetic field strength as moderator, LME proportions were higher in studies imaging at 7T (0.79 [95%-CI 0.64–0.89]; 3 publications) compared to studies imaging at 1.5/3T (0.21 [95%-CI 0.15–0.29], p<0.001; 11 publications) (**Figure 4**).

All 17 studies investigating LME in MS employed both a postcontrast 3D T2w-FLAIR and a postcontrast 3D T1w sequence to detect LME. Of note, 1 study acquired 2D scans instead of 3D, showing the lowest LME proportion among all MS studies (< 0.01, 1/112 cases).(28) Of note, is has been shown that 3D T2w-FLAIR can have higher sensitivity to detect gadolinium enhancement compared to T1w imaging, particularly for superficial enhancement.(29) The acquisition of the 3D T2w-FLAIR sequence was mostly 10 minutes after Gd-injection, with two studies reporting either earlier (3 minutes)(20) or later acquisition (up to 54 minutes).(15) It is noteworthy that latter study suggested that individual LME foci might have different enhancement kinetics and thus different peak enhancement time points, and that differences in LME detection between scanner types could be due to differences in 3D T2w-FLAIR sequences.

One study found lower sensitivity for LME detection using T1w sequences compared to T2w-FLAIR.(19) One study compared LME detection on 3D T2w-FLAIR postcontrast images in native space (method 1), on pre- and postcontrast 3D T2w-FLAIR images in native space (method 2), and on pre-/postcontrast 3D T2w-FLAIR co-registered and subtracted images (method 3).(3) In total, 51 (20%) MS cases showed LME using method 1; 39 (15%) using method 2; and 39 (15%) using method 3. The mean time to analyze the 3D T2w-FLAIR images was lower with method 2 compared to the other 2 methods.

3.2.3. LME phenotypes

Overall, two configurations of LME have been described, albeit with inhomogeneous nomenclature: nodular and “spread-fill” (subsuming linear, laminar, and plate-like). Most studies described these two phenotypes (12/17 publications).(2, 13-20, 30) 1 early publication with a very low LME proportion of <0.01 exclusively described a nodular LME phenotype.(28) 5 publications did not declare the LME pattern.

The prevalence of these patterns varied considerably between studies: 4 publications observed higher frequencies of nodular LME: 60% (vs. spread 40%),(18) 54% (vs. 13% linear and 31% plate-like),(19) 80% (vs. linear and plate-like),(17) and 89% (vs. 11% filling-like).(14) Two publications described higher frequencies of linear/spread-fill LME: 59–61% for spread-fill sulcal or gyral (vs. nodular)(13) and 76% spread-fill (vs. 15% nodular).(16) The latter study observed simultaneous presence of both LME phenotypes in 38% of MS cases.

3.2.4. Temporal evolution of LME

Eleven studies did not include longitudinal MRI data and did thus not assess temporal evolution of LME. The remaining 6 studies consistently reported mostly stable LME foci over several years. One study with a follow-up period of up to 5.5 years reported that 85% of LME foci remained stable and that only 6 new LME foci were detected over this observation period (in 4 of 299 MS patients).(30) Similar high percentages of stable LME foci have been observed in other studies: 75% stable over 24 months (and 2 new LME foci in 2/120 patients),(31) 90% stable over 18 months (and 14 new LME foci),(14) 100% stable over 24 weeks (32), and 73–100% stable over 24 months (33). The last study also included non-LME enhancement patterns and found that subarachnoid nodular and spread/fill LME patterns persisted less often than dural or vessel wall foci, and that MS patients with EDSS progression showed more persistent LME foci.

3.2.6. Association of LME with clinical and imaging parameters

Three studies found an association between LME and age and/or disease duration (14, 19, 30), which was not confirmed for disease duration by 1 study.(3) An association between LME and Expanded Disability Status Scale (EDSS) was described in 3 studies (3, 19, 30) but was not confirmed in 1 study after adjusting for age.(14) MS relapse rate was not associated with LME in 2 studies.(17, 19)

Five studies assessed the association between LME and T1 or T2 lesion volume. Three studies found such an association,(3, 17, 18) whereas 2 did not.(14, 16) Six studies consistently reported lower cortical gray matter volume and/or thickness in MS cases with LME.(13, 16-19, 34) Two studies assessed the association of LME with cortical MS lesions at 7T; one of these studies found no such association (13) while the other did.(18) Both studies found an association of LME with subcortical gray matter MS lesions (thalamic and hippocampal, respectively).

In view of the inconsistency regarding the association of LME with clinical and imaging parameters in MS, we assessed these effects in a meta-analysis (for all outcomes reported in ≥3 publications). In this analysis, the presence of LME was associated with longer disease duration (mean difference 2.2 years [95%-CI 0.2–4.2], p=0.03, **Figure 5A**) and higher EDSS (mean difference 0.6 EDSS points [95%-CI 0.2–1.0], p=0.006, **Figure 5B**). Additionally, MS cases with LME showed higher T1 lesion volume (mean difference 1.5 ml [95%-CI 0.1–3.0], p=0.04, **Figure 5C**), higher T2 lesion volume (mean difference 5.9 ml [95%-CI 3.2–8.6], p<0.001, **Figure 5D**), and lower cortical volume (mean difference -21.3 ml [95%-CI -34.7–-7.89], p=0.002, **Figure 5E**).

3.2.7. Histopathological validation of LME

One study performed histopathological validation of three LME foci in two progressive MS cases.(30) The gyri adjacent to the LME foci were affected by confluent cortical demyelination and/or subpial cortical demyelination. Leptomeningeal perivascular inflammation, including T cells, B cells, and macrophages, was detected in these areas.

3.2.7. LME in MS animal models

We included two studies that assessed LME in mouse EAE using postcontrast T2w-FLAIR. The first study employing 9.4T MRI found that all 13 inoculated mice showed LME foci, compared to none of the control mice.(35) Peak LME intensity was at 10 days post induction and correlated with weight loss and clinical symptoms. In a histopathological analysis, LME foci were associated with high densitiy of Iba-1 positive microglia cells as well as T and B cells, which were absent in control mice. Another recent study also emplyoing mouse EAE and MRI at 11.7T with pathological correlation showed that mice treated with a Bruton tyrosine kinase-inhibitor (evobrutinib) had a reduced number of LME foci, while anti-CD20 therapy had no effect on LME.(36) The pathological tissue substrate showed that this corresponded to a reduction in B cells within regions of meningeal inflammation as well as reduced astrocytosis in the adjacent cortex. Interestingly, myeloid cell infiltrates seemed to persist despite B cell depletion.

3.2.5. Therapeutic impact on LME

Four studies assessed the impact of drug treatment on LME resolution. One study found similar LME persistence rates in MS patients with/without disease-modifying therapy (DMT).(33) Another study assessing the efficacy of dimethyl fumarate or teriflunomide on LME reported no differences in LME resolution between treatment groups (8 of 12 patients showed stable LME, and 2 patients developed new LME).(31) One study assessing intrathecal rituximab treatment in 15 progressive MS patients observed stable LME foci in all patients over the 24-week follow-up period.(32) In contrast to these studies with relatively small sample size and consequently lower statistical power, one study including 241 MS patients observed resolution of LME in 2 patients after high-dose steroid treatment within 6 months follow-up.(15)

4. Leptomeningeal enhancement in other neurological diseases

4.1. Infectious CNS diseases

4.1.1. Diseases

Studies reported on LME in infectious (encephalo-)meningitis caused by various pathogens: bacterial (36 publications: tuberculosis, Bacillus anthracis/Anthrax, Borrelia, Clostridium, E. coli, group B streptococcus, Listeria), parasitic (20 publications, among them Angiostrongylus cantonensis, amoeba, Cryptococcus, Toxocariasis, and Toxoplasmosis), viral (18 publications: HIV, HTLV, SARS-CoV-2, Epstein-Barr virus, Murray Valley encephalitis, tick-borne encephalitis virus, Nipah virus, respiratory viruses [various strains], West Nile virus, and Enterovirus), and fungal (14 publications: Candida, Coccidioides, Blastomyces, and Histoplasma).

4.1.2. LME pattern

32 studies did not report on the LME pattern, while 4 studies did (2 spread, 2 nodular). 33 studies did not report on the LME evolution over follow-up, while 3 studies reported LME increase with clinical worsening, and 1 study in cryptococcal meningitis reported LME resolution with clinical improvement.(37)

4.1.3. Imaging

Most studies employed a postcontrast T1w sequence to visualize LME (16 publications), followed by T2w-FLAIR (7 publications). 11 studies did not report which sequences were used for LME detection.

4.1.4. Meta-analysis

A meta-analysis on LME in infectious diseases, including a total of 831 cases, showed an overall proportion of 0.59 [95%-CI: 0.47–0.69] with substantial heterogeneity across studies (I^2=86%, p<0.01) (**Figure 6**). COVID-19 had the lowest proportion of LME with 0.24 [95%-CI 0.13–0.41].

4.2. Neoplastic CNS diseases

4.2.1. Diseases

Studies reporting on LME in neoplastic CNS diseases were caused by primary CNS tumors (92 publications: CNS lymphoma, choroid plexus papilloma, meningioma, germinoma, lipoma, primitive neuroectodermal tumors (PNET), diffuse leptomeningeal glioneuronal tumor, midline glioma, hemangioblastoma, glioblastoma/high-grade astrocytoma, Hodgkin lymphoma, xanthogranuloma, medulloblastoma, melanoma, oligodendroglioma, pilocytic astrocytoma, anaplastic astrocytoma, and giant cell astrocytoma), leptomeningeal metastases (47 publications: metastases from breast cancer, acute myeloid leukemia, rhabdomyosarcoma, gastric cancer, lung adenocarcinoma, small-cell lung cancer, pancreatic cancer, melanoma, Waldenstrom macroglobulinemia [Bing-Neel syndrome], and multiple myeloma), and hereditary tumor syndromes (5 publications: von Hippel-Lindau syndrome, Klippel-Trenaunay-Weber syndrome, and tuberous sclerosis).

4.2.2. LME pattern

28 studies did not report on the LME pattern, while 8 studies did (3 spread, 3 nodular, 2 laminar/linear). 33 studies did not report on LME evolution over follow-up, while 1 study in glioblastoma reported persistent LME at follow-up MRI (up to two years later).(38)

4.2.3. Imaging

Most studies employed a postcontrast T1w sequence to visualize LME (18 publications) followed by a postcontrast T2w-FLAIR (6 publications). 14 studies did not report which sequences were used for LME detection.

4.2.4. Meta-analysis

A meta-analysis on LME in neoplastic diseases, including a total of 1393 cases, showed an overall proportion of 0.47 [95%-CI: 0.37–0.57] with substantial heterogeneity across studies (I^2=90%, $p<0.01$) (**Figure 7**). CNS leukemia had the lowest proportion of LME with 0.24 [95%-CI 0.13–0.39]. Bing-Neel syndrome (Waldenstrom macroglobulinemia) showed the highest proportion of LME with 0.74 [95%-CI 0.60–0.84].

4.3. Other neurological diseases including vascular diseases

4.3.1. Diseases

Of 147 publications, the most notable neurological diseases not belonging to the classes above were: rheumatoid arthritis with meningitis (16 publications), Sturge-Weber syndrome (16 publications), familial leptomeningeal amyloidosis/polyneuropathy (11 publications), ischemic (reversible cerebral vasoconstriction syndrome, stroke, post interventional revascularization, severe carotid stenosis, 11 publications), cerebral amyloid angiopathy (7 publications), epileptic seizures (6 publications), Moyamoya disease (6 publications), intoxications/drug-induced LME (abrin, ibuprofen, ipilimumab, propofol [in children], tacrolimus; 5 publications), hemophagocytic lymphohistiocytosis (4 publications), posterior reversible encephalopathy syndrome (PRES) (4 publications), Rosai-Dorfman disease (3 publications), hepatic encephalopathy (3 publications), Sjogren syndrome (2 publications), traumatic brain injury (2 publications).

4.3.2. Meta-analysis

A meta-analysis on diseases with ≥2 publications and ≥10 subjects per study, including a total of 187 cases, showed an LME proportion of 0.31 [95%-CI 0.16–0.52] in cerebral amyloid angiopathy and 0.69 [95%-CI 0.27–0.93] in reversible cerebral vasoconstriction syndrome (**Figure 8A**).(39, 40)

5. Leptomeningeal enhancement in healthy controls

LME has also been reported in healthy control subjects (6 publications). A meta-analysis of 4 publications, including a total of 163 individuals, corroborated the presence of LME in this group, albeit at a low overall proportion of around 0.06 [95%-CI 0.03–0.11] (**Figure 8B**). In addition to the low proportions of LME, it has been shown in 2 publications that none of the LME-positive control subjects had more than 1 LME focus.(13, 18) In another publication, the 2 LME-positive controls exclusively presented with more than one nodular LME foci.(41)

Discussion

Main findings

This study aims to provide systematic evidence on LME proportion in neurological diseases, including MS, and to determine whether the role of LME as a prognostic biomarker for MS is substantiated in the literature. Overall, primary neuroinflammatory diseases showed lower LME proportion (0.26 [95%-CI: 0.20–0.35]) compared to neoplastic (0.47 [95%-CI: 0.37–0.57]) or infectious neurological diseases (0.59 [95%-CI: 0.47–0.69]). Additionally, the presence of LME was associated with worse clinical and imaging parameters in MS, that is, on average MS patients with LME had 2 years longer disease duration (p=0.03), higher EDSS by 0.7 points (p=0.006), 21 ml less cortical volume (p=0.002), 5.9 ml more T2 lesion volume (p<0.001), and 1.6 ml more T1 lesion volume (p=0.04) compared to MS patients without LME. Finally, based on a few histopathological validation studies in MS and neuroinflammatory animal models, LME corresponds to meningeal inflammatory infiltrates as well as microglial activation in the adjacent cortex. However, the evidence supporting the association of LME with cortical MS pathology remains conflicting.

Findings in the context of existing evidence

A wide variety of disorders may present with LME, including neoplastic and infectious neurological diseases, making LME a highly nonspecific imaging finding. However, proportions of LME have considerable ranges across different neurological diseases. High LME proportions (on the order of 0.75), have been observed in Bing-Neel syndrome (a rare complication of Waldenstrom's macroglobulinemia) (42, 43) and infectious meningitis. (44-46) Interestingly, a subset of smaller studies employing ultrahigh-field (7T) static magnetic field strengths also found proportions of LME around 0.8 in MS,(13, 16, 18) which may indicate a need for higher static magnetic field strengths to facilitate LME detection.

Other notable diseases with LME include ischemic neurological diseases, such as reversible cerebral vasoconstriction syndrome (proportion around 0.7) (39, 40) and stroke (not included in the meta-analysis).(47-49) The presence of LME in brain ischemia also suggests a relevant role of the leptomeningeal compartment in its pathogenesis. Here, LME on post-contrast T2w-FLAIR has been attributed to early blood-brain-barrier disruption,(47) also being associated with hemorrhagic transformation and worse clinical outcomes.(47) Furthermore, poor leptomeningeal collateral flow has been associated with worse clinical outcome in acute stroke.(50) Finally, also COVID-19 has been reported to present with LME, albeit with low proportions around 0.25.(51-53)

Several primary neuroinflammatory diseases can also present with LME, among them neurosarcoidosis (proportion around 0.4), MS (0.3), primary angiitis of the CNS (0.2), NMOSD (0.06), and Susac's syndrome (not included in the meta-analysis).(54) Of note, in MS, LME proportions seem to vary among clinical phenotypes, with relapsing-remitting having lower proportions than progressive MS (0.2 vs. 0.4). However, overall longer disease duration in progressive MS could be a confounding factor.

Our meta-analysis substantiates the role of LME as prognostic biomarker in MS. The presence of LME was associated with worse physical disability and higher lesion burden as well as lower cortical volumes — the latter being also associated with worse clinical MS outcomes (reviewed in (55)). With this, our study highlights the relevance of including LME in routine clinical imaging. Along these lines, ultrahigh-field imaging at 7T might substantially improve the sensitivity to detect LME in MS in the clinical setting, as shown by our meta-analysis.

One major remaining question about LME is its underlying tissue signature. In neoplastic and infectious CNS diseases, LME likely corresponds to increased local blood supply and/or extravasation of gadolinium. However, in neuroinflammatory diseases, the pathological substrate of LME is much less clear. Based on limited EAE and MS histopathology data, LME corresponds to meningeal inflammatory infiltrates and/or tertiary lymphoid follicles (reviewed in (1)).(30, 36) Despite conflicting evidence as to whether LME is spatially associated with cortical pathology in MS,(5) there has been a consistent association of LME with low cortical volumes across studies, also substantiated by our meta-analysis. This indicates that the pathology underlying LME could exert a diffusely deleterious effect on cortical gray matter.

Different LME patterns have been described in MS (41) and, to a much lesser extent, in other neuroinflammatory diseases such as Susac syndrome,(20) neurosarcoidosis,(21, 22) and NMOSD.(23-25). LME patterns were very rarely reported in non-inflammatory neurological diseases, mostly as diffuse LME in neoplastic neurological diseases.(56) In MS, the prevalence of different LME patterns, their nomenclature as well as their association to clinical measures were highly inconsistent among studies. Nevertheless, different LME patterns could represent distinct pathophysiological features, also emphasized by the observation that healthy controls may present with nodular but not non-nodular LME.(41) With this, more data and a more stringent nomenclature is needed to describe LME phenotypes and their potential association to clinical disability and disease phenotypes. Regarding nomenclature, we favor the convention of nodular versus linear LME.

