Advances in diagnosis and management of intracranial and spinal menigiomas in dogs and cats

Avanços no diagnóstico e no tratamento do menigioma intracraniano e medular de cães e gatos

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Resumo: O meningioma é a neoplasia primária do sistema nervoso central mais comum nos cães e gatos. Origina-se de uma das três camadas das meninges e pode comprimir o tecido nervoso adjacente, resultando em sinais clínicos neurológicos progressivos, que muitas vezes podem comprometer a vida dos animais. Com isso, o objetivo dessa revisão é abordar as principais novidades em relação ao diagnóstico, prognóstico e tratamento dos menigiomas para melhorar a sobrevida dos pacientes.

Summary: Meningioma is the most common primary central nervous system neoplasm reported in dogs and cats. It originates from mesenchymal cells covering one of the three meningeal layers compressing the adjacent neural tissue, and resulting in progressive, life threatening neurological signs. Therewith, the aim of this review is to inform veterinary practitioners about current aspects related to diagnosis, prognosis and treatment options for menigiomas in order to provide the best advice for the pet owners.

Introduction

Meningioma is the most common primary central nervous system tumour (CNS) neoplasm reported in dogs and cats (Peterson et al., 2008; Motta et al., 2012). It occurs most frequently in the brain, and when localized in the spinal cord, is mostly found in the cervical segments followed by the lumbar segments (Levy et al., 1997). Since it does not arise from the brain tissue or spinal cord parenchyma, it is classified as an extra axial or extradural neoplasm. It originates from mesenchymal cells covering one of the three meningeal layers. Arachnoid layer is most commonly affected, particularly at the point where it projects into the venous sinuses (Babicsak et al., 2011). A presumptive diagnosis of meningiomas can be made based on advanced imaging findings and cerebrospinal fluid analysis, but histologic evaluation is essential for a definitive diagnosis. Once the diagnosis is established, the decision whether and how to treat and the choice of protocol must be considered (Sessums and Mariani, 2009). Therewith, the aim of this review is to inform veterinary practitioners about current aspects related to diagnosis, prognosis and treatment options for menigiomas in order to provide the best advice for the pet owners.

Epidemiology

Menigioma is the most commonly reported primary CNS tumour in dogs and cats (Troxel et al., 2003). Canine and feline brain menigioma accounts for about 45% to 85% of all primary intracranial neoplasms and 22% to 59% of all intracranial tumours (Troxel et al., 2003; Snyder et al., 2006). Spinal menigiomas have been reported sporadically (Fingeroth et al., 1987). In dogs, about 14% of all CNS menigiomas involve the spinal cord, whereas in cats only 4%. In one study of spinal menigiomas in 13 dogs, 10 were located in the cervical region and three were found in the lumbar area (Fingeroth et al., 1987). An extensive spinal menigioma from the cervical to the lumbosacral spine has been reported in a five month-old dog (Yeomans, 2000). Generally, menigioma affects dolichocephalic breeds, especially German Shepherds, Golden Retrievers and Labrador Retrievers, with a female sex predisposition and a male:female ratio of 0.6, which is similar to that among people with these tumours (Adamo et al., 2004). Other studies report Boxers as an overrepresented breed (Peterson et al., 2008). Domestic shorthaired cats seem to be predisposed to development of menigioma, although it was also described in Persian, Siamese and Maine coon cats (Adamo et al., 2004). Menigioma has been reported to occur most commonly in dogs over seven years of age and in cats over nine years of age, however there are reports in veterinary medicine describing menigioma in younger dogs and cats, which has been associated with a hereditary metabolic disease- mucopolysaccharidosis type 1 (Keller and Madewell, 1992; Lobetti et al., 1997).
Biological behavior

Meningioma is a neoplastic transformation of arachnoid cells. These cells are characterized by a wide range of functions, which partially explain the highly variable morphological and immunophenotypic patterns exhibited by meningiomas (Louis et al., 2007).

