INTRODUCTION

Inflammatory brain disease affecting the parenchyma (encephalitis) and/or meninges (meningitis) is common in small animals and may be non-infectious or infectious. MRI abnormalities are similar and are often non-specific. CSF analysis is considered by some authorities to be more sensitive than MRI for the detection of inflammatory brain disease, and a normal MRI scan does not rule out the possibility that inflammatory disease is present. Several studies have given false negative rates for MRI of 24-29%. However, MRI often gives important information about the degree and distribution of abnormality, its morphology and secondary changes such as obstructive hydrocephalus. In some cases, the cause of the brain inflammation may be evident since MRI also allows assessment of other head structures which may be involved in an inflammatory process, e.g. tympanic bullae, muscles. MRI also shows whether it is safe to obtain CSF or not. Surprisingly, CSF may sometimes be normal even when MRI changes suggestive of inflammatory disease are present (especially if they are deep in the parenchyma). The two techniques are therefore complementary and in our clinic CSF is always obtained after the MRI scan unless tapping is contraindicated based on the sagittal MRI images. Other diagnostic tests used may also include general thoracic and abdominal imaging, serology/PCR testing and even brain biopsy. MRI and CSF are also useful for monitoring progression or otherwise of lesions following treatment.

The changes due to inflammatory parenchymal brain disease are most often multifocal/diffuse and ill-defined, and in some cases involve the meninges as well as brain parenchyma. Meningitis alone also occurs. Mass effect is variable and may be absent. White matter (WM) is often principally affected although grey matter (GM) may also be involved. MRI features reflect inflammation, oedema, demyelination, gliosis and vascular permeability changes. The multifocal and ill-defined pattern usually helps to differentiate inflammatory disease from neoplasia, since brain tumours are usually focal and fairly well-defined masses. However, there is considerable variation, and for many inflammatory diseases it is not possible to describe a “typical” appearance. Therefore interpretation of MRI scans and planning of further work-up should follow general principals, taking into account signalment and history too. Differential diagnosis of suspected inflammatory brain lesions on MRI includes diffuse neoplasia (e.g. lymphoma), metabolic disease (usually symmetrical), post-seizure oedema, infarction and radiation necrosis. Further diagnostic tests and follow-up MRI scans after empirical treatment may be required to monitor progression or otherwise of pathology; for example neoplasia is likely to progress whereas post-seizure changes may improve or resolve.
Technique

Ensure careful anaesthesia and monitoring, as these can be at-risk patients due to the often acute nature of the disease. Consider administering mannitol and/or steroids prior to the study if there is concern over the possibility of raised intra-cranial pressure (ICP).

For suspected raised ICP, perform a sagittal T2W scan first, for detection of subtentorial or foraminal herniation. This will also show whether or not it is safe to perform a CSF tap as well as including the cranial part of the cervical spinal cord, which can show changes secondary to herniation and impairment of CSF flow.

Multiple sequences are required but the two most helpful are FLAIR (especially for parenchymal lesions) and post-contrast T1W (especially for meningeal enhancement):

**FLAIR**
- shows hyperintense lesions close to CSF in ventricles or subarachnoid space which might otherwise be overlooked
- avoids over-diagnosis of ill-defined T2W hyperintensities which are actually partial volume averaging artefacts created by sulci running obliquely through the scan slice; therefore gives confidence to a negative diagnosis on T2W.
- shows whether fluid in “cystic” areas is CSF-like or not.
  BUT
  - may give false positive diagnoses.
  - poorer resolution that with other sequences.
  - less sensitive for meningeal pathology than T1/contrast.

**T1/contrast**
- good for meningeal inflammation (meningitis) although increased enhancement may be subtle and is a subjective diagnosis
- if adjacent to the sphenoid bones, additional fat suppression is helpful to remove the hyperintense signal from bone marrow fat and render enhancement more obvious
- parenchymal lesions often enhance only weakly or not at all, and enhancement is often ill-defined
- increased doses of contract medium are sometimes recommended
- an extra scan performed a few minutes later is used to look for delayed enhancement
- identical pre-contrast slices must be obtained, for comparison and to allow subtraction.

One study (Cherubini and others 2008) found that the sensitivity of different sequences for detection of inflammatory brain lesions was:

FLAIR 84% > T2W 63% ~ T1/contrast 62% > T1W 23%.

