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Canine and Feline Meningiomas: Diagnosis, Treatment, and Prognosis

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ABSTRACT:

Meningiomas are the most common primary tumor of the central nervous system in dogs and cats. Meningiomas are usually histologically benign; however, their biologic behavior may be malignant. Differences between canine and feline meningiomas include histopathologic appearance, prognosis, and therapy. Meningiomas in cats are often fibrotic and usually do not infiltrate the brain tissue; their surgical removal is easier compared with that in dogs, radiation therapy may not be necessary in cats after complete gross tumor resection, and the long-term prognosis is better than that in dogs. This article reviews the origin, histologic subtypes, therapeutic response, and outcome of meningiomas in dogs and cats.

Meningiomas are the most common primary intracranial tumor in dogs and cats.[1–5] Spinal meningiomas occur less commonly.[6–10] Orbital meningiomas have been described in dogs.[11] Canine and feline meningiomas have several dissimilarities, and the prognosis and treatment of these tumors differ between species.

ORIGIN

Meningiomas can arise from any of the three meningeal layers; however, cerebral meningiomas are considered to originate from arachnoid cap cells or granulations (projections of arachnoid mater through dura mater into the superior sagittal sinus), particularly from arachnoid cells that are associated with the venous sinus of the dura.[1,12] Intraventricular meningiomas are presumed to arise from the thela choroidea, pia mater, or choroid plexus.[1]

HISTOLOGIC CLASSIFICATION

Canine and feline meningiomas are classified as meningothelial, fibroblastic, transitional, psammomatous, angioblastic, papillary, granular cell, myxoid, or anaplastic.[13] Mild nuclear pleomorphism, rare mitosis among cells, absence of tumor infiltration in the neuroparenchyma, and extensive hemorrhage and necrosis are histologic features of benign meningiomas. In contrast, malignant meningiomas have high numbers of cells undergoing mitosis, necrosis, loss of normal cell architecture, and, rarely, metastasis.[13–15] Meningiomas in dogs and cats are usually histologically benign, but their biologic behavior may be malignant.[13,16] According to immunohistochemical studies used to evaluate hormonal receptors, meningiomas in dogs and cats have a high number of cells that express intranuclear progesterone receptors and a low to absent number of cells that express estrogen receptors.[17–19] Two separate studies using two different proliferative index markers, PFPCNA and

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Ki67, respectively, showed an inverse correlation between progesterone receptor expression and tumor behavior.[17,19] High progesterone receptor expression is more common in benign meningioma, whereas decreased or absent progesterone receptor expression seems to be more common in malignant meningiomas.[18] Tumors with a high PFPCNA index were 9.1 times as likely to recur as were tumors with a low PFPCNA index.[17]

INCIDENCE, PREDISPOSITION, LOCALIZATION, AND HISTOLOGIC FEATURES

Canine Cerebral Meningiomas

In dogs, intracranial meningiomas account for 33% to 49% of all primary brain tumors and are the most common central nervous system (CNS) tumor in this species.[1–5] Intracranial meningiomas can develop in dogs 16 months to 14 years of age (mean age: 9 years); occurrence has been reported in dogs older than 7 years of age in 95% of cases, and dolichocephalic breeds, especially German shepherds and collies, are more frequently affected.[11,16,20–23] In another study, such breed predisposition was not reported.[24] In dogs with intracranial meningiomas, a female sex predisposition has been reported, with a male:female ratio of 0.6, which is similar to that among humans with these tumors.[12,25]

In dogs, most intracranial meningiomas are located over the cerebral convexities and below the brain stem; other locations include the midline attached to the falx cerebri, or the tentorium cerebelli or an intraventricular location associated with a choroid plexus.[1] On gross examination, meningiomas in dogs are usually more friable and red compared with meningiomas in cats.[1] In contrast to histologic findings in meningiomas of humans and cats, many meningiomas in dogs have areas of focal necrosis with pools of neutrophils, and some have been invaded along the perivascular spaces around the veins and arteries of the CNS.[1] Attachment of meningiomas to the dura or leptomeninges may be broad (sessile), narrow (pedunculated), or total (meningioma en plaque).[1] Ultrastructurally, canine meningiomas have extensive folds in the cytoplasm and desmosomal junctions, as do human meningiomas.[1] Immunohistochemically, canine meningiomas commonly express vimentin-intermediate filaments.[1,26]

Cystic meningiomas are occasionally reported in dogs.[27–29] Cystic meningioma accounts for 2% to 4% of all intracranial meningiomas in humans, and it is even less common in dogs.[27–30] Meningioma cysts can be peritumoral or intratumoral.[31,32] Intratumoral cyst formation can be due to ischemic necrosis and aggregation of microcytes. Peritumoral cyst formation can be due to development of peritumoral edema or secondary dilation of the subarachnoid cavity being compressed by the tumor.[33] Active secretion from the tumor may also be a factor in cyst formation.[32]

Feline Cerebral Meningiomas

In cats, intracranial meningiomas are the most common primary intracranial tumor and account for 56% of neoplasms of the CNS in this species.[34–38] These tumors develop mainly in geriatric patients (i.e., older than 10 years of age).[35–38] Development of meningiomas in young cats (i.e., younger than 3 years of age) has been associated with mucopolysaccharidosis type 1.[1] There is no breed predisposition for development of meningiomas; among affected cats, domestic shorthaired and longhaired cats are overrepresented, although meningiomas are also reported in Siamese, Persians, and Maine coons.[37,38] There is a slight predominance of males among cats with meningiomas.[37,38]

In dogs, the main initial clinical sign associated with forebrain meningiomas is seizures. In contrast, the most common initial clinical signs in cats are lethargy and behavior changes.

Cerebral meningiomas in cats are located mainly in the rostral fossa, particularly on the cerebral falx or in the transverse fissure.[34,37,38] Microscopically, these tumors in cats are much more stereotyped than those in dogs; most are meningotheliomatous or psammomatous, and many have cholesterol deposits.[1] Intracranial meningiomas in cats and humans share similarities; these tumors are often fibrotic and benign and do not usually infiltrate brain tissue.[12,35,38]

Multiple meningiomas reportedly occur in 14% to 17% of affected cats and as much as 20% of affected

humans.[12,37–39] Calvaria hyperostosis in cats has been reported in 50% of cases.[38]

Spinal Meningiomas

Spinal meningiomas in dogs and cats have been reported sporadically.[6,7,40–44] In dogs, about 14% of CNS meningiomas reportedly involve the spinal cord (27% in the cervical spinal cord, 47% in the thoracic cord, and 27% in the lumbar cord), whereas in cats, only 4% of all CNS meningiomas reportedly occur in the spinal cord.[45,46] Other studies in dogs also report that spinal meningiomas were mainly located in the cervical area.[7,18,19] In one study of spinal meningiomas in 13 dogs, 10 were located in the cervical region and three were found in the lumbar area.[6] Four of these meningiomas invaded the spinal cord.[6] An extensive spinal meningioma from the cervical to the lumbosacral spine has been reported in a 5-month-old dog.[40] In dogs with spinal meningiomas, there is no breed predisposition, the mean age at diagnosis is 9.5 years (range: 5 to 14 years of age), and there is a male:female predominance of 2:1.[6] These data contrast sharply with what is reported in humans, in whom spinal meningiomas demonstrate a distinct female predilection, ranging from 4:1 to 19:1.[12,47–49] Spinal meningiomas in cats have rarely been reported.[9,10,42,50]

