VETERINARY EVIDENCE

Veterinary Evidence (2026) Vol 11 Iss 4 | Page 1 of 10
DOI: https://doi.org/10.18849/ve.v11i4.759

KNOWLEDGE SUMMARY

Keywords: CEREBROSPINAL FLUID (CSF); HISTOPATHOLOGY; INFLAMMATORY CENTRAL NERVOUS SYSTEM (CNS); MENINGOENCEPHALOMYELITIS OF UNKNOWN ORIGIN (MUO); PLEOCYTOSIS; TOTAL NUCLEATED CELL COUNT (TNCC)

Diagnostic accuracy of cerebrospinal fluid pleocytosis in suspected meningoencephalomyelitis of unknown origin in canines

Zachary Horowitz, BS[1*]
[1] The Royal Veterinary College, London, United Kingdom, NW1 0TU
[*]Corresponding author email: zhorowitz23@rvc.ac.uk

Submitted: 22 January 2026; published: 16 November 2026

PICO question

In canines with suspected meningoencephalomyelitis of unknown origin (MUO) [population], does cerebrospinal fluid analysis for pleocytosis [intervention], compared with histopathology [comparison], accurately identify underlying noninfectious inflammatory central nervous system (CNS) disease [outcome]?

Clinical bottom line

Category of research	Diagnosis.
Number and type of study designs reviewed	Three retrospective case series and one systematic review.
Strength of evidence	Moderate.
Outcomes reported	The four studies included collectively reported the presence or absence of cerebrospinal fluid (CSF) pleocytosis in dogs with histopathologically confirmed noninfectious inflammatory CNS disease or clinically diagnosed MUO where applicable. Pleocytosis is defined as a total nucleated cell count (TNCC) above the laboratory reference interval. The first study reported pleocytosis in all 22 dogs evaluated with granulomatous meningoencephalomyelitis (GME). In contrast, the second paper identified normal TNCC in a substantial proportion of GME cases, a finding mirrored by the third study across a significant number of GME, necrotising meningoencephalitis (NME), and clinically diagnosed MUO patients. Finally, the fourth study documented normal repeat CSF TNCC levels in a single dog with progressive NME within a small sample size.
Conclusion	Cerebrospinal fluid pleocytosis supports a diagnosis of noninfectious inflammatory CNS disease in dogs with suspected MUO but does not exclude disease when absent. Furthermore, CSF findings cannot reliably distinguish between inflammatory disease subtypes. Therefore, CSF analysis should be interpreted alongside neurological examination, magnetic resonance imaging (MRI) findings, and exclusion of infectious diseases rather than used as a standalone diagnostic test.

This Open Access work is distributed under a Creative Commons Attributions 4.0 International License. You are free to share (copy and redistribute the material in any medium or format) or adapt (remix, transform, and build upon the material for any purpose, even commercially), provided you fully cite this original work.

Veterinary Evidence is an online, open access, peer-reviewed journal owned and published by RCVS Knowledge.

How to apply this evidence in practice

The application of evidence into practice should take into account multiple factors, not limited to: individual clinical expertise, patient's circumstances and owners' values, country, location or clinic where you work, the individual case in front of you, the availability of therapies and resources.

Knowledge Summaries are a resource to help reinforce or inform decision-making. They do not override the responsibility or judgement of the practitioner to do what is best for the animal in their care.

Clinical scenario

A 6-year-old Maltese presents with acute onset seizures. Neurological examination localises the lesion to the forebrain, and MRI reveals multifocal, poorly demarcated T2-weighted hyper-intense lesions consistent with inflammatory disease. The clinician suspects meningoencephalomyelitis of unknown origin (MUO) and collects cerebrospinal fluid (CSF) for analysis. The sample reveals mild mononuclear pleocytosis (8 cells/µL) and elevated protein. The clinician wonders whether the presence or absence of pleocytosis can reliably support or exclude noninfectious inflammatory central nervous system (CNS) disease before initiating immunosuppressive therapy.