It is interesting that LME has also been observed in healthy controls, albeit at low proportions (0.06).(18, 30, 57, 58) The etiology of LME foci in healthy subjects is still a matter of debate. However, one potential cause could be minor traumatic brain injuries. It should be emphasized that LME foci can be very subtle and can easily be misinterpreted on MRI. Hence, several imaging pitfalls for LME should be taken into account, among them: gadolinium leakage of indeterminate biological significance, enhancement related to slow blood flow of cortical veins, or anatomic structures as well as imaging artifacts.(15)

Finally, data from clinical studies do not suggest a therapeutic effect of DMTs on LME in MS. However, most of these studies have a small sample size and might thus be insufficiently powered to detect a potential therapeutic effect. In addition, newer DMTs such as Bruton tyrosine kinase inhibitors (59) have not been assessed in this regard, even though these drugs led to a resolution of LME in one rodent study employing a neuroinflammatory model.(36). Hence, more data is needed on potential therapeutic impact of DMT on LME resolution. However — in our opinion — LME should still be routinely assessed in MS patients in order to enhance the knowledge and experience of radiologists and referring neurologists on this matter.(60)

Limitations

Our study has some limitations: First, a wide variety of imaging methods have been employed to detect LME, for which we only partially corrected our analysis (e.g., static magnetic field strengths). Notably, the use of either T1w or T2w-FLAIR postcontrast sequences was not considered, with the latter generally having higher sensitivity to detect LME.(4, 19) This could have led to an underestimation of LME proportions in studies acquiring T1w sequences. However, studies in neoplastic and infectious neurological diseases with mostly bulk LME mainly employed T1w sequences, potentially counterbalancing this effect. Along these lines, we also point out that a substantial number of studies did not report on which MRI sequences they employed (T1w versus T2w-FLAIR). Second, for assessing the prognostic value of LME, we pooled studies with various methodological backgrounds for summary estimates. Nonetheless, the outcome measures reported were surprisingly uniform, even allowing for the use of mean differences in our meta-analysis.

Conclusions

Our study provides systematic evidence for LME proportions in a comprehensive panel of neurological diseases, including MS. This high-level evidence also corroborates the prognistic value of LME in MS, supporting the inclusion of LME as a standard imaging feature in clinical MS imaging. Furthermore, this systematic review indicates that future LME studies will need to control for static magnetic field strength, type of 3D T2w-FLAIR sequence, scanner type, timing of acquisition, prior steroid administration as well as exclusion of LME imaging mimicks. More evidence on the pathological substrate of LME and on its potential association with cortical pathology in neuroinflammation is needed to further improve our understanding for this MRI feature and to strengthen its role in the clinical and research settings.

Compliance with Ethical Standards

Conflicts of interest

The authors declare no conflict of interest related to this study.

Data sharing statement

Data available from the authors upon request.

Code availability

R code for meta-analysis available upon request.

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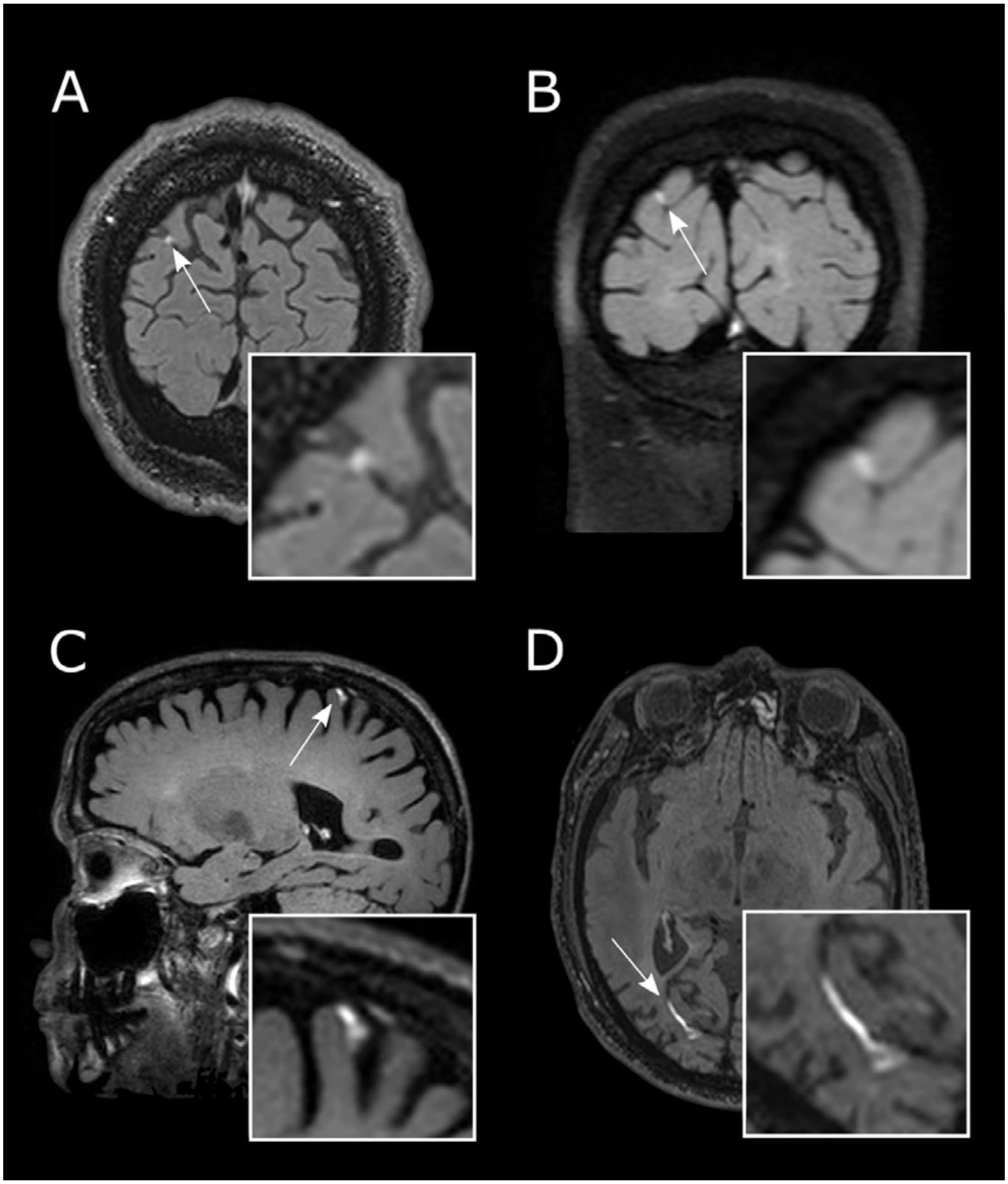
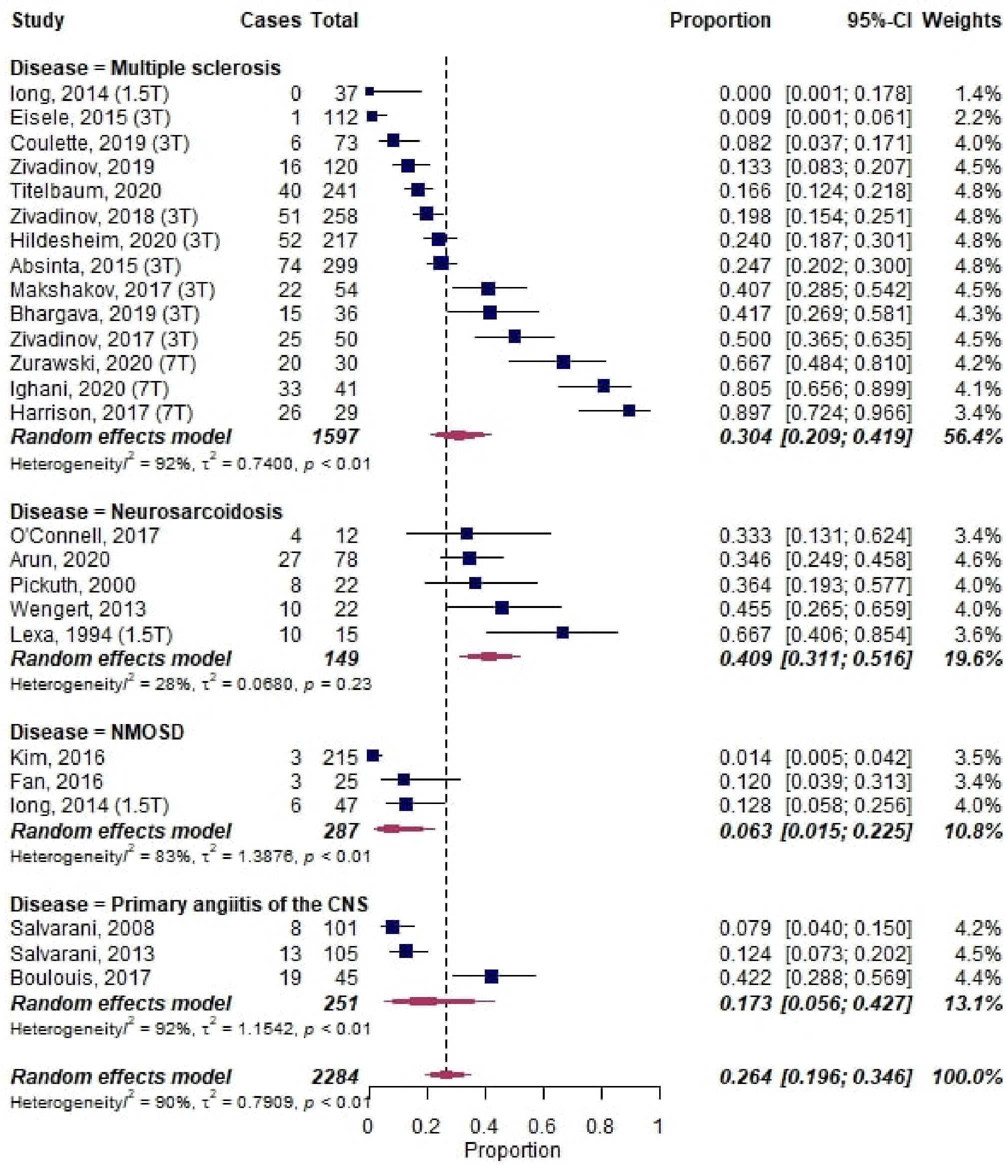
Figures

Figure 1: Leptomeningeal enhancement (LME) across the spectrum of neurological diseases.



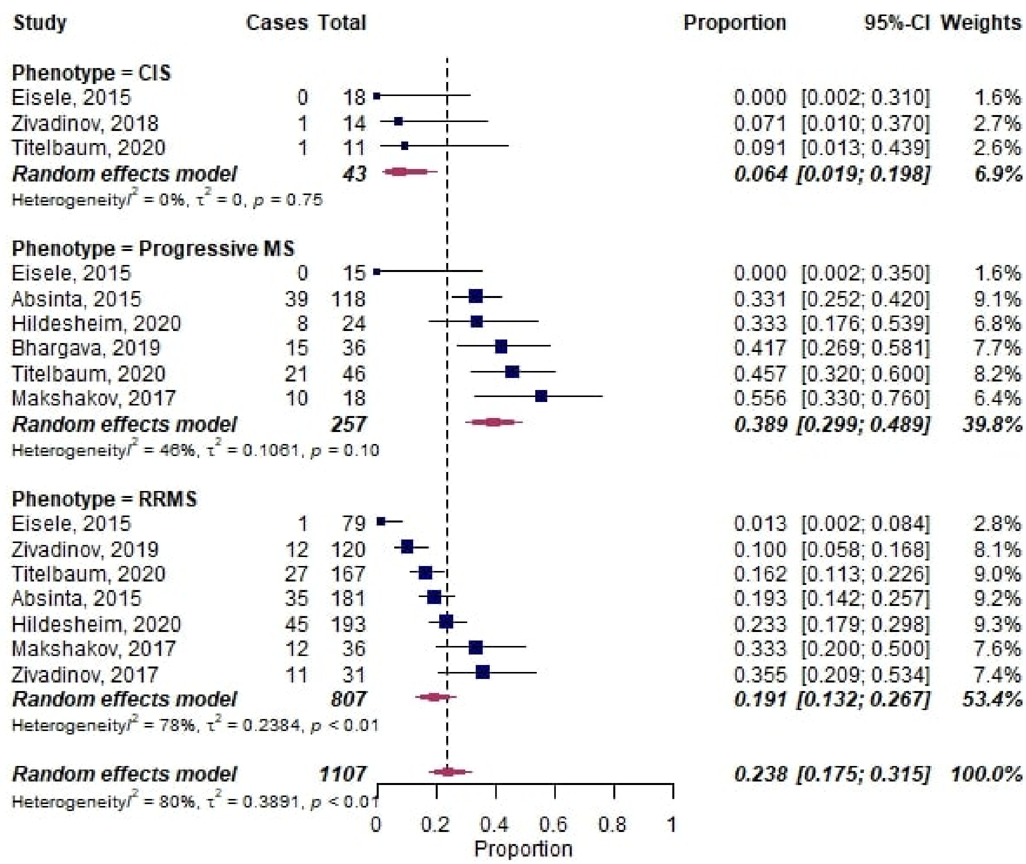
Leptomeningeal enhancement (LME, white arrows) can be detected using post-gadolinium fluid-attenuated inversion recovery (FLAIR) T2-weighted magnetic resonance imaging and can be present in viral diseases such as HIV (A, at 3T), in primary neuroinflammatory diseases such as Susac syndrome (B, at 3T) and multiple sclerosis (C, at 7T), and in aseptic meningitis (natalizumab-induced) (D, at 3T).

Figure 2: Forest plot of leptomeningeal enhancement (LME) proportions in primary neuroinflammatory diseases.



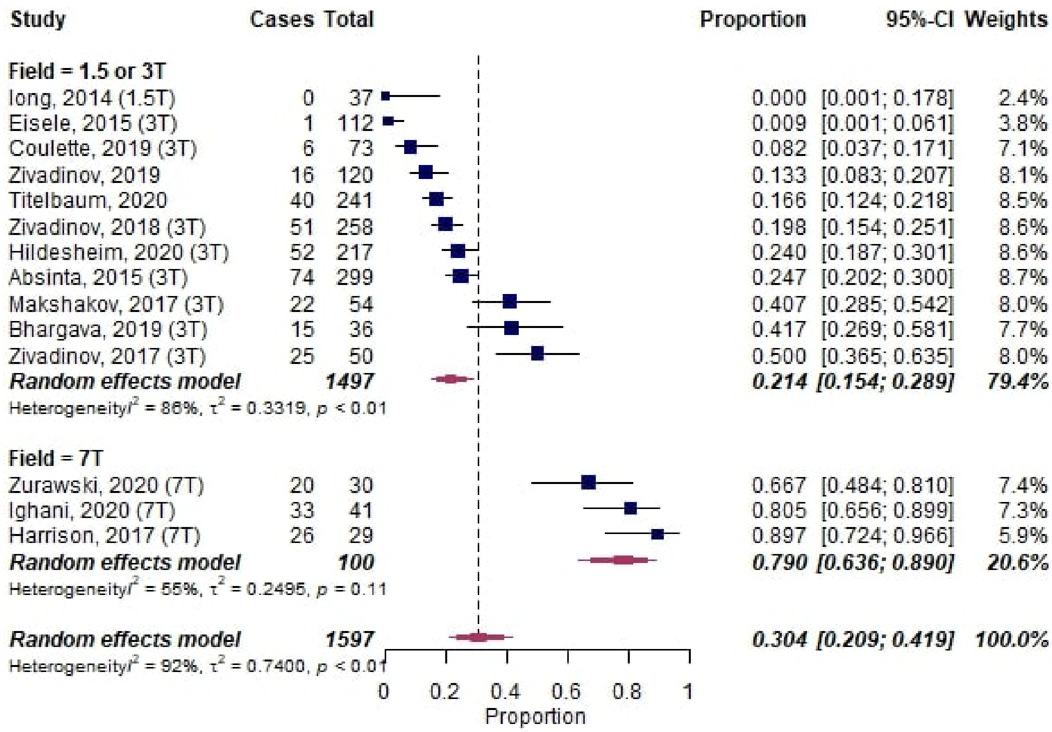
Pooled analyses of studies comparing the proportion of LME on MRI in neuroinflammatory diseases, stratified by diseases. Static magnetic field strength for MRI acquisition for respective studies are listed in brackets if reported. Proportions for LME were extracted and pooled using the random effects DerSimonian-Laird method. Abbreviations: CNS, central nervous system; CI, confidence interval; NMOSD, neuromyelitis optica spectrum disorder.

Figure 3: Forest plot of leptomeningeal enhancement (LME) proportions in multiple sclerosis (MS).