Orbital Meningiomas in Dogs

Orbital meningiomas can arise from secondary extension of an intracranial neoplasm along the optic nerve or, as in the case of primary orbital tumors, from neoplastic transformation of arachnoid cap cells surrounding the intraorbital optic nerve cells within the optic nerve sheath.[21,51–53] In dogs and humans, primary orbital meningiomas are generally thought to be slow growing and benign, but intraocular invasion and malignant variants with extracranial metastasis have been reported.[52–55] In a study of 22 cases, poodles, poodle crosses, Samoyeds, Samoyed crosses, German shepherds, and German shepherd crosses were the only breeds affected.[11] In the same study, the mean age at the time of diagnosis was 9 years (range: 3 to 7 years of age), and a male sex predisposition with a male:female ratio of 2:1 was also evident.[11] Although not highly invasive, canine orbital meningiomas are difficult to remove, and local regrowth or extension through the optic foramen leading to blindness is a common complication.[11] Histologically, canine orbital meningiomas often have multiple foci of myxomatous, chondromatous, or osseous metaplasia, a distinctive feature in these tumors.[11]

Paranasal Meningioma

Paranasal meningiomas have been reported in 10 dogs.[56] These meningiomas may occur as primary extracranial masses as a result of embryonic displacement of arachnoid cells or meningocytes. Paranasal meningiomas differ from intracranial meningiomas mainly because paranasal meningiomas are more anaplastic, malignant, and aggressive.[56]

Once neurologic deficits have been detected in dogs with meningiomas of the brain, survival time is short if animals are not treated with primary therapy, which includes surgical excision, radiation therapy, or a combination of the two.

CLINICAL SIGNS

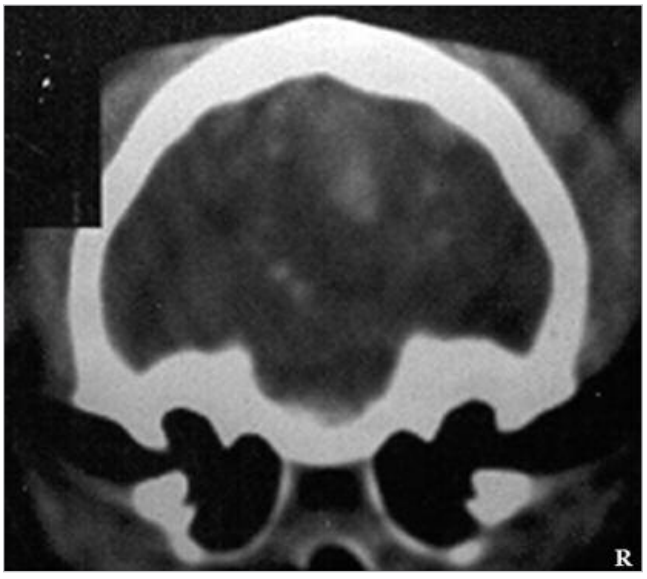
Intracranial Meningiomas

Slow tumor growth accounts for the typically insidious and progressive onset of neurologic dysfunction.[4,35,38] Clinical signs depend on the location and size of the tumor. If affected animals are not treated with primary therapy, survival time is usually short once neurologic deficits are observed.[4] In one study, dogs with seizures and/or behavioral abnormalities developed demonstrable neurologic deficits within 3 months; once these deficits were detected, the clinical course was short (mean time from deficits to necropsy: less than 2 weeks).[4]

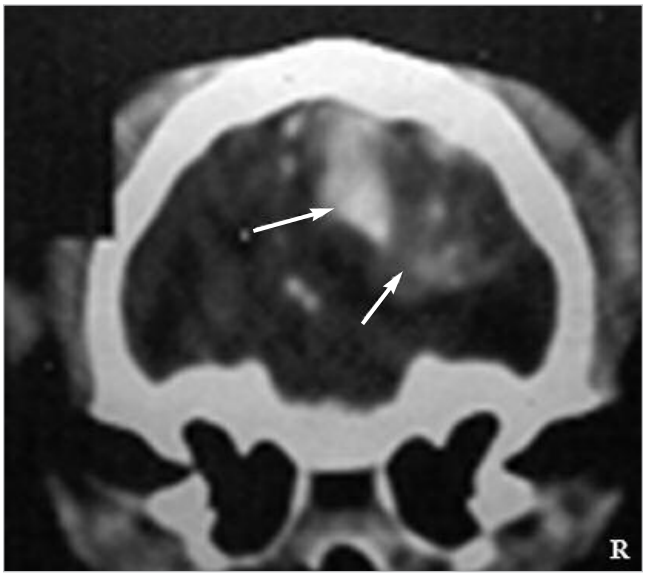
Forebrain Meningiomas

Unilateral forebrain (diencephalon–cerebral hemispheres) meningiomas may be associated with any of the classic clinical signs seen in patients with any unilateral

Figure 1. CT transverse section of a 16-year-old domestic shorthaired castrated cat with a parieto-occipital transitional meningioma. Note the large, partially mineralized mass in the right dorsal cerebrum, which is obliterating the right lateral ventricle. There is cortical bone thickening of the calvarium adjacent to the mass. On postcontrast images, there is evidence of marked deviation of the falx to the left and patchy contrast enhancement of the mass *(arrows)*. At presentation, neurologic clinical signs included dullness, circling, pacing, left eye blindness, and a proprioceptive deficit on the left front limb, suggesting a focal right forebrain lesion. The tumor was surgically removed, and there were no clinical signs of recurrence at follow-up 2 years later.



Precontrast CT transverse section.



Postcontrast CT transverse section.

forebrain disease, including change in mental status, behavior changes, circling (usually toward the side of the lesion), central blindness (amaurosis), contralateral menace deficit with normal pupillary light reflexes and palpebral reflexes, contralateral decreased conscious proprioception, and contralateral facial sensation deficit.[18,24,57] Papilledema (often bilateral) may also be present and is considered to arise from a generalized increase in intracranial pressure (ICP).[58] The cause of circling secondary to cerebral disease is unknown; however, thalamic dysfunction is believed to be involved.[37] Circling and visual abnormalities are also probably part of hemineglect or hemiinattention syndrome.[59] Dogs with this syndrome are slightly to moderately obtunded; have the tendency to walk in circles; and have unique, unilateral disturbances in response to somatosensory, auditory, and visual stimuli and in eating and drinking behavior.[59]

The anatomic basis for hemineglect syndrome is hypothesized to be located in a corticolimbic-reticular loop, which helps explain the association of the syndrome with lesions at widely separated locations.[59] Visual abnormalities are generally attributed to pressure on the optic radiation as it ascends or to pressure on the occipital lobe of the cerebrum.[37] In meningiomas involving the rostral cerebrum (e.g., olfactory and frontal lobes), the initial abnormalities may be restricted to seizures and behavioral changes, and meningiomas in the frontal and prefrontal lobe may be clinically silent.[4,57] In dogs, the main initial clinical sign associated with forebrain meningiomas is seizure activity; in contrast, seizures in cats were reported in only 11% of cases (36 cats) in one study and in 29% (17 cats) in another study.[18,24,37,57,60] In cats, the most common initial clinical signs seen with forebrain meningiomas are lethargy and behavioral changes.[37]