Most human meningiomas, as well as canine and feline meningiomas, are histologically benign, with very low metastatic potential, mostly associated with anaplastic types. If metastases occur, they are most commonly reported in the lungs and heart of the dog (Schulman et al., 1992; Pérez et al., 2005), and kidneys and uterus in the cat (Dahme, 1957). This is probably related to the nature of these tumours, characterized by endless interdigitations and desmossomal interconnections that reduce their exfoliation, common low proliferation index and lack of ability to colonize distant sites (Kepes, 1986). However, exfoliation of neoplastic cells into the subarachnoid space may predispose the spread of a primary focus to adjacent intracranial or spinal regions in a mechanism known as drop metastasis. Furthermore, this neoplasm may exhibit a marked biological malignancy as it may result in high secondary morbidities including herniation, edema, hemorrhage, obstruction to cerebral spinal fluid (CSF) and increased intracranial pressure (ICP). These effects are particularly common in dogs, while 22.6% of meningiomas in cats are considered incidental findings, since they are not associated with any clinical signs, even in a multiple presentation (Troxel et al., 2003). Therefore the terms benign and malignant must be carefully applied in the context of an intracranial or spinal neoplasm (McEntee and Dewey, 2013).

This neoplasm grows within the meninges, but outside the brain and spinal cord parenchyma, however direct invasion of the nervous tissue can occur. Most canine meningiomas are adjacent to the calvarium and a significant number of these involve the olfactory/frontal region, the floor of the cranial cavity, the optic chiasm or the suprasellar and parasellar regions (Snyder et al., 2006; Sturges et al., 2008). Other uncommon intracranial localizations include the cerebello-pontomedullary region, retrobulbar space and middle ear cavity. In cats, common locations include the third ventricle, the supratentorial meninges, and rarely, the cerebellar meninges. Feline multiple meningiomas are common, occurring in approximately 17% of affected cats, however this is very unusual in dogs (McEntee and Dewey, 2013).

Clinical signs

Intracranial meningiomas

Neurological signs caused by intracranial tumours depend on the location, size and rate of growth of the tumour. Although the primary neoplasms most often occur as solitary nodules, clinical signs are usually multifocal due to increased ICP (Walmsley et al., 2006).

Dogs and cats with intracranial tumours may have a long history of inaccurate signals that are often neglected by the owner or veterinarian. Behavioral changes related to an intracranial tumour may progress slowly over several months until the appearance of the most obvious signs of a brain dysfunction. In dogs, the main initial clinical sign is seizure, while in cats seizures are only reported in 11% to 29% of cases (Nafe, 1979; Bagley and Gavin, 1998). Unilateral forebrain meningiomas may be associated with any of the classical asymmetrical clinical signs seen in patients with any unilateral forebrain disease such as mental status and behavior changes, circling (usually toward the side of the lesion), central blindness (amaurosis), contralateral menace deficit, facial sensation, conscious proprioceptive and nasal hypalgesia. If the rostral forebrain (olfactory and frontal lobes) is involved, the initial abnormalities may be restricted to seizures only or may be clinically silent (Bagley and Gavin, 1998; Thomas, 2010).

Meningiomas that arise from brainstem can result in mental status depression, vestibular ataxia, circling, head tilt, hemi- or tetraparesis and ipsilateral cranial nerve deficits. Cerebellar meningiomas may cause hypermetria, cerebellar ataxia and intentional head tremor. If located in the cerebellopontine angle, it could be associated with paradoxical vestibular syndrome (Adamo et al., 2004).

However in some cases, precise neuroanatomical localization can be obscured by secondary effects induced by the tumour, such as cerebral edema, obstructive hydrocephalus, and brain herniation (Snyder et al., 2006; Motta et al., 2012).

Spinal cord meningiomas

Spinal meningiomas can result in upper motor neuron (UMN) or lower motor neuron (LMN) signs depending on the location of the tumour. Generally, common clinical signs include mild to moderate progressive spinal pain, weakness, proprioceptive ataxia, urine incontinence and non-ambulatory states. Tumours of the C6-T2 spinal segments may be associated with ipsilateral Horner’s Syndrome and ipsilateral loss of cutaneous trunci contraction (Parent, 2010).

Presumptive diagnosis

The history of progressive and generally focal CNS signs in the middle-aged or older dogs and cats should raise the suspicion of neoplasia. A minimum database should include haemogram, serum biochemistry panel, and urinalysis (McEntee and Dewey, 2013). Survey radiographs of the thorax and abdominal ultrasound should be performed to rule out a primary tumour el-
severe in the body since 55% of intracranial tumours can be metastatic in origin (Snyder et al., 2006). Survey radiographs of the spinal column can exclude other disorders that can cause spinal cord damage such as bone tumour, dyscoypondylitis and intervertebral disc disease. Furthermore, in few cases of meningioma, the spinal tumour expansion may enlarge an intervertebral foramen, widening the vertebral canal and thinning surrounding bone (Sessums and Mariani, 2009).