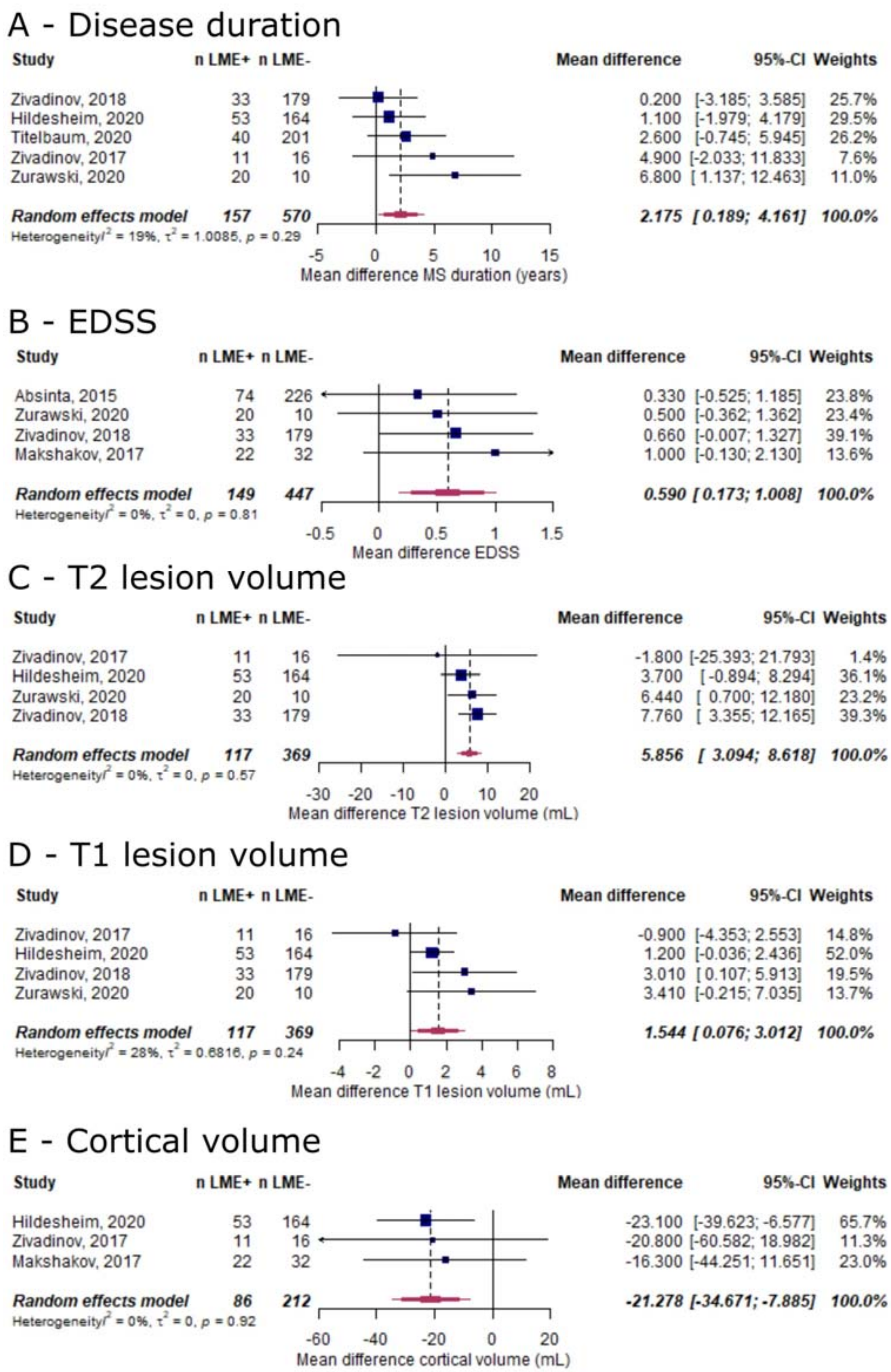
Pooled analyses of studies comparing the proportion of LME on MRI in MS, stratified by clinical phenotype. Proportions for LME were extracted and pooled using the random effects DerSimonian-Laird method. Abbreviations: *CI* confidence interval; CIS, clinically isolated syndrome; RRMS, relapsing-remitting multiple sclerosis.

Figure 4: Forest plot of leptomeningeal enhancement (LME) proportions in multiple sclerosis (MS) with B_0 magnetic field strength as moderator.



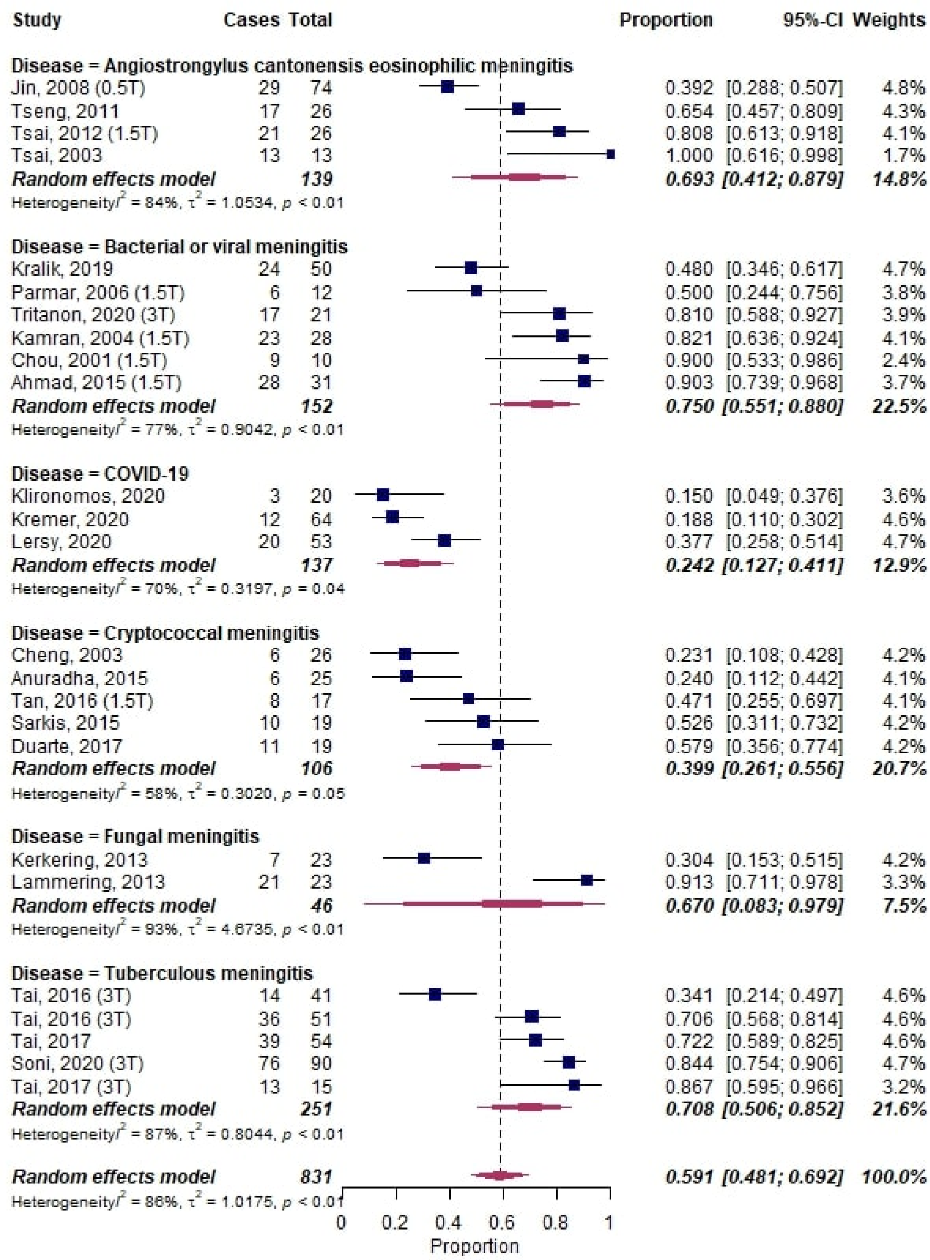
Pooled analyses of studies comparing the proportion of LME on MRI in MS, stratified by magnetic field strength. Proportions for LME were extracted and pooled using the random effects DerSimonian-Laird method. Abbreviations: *CI* confidence interval.

Figure 5: Forest plot for subgroup analysis for the association between LME and clinical and imaging outcomes in MS.



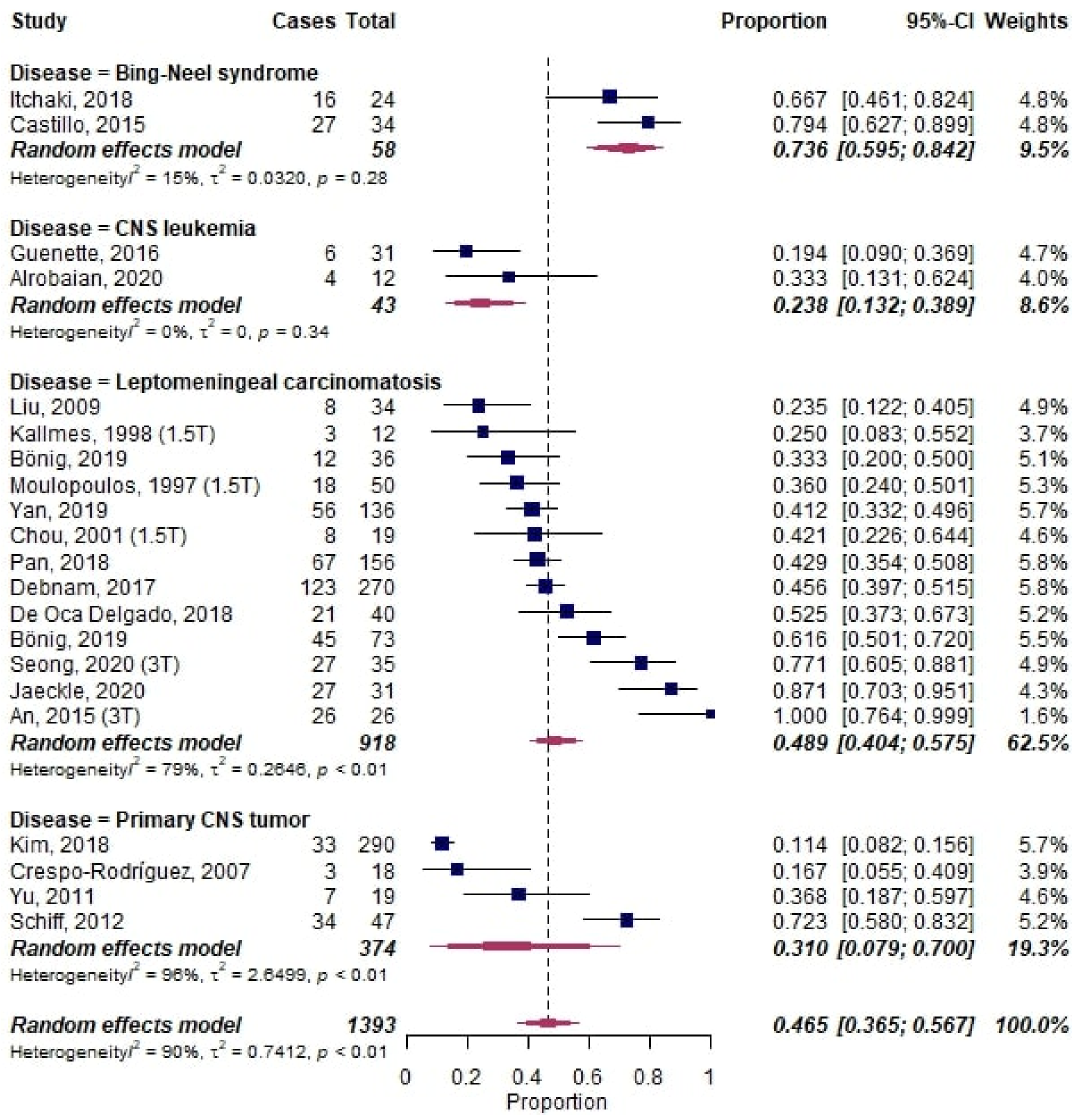
Pooled analyses of studies comparing the mean differences of clinical and imaging outcomes between LME-positive and LME-negative MS cases (A, disease duration [years]; B, EDSS [in EDSS points]; C, T1 lesion volume [in mL]; D, T2 lesion volume [in mL]; E, cortical gray matter volume [in mL]). Mean differences were extracted and pooled using the random effects DerSimonian-Laird method. Abbreviations: CI confidence interval; EDSS, Expanded Disability Status Scale; LME, leptomeningeal enhancement.

Figure 6: Forest plot of leptomeningeal enhancement (LME) proportions in infectious neurological diseases.



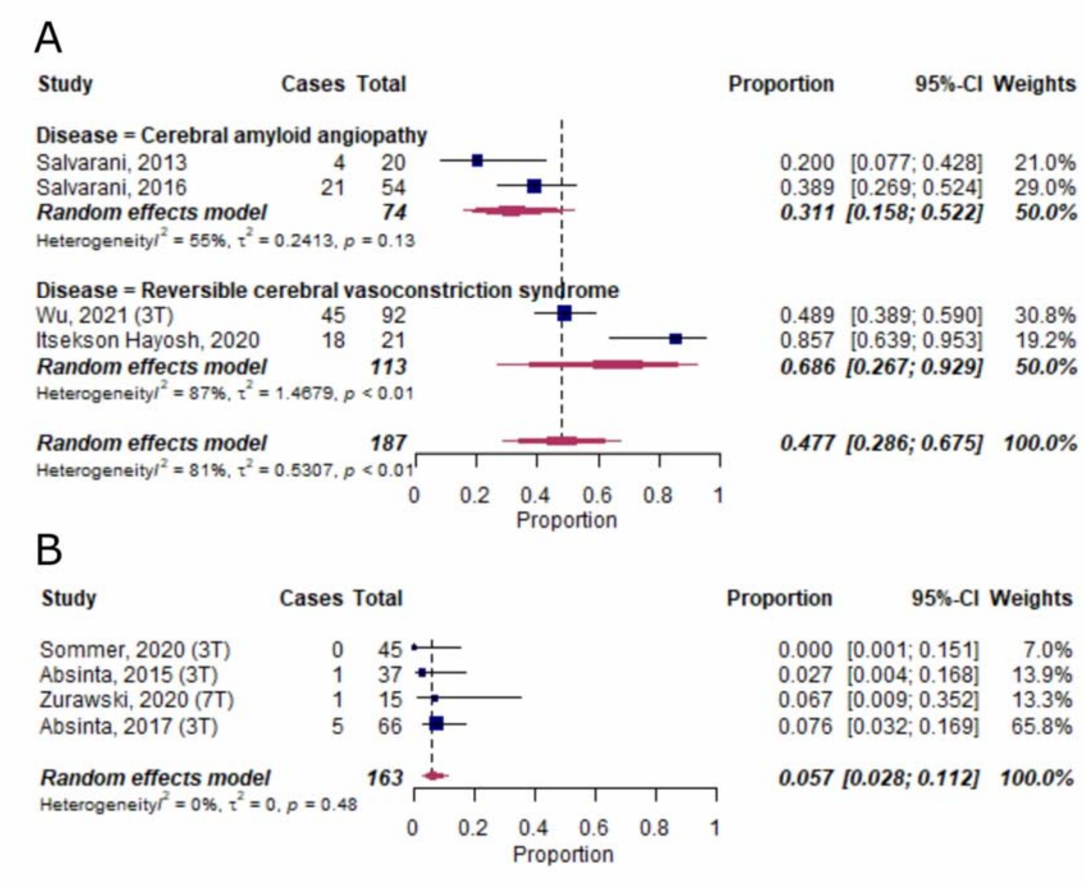
Pooled analyses of studies comparing the proportion of LME on MRI in infectious neurological diseases. Static magnetic field strength for MRI acquisition for respective studies are listed in brackets if reported. Proportions for LME were extracted and pooled using the random effects DerSimonian-Laird method. Abbreviations: CI confidence interval.

Figure 7: Forest plot of leptomeningeal enhancement (LME) proportions in neoplastic neurological diseases.



Pooled analyses of studies comparing the proportion of LME on MRI in infectious neurological diseases. Static magnetic field strength for MRI acquisition for respective studies are listed in brackets if reported. Proportions for LME were extracted and pooled using the random effects DerSimonian-Laird method. Abbreviations: CI confidence interval; CNS, central nervous system.

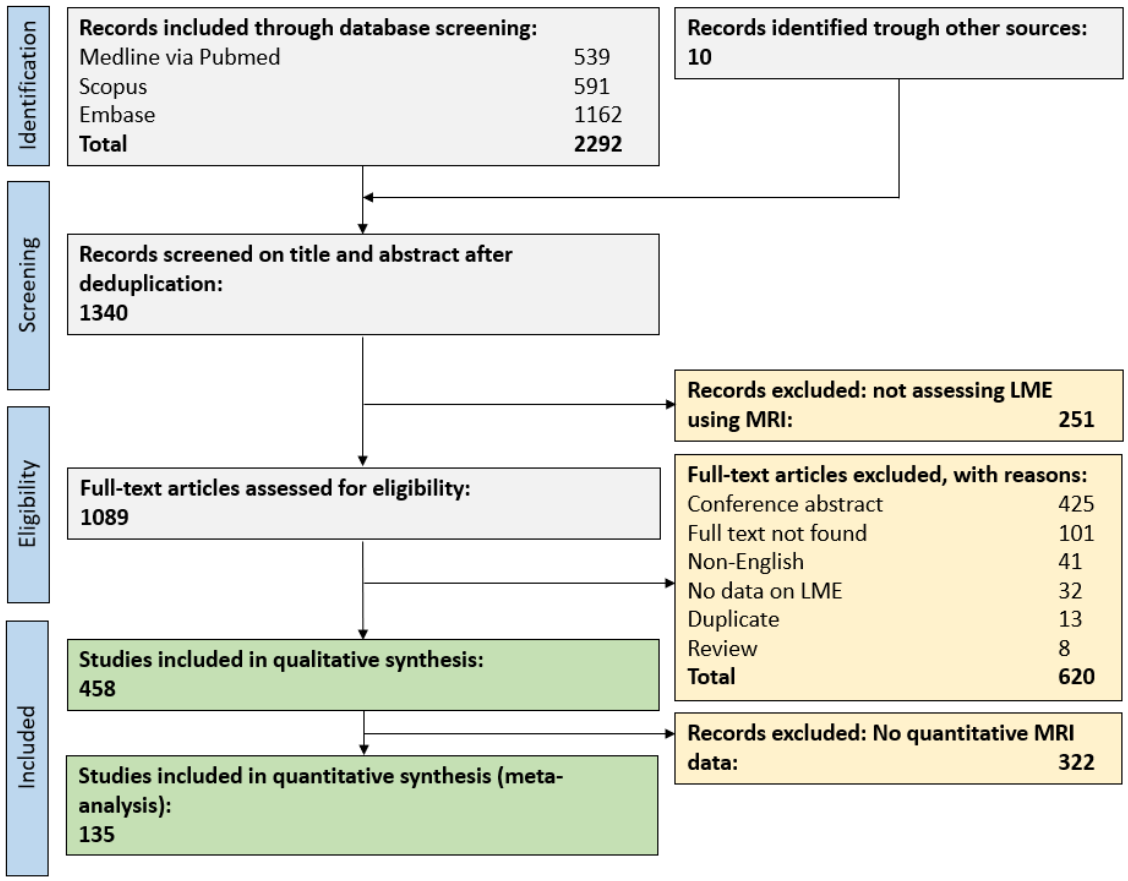
Figure 8: Forest plot of leptomeningeal enhancement (LME) proportions in neurological diseases not classified into the other groups and in healthy controls.