Falzone (2008a) found even greater sensitivity for contrast-enhanced FLAIR, using low field MRI.
The STIR sequence (a fat-suppressed, T2W inversion recovery sequence) is very useful for soft tissue inflammation, e.g. myositis, which is sometimes seen with inflammatory CNS disease, especially neosporosis.

**General MRI features**

- MRI may be normal
- Non-specific and more variable than with neoplasia.
- Multifocal or diffuse, ill-defined parenchymal lesions; spinal cord may also be affected.
- Usually asymmetrical or even unilateral (*cf* metabolic disease is usually symmetrical).
- Cerebral hemispheres and brainstem affected more often than cerebellum.
- +/- or meningeal involvement.
- Lesions often more obvious in WM, and blurring of WM:GM differentiation results.
- Lesions hyperintense on T2W and FLAIR.
- Lesions isointense or mildly hypointense on T1W.
- Contrast enhancement (due to BBB breakdown, changes in vascular permeability or neovascularisation) is usually absent, or weak and ill-defined, although sometimes is more intense and focal.
- Very variable mass effect but usually milder than with neoplasia.
- Hydrocephalus can occur due to impaired CSF flow as a result of brain swelling or reduced absorption resulting from ependymitis (e.g. canine distemper, FIP).
- Late or healed disease may result in parenchymal shrinkage and hydrocephalus *ex vacuo* (*negative mass effect*) or formation of CSF-filled parenchymal cavities.
- Haemorrhage is not usually a feature.

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**NON-INFECTIONOUS INFLAMMATORY BRAIN DISEASE**

Pathologically, there is overlap with neoplasia:

- Chronic, severe inflammation may show some histopathological characteristics of neoplasia.
- Neoplastic processes can give rise to a significant inflammatory response.

Non-infectious causes of meningoencephalitis can be divided into granulomatous meningoecephalomyelitis (GME) and necrotising meningo- or leucoencephalitis (NME and NLE). The necrotising forms cause tissue necrosis and secondary cavitation, which may be visible on MRI, whereas GME does not. Differentiation is usually made on histopathology rather than on clinical or MRI findings, but MRI helps to distinguish diseases by topographical distribution (e.g. NME vs NLE) and presence or absence of necrosis (e.g. NLE vs GME). Since definitive diagnosis cannot be made *ante mortem*, these conditions are now usually referred to as meningoencephalitis of unknown origin (MUO).
Granulomatous meningoencephalomyelitis (GME)

Aetiology
First documented in 1978, although possibly constituted the disease “reticulosis” which was described in the early 1960s. Aetiology unknown but thought to be autoimmune (T-cell-mediated, delayed-type, organ-specific hypersensitivity).

Pathology
- Characteristic perivascular cuffing by mononuclear inflammatory cells; these may coalesce to form nodular granulomas which can appear as space-occupying lesions, mimicking tumours.
- Adjacent parenchyma is compressed or obliterated and infiltration is minimal.
- Especially white matter in brain and cervical spinal cord.
- Lacks the tissue necrosis and cavitation of NME and NLE.
- Overlying meningeal inflammation may occur, though is usually not visible on MRI.
- CSF shows raised protein (esp. globulins) and mononuclear pleocytosis.
- Diagnosis confirmed by biopsy or post mortem examination.
- Three basic forms:
  a. disseminated/generalised.
  b. focal (cerebrum and brainstem >> cerebellum).
  c. ocular (optic neuritis).

Signalment
Usually young-to-middle-aged small breed dogs with female preponderance, but any signalment is possible.

Clinical signs
Multifocal clinical signs, often vestibular
  (i) acute form with rapid onset and clinical course
  (ii) chronic form with insidious onset (≈ neoplasia)
May respond to corticosteroids and immunosuppressive drugs.