Advanced techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) are considered the main diagnostic tools available for identifying the precise anatomical location and relationships between brain and spinal cord tumours and the surrounding tissues; these imaging modalities are useful whenever surgical excision or biopsy is planned (Sturges et al., 2008). However, other intracranial neoplasms and inflammatory disease may have the same appearance (Vite and Cross, 2011). MRI is superior to CT in detecting many of the features associated with brain tumours, such as edema, cyst formation, change in vascularity, and necrosis (Turrel et al., 1986). The MRI appearance of intracranial and spinal meningiomas is characterized by extreme contrast enhancement, resulting in increased contrast between the tumour and normal tissue. Increased signal intensity on T2-weighted images, decreased signal intensity on T1-weighted images and the presence of edema is commonly seen (Sutherland-Smith et al., 2011). On CT, meningiomas are usually characterized by extensive contrast enhancement throughout the lesion associated with calvaria hyperostosis in the brain (Figure 1). Characteristics of CT and MRI images of intracranial meningiomas in dogs and cats are summarized in Table 1. New imaging techniques as magnetization transfer imaging (Vite and Cross, 2011), diffusion-weighted imaging (Sutherland-Smith et al., 2011), magnetic resonance spectroscopy (Vite and Cross, 2011) and dynamic contrast-enhanced MRI (Zhao et al., 2010) may improve characterization of brain lesions. See the references for full details.

For spinal cord meningiomas, myelography is generally an accurate alternative in localizing and evaluating the extension of the tumour (Bagley, 2010). However these tumours often have mixed appearance and myelographic findings may be misleading. CT or MRI is more useful in providing information on the exact localization and extension of the lesion (Sturges et al., 2008).

Analysis of CSF is important when diagnosing meningiomas (Rossmeisl and Pancotto, 2012). Albuminocytological dissociation (increased CSF protein content and a normal CSF leukocyte count) is found in about 30% of canine and feline intracranial meningiomas. In cats a mild to moderate, predominantly neutrophilic pleocytosis may be observed with increased total protein concentration. In dogs CSF may reveal a polymorphonuclear pleocytosis, especially when the tumour is located within the caudal portion of the cranial fossa (Rossmeisl and Pancotto, 2012). However, if the lesion on CT or MRI appears to show increased ICP, a CSF should be avoided, because the pressure alteration associated with CSF drainage may lead to brain herniation (Rossmeisl and Pancotto, 2012).

Table 1 - Characteristics of CT and MRI images of intracranial meningiomas in dogs and cats

<table>
<thead>
<tr>
<th></th>
<th>CT</th>
<th>MRI</th>
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<tr>
<td></td>
<td>Dogs</td>
<td>Cats</td>
</tr>
<tr>
<td></td>
<td>Dogs</td>
<td>Cats</td>
</tr>
<tr>
<td><strong>Margins</strong></td>
<td>Well-defined</td>
<td>Well-defined</td>
</tr>
<tr>
<td><strong>Pre-contrast image features</strong></td>
<td>Isodense to Hyperdense</td>
<td>T1 – weighted: Iso to Hypointensity. Uncommonly hyperintensity. T2 – weighted: hyperintensity. Uncommonly isointensity, hypointensity and mixed signal</td>
</tr>
<tr>
<td><strong>Pattern of contrast enhancement</strong></td>
<td>Usually marked, uniformly contrast enhancing; occasionally ring enhancing</td>
<td>Usually marked, uniformly contrast enhancing; occasionally ring enhancing</td>
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<tr>
<td><strong>Tumour associated oedema</strong></td>
<td>Mild/moderate</td>
<td>Mild</td>
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<tr>
<td><strong>Hyperostosis</strong></td>
<td>Rare</td>
<td>Rare</td>
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<tr>
<td><strong>Dural tail</strong></td>
<td>May be visualized</td>
<td>May be visualised</td>
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<tr>
<td><strong>Visualization of mass effect</strong></td>
<td>Usually visualized</td>
<td>Usually visualized</td>
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All MRI intensity are referred to the grey matter, All CT density are referred to the rest of the brain parenchyma
1 – Troxel et al., 2004; 2 – Rodenas et al., 2011; 3 – Hathcock, 1996; 4 – Sturges et al., 2008; 5 – Graham et al., 1998; 6 – Cherubini et al., 2005.
Definitive diagnosis and prognosis

Despite practical features and efficacy of a presumptive diagnosis, a definitive diagnosis may only be obtained through histopathology. Biopsy specimens should be obtained during de-bulking surgery or stereotactic techniques. Cytological examination may also be performed since it can contribute to a fast diagnosis and are represented by epithelial-like appearance of spindle cells which are arranged in storiform pattern (Vernau et al., 2001).