Pooled analyses of studies comparing the proportion of LME on MRI in other neurological diseases (A) and in healthy control subjects (B). Static magnetic field strength for MRI acquisition for respective studies are listed in brackets if reported. Proportions for LME were extracted and pooled using the random effects DerSimonian-Laird method. Abbreviations: CI confidence interval.

Supplementary figure

Figure S1: Flow chart for study inclusion.



Tables

Table 1: Synopsis of studies assessing leptomeningeal enhancement (LME) in multiple sclerosis (MS).

Study	n	Controls	Location	MRI field	MRI sequence	Pattern	Main findings
Eisele, 2015	112	5 stroke	Convexities	3T	Pc. 2D T2w-FLAIR (at least 10 min delay), pc. 2D T1w	Nodular	Only 1/112 patient (<1%) showed evidence of LME in the right temporal lobe. This patient also showed 6 parenchymal contrast-enhancing lesions. <u>Association of LME with imaging/clinical parameters:</u> N/A <u>Temporal dynamics of LME:</u> N/A
Absinta, 2015	171 RRMS, 74 PPMS, 44 SPMS, 10 CIS	37 HC	N/A	3T in vivo, 7T postmortem	Pc. 3D T2w FLAIR (at least 10 min delay), pc. 3D T1w	Laminar, nodular	Focal LME was detected in the leptomeningeal compartment in 74 of 299 MS cases (25%) and in only 1 of 37 neurologically healthy controls (2.7%). Progressive MS showed around twice as much enhancement (39/118 cases, 33%) compared to relapsing-remitting MS (35/181, 19%). <u>Association of LME with imaging/clinical parameters:</u> Median age, disease duration, and EDSS were higher in LME+ MS patients compared to LME- patients. Relapsing or progressive phenotype were not associated with presence of LME. Whole brain and cortical volumes were lower in LME+ MS patients, but no difference for white matter lesions or white matter lesion volume. No association of LME with oligoclonal bands. Correlative histopathology showed perivascular lymphocytic and mononuclear infiltration in the enhancing areas in association with adjacent subpial cortical demyelination. <u>Temporal dynamics of LME:</u> Most LME foci (53/62, 85%) remained stable in shape and size throughout the evaluation period (up to 5.5 years). 1 LME focus disappeared; 6 new foci were detected in 4 patients.

Harrison, 2017	21 RRMS, 4 SPMS, 4 PPMS	3 HC	Convexities	7T	Pc. 3D T2w-MPFLAIR (20 min delay), pc. 3D T1w MP2RAGE (3 min delay)	Spread/fill (76% of subjects) and nodular (15% of subjects). Both types occurred simultaneously in 38% of patients.	LME on postcontrast 7T MPFLAIR is more prevalent than prior reports at 3T. T1w MP2RAGE images were consulted to exclude leptomeningeal vessels. Of note, no instances of spread/fill foci were seen in healthy controls. However, 2/3 healthy controls had nodular LME (normal variant?). <u>Association of LME with imaging/clinical parameters:</u> Spread/fill foci were associated with reduced cortical gray matter volumes. There were no differences in WM lesion, cerebral WM volume, thalamus, caudate, or putamen volume in those with and without spread/fill LME foci. <u>Temporal dynamics of LME:</u> N/A
Absinta, 2017	299 MS patients (same as in Absinta, 2015),	189 non-MS, 66 HC	Convexities, infratentorial	3T	Pc. 3D T2w FLAIR (at least 10 min delay), pc. 3D T1w	Nodular or linear	LME was observed in 56/254 non-MS patients (22%) compared to 74/299 (25%) of MS patients. LME was around 4-fold more common in non-MS inflammatory neurologic diseases (18/51 cases, 35%) than in noninflammatory neurologic diseases (3/38, 8%) and healthy volunteers (5/66, 8%). The highest prevalence of LME was detected in HTLV infections (17/38 cases, 45%), particularly in the setting of HTLV-associated myelopathy (14/25 cases, 56%). LME was also frequently detected in HIV infection (13/61 cases, 21%). <u>Association of LME with imaging/clinical parameters:</u> Unlike in MS, LME was not associated with lower brain and cortical volumes in non-MS inflammatory neurologic conditions, including HTLV and HIV infection. <u>Temporal dynamics of LME:</u> N/A
Makshakov, 2017	54	0	Convexities (Mainly sulcal (65%) and less brain surface (35%)	3T	Pc. 3D T2w-FLAIR (after T1w), pc. 3D T1w (lower sens for LME detection than T2w-FLAIR)	Linear (13%), plate-like (31%), nodular (54%)	LME was detected in 41% of MS patients. <u>Association of LME with imaging/clinical parameters:</u> LME+ patients had longer disease duration and higher EDSS score, but an equal relapse rate. No association of LME with higher frequency of contrast-enhancing lesions. LME+ patients had lower cortical volume, the total grey matter volume as well as total ventricular volume. No difference in oligoclonal bands or kappa-FLC.

							Temporal dynamics of LME: N/A
Xia, 2017	100 first-degree relatives of MS	0	N/A	3T	Pc. 3D T2w FLAIR (at least 10 min delay), pc. 3D T1w	N/A	Higher-risk asymptomatic family members of patients with MS are more likely to have early subclinical manifestations of MS and deserve further monitoring. A subset of participants harboured LME, consistent with the hypothesis that these subjects ar at higher risk for developing MS. Association of LME with imaging/clinical parameters: N/A Temporal dynamics of LME: N/A
Zivadinov, 2017	27 RRMS, 23 SPMS	0	Convexities (mainly surface (79%) and less sulcal (21%)	3T	Pc. 3D T2w-FLAIR (10 min delay), pc. 2D FLAIR (after Gd-injection), pc. 2D T1w	Most of the LME foci were nodular (49, 80%), linear, plate-like	In total, 25/50 MS patients (50%) showed LME at the 5-year follow-up. Of note, No LME foci were detected on 2D T2w-FLAIR or 2D T1w sequences. Association of LME with imaging/clinical parameters: SPMS presented with significantly more LME foci (12, 85.7%) compared to RRMS (2, 18.2%). LME+ MS patients had greater percentage decrease in total GM (−3.6% vs −2%) and cortical (−3.4% vs −1.8%) volumes and greater percentage increase in ventricular cerebrospinal fluid volume (22.8% vs 9.9%) over the follow-up compared to LME- MS patients. SPMS patients with LME showed higher T1 lesion volume increase compared to patients without LME. No difference in annual relapse rate or DMT. Temporal dynamics of LME: N/A
Jonas, 2018	21 RRMS, 7 SPMS, 3 PPMS	0	Convexities, mostly frontoparietal (75%)	7T	Pc. 3D MP2RAGE (3 min delay, pc. MPFLAIR (20 min delay)	Subarachnoid spread/fill or nodular, vessel wall, dural (also including non-LME enhancement patterns)	2-year follow up study. At baseline, 284 LME foci among 31 MS patients are reported., 25/50 MS patients (50%) showed LME at the 5-year follow-up. Of note, No LME foci were detected on 2D T2w-FLAIR or 2D T1w sequences. Association of LME with imaging/clinical parameters: No difference in the total number/proportion of longitudinally persistent LME foci between those on or off treatment or between pro-

							gressive versus relapsing MS. More persistent LME foci were present in EDSS progressors compared to non-EDSS progressors (median, 12; range 1–15 versus median, 7.5; range 1–24). Temporal dynamics of LME: In total, 15 additional LME foci developed within the follow-up period. LME patterns were: 6 subarachnoid spread/fill, 4 subarachnoid nodular, 2 vesselwall, and 3 dural foci.
Zivadinov, 2018	212 RRMS, 32 SPMS, 14 CIS	0	Convexities	3T	Pc. 3D T2w-FLAIR (10 min delay; native, sub-tracted, or co-registered)	N/A	Study compared LME detection on 3D T2w-FLAIR postcontrast images in native space (method 1), on pre- and postcontrast 3D T2w-FLAIR images in native space (method 2), and on pre-/postcontrast 3D T2w-FLAIR co-registered and subtracted images (method 3). In total, 51 (20%) patients with MS showed LME using method 1; 39 (15%) using method 2; and 39 (15%), The mean time to analyze the 3D T2w-FLAIR images was lower with method 2 compared to the other 2 methods. Association of LME with imaging/clinical parameters: Higher T1 and T2 lesion volume in LME+ MS patients. Similar volume of contrast-enhancing lesions between LME+ and LME- patients. LME+ patients had higher EDSS and age but similar MS disease duration. Temporal dynamics of LME: N/A
Bergsland, 2019	43 RRMS, 15 SPMS	0	N/A	3T	Pc. 3D T2w-FLAIR (10 min delay), Pc. 3D T1w	N/A	Focal LME is associated with reduced thickness of the adjacent cortex in patients with RRMS, but not in those with secondary-progressive MS. Association of LME with imaging/clinical parameters: See above. Temporal dynamics of LME: N/A
Bhargava, 2019	36 progressive MS	0	N/A	3T	Pc. 3D T2w-FLAIR (10 min delay), Pc. 3D T1w	N/A	Study assessed safety of intrathecal rituximab on progressive MS and its potential effect on LME. Out of 36 screened patients, 15 had LME (42%). LME frequency did not change following intrathecal rituximab treatment. Association of LME with imaging/clinical parameters:

							N/A Temporal dynamics of LME: There was no change in the number or shape of LME during the 24-week follow-up period. There was no appearance of new LME over the course of the study.
Coulette, 2019	73 MS	9 Susac	Convexities (MS), Cerebellar (Susac)	3T	Pc. 3D T2w-FLAIR (3 min delay), Pc. 3D T1w	2 nodular, 4 linear	Susac syndrome patients were more likely to present with LME: 5/9 (56%) versus 6/73 (8%) in the MS group. Association of LME with imaging/clinical parameters: N/A Temporal dynamics of LME: No evolution data on MS.
Zivadinov, 2019	120 RRMS	0	N/A	3T	Pc. 3D T2w-FLAIR (10 min delay), pc. 3D T1w	N/A	No significant difference in LME between teriflunomide or dimethylfumarate treated patients. 12 out of 120 patients with LME (10%). Association of LME with imaging/clinical parameters: N/A Temporal dynamics of LME: Out of 8 dimethylfumarate-treated patients who presented with LME at baseline, 6 continued to show the same LME foci at 12 and 24 months, one other patient under dimethylfumarate developed a new LME focus over the follow-up. Of 4 teriflunomide-treated patients who presented with LME foci at baseline, 2 patients continued to present the same LME foci at follow-up, whereas one patient treated with teriflunomide devel-oped a new LME focus over the follow-up
Bonnan, 2020	1 prog MS	0	Convexities	3T	Pc. 3D T2w-FLAIR	laminar	Spontaneously remitting LME focus in 1 SPMS patient. Association of LME with imaging/clinical parameters: LME focus was associated with adjacent cortical thinning. Temporal dynamics of LME: Disappearance of focus at 3 and 9 months follow-up.
Hildesheim,	193 RRMS,	0	Convexities	3T	Pc. 3D T2w-	Nodular (89%),	Fifty-three out of 217 MS patients (24%) had at least one LME focus.

2020	24 progressive MS				FLAIR (10 min delay, subtraction), Pc. 3D T1w	filling-like (11%)	Association of LME with imaging/clinical parameters: No difference in LME between relapsing and progressive MS (23% vs. 33%). No difference in EDSS between LME+ and LME- MS patients when adjusted for age. LME+ MS patients showed higher CSF volume and more contrast-enhancing lesions compared to LME- patients at baseline. LME frequency was associated with higher age. LME was not associated with clinical or imaging markers of MS severity (T1 and T2 lesion volume). Temporal dynamics of LME: analyzed for persistence over 18 months follow-up. Of the 76 LME foci at baseline, 68 foci (90%) remained stable in shape and size. 8 LME foci resolved during the follow-up interval, a total of 14 LME foci were newly detected.
Ighani, 2020	31 RRMS, 5 SPMS, 5 PPMS	5 HC	N/A	7T	Pc. 3D T2w-MPFLAIR (unclear delay), pc. 3D MP2-RAGE T1w	Nodular, spread/fill-sulcal (59%), spread/fill-gyral (61%), spread/fill-infratentorial	33/41 MS patients had LME (81%) and 27 had > 1 LME focus. One LME focus was found in 3/5 healthy controls (60%), one subject with a nodular LME and two cases with one spread/fill-sulcal LME each. None of the control subjects had >1 LME focus. Association of LME with imaging/clinical parameters: There was an association between spread/fill-sulcal LME and hippocampal lesion count in RRMS. Participants with RRMS had no correlation with cortical lesions, but significant correlations were detected between LME and hippocampal lesion count, normalized cortical gray matter volume, and mean cortical thickness. WM lesion volume was greater in patients with >1 focus of spread/fill-sulcal LME compared to those with ?1 focus. Temporal dynamics of LME: N/A
Titelbaum, 2020	241	100 non-MS	Convexities, rarely cerebellar	1.5T (most patients), 3T	Pc. 3D T2w-FLAIR (delay 7 – 54 min)	Nodular, curvilinear	A total of 16.6% (40/241) of MS patients had LME compared to 8% (8/100) in non-MS patients. There was no association with MS subtype, therapy, or disease activity. General Electric's version of 3D T2w-FLAIR (29%) was greater than with Siemen's 3D T2w-FLAIR (12%) at 1.5T. LME kinetics were heterogeneous, even within patients, without uniform optimal time for acquisition. Of note, Imaging pitfalls fell into three categories: contrast leakage of

							uncertain medical significance; enhancements related to cortical veins/anatomic structures; and imaging artifacts <u>Association of LME with imaging/clinical parameters:</u> N/A <u>Temporal dynamics of LME:</u> LME foci were overall persistent over the observation period (0.5 – 6 months) but resolved in 2 patients following high-dose steroids.
Zurawski, 2020	30 RRMS	15 HC	Convexities	7T	Pc. 3D T2w-FLAIR (10 min delay), pc. 3D MP2RAGE T1w	nodular only n=12 (60%), any spread n=8 (40%)	Two thirds of MS patients had LME. Patients had a mean of 2.7 (± 1.5) LME foci. <u>Association of LME with imaging/clinical parameters:</u> LME+ patients had longer disease duration, a sixfold higher cortical lesion volume, and a higher T1/T2 lesion volume compared to LME-patients. The number of LME foci correlated with cortical and thalamic lesions. Patients with spread LME significantly higher cortical lesion volumes. <u>Temporal dynamics of LME:</u> N/A

Studies are in chronological and alphabetical order. *Abbreviations: FLAIR, fluid-attenuated inversion recovery (MP, magnetization-prepared); HC, healthy control; LME, leptomeningeal enhancement; MS, multiple sclerosis (RR relapsing-remitting, SP, secondary progressive; PP, primary progressive); pc, post-contrast.*

Supplementary data

Supplementary search string

In alphabetical order

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Supplementary tables

Table S1: Predefined systematic search strings in PubMed Ovid EMBASE and Web of Science. Last search 2nd of February, 2021.

Databases	Studies produced	Search string
Pubmed via Medline:	539	Leptomeningeal Enhancement OR Leptomeningeal Contrast Enhancement OR Leptomeningeal Inflammation OR pericortical enhancement
Scopus:	591	Leptomeningeal Enhancement OR Leptomeningeal Contrast Enhancement OR Leptomeningeal Inflammation OR pericortical enhancement
Web of Science:	1162	Leptomeningeal Enhancement OR Leptomeningeal Contrast Enhancement OR Leptomeningeal Inflammation OR pericortical enhancement

Table S1: Risk of bias assessment of the included studies with 10 or more included human subjects according to the Newcastle-Ottawa scale for nonrandomized studies. S1-S4: Selection domain (only S1-S3 for cohort studies, case series and retrospective studies), C1a and C1b: comparability domain, E1-E2: exposure domain. In alphabetical order and sorted for disease class.