MRI features
Lesions are usually seen but are non-specific.
  a. Disseminated form of GME
    - MRI may be normal in some cases.
    - Multifocal lesions or diffuse pathology over a larger area, often including the brainstem; asymmetrical and may be unilateral; mainly WM therefore giving lack of WM:GM differentiation.
    - Ill-defined, hyperintense lesions on T2W and FLAIR; isointense, mildly hypointense or even mildly hyperintense on T1W; variable and usually ill-defined contrast enhancement which may be patchy, nodular or ring-like.
    - No evidence of haemorrhage.
    - Variable mass effect (none – mild – severe); chronic cases may show focal brain atrophy. Foramen magnum herniation and loss of sulci due to mass effect are poor prognostic signs.
    - Obstructive hydrocephalus may be seen if the mesencephalic aqueduct is compressed.
• Meningeal pathology is not usually an obvious feature, although is found on histopathology.

b. Focal form of GME - unusual
• Much less common than the disseminated form.
• Clinical course is slower, more like that of neoplasia.
• Solitary, well-defined, contrast-enhancing lesion on MRI.
• Relatively little mass effect cf. neoplasia.
• Any location (cerebrum, cerebellum, brainstem).
• CSF is unreliable.
• Disseminated lesions are found at PM.
• DDx neoplasia (although intra-axial tumours are often cavitary).

c. Ocular form of GME - optic neuritis +/- severe uveitis (acute blindness, mydriasis, papilloedema, retinal haemorrhages)
• A specific localisation of GME.
• Uncommon.
• Usually bilateral, although may be unilateral.
• Optic neuritis +/- severe uveitis.
• Swollen, contrast-enhancing optic nerves on MRI: thin, oblique sagittal T1/C images aligned with the nerve are best.
Necrotising encephalitis

“There is a growing body of evidence that immune-mediated encephalitis, and thus possibly NE in dogs, may not be single different entities in different small breeds but more likely represents different expression of a single disease under variable genetic backgrounds.”

Two distinct disease entities of non-suppurative, necrotising encephalitis are differentiated by histopathology:

a. *Necrotising meningoencephalitis (NME)*; especially pugs, in which a DNA mutation has been found (“pug dog encephalitis”), and Maltese; also other small breeds. Affects GM and WM of cerebrum, with intense meningitis, but there are rarely any significant lesions in the brainstem. Clinical signs are therefore of the forebrain (e.g. seizures, visual deficits).

b. *Necrotising leucoencephalitis (NLE)*; mainly Yorkshire terriers – seen from 1986 although first described in 1993, when it was thought to be viral; similar to NME but is confined to WM and affects the brainstem as well as the cerebrum. Clinical signs are therefore more variable.

**MRI features**

As GME PLUS:
Malacia, necrosis, liquefaction and cavitation produce small, discrete pockets of fluid seen as well-defined foci of T2W hyperintensity and T1W hypointensity which suppress variably on FLAIR and which have peripheral contrast enhancement.

a. *NME*
   - Almost exclusively cerebrum (especially parietal, temporal and occipital lobes).
   - Meninges, GM and WM all affected.
   - Often at GM:WM interfaces, resulting in loss of sharp demarcation.
   - Focal cavitary (fluid-filled) lesions with peripheral enhancement; contents may suppress on FLAIR.
   - Often “negative mass effect” i.e. loss of parenchymal volume and hydrocephalus *ex vacuo*.
   - “Ventriculomegaly” described by some authors is probably just breed variant.
   - +/- meningitis.

b. *NLE*
   - As NME but only WM; brainstem as well as cerebrum; minimal meningeal change.
**Non-infectious meningitis (e.g. SRMA)**

Similar MRI appearance to infectious causes but is predominantly cervical spine rather than brain and may be associated with muscle changes (see spine lecture). The signalment of the patient is often suggestive (often young). Eosinophilic meningitis has also been recognised.

**Non-suppurative meningoencephalitis in greyhounds**

Idiopathic (unknown aetiology), breed-associated disease seen only in Ireland. Affected dogs are usually less than one year of age, and several from a litter may develop the disease. The clinical signs are acute or chronic and progressive, with a range of non-specific signs and a severe clinical course, which is usually fatal. It can only be confirmed on post mortem examination, although the topographic distribution of lesions on MRI is said to be characteristic. CSF shows a mild mononuclear/lymphocytic pleocytosis.

**MRI features**
- Lesions are mainly rostral and ventral in the brain, including the olfactory lobes and caudate nuclei.
- Affects grey matter and subcortical white matter.
- Often symmetrical or nearly so.
- No mass effect.
- Ill-defined hyperintense areas on T2W and FLAIR.
- Isointense on T1W with absent or minimal contrast enhancement.