Brain Stem and Cerebellar Meningiomas

Meningiomas that arise from the brain stem can result in cranial nerve deficits or hemi- or tetraparesis, and cerebellar meningiomas may cause dysmetria, circling, ataxia, and intention tremors.[21,24,61] Meningiomas located in the cerebellopontine angle are often associated with clinical signs of paradoxical vestibular syndrome.[62,63] However, in some cases, precise neuroanatomic localization can be obscured by the secondary effects induced by the tumor, such as cerebral edema, obstructive hydrocephalus, and brain herniation.[1,64]

Spinal Meningiomas

According to tumor location, any of the four spinal cord syndromes (i.e., cervical, cervicothoracic, thoracolumbar, lumbosacral) can be anticipated.[64] Common clinical signs associated with spinal meningiomas in dogs include mild to moderate spinal pain, weakness, or sensory or motor deficits from ataxia to nonambulatory states and urinary incontinence. Motor deficits are usually insidious and progressive, with a mean duration between onset of clinical signs and diagnosis of 5.8 months (range: 3 days to 14 months).[6]

Orbital Meningiomas

Common clinical signs are usually indicative of a retrobulbar mass (i.e., exophthalmos, orbital swelling, prolapsed globe). Fundic abnormalities in the posterior segment may show papilledema, an abnormal optic disk, or retinal hemorrhage, and animals are often blind in the affected eye.[11]

DIAGNOSTIC WORKUP

Intracranial Meningiomas

History

The history of CNS signs in middle-aged or older dogs or cats should raise the possibility of neoplasia. Neoplasia should be first on the differential list in animals older than 7 years of age with seizures and no other clinical signs.[65] A progressive course and focal neurologic signs are initial clues to the presence of a brain tumor.[65]

Minimum Database

A minimum database for a dog or cat with clinical signs of brain dysfunction should include a hemogram, serum chemistry panel, and urinalysis.[66] Survey radiography of the thorax and an abdominal ultrasonographic examination can help rule out a primary tumor or malignancy elsewhere in the body.[65,66] In one study, 55% of pathologically confirmed intracranial tumors were metastatic in origin.[67] This same screening may reveal evidence of metastasis from a primary CNS neoplasm.[15]

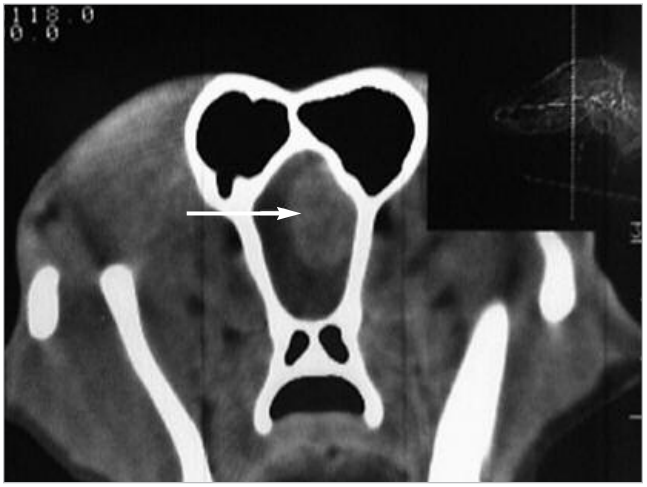


Figure 2. Postcontrast CT transverse section of an olfactory bulb meningioma in a 12-year-old German shepherd mix. Seizure was the only neurologic complaint. Note the contrast-enhancing mass in the right dorsal frontal lobe *(arrow)*. The peripheral location and uniform enhancement are typical of meningiomas. The tumor was surgically removed, and the dog was treated with phenobarbital. The dog died 1 year later because of an unrelated neurologic cause.

Advanced Imaging Techniques

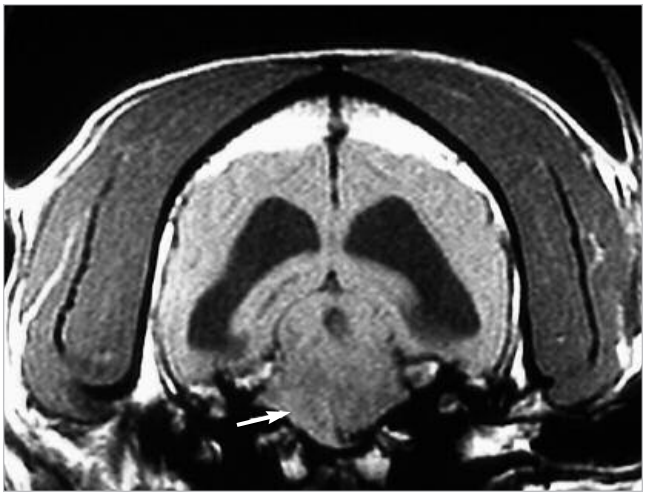
Findings on computed tomography (CT) and magnetic resonance imaging (MRI) are often highly suggestive of intracranial meningioma.[28,68,69] On CT and MRI, meningiomas usually present as a mass effect with distortion of brain symmetry.[68,70,71]

Meningiomas on CT are usually characterized by extensive contrast enhancement throughout the lesion (Figures 1 and 2); however, other intracranial neoplasms (e.g., choroid plexus tumor) and inflammatory diseases (e.g., granulomatous meningoencephalomyelitis) may have the same appearance.[72,73] Calvaria hyperostosis is a common finding on CT scans in cats with intracranial meningiomas. Hyperostosis is associated with bone erosion caused by pressure atrophy of the calvaria, and subsequent bony thickening is associated with the presence of clumps of tumor cells in the medullary spaces.[21,38]

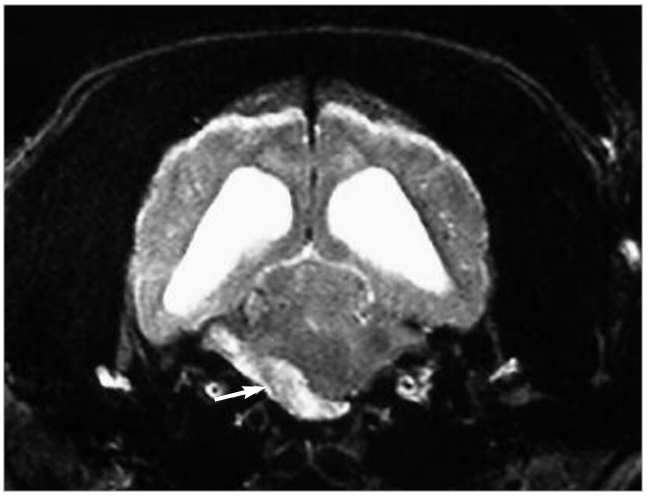
MRI is superior to CT in detecting many of the fea-

The standard of care for intracranial and spinal meningiomas in dogs includes surgical excision when viable, followed by postoperative fractionated radiation therapy.