Author, year	Title	Type	S1	S2	S3	S4	C1a	C1b	E1	E2
Ahmad, 2015	Differentiation of leptomeningeal and vascular enhancement on post-contrast FLAIR MRI sequence: Role in early detection of infectious meningitis	Infectious	*	*	N/A	*	*	-	-	N/A
Anuradha, 2015	Cryptococcal meningitis in people living with hiv (PLHIV): A comparative analysis of the diagnostic tests	Infectious	*	*	N/A	-	-	-	N/A	N/A
Cheng, 2003	Radiological manifestations of cryptococcal infection in central nervous system	Infectious	*	*	N/A	-	-	-	N/A	N/A
Crete, 2018	Spinal coccidioidomycosis: MR imaging findings in 41 patients	Infectious	*	*	N/A	-	*	*	N/A	N/A
Deuble, 2013	Neurologic melioidosis	Infectious	*	*	N/A	*	*	-	N/A	N/A
Duarte, 2017	Magnetic resonance imaging findings in central nervous system cryptococcosis: comparison between immunocompetent and immunocompromised patients	Infectious	*	*	N/A	-	-	-	N/A	N/A
Gupta, 2016	A prospective study of magnetic resonance imaging patterns of central nervous system infections in pediatric age group and young adults and their clinico-biochemical correlation	Infectious	*	*	N/A	*	-	-	N/A	N/A
Imam, 2015	Adult tuberculous meningitis in Qatar: a descriptive retrospective study from its referral center	Infectious	*	*	N/A	-	N/A	-	*	N/A
Jin, 2008	Magnetic resonance imaging of eosinophilic meningoencephalitis caused by Angiostrongylus cantonensis following eating freshwater snails	Infectious	*	*	N/A	*	-	-	N/A	N/A
Kamran, 2004	Role of Fluid-Attenuated Inversion Recovery in the Diagnosis of Meningitis: Comparison with Contrast-Enhanced Magnetic Resonance Imaging	Infectious	*	*	-	*	*	*	N/A	N/A
Kerkering, 2013	Early clinical observations in prospectively followed patients with fungal meningitis related to contaminated epidural steroid injections	Infectious	*	*	N/A	-	-	-	N/A	N/A
Klironomos, 2020	Nervous System Involvement in Coronavirus Disease 2019: Results from a Retrospective Consecutive Neuroimaging Cohort	Infectious	*	*	N/A	*	*	-	N/A	N/A
Kralik, 2019	Comparison of CSF and MRI Findings among Neonates and Infants with E coli or Group B Streptococcal Meningitis	Infectious	*	*	N/A	*	-	-	N/A	N/A
Kremer, 2020	Neurologic and neuroimaging findings in patients with COVID-19: A retrospective multicenter study	Infectious	*	*	N/A	*	*	*	N/A	N/A

Kumar, 2010	Revisiting the CNS tuberculosis with emphasis on giant tuberculomas and introducing the outer RIM excrescence sign	Infectious	-	-	N/A	-	-	-	N/A	N/A
Lammering, 2013	Imaging spectrum of CNS coccidioidomycosis: Prevalence and significance of concurrent brain and spinal disease	Infectious	*	*	N/A	-	-	-	N/A	N/A
Lersy, 2020	Cerebrospinal fluid features in COVID-19 patients with neurologic manifestations: correlation with brain MRI findings in 58 patients	Infectious	*	*	N/A	-	-	-	N/A	N/A
Oliveira, 2014	Brain magnetic resonance imaging of infants with bacterial meningitis	Infectious	*	*	N/A	-	-	-	N/A	N/A
Parmar, 2006	Contrast-enhanced flair imaging in the evaluation of infectious leptomeningeal diseases	Infectious	*	*	*	*	-	-	N/A	N/A
Sarkis, 2015	MRI findings in cryptococcal meningitis of the non-HIV population	Infectious	-	*	N/A	-	-	-	N/A	N/A
Soni, 2020	Cerebrovascular complications in tuberculous meningitis—A magnetic resonance imaging study in 90 patients from a tertiary care hospital	Infectious	*	*	N/A	*	-	-	N/A	N/A
Speers, 2013	Clinical and radiological predictors of outcome for murray valley encephalitis	Infectious	*	*	N/A	*	-	-	N/A	N/A
Tai, 2016	Cerebral infarction pattern in tuberculous meningitis	Infectious	*	*	N/A	*	-	-	N/A	N/A
Tai, 2016	Paradoxical manifestation is common in HIV-negative tuberculous meningitis	Infectious	*	*	N/A	*	*	-	N/A	N/A
Tai, 2017	Neuroimaging findings are sensitive and specific in diagnosis of tuberculous meningitis	Infectious	*	*	N/A	*	*	-	N/A	N/A
Tai, 2017	Role of cytokines in the assessment of clinical outcome and neuroimaging findings in patients with tuberculous meningitis	Infectious	*	*	N/A	-	-	-	N/A	N/A
Tai, 2017	Spinal tuberculous disease is common in tuberculous meningitis	Infectious	*	*	N/A	*	*	-	N/A	N/A
Tai, 2018	Tuberculous optochiasmatic arachnoiditis and optochiasmatic tuberculoma in Malaysia	Infectious	*	*	N/A	*	*	-	N/A	N/A
Tan, 2016	Spectrum of neuroimaging findings in cryptococcal meningitis in immunocompetent patients in China — A series of 18 cases	Infectious	*	*	N/A	*	-	-	N/A	N/A
Thurnher, 2000	MRI of infections and neoplasms of the spine and spinal cord in 55 patients with AIDS	Infectious	*	*	N/A	*	*	-	N/A	N/A
Tritanon, 2020	Infectious leptomeningitis: Diagnostic value of contrast-enhanced 3D fluid-attenuated inversion recovery with fat suppression, contrast-enhanced 3D spoiled gradient-echo high resolution T1-weighted and contrast-enhanced T1-weighted images correlated with CSF analysis	Infectious	*	*	N/A	*	-	-	N/A	N/A
Tsai, 2003	Eosinophilic meningitis caused by Angiostrongylus cantonensis associated with eating raw snails: correlation of brain magnetic resonance imaging scans with clinical findings	Infectious	*	*	N/A	-	-	-	N/A	N/A
Tsai, 2012	Brain magnetic resonance imaging abnormalities in eosinophilic meningitis caused by angiostrongylus	Infectious	*	*	N/A	-	-	-	N/A	N/A

	cantonensis infection									
Tseng, 2011	Clinical manifestations of eosinophilic meningitis caused by Angiostrongylus cantonensis: 18 years' experience in a medical center in southern Taiwan	Infectious	*	*	N/A	-	-	-	N/A	N/A
Guo, 2018	Brain MRI findings in acute hepatic encephalopathy in liver transplant recipients	Metabolic	*	*	N/A	-	-	*	N/A	N/A
Alrobaian, 2020	Neuro-Ophthalmic Manifestations of Acute Leukemia	Neoplastic	*	*	N/A	*	*	-	N/A	N/A
An, 2015	An NMR metabolomics approach for the diagnosis of leptomeningeal carcinomatosis in lung adenocarcinoma cancer patients	Neoplastic	*	*	*	*	*	-	N/A	N/A
Andica, 2019	Aberrant myelination in patients with Sturge-Weber syndrome analyzed using synthetic quantitative magnetic resonance imaging	Neoplastic	*	*	N/A	*	N/A	-	N/A	N/A
Arulrajah, 2010	MRI with diffusion-weighted imaging in children and young adults with simultaneous supra- and infratentorial manifestations of Sturge-Weber syndrome	Neoplastic	*	*	N/A	*	*	-	N/A	N/A
Bar, 2020	Early magnetic resonance imaging to detect presymptomatic leptomeningeal angioma in children with suspected Sturge–Weber syndrome	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Bönig, 2019	Leptomeningeal metastasis: The role of cerebrospinal fluid diagnostics	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Castillo, 2015	Bing-neel syndrome: A multi-institutional retrospective study	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Chou, 2001	Intracranial Meningeal Carcinomatosis and Non-neoplastic Meningeal Diseases: Evaluation with Contrast-Enhanced MR Imaging	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Crespo-Rodríguez, 2007	MR and CT imaging of 24 pleomorphic xanthoastrocytomas (PXA) and a review of the literature	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
De Oca Delgado, 2018	The comparative treatment of intraventricular chemotherapy by Ommaya reservoir vs. Lumbar puncture in patients with leptomeningeal carcinomatosis	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Debnam, 2017	Most common sites on MRI of intracranial neoplastic leptomeningeal disease	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Griffiths, 1996	Choroid plexus size in young children with Sturge-Weber syndrome	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Griffiths, 2003	Contrast-enhanced fluid-attenuated inversion recovery imaging for leptomeningeal disease in children	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Guenette, 2016	MRI findings in patients with leukemia and positive CSF cytology: A single-institution 5-year experience	Neoplastic	*	*	N/A	*	*	-	N/A	N/A
Ho, 2015	Cytomorphologic and clinicoradiologic analysis of primary nonhematologic central nervous system tumors with positive cerebrospinal fluid	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Itchaki, 2018	Ibrutinib for the treatment of bing-neel syndrome	Neoplastic	*	-	-	N/A	-	-	N/A	N/A

Jaeckle, 2020	Intra-CSF topotecan in treatment of breast cancer patients with leptomeningeal metastases	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Kallmes, 1998	High-dose gadolinium-enhanced MRI for diagnosis of meningeal metastases	Neoplastic	*	*	*	N/A	*	-	N/A	N/A
Kim, 2018	Leptomeningeal enhancement on preoperative brain MRI in patients with glioblastoma and its clinical impact	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Le Rhun, 2019	The RANO Leptomeningeal Metastasis Group proposal to assess response to treatment: lack of feasibility and clinical utility and a revised proposal	Neoplastic	-	*	N/A	*	*	*	N/A	N/A
Liu, 2009	Cerebrospinal fluid cytology and clinical analysis of 34 cases with leptomeningeal carcinomatosis	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Marcrom, 2017	Focal management of large brain metastases and risk of leptomeningeal disease	Neoplastic	*	*	*	N/A	*	-	N/A	N/A
Moulopoulos, 1997	A second look at unenhanced spinal magnetic resonance imaging of malignant leptomeningeal disease	Neoplastic	-	-	N/A	*	*	-	N/A	N/A
Oh, 2009	Gastric leptomeningeal carcinomatosis: Multi-center retrospective analysis of 54 cases	Neoplastic	*	-	*	N/A	*	-	N/A	N/A
Pan, 2018	Leptomeningeal metastasis from solid tumors: Clinical features and its diagnostic implication	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Porto, 2010	Leptomeningeal metastases in pediatrics: magnetic resonance image manifestations and correlation with cerebral spinal fluid cytology	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Rinaldo, 2018	Outcomes following cerebrospinal fluid shunting in high-grade glioma patients	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
River, 1996	Clinical significance of diffuse dural enhancement detected by magnetic resonance imaging	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Rodriguez, 2012	Disseminated oligodendroglial-like leptomeningeal tumor of childhood: A distinctive clinicopathologic entity	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Salvarani, 2018	Primary central nervous system vasculitis associated with lymphoma	Neoplastic	*	*	*	-	*	*	N/A	N/A
Schiff, 2012	Presentation, diagnostics and treatment of primary leptomeningeal lymphoma: An international primary cns lymphoma collaborative group series	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Schluterman, 2004	Multiple myeloma invasion of the central nervous system	Neoplastic	*	*	N/A	-	*	-	N/A	N/A
Seong, 2020	Diagnostic accuracy of MR imaging of patients with leptomeningeal seeding from lung adenocarcinoma based on 2017 RANO proposal: added value of contrast-enhanced 2D axial T2 FLAIR	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Sommer, 2020	Contrast-enhanced modified 3D T1-weighted TSE black-blood imaging can improve detection of infectious and neoplastic meningitis	Neoplastic	*	*	N/A	*	*	-	N/A	N/A
Wagner, 2017	Radiation injury versus malignancy after stereotactic radiosurgery for brain metastases: Impact of time-dependent changes in lesion morphology on MRI	Neoplastic	*	*	N/A	*	*	-	N/A	N/A
Whitehead, 2015	Osseous intramedullary signal alteration and enhancement in Sturge-Weber syndrome: an early diagnostic clue	Neoplastic	*	*	N/A	*	-	-	N/A	N/A

Yan, 2019	The clinical characteristic and prognostic factors of leptomeningeal metastasis in patients with non-small-cell lung cancer-a retrospective study from one single cancer institute	Neoplastic	*	*	N/A	-	-	-	N/A	N/A
Yeom, 2013	Distinctive MRI features of pediatric medulloblastoma subtypes	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Yu, 2011	Pleomorphic xanthoastrocytoma: MR imaging findings in 19 patients	Neoplastic	*	*	N/A	*	-	-	N/A	N/A
Agarwal, 2015	Childhood posterior reversible encephalopathy syndrome: Magnetic resonance imaging findings with em-phasis on increased leptomeningeal FLAIR signal	Other	*	*	N/A	*	-	-	N/A	N/A
Alonso, 2015	Leptomeningeal contrast enhancement and blood-CSF barrier dysfunction in aseptic meningitis	Other	*	*	N/A	*	-	-	N/A	N/A
Arun, 2020	Distinguishing neurosarcoidosis from multiple sclerosis based on CSF analysis: A retrospective study	Other	*	*	N/A	-	-	-	N/A	N/A
Blevins, 2003	Oculoleptomeningeal amyloidosis in a large kindred with a new transthyretin variant Tyr69His	Other	*	*	N/A	-	-	-	N/A	N/A
Cianfoni, 2013	Seizure-induced brain lesions: A wide spectrum of variably reversible MRI abnormalities	Other	*	*	N/A	*	*	-	N/A	N/A
George, 2020	Central Nervous System Fungal Infection-Related Stroke: A Descriptive Study of Mold and Yeast-Associated Ischemic Stroke	Other	*	*	N/A	-	-	-	N/A	N/A
Henegar, 1996	Early postoperative magnetic resonance imaging following nonneoplastic cortical resection	Other	*	*	N/A	*	-	-	N/A	N/A
Higuera-Ortiz, 2017	Pachymeningitis in granulomatosis with polyangiitis: case series with earlier onset in younger patients and literature review	Other	*	*	N/A	-	-	-	N/A	N/A
Hong, 2015	Leptomeningeal enhancement on magnetic resonance imaging as a predictor of hemodynamic insufficiency	Other	*	*	N/A	*	-	-	N/A	N/A
Itsekson Hayosh, 2020	Early Fluid Attenuation Inversion Recovery Sulcal Contrast Enhancement Correlates with Severity of Reversi-ble Cerebral Vasoconstriction Syndrome	Other	*	*	N/A	*	-	-	N/A	N/A
Jakchairoongruang, 2018	New insights into neurocutaneous melanosis	Other	*	*	N/A	*	-	-	N/A	N/A
Junger, 1993	Intramedullary spinal sarcoidosis: Clinical and magnetic resonance imaging characteristics	Other	*	*	N/A	-	-	-	N/A	N/A
Kim, 2012	Central nervous system (CNS) involvement is a critical prognostic factor for hemophagocytic lymphohistiocytosis	Other	*	*	N/A	-	-	-	N/A	N/A
Kim, 2020	The timelines of MRI findings related to outcomes in adult patients with new-onset refractory status epilepticus	Other	*	*	N/A	*	*	*	N/A	N/A
Kioumehr, 1995	Postcontrast mri of cranial meninges: Leptomeningitis versus pachymeningitis	Other	*	*	N/A	*	-	-	N/A	N/A
Komiyama, 2001	Leptomeningeal contrast enhancement in moyamoya: Its potential role in postoperative assessment of circu-lation through the bypass	Other	*	*	*	*	-	-	N/A	N/A