The evidence

Three retrospective case series (Bailey & Higgins, 1986; Demierre et al., 2001; Stalis et al., 1995) and one systematic review (Granger et al., 2010) provide evidence relevant to the PICO. None were designed as formal diagnostic accuracy studies, and all are limited by their retrospective design.

Granger et al. (2010) provides the strongest evidence by reviewing 56 publications describing dogs with noninfectious inflammatory CNS disease. The review found that normal CSF total nucleated cell count (TNCC) occurred in a clinically important minority of dogs with granulomatous meningoencephalomyelitis (GME), necrotising meningoencephalitis (NME), and clinically diagnosed MUO, demonstrating that normal CSF TNCC may occur in inflammatory CNS disease. However, the retrospective nature of the data, methodological heterogeneity, and lack of formal diagnostic accuracy analyses prevent reliable estimation of sensitivity and specificity.

The three histopathologically confirmed case series (Bailey & Higgins, 1986; Demierre et al., 2001; Stalis et al., 1995) provide supporting evidence that CSF pleocytosis is common but not universal in inflammatory CNS disease, but are limited by small sample sizes, retrospective methodology, and variation in CSF collection methods.

Taken together, the available evidence provides moderate evidence that CSF pleocytosis supports, but does not confirm or exclude, noninfectious inflammatory CNS disease. Consequently, CSF analysis should not be used as a standalone diagnostic test.

Summary of the evidence

Bailey & Higgins (1986)

Characteristics of cerebrospinal fluid associated with canine granulomatous meningoencephalomyelitis: A retrospective study

Aim: To retrospectively characterise the cerebrospinal fluid (CSF) abnormalities in dogs with histopathologically confirmed granulomatous meningoencephalomyelitis (GME).

Study design	Retrospective case series.
Intervention details	Target Population: Dogs with histopathologically confirmed GME that underwent CSF analysis.

	Sample: 22 dogs identified from the medical records of the University of California, Davis Veterinary Medical Teaching Hospital (United States) between 1970 and 1984. Inclusion criteria were histopathological confirmation of GME and CSF analysis performed at the institution. 17 dogs underwent cisternal CSF collection, 4 underwent lumbar CSF collection, and one underwent both.
Outcome studied	CSF total nucleated cell count (TNCC), protein concentration, differential cell count, and CSF pressure in dogs with histopathologically confirmed GME.
Main findings (relevant to PICO question)	• Normal CSF TNCC (0–5 cells/µL) was not reported in any dogs with histopathologically confirmed GME (0/22, 0%). • CSF TNCC ranged from 9–5,400 cells/µL for cisternal samples and 12–1,600 cells/µL for lumbar samples. • CSF pleocytosis was predominantly characterised by mononuclear pleocytosis, although polymorphonuclear cells were also present.
Limitations	• Retrospective cases series design with no control or comparison group. • Formal diagnostic accuracy analysis (e.g., sensitivity and specificity) was not performed. • Small sample size (22 dogs). • Inclusion of only histopathologically confirmed GME cases.
Strengths	• All cases were histopathologically confirmed. • CSF findings were reported comprehensively, including TNCC, protein concentration, and differential cell counts.
Strength of evidence	Moderate.

Demierre et al. (2001)

Correlation between the clinical course of granulomatous meningoencephalomyelitis in dogs and the extent of mast cell infiltration

Aim: To investigate the clinical, morphological, and cellular parameters associated with the acute and chronic clinical course of histopathologically confirmed granulomatous meningoencephalomyelitis (GME), with particular focus on mast cell infiltration.

Study design	Retrospective case series.
Intervention details	Target population: Dogs with histopathologically confirmed GME. Sample: 20 dogs with histopathologically confirmed GME referred to the Institute of Animal Neurology, University of Bern (Switzerland), between 1982 and 1999. 13 dogs had an acute clinical course and 7 had a chronic clinical course. Cerebrospinal fluid (CSF) analysis was performed in 16 dogs.
Outcome studied	CSF total nucleated cell count (TNCC), protein concentration, and differential cell counts in dogs with histopathologically confirmed GME.