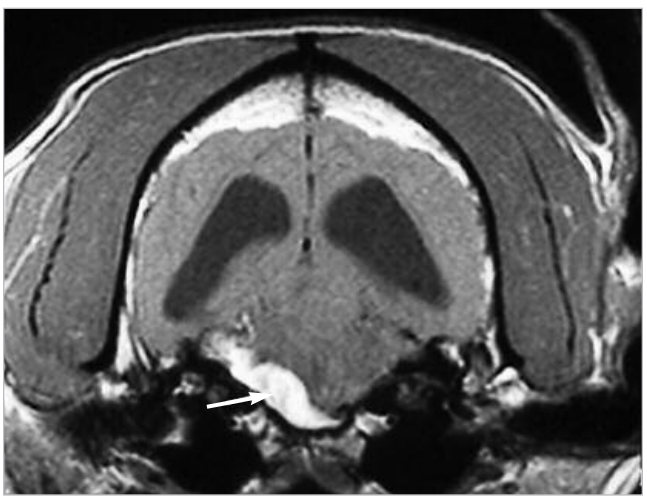
Figure 3. MRI of the brain of a 12-year-old castrated Labrador mix that presented with neurologic signs of central vestibular disease. The histologic diagnosis on necropsy was transitional meningioma.



Transverse T1-weighted MRI. Note the subtle heterogeneous hyperintensity in the left pontine-cerebellar angle *(arrow)*.



Transverse T2-weighted MRI. Note the subtle heterogeneous hyperintensity in the left pontine-cerebellar angle *(arrow)*.



Transverse T1-weighted postcontrast MRI. After administering gadolinium, there is intense contrast enhancement *(arrow)*.

tures associated with brain tumors, such as edema, cyst formation, change in vascularity, hemorrhage, and necrosis, and MRI provides superior soft tissue detail compared with CT.[66] Images obtained by MRI are superior to those from CT in certain brain regions (e.g., the caudal fossa) because beam-hardening artifact, which often obscures the brain stem, does not occur with MRI.[66]

The MRI appearance of intracranial meningiomas in dogs and cats is characterized by extreme contrast enhancement, resulting in increased contrast between the tumor and normal tissue.[71,74] The enhancement may be homogeneous but is often heterogeneous.[71,74] The most common changes seen with MRI are increased signal intensity on T2-weighted images, decreased signal intensity on T1-weighted images, marked homogeneous or nonhomogeneous contrast enhancement with well-defined margins, and the presence of edema[71,74] (Figures 3 and 4). Cysts within or associated with meningiomas have been reported[28,29,63] (Figure 5).

Intratumoral calcification may be responsible for nonhomogeneous enhancement in some cases[71] (Figure 6). Tumor mass effect and edema can be mild or quite prominent.[71,75] The size of the tumor as well as location and severity of associated edema can influence the appearance of the mass effect[71] (Figure 7). Changes in adjacent bone (i.e., lack of fatty bone marrow in the diploë or indistinct bony margins) may be present as a result of the compressive effect of the tumor.[71] The "dural tail" sign (a linear enhancement of thickened dura mater adjacent to an extraaxial mass seen on Gd-DTPA–enhanced T1-weighted images), although not necessarily specific, is often associated with meningioma.[76] The dural tail may be due to either neoplastic infiltration of meninges beyond the margin of the meningioma or hypervascularity of the dura mater.[71,77]

Cerebrospinal Fluid Analysis

Analysis of cerebrospinal fluid (CSF) is recommended as an aid in diagnosing a brain tumor.[60,66] However, if the lesion on CT and MRI appears to fit the criteria for

meningioma, a CSF tap can be avoided. Care should be used in collecting CSF because increased ICP may often be present in association with a brain tumor, and pressure alteration associated with CSF drainage may lead to brain herniation.[78] CSF collection is usually delayed until CT or MRI has been completed to evaluate factors such as the presence of cerebral edema and hemorrhage. Administering mannitol may help decrease elevated ICP before CSF collection.[66] In general, increased CSF protein content and a normal to increased CSF leukocyte count are present with intracranial meningioma.[65,79] CSF from dogs with meningiomas often has an elevated leukocyte count (more than 50/µl), with more than 50% of these cells being polymorphonuclear leukocytes.[80] It appears that CSF alterations seen in association with feline brain tumors are similar to those described for dogs.[81]

Biopsy

Although MRI or CT abnormalities can be highly suggestive of meningiomas, definitive diagnosis should be made by histopathology. Antemortem diagnosis may be obtained by CT-guided stereotactic brain biopsy or by collecting a diagnostic sample during surgery.[82–84]

Spinal Meningiomas

Meningiomas are the most frequently diagnosed primary spinal neoplasia in dogs.[6,85] As with intracranial meningiomas, diagnosing spinal meningiomas requires a systematic approach that includes obtaining a detailed history and minimum database.[66,86] Additional neurodiagnostics should include spinal radiography, CSF analysis, myelography, and CT/myelogram (i.e., CT immediately following a myelogram while contrast medium is in the subarachnoid space) or MRI.[66]

Radiography

Survey radiography of the spinal column should be conducted with the patient under general anesthesia to permit accurate positioning of the spine.[66] Plain-film radiographic abnormalities are uncommon; however, spinal tumor expansion may enlarge an intervertebral foramen, widening the vertebral canal and thinning surrounding bone.[66]

Cerebrospinal Fluid Analysis

CSF is usually collected before injecting the contrast medium. CSF may be normal or may have an increased protein concentration and/or a mildly elevated leukocyte count.[65] Polymorphonuclear cells may predominate as a result of meningeal inflammation and necrosis.[65] It is important to remember that, in dogs and cats, the normal protein concentration in CSF collected from a lumbar puncture may be as high as 45 mg/dl, whereas that from the cerebellomedullary cisterna is usually less than 25 mg/dl.[65]

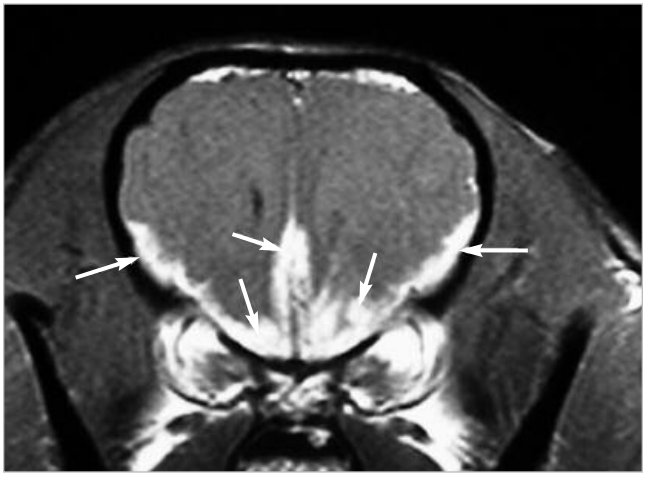
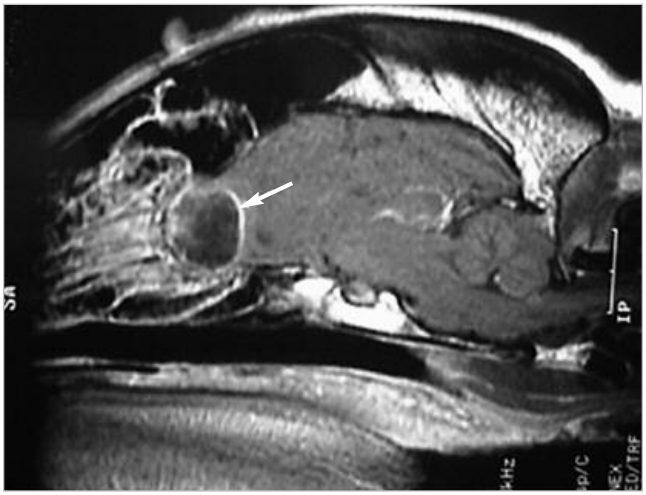


Figure 4. Transverse T1-weighted, postcontrast administration MRI of the brain of a 16-year-old castrated beagle with a history of seizures. Note the intense contrast enhancement centrally along the falx cerebri and ventrally extending along the meninges *(arrows)*. Final diagnosis on necropsy was papillary meningioma "en plaque."