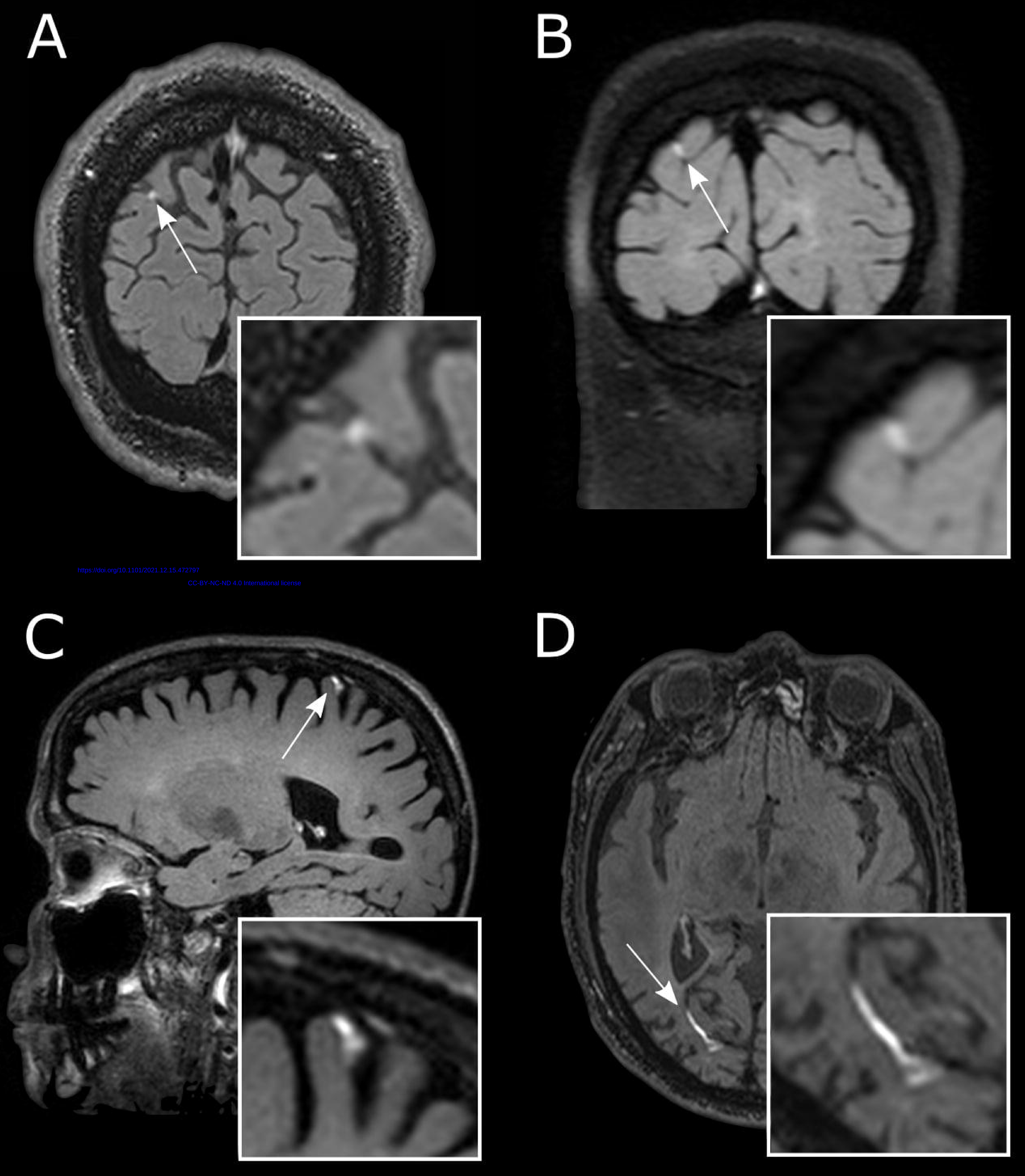
Kumar, 2019	To compare diagnostic ability of contrast-enhanced three-dimensional T1-SPACE with three-dimensional fluid-attenuated inversion recovery and three-dimensional T1-magnetization prepared rapid gradient echo magnetic resonance sequences in patients of meningitis	Other	*	*	N/A	*	*	-	N/A	N/A
Lee, 2002	Subcortical low intensity on MR images of meningitis, viral encephalitis, and leptomeningeal metastasis	Other	*	*	N/A	*	*	-	N/A	N/A
Lee, 2014	Usefulness of contrast enhanced FLAIR imaging for predicting the severity of meningitis	Other	*	*	N/A	*	*	-	N/A	N/A
Lee, 2016	Characteristic MRI findings in hyperglycaemia-induced seizures: diagnostic value of contrast-enhanced fluid-attenuated inversion recovery imaging	Other	*	*	N/A	*	-	-	N/A	N/A
Lexa, 1994	MR of sarcoidosis in the head and spine: Spectrum of manifestations and radiographic response to steroid therapy	Other	*	*	N/A	*	-	-	N/A	N/A
Li, 2018	Leptomeningeal Enhancement Is Associated with Transient Neurologic Deficits after Flow Diversion of Intra-cranial Aneurysms	Other	*	*	N/A	*	-	-	N/A	N/A
Lu, 2000	Leptomeningeal enhancement after carotid stenting	Other	*	*	N/A	*	-	*	N/A	N/A
McKinney, 2018	Pseudo-leptomeningeal contrast enhancement at 3T in pediatric patients sedated by propofol	Other	-	*	N/A	*	*	-	N/A	N/A
O'Connell, 2017	Neurosarcoidosis: clinical presentations and changing treatment patterns in an Irish Caucasian population	Other	*	*	N/A	*	*	-	N/A	N/A
Parsons, 2020	Rheumatoid Meningitis: Clinical Characteristics, Diagnostic Evaluation, and Treatment	Other	*	-	N/A	-	-	-	N/A	N/A
Pektezel, 2020	Pediatric headache and neuroimaging: experience of two tertiary centers	Other	*	*	N/A	-	-	-	N/A	N/A
Pickuth, 2000	Role of radiology in the diagnosis of neurosarcoidosis	Other	*	-	N/A	*	*	-	N/A	N/A
Salvarani, 2016	Imaging Findings of Cerebral Amyloid Angiopathy, Aβ-Related Angiitis (ABRA), and Cerebral Amyloid Angiopathy-Related Inflammation	Other	*	*	N/A	-	-	-	N/A	N/A
Soni, 2019	Imaging findings in spinal sarcoidosis: a report of 18 cases and review of the current literature	Other	*	*	*	N/A	*	-	N/A	N/A
Spencer, 2005	Clinical and magnetic resonance imaging manifestations of neurosarcoidosis	Other	*	*	N/A	*	-	-	N/A	N/A
Wengert, 2013	Neurosarcoidosis: Correlation of cerebrospinal fluid findings with diffuse leptomeningeal gadolinium en-hancement on MRI and clinical disease activity	Other	*	*	N/A	*	-	-	N/A	N/A
Wilkinson, 2003	Short-term changes in cerebral microhemodynamics after carotid stenting	Other	*	-	N/A	*	*	-	N/A	N/A
WilkinsonI, 2000	Unilateral leptomeningeal enhancement after carotid stent insertion detected by magnetic resonance imag-ing	Other	*	-	N/A	*	*	-	N/A	N/A
Wu, 2021	Noninvasive Characterization of Human Glymphatics and Meningeal Lymphatics in an in vivo Model of Blood–Brain Barrier Leakage	Other	*	*	*	*	-	-	N/A	N/A

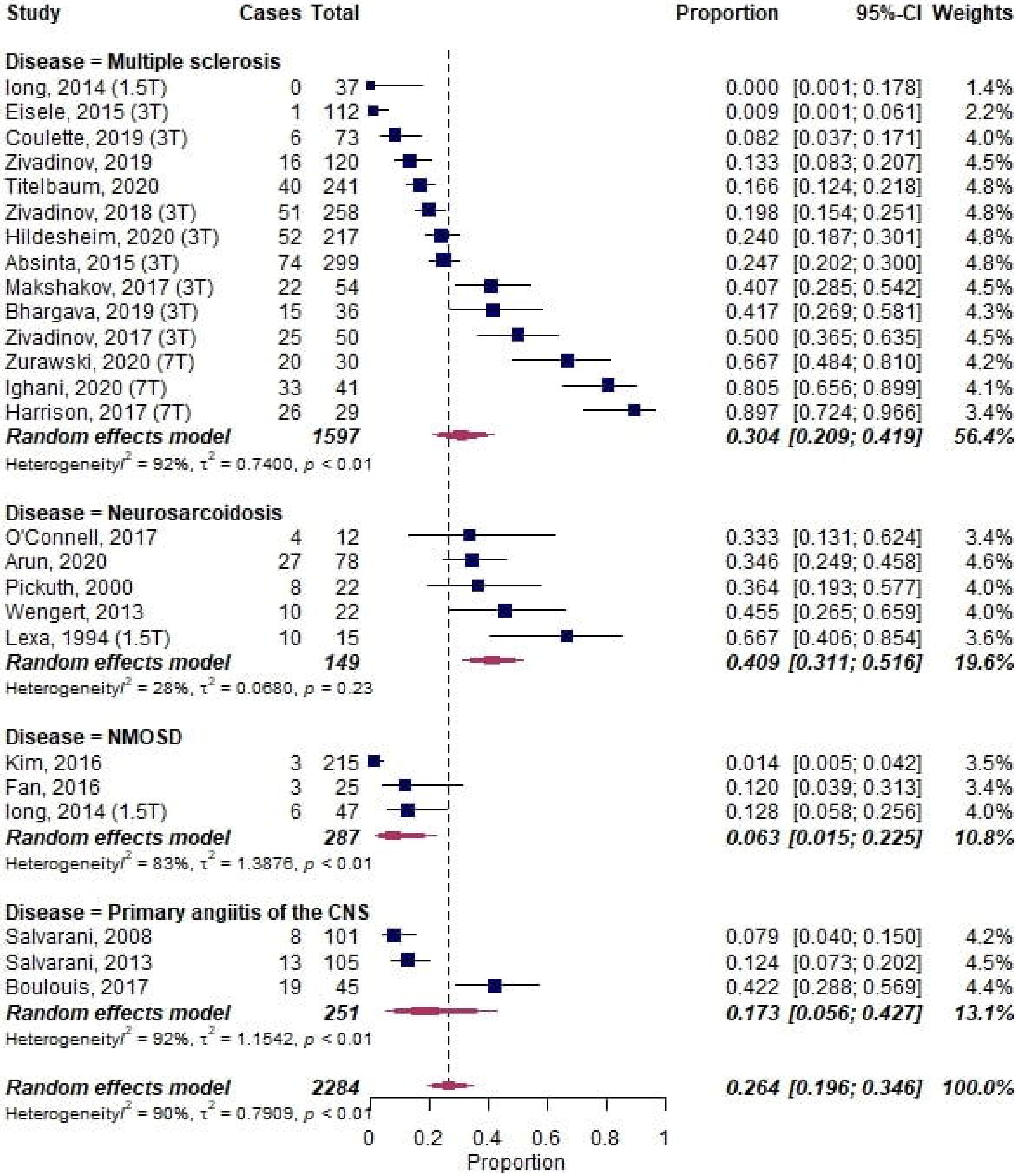
Absinta, 2015	Gadolinium-based MRI characterization of leptomeningeal inflammation in multiple sclerosis	Neuroinflammatory	*	*	*	*	*	-	N/A	N/A
Absinta, 2017	Leptomeningeal gadolinium enhancement across the spectrum of chronic neuroinflammatory diseases	Neuroinflammatory	*	*	*	*	*	-	N/A	N/A
Asgari, 2017	Disruption of the leptomeningeal blood barrier in neuromyelitis optica spectrum disorder	Neuroinflammatory	*	*	N/A	*	-	-	N/A	N/A
Bergsland, 2019	Leptomeningeal contrast enhancement is related to focal cortical thinning in relapsing-remitting multiple sclerosis: A cross-sectional MRI study	Neuroinflammatory	*	*	N/A	*	-	-	N/A	N/A
Bhargava, 2019	Trial of intrathecal rituximab in progressive multiple sclerosis patients with evidence of leptomeningeal contrast enhancement	Neuroinflammatory	*	*	N/A	*	*	-	N/A	N/A
Boulouis, 2017	Primary Angiitis of the Central Nervous System: Magnetic Resonance Imaging Spectrum of Parenchymal, Meningeal, and Vascular Lesions at Baseline	Neuroinflammatory	*	*	N/A	-	*	-	N/A	N/A
Coulette, 2019	Diagnosis and prediction of relapses in susac syndrome: A new use for MR postcontrast FLAIR leptomeningeal enhancement	Neuroinflammatory	*	*	N/A	*	*	-	N/A	N/A
Dubey, 2018	Autoimmune GFAP astrocytopathy: Prospective evaluation of 90 patients in 1 year	Neuroinflammatory	*	*	N/A	-	-	-	N/A	N/A
Eiden, 2019	High-resolution contrast-enhanced vessel wall imaging in patients with suspected cerebral vasculitis: Prospective comparison of wholebrain 3D T1 SPACE versus 2D T1 black blood MRI at 3 Tesla	Neuroinflammatory	*	*	*	N/A	*	-	N/A	N/A
Eisele, 2015	Investigation of leptomeningeal enhancement in MS: A postcontrast FLAIR MRI study	Neuroinflammatory	*	*	N/A	*	-	*	N/A	N/A
Fan, 2016	Dynamic change in magnetic resonance imaging of patients with neuromyelitis optica	Neuroinflammatory	*	*	N/A	-	-	-	N/A	N/A
Harrison, 2017	Leptomeningeal Enhancement at 7T in Multiple Sclerosis: Frequency, Morphology, and Relationship to Cortical Volume	Neuroinflammatory	*	*	*	*	-	*	N/A	N/A
Hildesheim, 2020	Leptomeningeal, dura mater and meningeal vessel wall enhancements in multiple sclerosis	Neuroinflammatory	*	*	N/A	*	-	*	N/A	N/A
Ighani, 2020	No association between cortical lesions and leptomeningeal enhancement on 7-Tesla MRI in multiple sclerosis	Neuroinflammatory	*	*	*	*	*	*	N/A	N/A
Kim, 2016	Cerebral cortex involvement in neuromyelitis optica spectrum disorder	Neuroinflammatory	*	*	N/A	-	-	-	N/A	N/A
Kunchok, 2020	MOG-IgG1 and co-existence of neuronal autoantibodies	Neuroinflammatory	*	*	N/A	-	-	-	N/A	N/A
long, 2014	Brain gadolinium enhancement along the ventricular and leptomeningeal regions in patients with aquaporin-4 antibodies in cerebral spinal fluid	Neuroinflammatory	*	*	N/A	-	-	-	N/A	N/A
Makshakov, 2017	Leptomeningeal Contrast Enhancement Is Associated with Disability Progression and Grey Matter Atrophy in Multiple Sclerosis	Neuroinflammatory	*	*	N/A	*	-	-	N/A	N/A
Neo, 2020	Acute radiological features facilitate diagnosis and prognosis of anti-N-methyl-d-aspartate receptor (NMDAR)	Neuroinflammatory	*	*	N/A	*	N/A	-	N/A	N/A

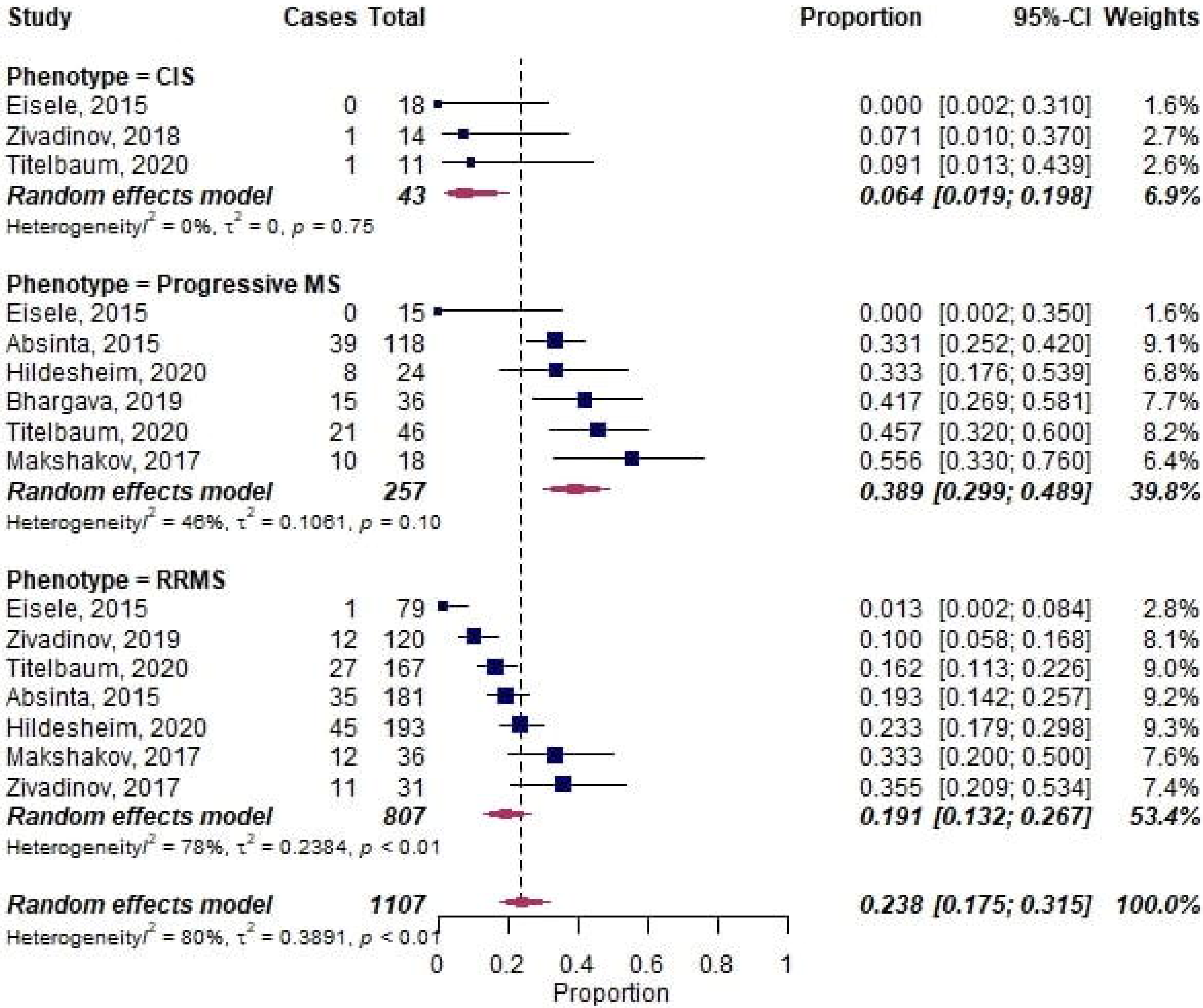
	and anti-voltage-gated potassium channel (VGKC) encephalitis in adults									
Pino-Lopez, 2019	Contrast-enhanced fat-suppressed FLAIR for the characterization of leptomeningeal inflammation in optic neuritis	Neuroinflammatory	*	*	N/A	*	-	-	N/A	N/A
Salvarani, 2008	Primary central nervous system vasculitis with prominent leptomeningeal enhancement: a subset with a benign outcome	Neuroinflammatory	*	*	N/A	-	-	-	N/A	N/A
Salvarani, 2013	Aβ-related angiitis: comparison with CAA without inflammation and primary CNS vasculitis	Neuroinflammatory	*	*	N/A	-	-	-	N/A	N/A
Susac, 2003	MRI findings in Susac's syndrome	Neuroinflammatory	-	-	N/A	*	-	-	N/A	N/A
Titelbaum, 2020	Leptomeningeal Enhancement on 3D-FLAIR MRI in Multiple Sclerosis: Systematic Observations in Clinical Practice	Neuroinflammatory	*	*	*	*	*	*	N/A	N/A
Xia, 2017	Assessment of early evidence of multiple sclerosis in a prospective study of asymptomatic high-risk family members	Neuroinflammatory	*	*	N/A	*	N/A	N/A	N/A	N/A
Zivadinov, 2017	Leptomeningeal contrast enhancement is associated with progression of cortical atrophy in MS: A retrospective, pilot, observational longitudinal study	Neuroinflammatory	*	*	N/A	*	-	-	N/A	N/A
Zivadinov, 2018	Evaluation of leptomeningeal contrast enhancement using pre-and postcontrast subtraction 3D-FLAIR imaging in multiple sclerosis	Neuroinflammatory	*	*	N/A	*	*	-	N/A	N/A
Zivadinov, 2019	Effect of teriflunomide and dimethyl fumarate on cortical atrophy and leptomeningeal inflammation in multiple sclerosis: A retrospective, observational, case-control pilot study	Neuroinflammatory	*	*	N/A	*	*	*	N/A	N/A
Zurawski, 2020	7T MRI cerebral leptomeningeal enhancement is common in relapsing-remitting multiple sclerosis and is associated with cortical and thalamic lesions	Neuroinflammatory	*	*	*	*	*	*	*	*

Table S3: Risk of bias assessment of the included animal studies according to the risk of bias assessment checklist for good research practice and whether a study was in accordance with the ARRIVE guidlines. A, in accordance with ARRIVE guidelines; B, blinding; C, conflict of interest; R, randomization; S, prior sample size calculation; W, animal welfare statement.