Main Findings (relevant to PICO question)

- Normal CSF TNCC (0–5 cells/µL) was reported in 12.5% (2/16) of dogs with histopathologically confirmed GME.
- CSF TNCC ranged from 0–5000 cells/µL.
- CSF cytomorphology was available for 13/16 dogs. Mixed pleocytosis (up to 50% neutrophils, 20% macrophages) predominated in acute cases (7/8 dogs, 87.5%), whereas chronic cases showed an equal split between mixed (2/4) and mononuclear pleocytosis (2/4; up to 99% mononuclear cells).

Limitations

- Retrospective case series design with no control or comparison group.
- Formal diagnostic accuracy analysis (e.g., sensitivity and specificity) was not performed.
- Small sample size (20 dogs) with incomplete CSF data for all dogs.

Strengths

- All cases were histopathologically confirmed.
- CSF findings were reported comprehensively, including TNCC, protein concentration, and differential cell counts.
- CSF findings were compared between dogs with acute and chronic clinical courses.

Strength of evidence

Moderate.

Granger et al. (2010)

Clinical findings and treatment of non-infectious meningoencephalomyelitis in dogs: a systematic review of 457 published cases from 1962 to 2008

Aim: To compare published diagnostic and clinical findings from dogs with histopathologically confirmed granulomatous meningoencephalomyelitis (GME) or necrotising meningoencephalitis (NME), and dogs clinically diagnosed with meningoencephalomyelitis of unknown origin (MUO).

Study design

Systematic review.

Intervention details

Target population: Dogs with noninfectious meningoencephalomyelitis, including histopathologically confirmed GME or NME, and clinically diagnosed MUO.

Sample: 56 papers containing 457 dogs were included: 244 with GME, 138 with NME, 59 with MUO, 2 with eosinophilic meningoencephalomyelitis, and 14 with Greyhound meningoencephalomyelitis.

- Histopathologically confirmed cases were included if at least one clinical or diagnostic variable (signalment, neurological localisation, cerebrospinal fluid (CSF) analysis, advanced imaging, and exclusion of infectious disease) was reported.
- Clinically diagnosed MUO cases were required to report signalment, neurological localisation, CSF analysis, imaging results, and exclusion of infectious disease. Studies not meeting these criteria were excluded. As not all included studies reported CSF findings, analyses were performed using smaller subsets of dogs with available data (145 GME, 45 MUO, 32 NME).

Outcome studied	CSF total nucleated cell count (TNCC), presence or absence of pleocytosis (TNCC > 5 cells/µL), CSF protein concentration, and predominant CSF cell type in dogs with GME, NME, or MUO.
Main Findings (relevant to PICO question)	• Normal CSF TNCC (0–5 cells/µL) was reported in 15.9% (23/145) of GME cases, 22.2% (10/45) of MUO cases, and 12.5% (4/32) of NME cases. • CSF TNCC ranged from 0–11,840 cells/ µL in GME cases, 0–6860 cells/ µL in MUO cases, and 0–630 cells/µL in NME cases. • CSF findings were highly variable within disease groups (GME, MUO and NME) but had significant overlap between groups. Mononuclear pleocytosis predominated, although other cell types were also reported.
Limitations	• The authors stated that inconsistencies in the available data prevented a meta-analysis. • Retrospective data extraction from heterogeneous sources. • Inconsistent CSF reference intervals, sampling sites, and reporting standards across decades. • Diagnostic accuracy measures (e.g., sensitivity and specificity) were not evaluated.
Strengths	• Includes a substantial number of histopathologically confirmed cases. • Largest available dataset addressing CSF findings in noninfectious inflammatory CNS disease. • Systematic methodology with predefined inclusion criteria.
Strength of evidence	Moderate.

Stalis et al. (1995)

Necrotizing Meningoencephalitis of Maltese Dogs

Aim: To describe the clinical and pathological features of necrotizing meningoencephalitis (NME) in Maltese dogs and compare them with previously reported cases in Pug dogs.