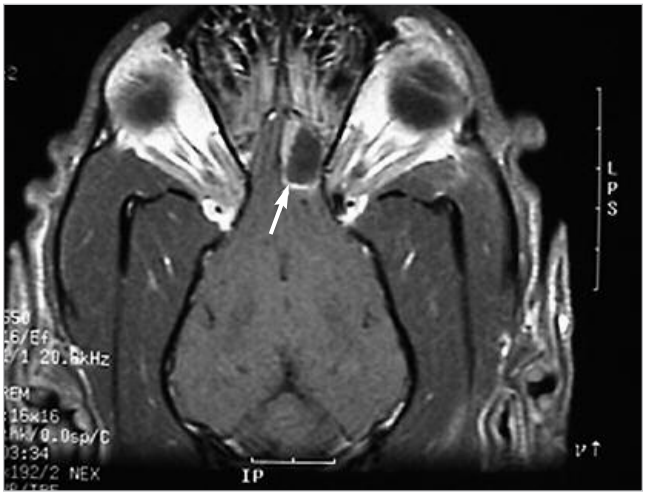
Myelography

Myelography is generally accurate in localizing and evaluating the extension of the tumor. In some cases, both cisternal and lumbar injection of contrast material may be necessary to outline both the cranial and caudal extent of a tumor.[87] Spinal meningiomas are classified as intradural–extramedullary tumors.[85] Myelographic features of spinal meningiomas are not different from those of any other intradural–extramedullary spinal lesions or primary spinal tumors. Nerve sheath tumors, neuroepithelioma (in young dogs), sarcoma, and lymphoma or secondary metastatic tumors should also be considered in the differential diagnosis.[1,88] Because intradural–extramedullary spinal cord lesions occupy space within the subarachnoid volume, they displace contrast agent and appear as a filling defect.[89] The leading edge of the contrast column tends to taper toward the spinal cord and bony margin of the vertebral canal.[89] When captured in this tangent projection, this pattern is described as a "golf tee" appearance[89] (Figure 8). Multiple radiographic views are necessary to determine whether a

Figure 5. T1-weighted, postcontrast administration MRI of the brain of a 9-year-old spayed golden retriever with a history of seizures. Note the hypointense mass with a rim of contrast enhancement *(arrows)* in the olfactory bulb. This tumor was a cystic meningioma and was treated with surgery followed by radiation therapy.



Sagittal view.



Dorsal view.

lesion is intramedullary, intradural–extramedullary, or extradural.[87] However, intradural–extramedullary tumors often have a mixed myelographic appearance and myelographic findings may be misleading.[87] In such cases, CT or MRI may provide more information on the exact localization of the lesion.[70,74,87] Possible adverse effects associated with myelography include temporary worsening of a patient's neurologic status and seizures.[87]

Magnetic Resonance Imaging

On MRI, spinal cord meningiomas have features similar to those of intracranial meningiomas (Figure 9). Advantages of MRI over myelography include minimal complications associated with injection of contrast medium and more accurate determination of the extension of the tumor.[87]

Biopsy

Biopsy is necessary for a definitive diagnosis.[87]

Orbital Meningiomas

The history and clinical presentation are usually suggestive of a retrobulbar mass. As with cerebral meningiomas, a minimum database should be obtained. Ultrasonography, CT, and MRI provide excellent morphologic details of the eye and its associated structures.[90]

THERAPY

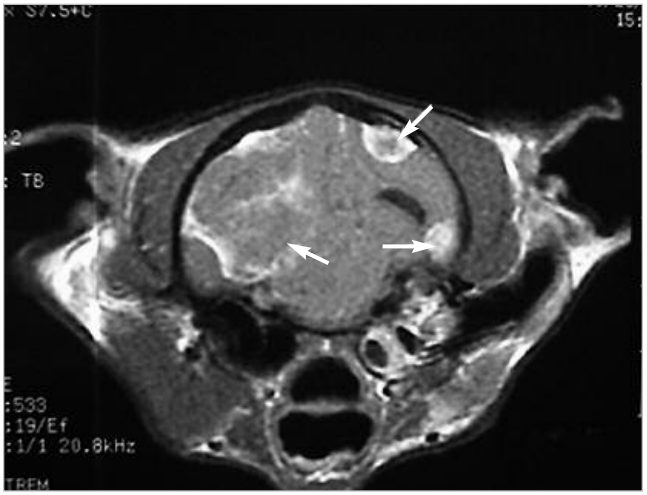
Intracranial Meningiomas

Current management strategies for meningiomas include palliative and primary therapies. Palliative therapy usually consists of administering glucocorticosteroids and antiepileptics, depending on clinical signs.[65,66] Clinical effects of corticosteroids appear to be directed at decreasing the permeability of tumor capillaries. Steroid administration decreases the blood supply to a tumor by 29% within 6 hours of administration and further decreases tumor blood volume by 21% within 24 hours.[91] These changes can result in reduced ICP, decreased brain edema, and reduced clinical signs.[65] Corticosteroid therapy is only palliative, and the reported median survival time in dogs is 2.5 months (range: 1 day to 13 months) and 3.9 months (range: 2.8 to 6 months).[4,92] Seizures in these patients also tend to be less responsive to anticonvulsant therapy.[65]

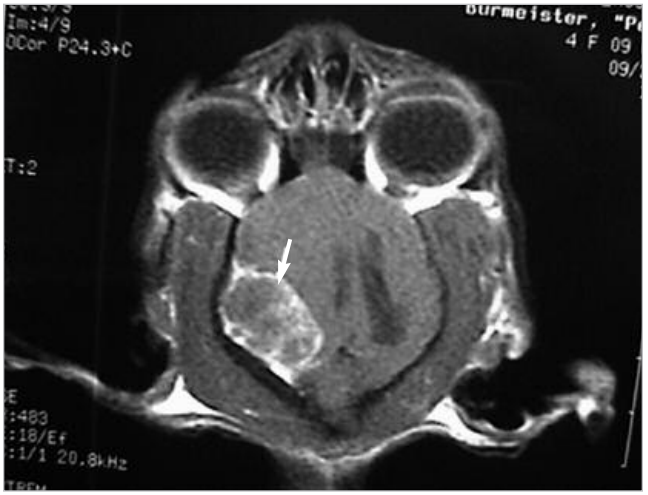
Surgical removal is the treatment of choice for operable intracranial meningiomas in cats.

Primary therapy for intracranial meningiomas in dogs

Figure 6. T1-weighted plus contrast images of a spayed domestic shorthaired cat with multiple meningiomas. The cat presented for behavioral change. The neurologic examination revealed obtundation, ataxia, and decreased proprioception in all four legs as well as clinical signs of right central vestibular disease. The tumors are all peripheral and vary in their contrast enhancement *(arrows)*.



Transverse plane.