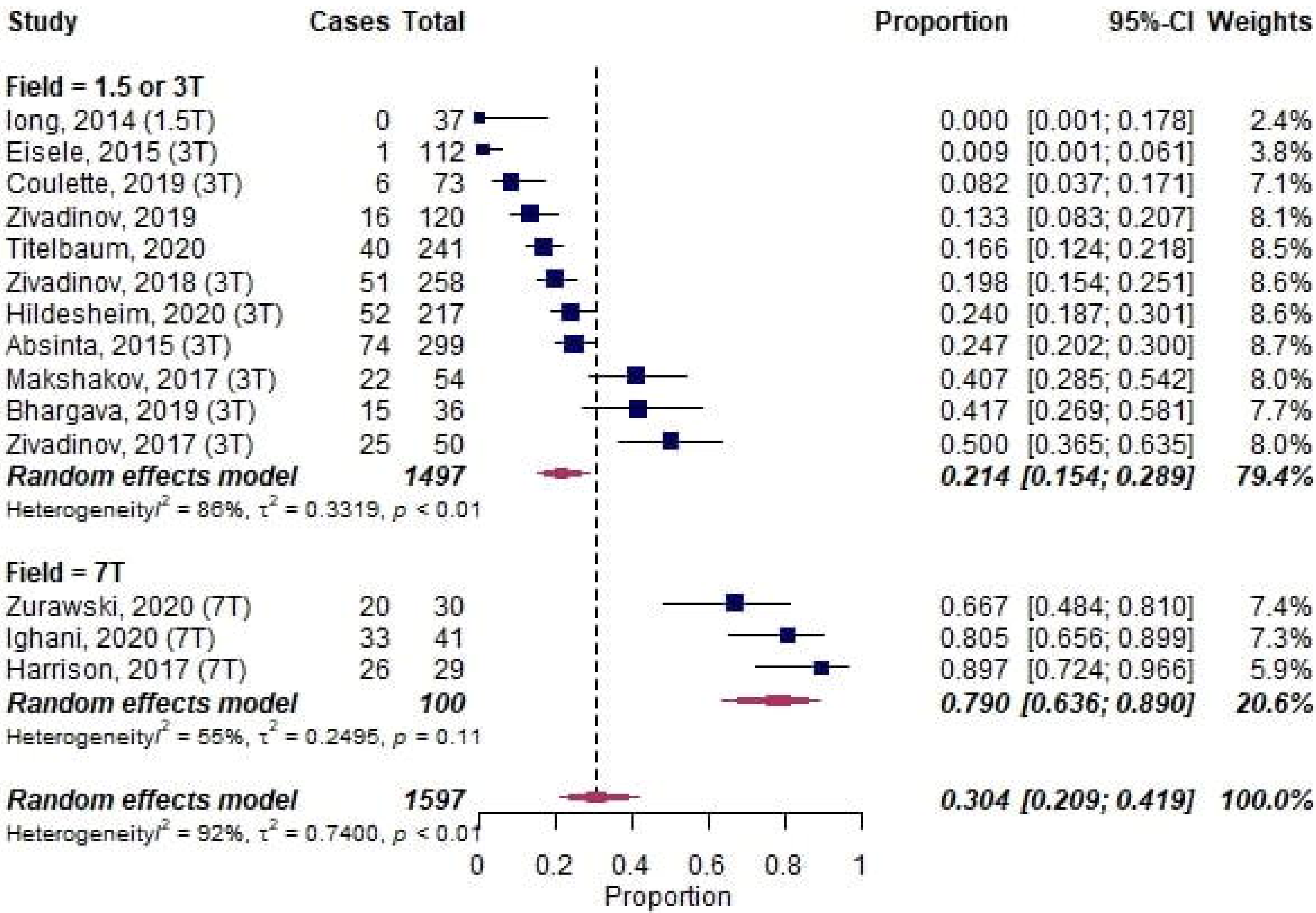
Author, year	Title	Type	R	B	S	W	C	A
Pol, 2019	Characterization of leptomeningeal inflammation in rodent experimental autoimmune encephalomyelitis (EAE) model of multiple sclerosis	Neuroinflammatory	-	-	-	*	*	*
Wiesmann, 2002	Experimental bacterial meningitis in rats: Demonstration of hydrocephalus and meningeal enhancement by mag-netic resonance imaging	Infectious	*	*	-	-	-	*
Young, 2009	Magnetic resonance imaging characteristics of necrotizing meningoencephalitis in pug dogs	Other	-	-	-	-	*	*


A
B
C
D

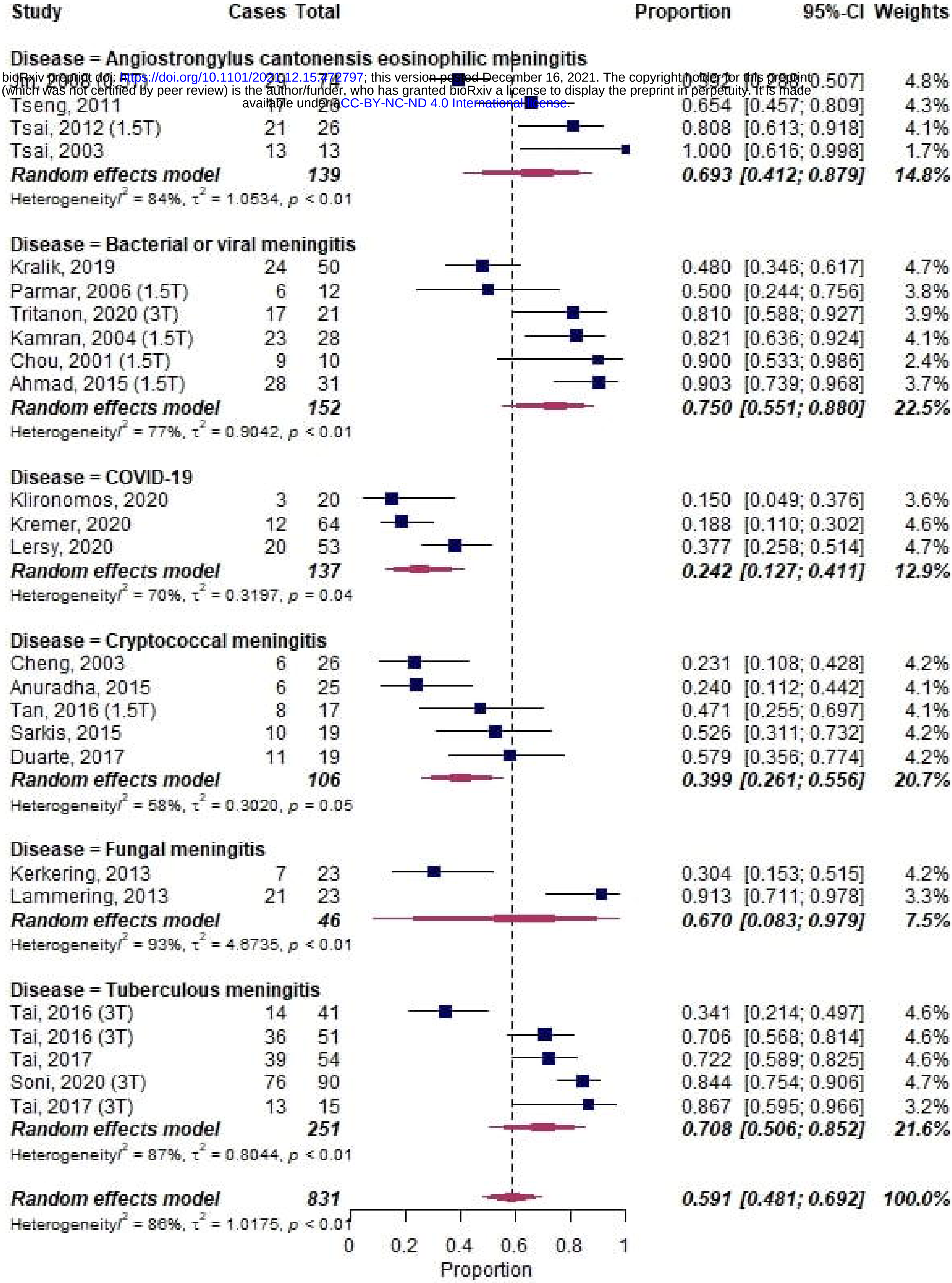

Study Cases Total Proportion 95%-CI Weights
Disease = Multiple sclerosis
Iong, 2014 (1.5T) 0 37 0.000 [0.001; 0.178] 1.4%
Eisele, 2015 (3T) 1 112 0.009 [0.001; 0.061] 2.2%
Coulette, 2019 (3T) 6 73 0.082 [0.037; 0.171] 4.0%
Zivadinov, 2019 16 120 0.133 [0.083; 0.207] 4.5%
Titelbaum, 2020 40 241 0.166 [0.124; 0.218] 4.8%
Zivadinov, 2018 (3T) 51 258 0.198 [0.154; 0.251] 4.8%
Hildesheim, 2020 (3T) 52 217 0.240 [0.187; 0.301] 4.8%
Absinta, 2015 (3T) 74 299 0.247 [0.202; 0.300] 4.8%
Makshakov, 2017 (3T) 22 54 0.407 [0.285; 0.542] 4.5%
Bhargava, 2019 (3T) 15 36 0.417 [0.269; 0.581] 4.3%
Zivadinov, 2017 (3T) 25 50 0.500 [0.365; 0.635] 4.5%
Zurawski, 2020 (7T) 20 30 0.667 [0.484; 0.810] 4.2%
Ighani, 2020 (7T) 33 41 0.805 [0.656; 0.899] 4.1%
Harrison, 2017 (7T) 26 29 0.897 [0.724; 0.966] 3.4%
Random effects model 1597 0.304 [0.209; 0.419] 56.4%
Heterogeneity: I^2 = 92%, τ^2 = 0.7400, p < 0.01
Disease = Neurosarcoidosis
O'Connell, 2017 4 12 0.333 [0.131; 0.624] 3.4%
Arun, 2020 27 78 0.346 [0.249; 0.458] 4.6%
Pickuth, 2000 8 22 0.364 [0.193; 0.577] 4.0%
Wengert, 2013 10 22 0.455 [0.265; 0.659] 4.0%
Lexa, 1994 (1.5T) 10 15 0.667 [0.406; 0.854] 3.6%
Random effects model 149 0.409 [0.311; 0.516] 19.6%
Heterogeneity: I^2 = 28%, τ^2 = 0.0680, p = 0.23
Disease = NMOSD
Kim, 2016 3 215 0.014 [0.005; 0.042] 3.5%
Fan, 2016 3 25 0.120 [0.039; 0.313] 3.4%
Iong, 2014 (1.5T) 6 47 0.128 [0.058; 0.256] 4.0%
Random effects model 287 0.063 [0.015; 0.225] 10.8%
Heterogeneity: I^2 = 83%, τ^2 = 1.3876, p < 0.01
Disease = Primary angiitis of the CNS
Salvarani, 2008 8 101 0.079 [0.040; 0.150] 4.2%
Salvarani, 2013 13 105 0.124 [0.073; 0.202] 4.5%
Boulouis, 2017 19 45 0.422 [0.288; 0.569] 4.4%
Random effects model 251 0.173 [0.056; 0.427] 13.1%
Heterogeneity: I^2 = 92%, τ^2 = 1.1542, p < 0.01
Random effects model 2284 0.264 [0.196; 0.346] 100.0%
Heterogeneity: I^2 = 90%, τ^2 = 0.7909, p < 0.01
0 0.2 0.4 0.6 0.8 1
Proportion