Study design	Retrospective case series.
Intervention details	Target population: Maltese dogs with histopathologically confirmed necrotising meningoencephalitis. Sample: 5 Maltese dogs with histopathologically confirmed NME. 5 total cerebrospinal fluid (CSF) samples were available from 3 dogs, including repeat CSF analysis in 2 of the 3 dogs.
Outcome studied	CSF total nucleated cell count (TNCC), protein concentration, and clinicopathological findings in Maltese dogs with histopathologically confirmed NME.
Main Findings (relevant to PICO question)	• Normal CSF TNCC (0–5 cells/µL) was reported in 20% (1/5) of CSF samples from 3 dogs with histopathologically confirmed NME. • CSF TNCC ranged from 50–247 cells/µL in abnormal CSF samples (4/5). • Normal CSF TNCC occurred on repeat sampling in a dog with progressive disease following an initially abnormal CSF analysis.

Limitations	• Retrospective case series design with no control or comparison groups. • Formal diagnostic accuracy analysis (e.g., sensitivity and specificity) was not performed. • Very small sample size (5 dogs). • CSF analysis was performed in only 3 dogs (5 CSF samples).
Strengths	• All cases were histopathologically confirmed. • CSF findings were reported alongside detailed clinicopathological and histopathological findings. • Included repeat CSF analysis in 2 dogs, demonstrating changes in CSF findings over the clinical course of disease.
Strength of evidence	Weak.

Appraisal, application and reflection

The body of evidence assessing the diagnostic accuracy of cerebrospinal fluid (CSF) pleocytosis for identifying noninfectious inflammatory central nervous system (CNS) disease in dogs is limited in both quantity and methodological strength. Only four studies met the inclusion criteria – (one systematic review (Granger et al., 2010) and three retrospective case series (Bailey & Higgins, 1986; Demierre et al., 2001; Stalis et al., 1995) – all of which evaluate CSF total nucleated cell count (TNCC) in cases with histopathological confirmation. Histopathology represents the most appropriate reference standard for assessing diagnostic accuracy and reduces the risk of diagnostic misclassification (Andersen-Ranberg et al., 2021). As meningoencephalomyelitis of unknown origin (MUO) is a clinical diagnosis, histopathological examination classifies cases according to the underlying inflammatory subtype (e.g., granulomatous meningoencephalomyelitis or necrotising meningoencephalitis). Among the included studies, the systematic review by Granger et al. (2010) provides the strongest evidence, as it synthesised the available literature on histopathologically confirmed inflammatory CNS disease.

Despite the limited evidence base, the included studies consistently demonstrated that CSF pleocytosis is common but not universally present in dogs with noninfectious CNS disease. Normal TNCC was reported in 2/16 (12.5%) of dogs by Demierre et al. (2001), in one of three dogs with progressive disease on repeat CSF analysis by Stalis et al. (1995), and in 23/145 (15.9%) of dogs with granulomatous meningoencephalomyelitis (GME), 10/45 (22.2%) of clinically diagnosed MUO cases, and 4/32 (12.5%) of dogs with necrotising meningoencephalitis in Granger et al. (2010). This indicates that CSF pleocytosis is a supportive rather than definitive diagnostic finding and should not be used in isolation to exclude noninfectious inflammatory CNS disease. Across studies, mononuclear (lymphocytic or lymphoplasmacytic) pleocytosis was the most reported cytological pattern, although mixed inflammatory and neutrophilic populations were also described in acute GME. While this pattern may support clinical suspicion, it does not confer diagnostic specificity and does not compensate for the limited sensitivity of TNCC, as similar cytological profiles occur in other inflammatory, infectious, neoplastic, and vascular CNS diseases (Andersen-Ranberg et al., 2021; Danciu et al., 2021; Granger et al., 2010).

Overall, the internal validity of the evidence is moderate to weak. Although findings are consistent in demonstrating that CSF pleocytosis is not universally present in dogs with noninfectious inflammatory disease, the absence of large, prospective diagnostic accuracy studies using histopathology as a reference standard limits confidence in precise sensitivity and specificity estimates, and precludes reliable estimation of positive and negative predictive values.