Dorsal plane.

includes surgical excision, radiation therapy, or a combination of surgery and radiation therapy (Table 1).

Surgical Excision

Median survival time for dogs treated with surgery alone is 7 months (mean: 7.6 months; range: 0.5 to 22 months).[24,93] Previous studies have also reported shorter median survival times: 6 months in 10 dogs and 4.5 months in four dogs with olfactory meningiomas.[94,95] Survival time may improve if regional cerebral resection is used in conjunction with gross tumor excision.[96]

Enucleation with excisional biopsy has reportedly been effective in 50% of cases with orbital meningiomas.

Radiation Therapy

Radiation therapy has been employed alone, in combination with corticosteroids, and as adjuvant therapy following surgical excision of intracranial meningiomas in dogs.[17,92,93,97–99] Reported protocols have included 5 weekly fractions (hypofractionation)[92,97] or 12 to 16 fractions given either daily (Monday through Friday) or on a Monday/Wednesday/Friday schedule.[100–102] Survival times for dogs treated with surgery and hypofractionated radiation therapy were significantly longer than survival times for dogs treated with surgery alone.[17,24,92] Best results were obtained when radiation therapy was started 3 weeks after surgery with a telecobalt-60 unit, with a planned total radiation dose of 48 Gy given in 12 fractions of 4 Gy during 4 weeks on an alternate-day schedule (i.e., Monday, Wednesday, Friday).[17] Good results were also obtained using a 6-megavolt linear accelerator.[24] It appears that a total dose of 45 Gy given in 15 equal fractions over 3 weeks is well tolerated by the tumor bed of cats and dogs.[66] Another study of 29 dogs (22 of 29 presumptive meningiomas) treated with radiation therapy alone reported a median survival of 250 days.[102] Further studies are needed to determine the optimal dose and number of fractions that should be given and the time over which radiation therapy should extend.[66] At this time, the standard of care for canine intracranial meningiomas should include surgical excision followed by postoperative fractionated radiation therapy.

An important consideration in using radiation therapy for intracranial meningiomas is adverse secondary delayed effects. Normal cells in the brain and spinal cord are either static or slowly dividing. Therefore, radiation effects on brain tissue are primarily delayed reac-

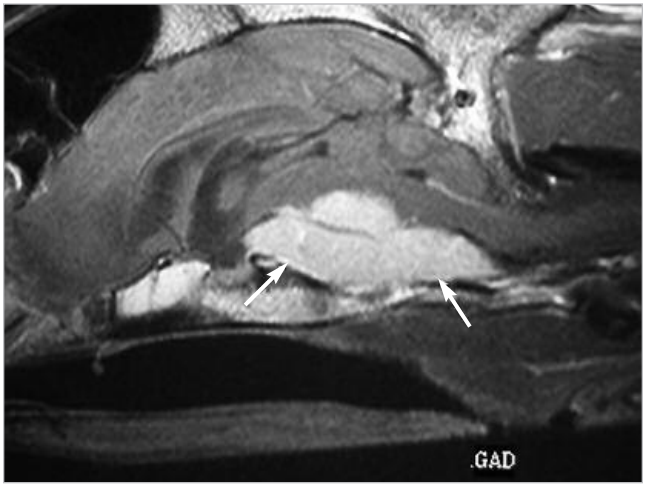


Figure 7. Sagittal T1-weighted, postcontrast administration MRI of an 8-year-old castrated golden retriever. Neurologic abnormalities included dullness, anisocoria, multiple cranial nerve abnormalities, and absent conscious proprioception in both pelvic limbs. Note the large contrast-enhancing mass in the brain stem *(arrows)*. The final diagnosis was brain stem meningioma.

tions with little dose-limiting acute toxicity other than edema. Because the CNS tissue is late-reacting, normal CNS parenchyma is sensitive to the size of individual radiation doses (dose per fraction). Early delayed effects can occur 2 weeks to 3 months after treatment and may be due to transient demyelination. Animals with early delayed effects may present with signs similar to those at initial presentation or may be generally stuporous. These signs are often transient and respond to systemic corticosteroids.[103] Acute radiation reactions of surrounding normal tissue are generally well tolerated and self-limiting. They include epilation and otitis and, when the eyes are included in the treatment field, conjunctivitis, keratoconjunctivitis, and corneal ulcers. These acute radiation side effects subside within 3 to 5 weeks after therapy has been completed. Late delayed effects can occur 6 months to years after treatment, with the most serious being brain necrosis.[103] These late effects are more likely with hypofractionated radiotherapy protocols in which the dose per fraction is larger. With long-term survival, the possibility of brain necrosis resulting in progression of neurologic abnormalities should be considered, and secondary effects cannot be differentiated from tumor recurrence on the basis of clinical signs alone.[24] Demyelination and reactive parenchymal gliosis were reported in two dogs, and a radiation-induced tumor was suspected in one dog that developed a glioma 39 months after radiation.[17]

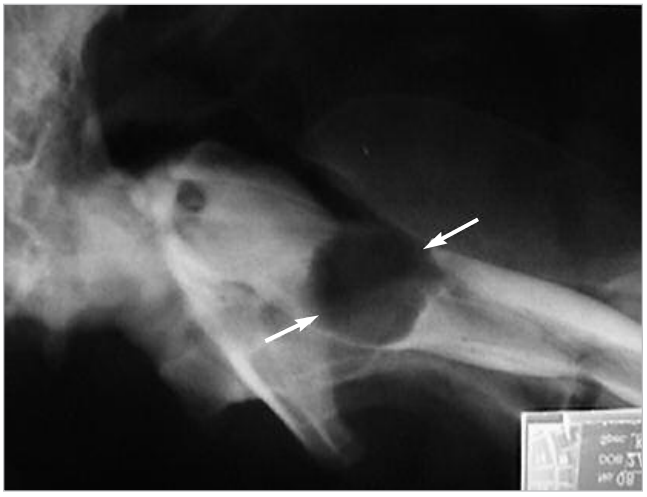
Intracranial Meningiomas in Cats

Surgical removal is the treatment of choice for operable intracranial meningiomas in cats.[34–36,38] The median survival time after surgical excision of meningiomas in cats is 26 months; at the 27-month follow-up in another study, 78.6% of cats did not develop evidence of local tumor recurrence.[34,38] Recurrence of meningiomas after surgical excision in cats is about 22% within a follow-up period ranging from 18 to 47 months.[34] A major question in treating cerebral meningiomas in cats is whether adjuvant radiation therapy should be routinely recommended after surgery. Although cats have been successfully treated with surgery and postoperative radiation therapy, a clear advantage of this method over surgery alone has not been demonstrated.[34] One study recommended treating cerebral meningiomas in cats with surgery alone and considering adjuvant radiation therapy only when surgical resection is incomplete or the tumor recurs.[34]