**Idiopathic hypertrophic pachymeningitis**

This condition is described in man as a rare, chronic, progressive, non-specific inflammatory and fibrotic disease of the dura mater. A series of six dogs with the condition has been described, interestingly comprising three greyhounds, one lurcher and two Labradors. CSF showed non-specific signs and was normal in one dog. The condition should be considered as a possible differential diagnosis for dogs with pachymeningeal thickening on MRI and no identifiable underlying cause.

**MRI features**
- Generalised or localised pachymeningeal thickening with intense contrast enhancement.
- May include falx and tentorium cerebelli.
- Hyperintense on T2W and not suppressing on FLAIR.
- Marked contrast enhancement, sometimes with a two-layered appearance.
- Leptomeninges unaffected; no extension into sulci.
- +/- mild mass effect.
Facial neuritis

MRI changes were first described using a 0.5T magnet on 2006, and now we realise that changes are usually easily seen!

Anatomy – the facial nerve emerges from the cranial cavity with the vestibulocochlear nerve and runs for a short distance through the internal acoustic meatus; it enters the initially straight part of the facial canal within the petrous temporal bone, then pursues a sigmoid course before emerging at the stylomastoid foramen. Between the first and second turns of this S-shaped canal it opens into the tympanic cavity. Varejao (2006) describes four segments of the intratemporal facial nerve: internal acoustic meatus, labyrinthine segment/geniculate ganglion, tympanic segment and mastoid segment. The labyrinthine segment is best seen on MRI as a narrow, horizontal band of soft tissue running through the petrous temporal bone just in front of the vestibulocochlear nerve.

**MRI features**

- Increased contrast enhancement of the affected nerve in some dogs compared with the non-affected side, if the disease is unilateral. Use thin slices in transverse and dorsal planes; subtraction images may help.
- In the Varejao article, 4/6 dogs showed enhancement (2 of all 4 segments, 1 of 3 segments and 1 of 1 segment). These dogs recovered in about 8 weeks or did not recover completely whereas the 2 dogs with no enhancement recovered in ~2w. Therefore the presence and extent of enhancement may be useful for prognosis.

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INFECTIONIOUS INFLAMMATORY DISEASE

Infectious inflammatory brain diseases are rare in small animals in the UK, especially since the climate does not predispose to fungal disease. As indicated above, MRI features are rarely specific for a given disease process, either because they are known to be variable or because too few reports exist in the literature to demonstrate a pattern. Disease is likely to be of haematogenous origin, giving rise to multifocal, pyogranulomatous lesions. Differential diagnosis usually includes lymphoma and histiocytic neoplasia, and other types of very diffuse neoplasia are also occasionally encountered. One exception is FIP, which often produces characteristic changes.
Feline infectious peritonitis, FIP

Neurological signs are usually seen with the dry (parenchymatous form), with insidious onset and lack of distinct clinical signs, which may be focal or multifocal. Exotic cat breeds are overrepresented. The pathology comprises multifocal pyogranulomatous lesions of the brain and spinal cord: meningitis, ependymitis, choroid plexitis, periventriculitis, encephalitis and myelitis. Viscous, proteinaceous CSF is produced and the viscosity of the CSF, exudate and inflammatory cells in the CSF, and ependymitis often lead to obstructive hydrocephalus.

MRI features
- May appear normal.
- Ventriculomegaly; varying severity and may cause herniation.
- Ependymal and periventricular hyperintensity on FLAIR with intense contrast enhancement delineating the ventricles on T1W.
- Marked meningitis, especially around the brainstem and cranial cervical spinal cord.
- Parenchymal lesions are not usually seen, although may be present on histopathology.

Toxoplasmosis

Toxoplasmosis, neosporosis and distemper may give rise to similar clinical and MRI findings. *Toxoplasma gondii* is an intracellular protozoon coccidian parasite, hard to differentiate from Neospora. The cat is the definitive host and any warm-blooded mammal (including the cat and man) or bird may be an intermediate host. It cycles in
the small intestine and is shed in faeces. The occurrence of infection in cats is high but most develop immunity unless immunosuppressed e.g. by FeLV or FIV. Disease in other species is also usually seen in young or immunosuppressed patients. Chronic infection may persist subclinically and re-activate later.

In intermediate hosts acute infection may pass unnoticed but an extra-intestinal cycle may produce cysts in many tissues including the CNS and skeletal muscle. Chronic and subclinical infection may persist life-long.