The applicability of these findings to clinical practice is moderate. The included studies primarily represent referral populations, where dogs underwent advanced diagnostics and, in some cases, postmortem examination. This differs from general practice settings, where histopathology is rarely available and diagnoses are commonly made on clinical presentation and exclusion of infectious diseases. As a result, the evidence may overrepresent more severe or atypical cases that progress to biopsy or necropsy.

Breed and age distributions across studies are consistent with recognised noninfectious inflammatory CNS epidemiology, supporting relevance to the typical clinical population. However, diagnostic case definitions vary substantially. This variation reflects real-world clinical practice, where diagnoses rely on combinations of clinical signs, magnetic resonance imaging (MRI) findings, and CSF analysis rather than definitive tissue diagnosis. However, it complicates evaluation of diagnostic accuracy because test performance cannot be separated from the criteria used to define disease. Differences in CSF collection site, disease chronicity at sampling, and laboratory reference intervals further limit generalisability across clinical settings (Lampe et al., 2020).

Despite these limitations, consistent findings across decades indicate that normal CSF TNCC does not exclude noninfectious inflammatory CNS disease. Cerebrospinal fluid pleocytosis remains a valuable supportive finding, particularly when mononuclear cells predominate, but it should not be considered a definitive or standalone diagnostic test. The widespread perception that cerebrospinal fluid pleocytosis is a sensitive diagnostic indicator for MUO appears to have arisen from pragmatic clinical heuristics rather than from formal diagnostic accuracy studies (Tipold, 1995). Although Granger et al. (2010) is frequently cited to support pleocytosis as a diagnostic criterion (Coates & Jeffery, 2014; Cornelis et al., 2019; Lowrie et al., 2013), the review itself reported normal TNCC in 22.2% of dogs classified as MUO. The pleocytosis guidelines proposed by Granger et al. (2010), intended to facilitate case recruitment in the absence of histopathology, require 'hypercellular CSF with > 50% mononuclear cells. While pragmatic in clinical settings, subsequent adoption of these recruitment thresholds as diagnostic or study inclusion criteria may have introduced circularity into later research by preferentially excluding non-pleocytic cases, thereby contributing to an overestimation of the diagnostic performance of CSF pleocytosis (Coates & Jeffery, 2014; Cornelis et al., 2019; Lowrie et al., 2013).

In practice, CSF analysis is most appropriately used as part of a risk-stratification and differential-narrowing process, rather than as confirmatory evidence of noninfectious inflammatory CNS disease. Reliance on pleocytosis as a mandatory diagnostic criterion risks false-negative diagnoses and may delay appropriate immunosuppressive treatment. Mononuclear pleocytosis may increase suspicion for inflammatory disease but lacks disease specificity (Andersen-Ranberg et al., 2021; Danciu et al., 2021). Cerebrospinal fluid findings should therefore be weighed alongside MRI results, neuroanatomical localisation, and clinical progression rather than interpreted in isolation. A presumptive diagnosis of MUO may still be appropriate when CSF analysis is unavailable, provided other clinical and imaging findings strongly support noninfectious inflammatory CNS disease.

Significant evidence gaps remain. Prospective diagnostic accuracy studies comparing CSF TNCC directly with histopathology are lacking due to ethical and practical constraints. Future research should focus on standardising pleocytosis thresholds, improving reporting transparency, and identifying adjunctive CSF biomarkers with greater disease specificity. Until such data are available, clinicians should recognise the limitations of CSF pleocytosis and avoid over-interpreting its diagnostic value in suspected noninfectious inflammatory CNS disease.