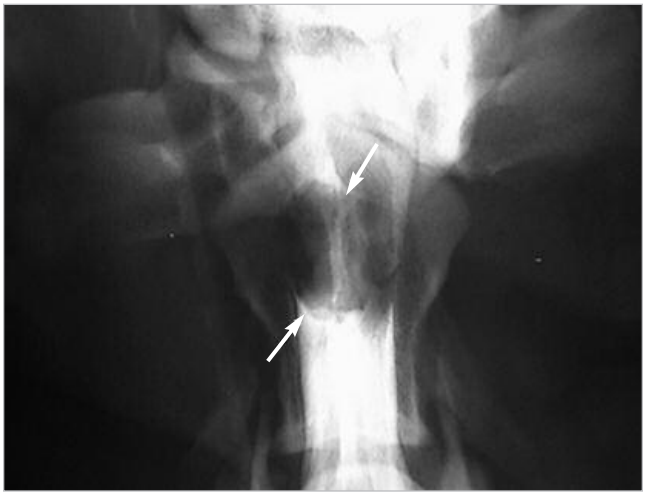
Spinal Meningiomas

An immediate goal of therapy is to relieve the deleterious effects of sustained spinal cord compression; this may be achieved medically (e.g., glucocorticoids) or surgically.[6,66,86] Surgery may permit complete removal or cytoreduction and biopsy of the tumor.[6,66] In one study, five of nine dogs survived for longer than 6 months following surgical resection of a meningioma, and of the five dogs, one was alive 3 years after surgery.[6] Complete resection of ventrally located tumors is more difficult.[6] An indistinct boundary between the tumor and spinal cord is usually a sign of tumor invasiveness.[6] Removing spinal meningiomas is more problematic than excising intracranial meningiomas.[6] Intracranial meningiomas seem much less adherent to the underlying neuroparenchyma compared with spinal meningiomas and may often be evacuated with minimal manipulation.[6] In addition, whereas brain tissue can be gently retracted with little deleterious effect, the spinal cord is much more sensitive to iatrogenic trauma of this kind.[6] Spinal meningiomas in dogs are usually friable, with tenacious adhesion to the pia mater or spinal cord parenchyma and arterial connections to the spinal cord.[6] Because of this intimate connection with the spinal cord, piecemeal excision of the tumor is recommended.[6] A study of nine dogs with spinal cord tumors irradiated after decompressive surgery reported a median survival time of 17

Figure 8. Cervical spine after myelography. There is a large filling defect in the contrast column on the left side and centrally at the level of the first and second cervical vertebral foramen *(arrows)*. Myelographic signs are consistent with an intradural, extramedullary mass. The final diagnosis was meningioma.



Lateral view. Note the caudal contrast column outlining the caudal margin of the mass ("golf tee" sign).



Ventrodorsal view.

months. Most (six of nine) of these tumors were meningiomas.[104] Although this was a small study, extrapolation of the favorable results of radiation therapy after surgical resection of intracranial meningiomas in dogs would be indicated.

Orbital Meningiomas

Enucleation with excisional biopsy has been reported as effective therapy in 50% of cases in one study of 22 dogs, with a 0.2- to 4.5-year follow-up.[11] Recurrence has been reported in 36% of the cases. In the same study, two dogs with recurrent neoplasms simultaneously developed central blindness in the opposite eye, suggesting infiltration of the tumor into the optic chiasm.[11]

ALTERNATIVE THERAPIES

Chemotherapy

Chemotherapy has generally not been shown to be efficacious in treating meningiomas in dogs.[105,106] In the few reported cases using BCNU (carmustine) or CCNU (lomustine), survival was short and not significantly longer than it was for historical control subjects treated with only corticosteroids and/or anticonvulsants.[107] Oral hydroxyurea (50 mg/kg three times weekly) given as adjuvant therapy after surgical resection of intracranial meningioma in dogs (as an alternative to radiation therapy) has recently been investigated, and the results seem promising.[107]

Radiosurgery

Radiosurgery is the application of radiation in a highly localized nature in a single fraction.[66,108] Radiosurgery uses multiple noncoplanar stereotactically focused beams of radiation in a series of arcs to deliver a single dose to the target with extreme accuracy. Two dogs with meningiomas treated with radiosurgery survived 56.7 and 14 months, respectively, and no complications were observed following use of this technique.[108]

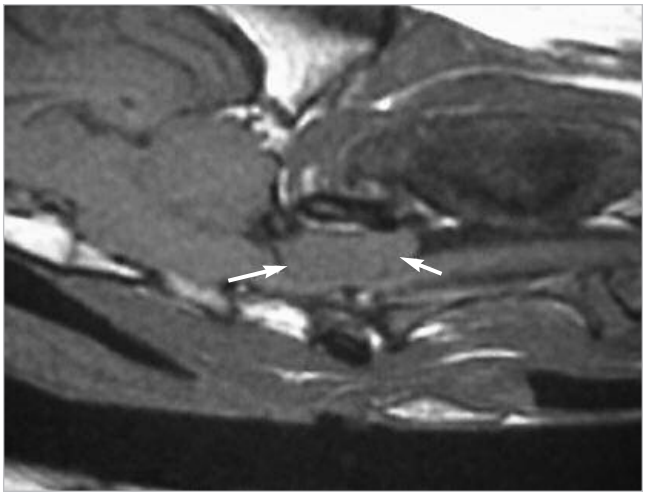
Gene Therapy

Gene therapy may be defined as a treatment modality in which DNA or RNA is transferred to target cells to modify their genetic makeup for therapeutic purposes.[66] A preliminary study of gene therapy of canine meningioma has been reported, but this practice has not gained widespread acceptance.[109]

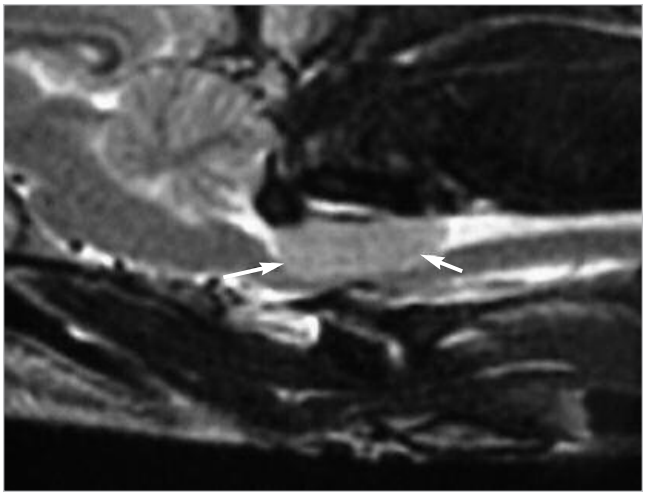
Immunotherapy

Immunotherapy is an approach that mobilizes cell immunity against a brain tumor by culturing and stimulating autologous lymphocytes and then returning them to the tumor.[110] Treating dogs with meningiomas using repeated intracisternal injection of stimulated lymphocytes resulted in clinical improvement and reduced tumor size.[110]

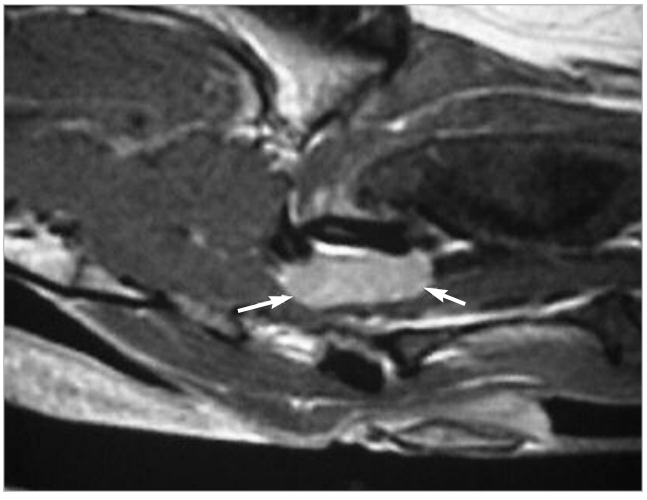
Figure 9. MRI of a dog with a spinal meningioma. Note the large dorsal, intradural, extramedullary mass at the level of the first cervical vertebra *(arrows)*.