Three clinical forms are seen:

a. *generalised* – CNS, lung, skeletal and heart muscle, lung, liver, pancreas lymph nodes, eyes etc. giving rise to numerous, vague, systemic clinical signs.

b. *CNS (brain and spinal cord)* – non-suppurative meningoencephalomyelitis GM>WM. Granulomas may give rise to focal lesions.

c. *Radiculoneuritis* – congenital, in puppies <3 months old; encephalomyelitis and myositis cause paresis and rigidity (DDx Neospora).

In cats, pneumonia is the commonest presentation.

*MRI features*

- In man, the changes are variable, with single or multiple lesions of (meningo)encephalitis. Often, typical ill-defined lesions progress to granulomata or abscesses, with ring enhancement around a necrotic centre.
- In veterinary patients few reports exist in the literature, but in both cats and dogs focal, enhancing lesions mimicking tumours (e.g. menigioma) have been reported.
- Microscopic lesions may be present without MRI changes.

**Neosporosis**

Protozoon, similar to Toxoplasma. First described in Norway in boxer puppies in 1984 and named in 1988. Probably many cases previously diagnosed as toxoplasmosis were actually due to Neospora; similar disease but more fulminating. Causes meningoencephalitis (usually GM in brain), meningomyelitis (usually WM in cord), radiculitis and myositis. Diagnosed on muscle biopsy.
Affects dogs and cattle but not cats or man. Dog is both definitive and an intermediate host. Usually affects young puppies via vertical transmission from dam, but can affect any age of dog especially those on a diet of raw meat (greyhounds, working dogs, foxhounds).

Young dogs – usually progressive, ascending paralysis with rigid pelvic limb hyperextension; often fatal.
Older dogs – multifocal neurological signs; also pain, dysphagia, stiff jaw, pyrexia, anorexia, myocarditis, pneumonia, pyogranulomatous ulcerative dermatitis.
Necrotising cerebellitis in older dogs was described in 1995, and is a severe, non-suppurative, necrotising inflammation affecting the cerebellar cortex and overlying leptomeninges; cortical folia atrophy and collapse.

**MRI features**
Two noteworthy MRI changes:
- Myositis, seen as patchy, ill-defined areas of T2W or STIR muscle hyperintensity with contrast enhancement. This appearance should raise the suspicion of neosporosis in a dog with neurological signs.
- Necrotising cerebellitis – T2W hyperintensity surrounding the cerebellum, due both to cortical inflammation and atrophy which widens the subarachnoid space.
Distemper

The CNS is affected 10-14 days after infection, so neurological signs may follow earlier, systemic disease. On histopathology, demyelination is a prominent feature (DDx NLE) and distemper is an animal model for multiple sclerosis in man. Two variants are seen:

a. acute distemper encephalitis and myelitis – multifocal lesions predominate in GM (polioencephalomyelitis).

b. subacute or chronic, milder WM disease – humoral immune response causes perivascular lymphocytic infiltration and extensive WM involvement.

In both cases, secondary inflammation, oedema and haemorrhage may be superimposed.

MRI features

- There are too few reports in the literature to be useful, but general principles of inflammatory disease changes apply.
- MRI does not accurately reflect histopathological distribution of lesions due to superimposed oedema; WM changes and loss of WM:GM differentiation may be due to primary demyelination or oedema, which is usually more severe in WM.

Other infectious CNS disease

Bacterial (numerous bacteria), fungal (e.g. blastomycosis, cryptococcosis, in endemic areas) and algal (prototheca) meningoencephalitides are reported. Abscesses and granulomas may also arise. Various non-specific MRI changes have been described.

MRI features

- General features of meningoencephalitis; usually multifocal, reflecting the haematogenous origin.
- Ependymal and periventricular inflammation with ventriculomegaly has been reported with some cases of blastomycosis.
- Focal lesions e.g. fungal granulomas can mimic tumours such as meningiomas when extra-axial, and gliomas when intra-axial.
- There is no single typical appearance and diagnosis rests on a combination of tests and exclusion of other diseases.
Meningitis

Intracranial meningitis is usually seen as a component of more generalised inflammatory brain disease and may also be part of SRMA. Eosinophilic meningitis is recognised. Diagnosis of meningeal inflammation on MRI is very subjective in milder cases. Production of subtraction images may help, and fat suppression is useful for meninges near medullary bone. Dura = pachymeninges; arachnoid and pia = leptomeninges. Only the pia extends into sulci. Dural and pial patterns of contrast enhancement are recognised, but whereas with man the dural form is usually neoplastic and the pial form inflammatory the same does not seem to hold true for dogs and cats.