Methodology

Search strategy	
Databases searched and dates covered	CAB Abstracts accessed via the OVID platform: 1973 to Week 37 of 2026 PubMed accessed via the NCBI website: 1920 to September 2026
Search strategy	CAB Abstracts: 1. dog or dogs or canine or canines 2. meningoencephalomyelitis or meningoencephalitis or encephalitis or encephalomyelitis or "granulomatous meningoencephalomyelitis" or GME or "necrotising encephalitis" or "necrotizing encephalitis" or "meningoencephalomyelitis of unknown origin" or MUO or "non-infectious inflammatory CNS disease" 3. "cerebrospinal fluid" or CSF 4. pleocytosis or "total nucleated cell count" or TNCC 5. histopathology or histopathological or post-mortem or postmortem or necropsy 6. 1 and 2 and 3 and 4 and 5

Search strategy (cont.)	
Search strategy	PubMed: (dog OR dogs OR canine OR canines) AND (meningoencephalomyelitis OR meningoencephalitis OR encephalitis OR encephalomyelitis OR "granulomatous meningoencephalomyelitis" OR GME OR "necrotising encephalitis" OR "necrotizing encephalitis" OR "meningoencephalomyelitis of unknown origin" OR MUO OR "non-infectious inflammatory CNS disease") AND ("cerebrospinal fluid" OR CSF) AND (pleocytosis OR "total nucleated cell count" OR TNCC) AND (histopathology OR histopathological OR "post-mortem" OR postmortem OR necropsy) Hand-searching: Reference lists of relevant review articles were hand-searched to identify seminal studies not consistently indexed under cerebrospinal fluid–specific search terms. Granger et al. (2010) and Bailey & Higgins (1986) were identified through this process and included due to their reporting of cerebrospinal fluid total nucleated cell counts in dogs with histopathologically confirmed noninfectious inflammatory CNS disease.
Dates searches performed	14 September 2026

Exclusion / Inclusion criteria	
Exclusion	• Narrative reviews, opinion pieces, or expert perspectives without original data. • Studies in which the species-of-interest was not canines. • Studies in which noninfectious inflammatory CNS disease was based solely on clinical criteria, imaging findings, or treatment response, without histopathological confirmation. • Studies evaluating CSF findings in other neurological conditions (e.g., idiopathic epilepsy, intervertebral disc disease, vestibular disease) without a histopathologically confirmed noninfectious inflammatory CNS disease subgroup. • Studies focusing exclusively on CSF biomarkers (e.g., cytokines, oligoclonal bands) without evaluating diagnostic accuracy of TNCC. • Studies failing to meet the minimum threshold for a case series (> 3 cases).
Inclusion	• Studies that reported CSF total nucleated cell count (TNCC) or pleocytosis data. • Studies including histopathological confirmation of disease via post-mortem examination or CNS biopsy, allowing comparison with a diagnostic reference standard. • Studies reporting original data from cohort, case–control, or case series study designs. • Studies that published in peer-reviewed journals in English.

Search outcome							
Database	Number of results	Excluded — other neurological condition	Excluded — incorrect species	Excluded — no histological data	Excluded — CSF biomarkers alone	Excluded — case study	Total relevant papers
CAB Abstracts	35	20	3	2	1	8	1
PubMed	35	21	2	4	1	5	2
Hand-searching	2	–	–	–	–	–	2
Total relevant papers when duplicates removed							4

Acknowledgements
Thank you to Dr. Steven De Decker at the Royal Veterinary College for his guidance and time in discussing this topic.

ORCiD
Zachary Horowitz: https://orcid.org/0000-0002-2066-8425

Conflict of Interest
The author declares no conflicts of interest.