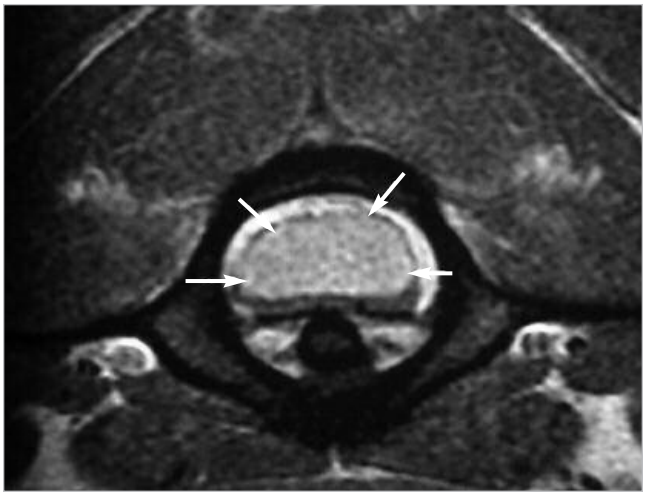
Sagittal T1-weighted administration.



Sagittal T2-weighted administration. Note that the hyperintense signal from the CSF forms a "golf tee" sign along the caudal edge of the mass.



Sagittal T1-weighted, postcontrast administration.



Transverse (axial) T1-weighted, postcontrast administration.

Hormonal Therapy

The abundant expression of progesterone receptors in human, canine, and feline meningiomas is well established.[17–19,111–114] Results of in vitro and in vivo studies indicate that these receptors may be functional and that meningioma growth can be inhibited with antiprogestinic drugs.[115,116] These findings may have clinical implications, particularly regarding antiprogesterone treatment. Antiprogestinic therapy has been used in human meningiomas with some success.[117,118] The efficacy of antiprogesterone treatment in dogs and cats with meningiomas warrants investigation. Hormonal therapy could be particularly beneficial, either alone or as adjuvant therapy for treating meningiomas that are unresectable or partially resectable or that recur after partial excision.

PROGNOSIS

Prognosis of meningiomas tends to be more favorable in cats than in dogs because of histologic features (i.e., fibrotic and not infiltrative), relative ease of surgical removal, occurrence in older patients, and longer survival

Table 1. Median Survival Times After Various Therapies for Intracranial Meningiomas in Dogs

Study	Therapy	Cases	Median Survival Time (mo)	Range
Foster et al[4]	Corticosteroids	13	2.5	1 day–13 mo
Platt et al[92]	Corticosteroids	10	3.9	2.8–6 mo
Platt et al[92]	Radiation therapy and corticosteroids	28	7.4	6–20 mo
Spugnini et al[102]	Radiation therapy and corticosteroids	22	8.3	NR
Kostolich and Dulisch[95]	Surgery	4 (olfactory bulb)	4.7	2.2–7.25 mo
Rossmeisl[96]	Surgery (cortical resection)	6	16.5	1.2–22.5 mo
Axlund et al[24]	Surgery	16	7	0.5–22 mo
Niebauer et al[94]	Surgery with or without radiation therapy or chemotherapy	10	7	—
Platt et al[92]	Surgery, radiation therapy, and corticosteroids	22	14.9	12.6–20.5 mo
Axlund et al[24]	Surgery and radiation therapy	11	18	3–58 mo
Theon et al[17]	Surgery and radiation therapy	20	30	21–39 mo

time reported with surgery alone. Dogs have a more guarded prognosis because their meningiomas tend to be infiltrative. Even after complete surgical removal and adjuvant radiation therapy, meningiomas tend to recur in dogs.[1,17,24,92,94–96] Surgical removal of benign intracranial meningiomas at the cranial base or that involve critical structures (e.g., the cavernous or sagittal sinuses) or are located ventral to the spinal cord is associated with high mortality and morbidity despite the histologically benign nature of these tumors.[5,6,18,24,34,35,37,38,94] For these tumors, surgical techniques and approaches may require reevaluation, and alternative treatments or multimodal therapies require further investigation.

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ARTICLE #4 CE TEST

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1. Which intracranial tumor is most common in dogs?
a. astrocytoma
b. oligodendroglioma
c. meningioma
d. ependymoma

2. Which statement regarding intracranial meningiomas in dogs is correct?
a. No sex predisposition has been reported in dogs with intracranial meningiomas.
b. A female sex predisposition (about 2:1) has been reported in dogs with intracranial meningiomas.
c. A male sex predisposition has been reported in dogs with intracranial meningiomas.
d. A male sex predisposition in dogs with intracranial meningiomas has been reported in only certain breeds.

3. Multiple intracranial meningiomas
a. are more frequent in cats than in dogs.
b. are more frequent in dogs than in cats.
c. have never been reported in dogs.
d. have never been reported in cats.

4. In dogs with spinal meningiomas, there is a
a. male:female predominance of 2:1.
b. female:male predominance of 10:1.
c. female:male predominance of 5:1.
d. male:female predominance of 1:1.

5. Paranasal meningiomas in dogs differ from intracranial meningiomas mainly because paranasal meningiomas
a. are more often benign and tend to be less aggressive.
b. have never been reported in dogs.
c. are more anaplastic, malignant, and aggressive.
d. have been reported only in cats.

6. Which clinical signs are most associated with intracranial meningiomas in the rostral cerebrum (e.g., olfactory and frontal lobes) in dogs?
a. seizures and behavioral changes
b. cranial nerve abnormalities
c. seizures and cranial nerve abnormalities
d. vestibular abnormalities

7. Which statement regarding brain tumors in dogs and cats is correct?
a. A minimum database in a dog or cat with a suspected brain tumor should include thoracic radiographs and an abdominal ultrasonogram because 55% of pathologically confirmed intracranial tumors are metastatic in origin.
b. Brain tumors in dogs are rarely metastatic in origin.
c. Brain tumors in cats are rarely metastatic in origin.
d. none of the above

8. Which statement regarding CSF abnormalities in dogs with meningiomas is correct?
a. CSF from dogs with meningiomas is often normal.
b. CSF from dogs with meningiomas may have an elevated leukocyte count (more than 200/µl, with more than 50% of these cells being lymphocytes) and a normal protein content.
c. CSF from dogs with meningiomas may often have an elevated leukocyte count (more than 50/µl, with more than 50% of these cells being polymorphonuclear leukocytes) and increased protein content.
d. none of the above

9. Standard care for canine intracranial and spinal meningiomas includes
a. surgical excision alone.
b. surgical excision followed by postoperative fractioned radiation therapy.
c. surgical excision followed by corticosteroid therapy.
d. postoperative fractioned radiation therapy and corticosteroids.

10. The prognosis of patients with meningiomas _____ because of histologic features, relative ease of surgical removal, occurrence in older patients, and longer survival time reported with surgery alone.
a. tends to be more favorable in dogs
b. tends to be more favorable in cats
c. is no different for cats or dogs
d. none of the above