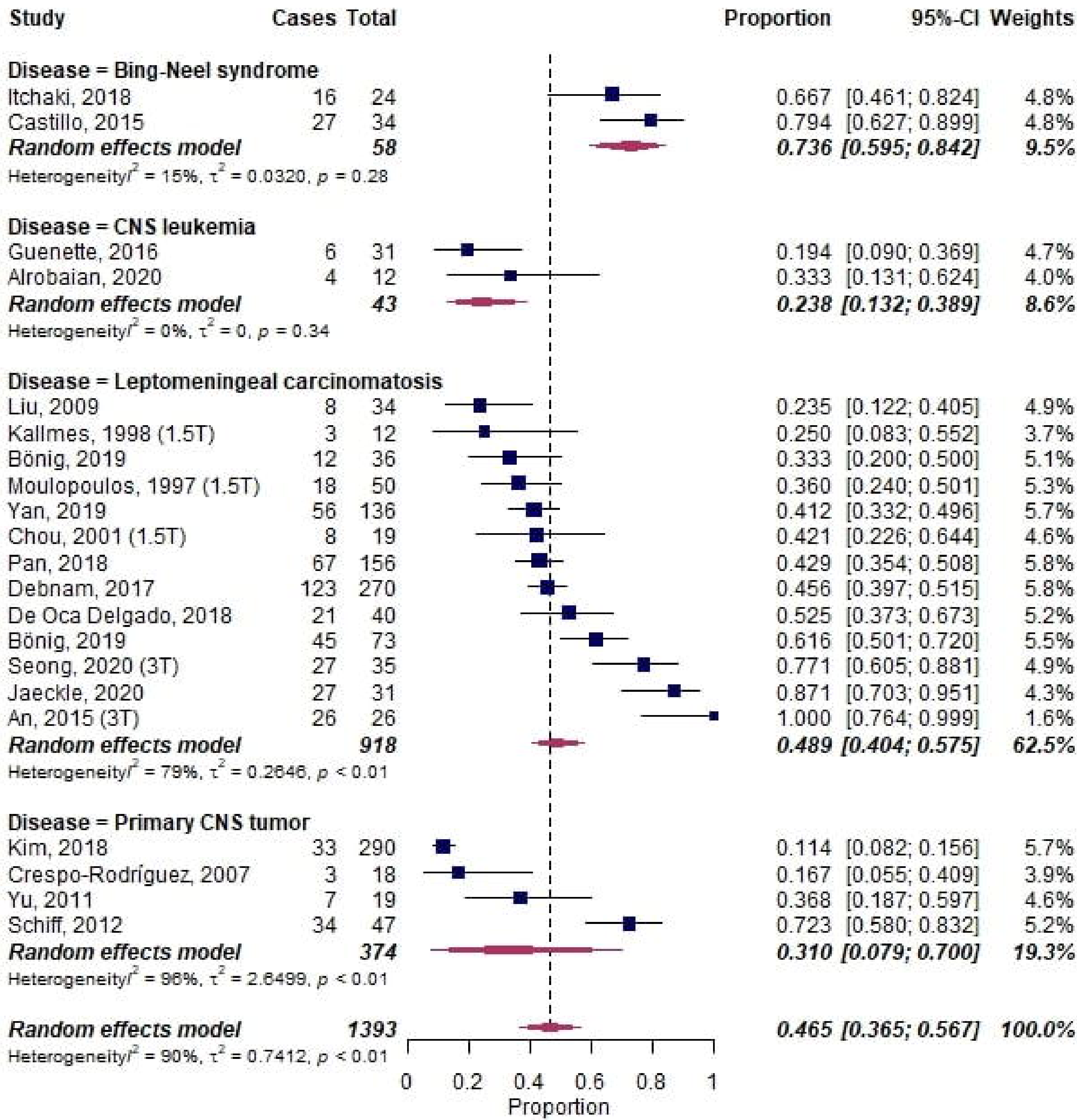


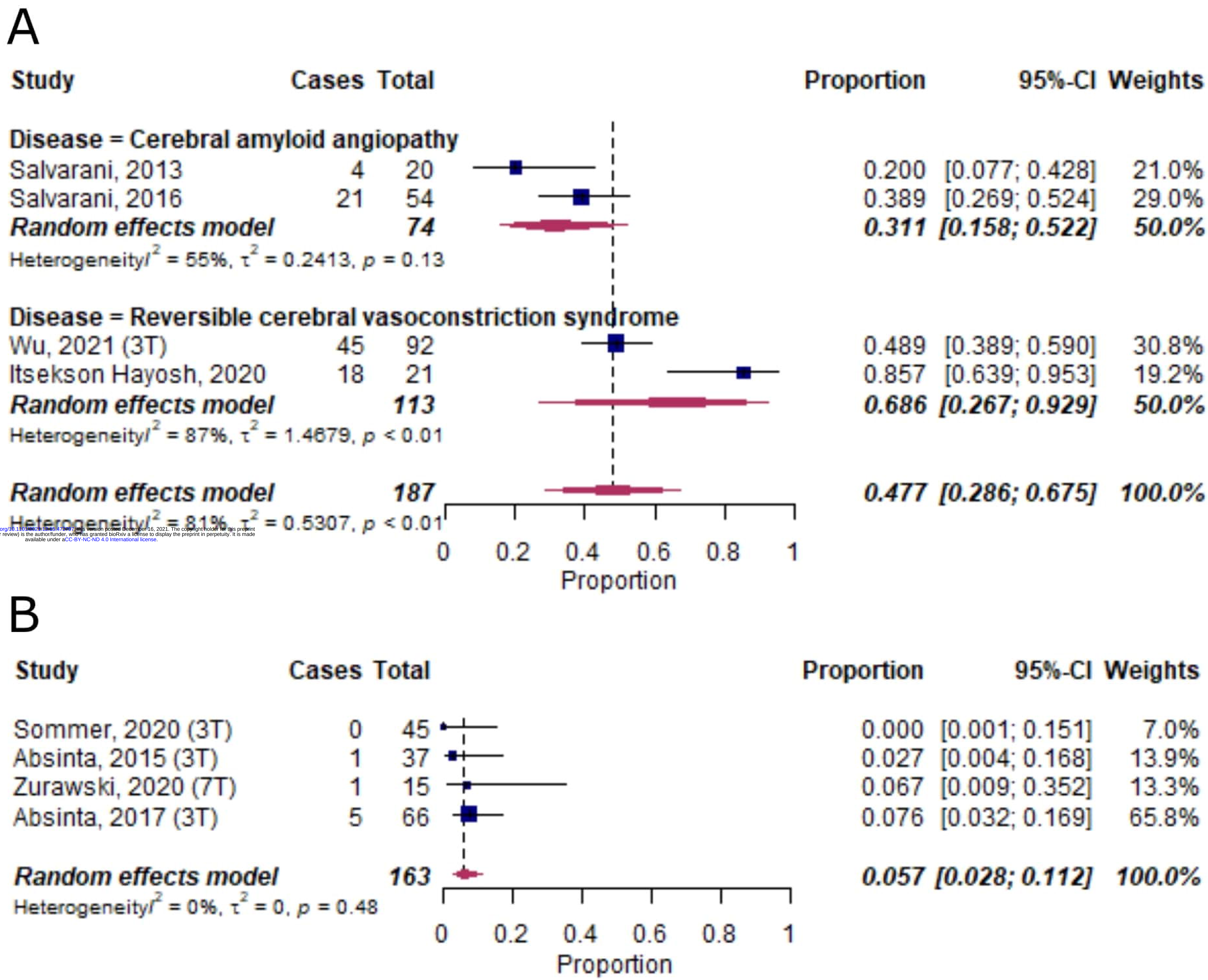
Study Cases Total Proportion 95%-CI Weights
Phenotype = CIS
Eisele, 2015 0 18 0.000 [0.002; 0.310] 1.6%
Zivadinov, 2018 1 14 0.071 [0.010; 0.370] 2.7%
Titelbaum, 2020 1 11 0.091 [0.013; 0.439] 2.6%
Random effects model 43 0.064 [0.019; 0.198] 6.9%
Heterogeneity: I^2 = 0%, τ^2 = 0, p = 0.75
Phenotype = Progressive MS
Eisele, 2015 0 15 0.000 [0.002; 0.350] 1.6%
Absinta, 2015 39 118 0.331 [0.252; 0.420] 9.1%
Hildesheim, 2020 8 24 0.333 [0.176; 0.539] 6.8%
Bhargava, 2019 15 36 0.417 [0.269; 0.581] 7.7%
Titelbaum, 2020 21 46 0.457 [0.320; 0.600] 8.2%
Makshakov, 2017 10 18 0.556 [0.330; 0.760] 6.4%
Random effects model 257 0.389 [0.299; 0.489] 39.8%
Heterogeneity: I^2 = 46%, τ^2 = 0.1061, p = 0.10
Phenotype = RRMS
Eisele, 2015 1 79 0.013 [0.002; 0.084] 2.8%
Zivadinov, 2019 12 120 0.100 [0.058; 0.168] 8.1%
Titelbaum, 2020 27 167 0.162 [0.113; 0.226] 9.0%
Absinta, 2015 35 181 0.193 [0.142; 0.257] 9.2%
Hildesheim, 2020 45 193 0.233 [0.179; 0.298] 9.3%
Makshakov, 2017 12 36 0.333 [0.200; 0.500] 7.6%
Zivadinov, 2017 11 31 0.355 [0.209; 0.534] 7.4%
Random effects model 807 0.191 [0.132; 0.267] 53.4%
Heterogeneity: I^2 = 78%, τ^2 = 0.2384, p < 0.01
Random effects model 1107 0.238 [0.175; 0.315] 100.0%
Heterogeneity: I^2 = 80%, τ^2 = 0.3891, p < 0.01
0 0.2 0.4 0.6 0.8 1
Proportion


Study Cases Total Proportion 95%-CI Weights
Field = 1.5 or 3T
Iong, 2014 (1.5T) 0 37 0.000 [0.001; 0.178] 2.4%
Eisele, 2015 (3T) 1 112 0.009 [0.001; 0.061] 3.8%
Coulette, 2019 (3T) 6 73 0.082 [0.037; 0.171] 7.1%
Zivadinov, 2019 16 120 0.133 [0.083; 0.207] 8.1%
Titelbaum, 2020 40 241 0.166 [0.124; 0.218] 8.5%
Zivadinov, 2018 (3T) 51 258 0.198 [0.154; 0.251] 8.6%
Hildesheim, 2020 (3T) 52 217 0.240 [0.187; 0.301] 8.6%
Absinta, 2015 (3T) 74 299 0.247 [0.202; 0.300] 8.7%
Makshakov, 2017 (3T) 22 54 0.407 [0.285; 0.542] 8.0%
Bhargava, 2019 (3T) 15 36 0.417 [0.269; 0.581] 7.7%
Zivadinov, 2017 (3T) 25 50 0.500 [0.365; 0.635] 8.0%
Random effects model 1497 0.214 [0.154; 0.289] 79.4%
Heterogeneity: I^2 = 86%, τ^2 = 0.3319, p < 0.01
Field = 7T
Zurawski, 2020 (7T) 20 30 0.667 [0.484; 0.810] 7.4%
Ighani, 2020 (7T) 33 41 0.805 [0.656; 0.899] 7.3%
Harrison, 2017 (7T) 26 29 0.897 [0.724; 0.966] 5.9%
Random effects model 100 0.790 [0.636; 0.890] 20.6%
Heterogeneity: I^2 = 55%, τ^2 = 0.2495, p = 0.11
Random effects model 1597 0.304 [0.209; 0.419] 100.0%
Heterogeneity: I^2 = 92%, τ^2 = 0.7400, p < 0.01
0 0.2 0.4 0.6 0.8 1
Proportion

A - Disease duration
Study n LME+ n LME- Mean difference 95%-CI Weights
Zivadinov, 2018 33 179 0.200 [-3.185; 3.585] 25.7%
Hildesheim, 2020 53 164 1.100 [-1.979; 4.179] 29.5%
Titelbaum, 2020 40 201 2.600 [-0.745; 5.945] 26.2%
Zivadinov, 2017 11 16 4.900 [-2.033; 11.833] 7.6%
Zurawski, 2020 20 10 6.800 [1.137; 12.463] 11.0%
Random effects model 157 570 2.175 [0.189; 4.161] 100.0%
Heterogeneity: I² = 19%, τ² = 1.0085, p = 0.29
-5 0 5 10 15
Mean difference MS duration (years)
B - EDSS
Study n LME+ n LME- Mean difference 95%-CI Weights
Absinta, 2015 74 226 0.330 [-0.525; 1.185] 23.8%
Zurawski, 2020 20 10 0.500 [-0.362; 1.362] 23.4%
Zivadinov, 2018 33 179 0.660 [-0.007; 1.327] 39.1%
Makshakov, 2017 22 32 1.000 [-0.130; 2.130] 13.6%
Random effects model 149 447 0.590 [0.173; 1.008] 100.0%
Heterogeneity: I² = 0%, τ² = 0, p = 0.81
-0.5 0 0.5 1 1.5
Mean difference EDSS
C - T2 lesion volume
Study n LME+ n LME- Mean difference 95%-CI Weights
Zivadinov, 2017 11 16 -1.800 [-25.393; 21.793] 1.4%
Hildesheim, 2020 53 164 3.700 [-0.894; 8.294] 36.1%
Zurawski, 2020 20 10 6.440 [0.700; 12.180] 23.2%
Zivadinov, 2018 33 179 7.760 [3.355; 12.165] 39.3%
Random effects model 117 369 5.856 [3.094; 8.618] 100.0%
Heterogeneity: I² = 0%, τ² = 0, p = 0.57
-30 -20 -10 0 10 20
Mean difference T2 lesion volume (mL)
D - T1 lesion volume
Study n LME+ n LME- Mean difference 95%-CI Weights
Zivadinov, 2017 11 16 -0.900 [-4.353; 2.553] 14.8%
Hildesheim, 2020 53 164 1.200 [-0.036; 2.436] 52.0%
Zivadinov, 2018 33 179 3.010 [0.107; 5.913] 19.5%
Zurawski, 2020 20 10 3.410 [-0.215; 7.035] 13.7%
Random effects model 117 369 1.544 [0.076; 3.012] 100.0%
Heterogeneity: I² = 28%, τ² = 0.6816, p = 0.24
-4 -2 0 2 4 6 8
Mean difference T1 lesion volume (mL)
E - Cortical volume
Study n LME+ n LME- Mean difference 95%-CI Weights
Hildesheim, 2020 53 164 -23.100 [-39.623; -6.577] 65.7%
Zivadinov, 2017 11 16 -20.800 [-60.582; 18.982] 11.3%
Makshakov, 2017 22 32 -16.300 [-44.251; 11.651] 23.0%
Random effects model 86 212 -21.278 [-34.671; -7.885] 100.0%
Heterogeneity: I² = 0%, τ² = 0, p = 0.92
-60 -40 -20 0 20
Mean difference cortical volume (mL)


Study Cases Total Proportion 95%-CI Weights
Disease = Angiostrongylus cantonensis eosinophilic meningitis
Jin, 2008 74 0.392 [0.288; 0.507] 4.8%
Tseng, 2011 17 26 0.654 [0.457; 0.809] 4.3%
Tsai, 2012 (1.5T) 21 26 0.808 [0.613; 0.918] 4.1%
Tsai, 2003 13 13 1.000 [0.616; 0.998] 1.7%
Random effects model 139 0.693 [0.412; 0.879] 14.8%
Heterogeneity: I² = 84%, τ² = 1.0534, p < 0.01
Disease = Bacterial or viral meningitis
Kralik, 2019 24 50 0.480 [0.346; 0.617] 4.7%
Parmar, 2006 (1.5T) 6 12 0.500 [0.244; 0.756] 3.8%
Tritanon, 2020 (3T) 17 21 0.810 [0.588; 0.927] 3.9%
Kamran, 2004 (1.5T) 23 28 0.821 [0.636; 0.924] 4.1%
Chou, 2001 (1.5T) 9 10 0.900 [0.533; 0.986] 2.4%
Ahmad, 2015 (1.5T) 28 31 0.903 [0.739; 0.968] 3.7%
Random effects model 152 0.750 [0.551; 0.880] 22.5%
Heterogeneity: I² = 77%, τ² = 0.9042, p < 0.01
Disease = COVID-19
Klironomos, 2020 3 20 0.150 [0.049; 0.376] 3.6%
Kremer, 2020 12 64 0.188 [0.110; 0.302] 4.6%
Lersy, 2020 20 53 0.377 [0.258; 0.514] 4.7%
Random effects model 137 0.242 [0.127; 0.411] 12.9%
Heterogeneity: I² = 70%, τ² = 0.3197, p = 0.04
Disease = Cryptococcal meningitis
Cheng, 2003 6 26 0.231 [0.108; 0.428] 4.2%
Anuradha, 2015 6 25 0.240 [0.112; 0.442] 4.1%
Tan, 2016 (1.5T) 8 17 0.471 [0.255; 0.697] 4.1%
Sarkis, 2015 10 19 0.526 [0.311; 0.732] 4.2%
Duarte, 2017 11 19 0.579 [0.356; 0.774] 4.2%
Random effects model 106 0.399 [0.261; 0.556] 20.7%
Heterogeneity: I² = 58%, τ² = 0.3020, p = 0.05
Disease = Fungal meningitis
Kerkering, 2013 7 23 0.304 [0.153; 0.515] 4.2%
Lammering, 2013 21 23 0.913 [0.711; 0.978] 3.3%
Random effects model 46 0.670 [0.083; 0.979] 7.5%
Heterogeneity: I² = 93%, τ² = 4.8735, p < 0.01
Disease = Tuberculous meningitis
Tai, 2016 (3T) 14 41 0.341 [0.214; 0.497] 4.6%
Tai, 2016 (3T) 36 51 0.706 [0.568; 0.814] 4.6%
Tai, 2017 39 54 0.722 [0.589; 0.825] 4.6%
Soni, 2020 (3T) 76 90 0.844 [0.754; 0.906] 4.7%
Tai, 2017 (3T) 13 15 0.867 [0.595; 0.966] 3.2%
Random effects model 251 0.708 [0.506; 0.852] 21.6%
Heterogeneity: I² = 87%, τ² = 0.8044, p < 0.01
Random effects model 831 0.591 [0.481; 0.692] 100.0%
Heterogeneity: I² = 86%, τ² = 1.0175, p < 0.01
0 0.2 0.4 0.6 0.8 1
Proportion


Study Cases Total Proportion 95%-CI Weights
Disease = Bing-Neel syndrome
Itchaki, 2018 16 24 0.667 [0.461; 0.824] 4.8%
Castillo, 2015 27 34 0.794 [0.627; 0.899] 4.8%
Random effects model 58 0.736 [0.595; 0.842] 9.5%
Heterogeneity: I^2 = 15%, τ^2 = 0.0320, p = 0.28
Disease = CNS leukemia
Guenette, 2016 6 31 0.194 [0.090; 0.369] 4.7%
Alrobaian, 2020 4 12 0.333 [0.131; 0.624] 4.0%
Random effects model 43 0.238 [0.132; 0.389] 8.6%
Heterogeneity: I^2 = 0%, τ^2 = 0, p = 0.34
Disease = Leptomeningeal carcinomatosis
Liu, 2009 8 34 0.235 [0.122; 0.405] 4.9%
Kallmes, 1998 (1.5T) 3 12 0.250 [0.083; 0.552] 3.7%
Bönig, 2019 12 36 0.333 [0.200; 0.500] 5.1%
Moulopoulos, 1997 (1.5T) 18 50 0.360 [0.240; 0.501] 5.3%
Yan, 2019 56 136 0.412 [0.332; 0.496] 5.7%
Chou, 2001 (1.5T) 8 19 0.421 [0.226; 0.644] 4.6%
Pan, 2018 67 156 0.429 [0.354; 0.508] 5.8%
Debnam, 2017 123 270 0.456 [0.397; 0.515] 5.8%
De Oca Delgado, 2018 21 40 0.525 [0.373; 0.673] 5.2%
Bönig, 2019 45 73 0.616 [0.501; 0.720] 5.5%
Seong, 2020 (3T) 27 35 0.771 [0.605; 0.881] 4.9%
Jaeckle, 2020 27 31 0.871 [0.703; 0.951] 4.3%
An, 2015 (3T) 26 26 1.000 [0.764; 0.999] 1.6%
Random effects model 918 0.489 [0.404; 0.575] 62.5%
Heterogeneity: I^2 = 79%, τ^2 = 0.2646, p < 0.01
Disease = Primary CNS tumor
Kim, 2018 33 290 0.114 [0.082; 0.156] 5.7%
Crespo-Rodríguez, 2007 3 18 0.167 [0.055; 0.409] 3.9%
Yu, 2011 7 19 0.368 [0.187; 0.597] 4.6%
Schiff, 2012 34 47 0.723 [0.580; 0.832] 5.2%
Random effects model 374 0.310 [0.079; 0.700] 19.3%
Heterogeneity: I^2 = 96%, τ^2 = 2.6499, p < 0.01
Random effects model 1393 0.465 [0.365; 0.567] 100.0%
Heterogeneity: I^2 = 90%, τ^2 = 0.7412, p < 0.01
0 0.2 0.4 0.6 0.8 1
Proportion


A
Study
Cases
Total
Proportion
95%-CI
Weights
Disease = Cerebral amyloid angiopathy
Salvarani, 2013 4 20 0.200 [0.077; 0.428] 21.0%
Salvarani, 2016 21 54 0.389 [0.269; 0.524] 29.0%
Random effects model 74 0.311 [0.158; 0.522] 50.0%
Heterogeneity: I² = 55%, τ² = 0.2413, p = 0.13
Disease = Reversible cerebral vasoconstriction syndrome
Wu, 2021 (3T) 45 92 0.489 [0.389; 0.590] 30.8%
Itsekson Hayosh, 2020 18 21 0.857 [0.639; 0.953] 19.2%
Random effects model 113 0.686 [0.267; 0.929] 50.0%
Heterogeneity: I² = 87%, τ² = 1.4679, p < 0.01
Random effects model 187 0.477 [0.286; 0.675] 100.0%
Heterogeneity: I² = 81%, τ² = 0.5307, p < 0.01
0
0.2
0.4
0.6
0.8
1
Proportion
B
Study
Cases
Total
Proportion
95%-CI
Weights
Sommer, 2020 (3T) 0 45 0.000 [0.001; 0.151] 7.0%
Absinta, 2015 (3T) 1 37 0.027 [0.004; 0.168] 13.9%
Zurawski, 2020 (7T) 1 15 0.067 [0.009; 0.352] 13.3%
Absinta, 2017 (3T) 5 66 0.076 [0.032; 0.169] 65.8%
Random effects model 163 0.057 [0.028; 0.112] 100.0%
Heterogeneity: I² = 0%, τ² = 0, p = 0.48
0
0.2
0.4
0.6
0.8
1
Proportion