References

1. Andersen-Ranberg, E., Berendt, M. & Gredal, H. (2021). Biomarkers of non-infectious inflammatory CNS diseases in dogs — Where are we now? Part I: Meningoencephalitis of unknown origin. *The Veterinary Journal*. 273, 105678. DOI: https://doi.org/10.1016/j.tvjl.2021.105678
2. Bailey, C.S. & Higgins, R.J. (1986). Characteristics of cerebrospinal fluid associated with canine granulomatous meningoencephalomyelitis: A retrospective study. *Journal of the American Veterinary Medical Association*. 188(4), 418–421. DOI: https://doi.org/10.2460/javma.1986.188.04.418
3. Coates, J.R. & Jeffery, N.D. (2014). Perspectives on Meningoencephalomyelitis of Unknown Origin. *Veterinary Clinics of North America: Small Animal Practice*. 44(6), 1157–1185. DOI: https://doi.org/10.1016/j.cvsm.2014.07.009
4. Cornelis, I., Volk, H.A. & De Decker, S. (2016). Clinical presentation, diagnostic findings and long-term survival in large breed dogs with meningoencephalitis of unknown aetiology. *Veterinary Record*. 179(6), 147. DOI: https://doi.org/10.1136/vr.103640
5. Danciu, C.G., Szladovits, B., Crawford, A.H., Ognean, L. & De Decker, S. (2021). Cerebrospinal fluid analysis lacks diagnostic specificity in dogs with vestibular disease. *Veterinary Record*. 189(10), e557. DOI: https://doi.org/10.1002/vetr.557
6. Demierre, S., Vandevelde, M., Jaggy, A., Griot-Wenk, M.E., Welle, M. & Tipold, A. (2001). Correlation between the clinical course of granulomatous meningoencephalomyelitis in dogs and the extent of mast cell infiltration. *The Veterinary Record*. 148(15), 467–472. DOI: https://doi.org/10.1136/vr.148.15.467
7. Granger, N., Smith, P.M. & Jeffery, N.D. (2010). Clinical findings and treatment of non-infectious meningoencephalomyelitis in dogs: A systematic review of 457 published cases from 1962 to 2008. *The Veterinary Journal*. 184(3), 290–297. DOI: https://doi.org/10.1016/j.tvjl.2009.03.031
8. Lampe, R., Foss, K.D., Vitale, S., Hague, D.W. & Barger, A. M. (2020). Comparison of cerebellomedullary and lumbar cerebrospinal fluid analysis in dogs with neurological disease. *Journal of Veterinary Internal Medicine*. 34(2), 838–843. DOI: https://doi.org/10.1111/jvim.15700
9. Lowrie, M., Smith, P.M. & Garosi, L. (2013). Meningoencephalitis of unknown origin: investigation of prognostic factors and outcome using a standard treatment protocol. *Veterinary Record*. 172(20), 527. DOI: https://doi.org/10.1136/vr.101431
10. Stalis, I.H., Chadwick, B. Dayrell-Hart, B., Summers, B.A. & Van Winkle, T.J. (1995). Necrotizing Meningoencephalitis of Maltese Dogs. *Veterinary Pathology*. 32(3), 230–235. DOI: https://doi.org/10.1177/030098589503200303
11. Tipold, A. (1995). Diagnosis of Inflammatory and Infectious Diseases of the Central Nervous System in Dogs: A Retrospective Study. *Journal of Veterinary Internal Medicine*. 9(5), 304–314. DOI: https://doi.org/10.1111/j.1939-1676.1995.tb01089.x

Contribute to the evidence

There are two main ways you can contribute to the evidence base while also enhancing your CPD:

- Tell us your information need
- Write a Knowledge Summary

Either way, you will be helping to add to the evidence base, and strengthen the decisions that veterinary professionals around the world make to give animals the best possible care. Learn more here: https://veterinaryevidence.org/index.php/ve/author-hub

Licence
Copyright (c) 2026 Zachary Horowitz

Intellectual property rights
Authors of Knowledge Summaries submitted to RCVS Knowledge for publication will retain copyright in their work, and will be required to grant to RCVS Knowledge a non-exclusive licence to publish including but not limited to the right to publish, re-publish, transmit, sell, distribute and otherwise use the materials in all languages and all media throughout the world, and to licence or permit others to do so.

Disclaimer
Knowledge Summaries are a peer-reviewed article type which aims to answer a clinical question based on the best available current evidence. It does not override the responsibility of the practitioner. Informed decisions should be made by considering such factors as individual clinical expertise and judgement along with patient's circumstances and owners' values. Knowledge Summaries are a resource to help inform and any opinions expressed within the Knowledge Summaries are the author's own and do not necessarily reflect the view of the RCVS Knowledge. Authors are responsible for the accuracy of the content. While the Editor and Publisher believe that all content herein are in accord with current recommendations and practice at the time of publication, they accept no legal responsibility for any errors or omissions, and make no warranty, express or implied, with respect to material contained within. For further information please refer to our Terms of Use.