MRI features

- Normal MRI does not rule out meningitis.
- Subjectively increased contrast enhancement on T1W.
- Severe cases may show hyperintensity on FLAIR.
- Differential diagnosis – leukaemic/lymphoma infiltrate, histiocytic sarcoma.

Otogenic infections

MRI is an excellent technique for investigation of intra-cranial extension of middle and inner ear disease, although CT is also useful for otitis media. Extension into the cranial cavity may occur. Various bacteria have been implicated and it is possible that plant material may also enter the cranial cavity. Intracranial changes may be localised (abscess) or more widespread (empyema). Intracranial infection secondary to ear disease is more common in cats than in dogs. The largest case series describes MRI changes in 11 cats and 4 dogs (Sturges 2006).

MRI features

- Middle ear disease – T2W-hyperintense lining to the bulla which enhances with contrast; +/- non-enhancing fluid material within the bulla; in chronic cases bulla wall thickening may be identified; para-aural cellulitis and abscessation in severe cases; poor modality for assessment of external ear. Do not overdiagnose the common retention of aural secretions in brachycephalic dogs especially the CKCS (so-called “secretory otitis media”) as significant MED.
• **Inner ear disease** – lesions are usually not seen but in severe cases loss of the normally T2W-hyperintense signal in the cochlea and semicircular canals may occur (use thin slices ~2mm) +/- contrast enhancement of this area.

• **Intracranial changes** - may be localised or more widespread. The Sturges study describes three groups of MR changes, acute, subacute and chronic:
  
  a. **acute disease** – meningitis seen as hyperintensity and thickening of meninges +/- changes in subjacent neuropil varying from mild to severe.
  
  b. **subacute disease** (3-7 days’ duration) – poorly-delineated, plaque-like, enhancing mass adjacent to the petrous temporal bone causing brainstem compression.
  
  c. **chronic disease** – well-defined, globular mass compressing the brainstem due to intracranial abscess (see below).
  
  d. we have also seen a number of cats with “**effusive meningitis**” or intracranial empyema (see below) – two layers of thickened, enhancing meninges separated by a fluid accumulation; presumably a walled-off abscess has not formed. May be contralateral to the affected ear.

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**Brain abscesses and intracranial empyema**

Rare in small animals. Due to penetrating bite injuries, extension from ear, sinus, orbital or dental disease, cranial fractures and migrating foreign bodies. Bite abscesses are seen dorsally on the head; the location of abscesses due to other causes varies. With abscesses the lesion is focal and with empyema there is diffuse, subdural accumulation of purulent material.

**MRI features: abscess (images overleaf)**

- Circumscribed mass, usually appearing extra-axial, which is hypointense on T1W, hyperintense on T2W, non-suppressing on FLAIR and with a thick, contrast-enhancing margin.
- Compression +/- extensive oedema or inflammation of adjacent brain.
- Significant mass effect.
- One report describes rupture of an intra-axial abscess into a ventricle causing pyocephalus seen as signal changes of the ventricular CSF (Seiler 2001).
- Depending on the cause, other MRI features may be seen too e.g. ear disease, cranial penetration and overlying soft tissue inflammation.
**MRI features: empyema**

- See description and images of ‘effusive meningitis’ above.

**Parasitic infection**

Cuterebrosis = CNS migration of *Cuterebra spp.* (visceral larva migrans), which typically causes a fatal meningoencephalitis in dogs and cats. Entry to brain is thought to be via the ethmoid foramen and other skull foramina, haematogenously via large vessels or via direct penetration through middle ear. One case report describes a presumed parasitic tract seen on MRI as a T2W hyperintense, contrast-enhancing cerebral band with overlying meningitis. *Angiostrongylus vasorum* infection has been reported to cause meningitis visible on MRI in a pug, with larvae found in CSF. An extra-axial mass with the appearance of a meningioma in a great Dane was found at surgery to be a granuloma containing an egg of the nematode *Eucoleus boehmi*, due to aberrant migration. Such cases are unusual, meriting individual case reports, and no typical appearance can be described.

**Migrating foreign bodies**

May exist, but hard to diagnose. Most likely to be plant material. Serial MR scans may show movement of lesion.
Further Reading