Macroscopically, canine and feline meningioma present as firm lobular, gray to pink mass. It grows within dura mater and is typically well encapsulated, particularly in cats, displacing the brain without its invasion. About 30% of canine meningiomas penetrate into Virchow-Robin spaces (perivascular spaces in the brain parenchyma), which results in the loss of distinction of the tumour from edematous brain tissue which compromises surgical excision. Canine meningiomas may also form large cystic cavities as a consequence of ischaemic events, necrosis, isolation of CSF, fluid production by the tumour itself or aggregation of secretions. Microscopically, meningiomas may be represented by two basic patterns that may coexist: (1) meningothelial or lobular pattern characterized by epithelial cells with abundant and homogeneous cytoplasm without defined borders and syncytial whorl-like formations; (2) fibroblastic pattern represented by spindle shaped cells disposed in streams separated by collagen fibers. Poorly differentiated tumours may be properly diagnosed by a basic immunohistochemical panel consisting of vimentin, CD34 and E-cadherin, widely preserved in different histological types (Ramos-Vara et al., 2010).

According to the World Health Organization, current histological classification for meningiomas in domestic animals includes two major groups: benign and malignant (anaplastic) meningiomas. Benign types are sub-classified according to histopathological features as meningothelialomatous (meningothelial pattern), fibrous (fibroblastic pattern), transitional (mixture of meningothelial and fibroblastic patterns), psammomatous (whorl formation with a core of central hyalinisation, necrosis and mineralisation), angiomatous (angioblastic), papillary, granular or myxoid. Special subtypes such as microcystic, chordoid, lipo- and secretory, similar to humans classification are also recognized in dogs (Louis et al., 2007). According to Greco et al. (2006), meningothelial, psammomatous and transitional meningiomas carry a better prognosis when compared to those patients with fibroblastic and anaplastic types.

A human grading system (Table 2) was currently used in dogs and cats, to reduce subjectivity of malignancy evaluation and to support a more accurate prognosis. It is based on a common malignancy criteria, mitotic index and growth fraction evaluated via immunohistochemical reaction for Ki-67. In the study performed on 112 dogs (Sturges et al., 2008), 56% of them were classified as grade I, 43% as grade II and 1% as grade III. Prevalence of atypical canine meningiomas (grade II) is much higher than in humans, while benign meningioma is less frequent (Sturges et al., 2008). A sheet-like growth pattern is thought to be enough to attribute a grade II to canine meningiomas (Mandara et al., 2010). Nevertheless, grade III meningiomas were not detected in cats which suggests that this grading system is not suitable for feline meningiomas and confirms its less aggressive biological behavior in this species (Mandara et al., 2010).

Table 2 - Human grading system for meningiomas adapted to dogs and cats (Sturges et al., 2008)

<table>
<thead>
<tr>
<th>GRADE</th>
<th>MITOTIC INDEX</th>
<th>Ki-67 INDEX</th>
<th>MALIGNANCY CRITERIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (Benign)</td>
<td>Less than four mitotic figures per 10 high-power fields</td>
<td>4-5%</td>
<td>-</td>
</tr>
<tr>
<td>II (Atypical)</td>
<td>Between four and 20 mitotic figures per 10 high-power fields</td>
<td>5-10%</td>
<td>At least three of malignancy criteria</td>
</tr>
<tr>
<td>III (Anaplastic)</td>
<td>20 or more mitotic figures per 10 high-power fields</td>
<td>&gt;15%</td>
<td>Obvious malignancy criteria</td>
</tr>
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Malignancy criteria includes increased cellularity, increase nucleus:cytoplasm ratio, sheet-like growth pattern, necrosis foci.
There is observed high predisposition for recurrence of meningiomas in pregnant women and the presence of progesterone receptors in the absence of oestrogen receptors was demonstrated in the majority of meningiomas in humans, dogs and cats (Hsu et al., 1997; Adamo et al., 2004). Loss of progesterone receptors is associated with more aggressive phenotypes characterized by high proliferation index and low degree of differentiation (Hsu et al., 1997; Mandara et al., 2002). Evidence of hormone-dependent growth suggests usefulness of antiprogestogenic drugs in the treatment of meningiomas (Serfaty, 1995).

In humans, over-expression of vascular endothelial growth factor (VEGF) is associated with elevated growth fraction ( Ki-67 ) and the ability of meningioma to produce peritumoral edema (Matiassek et al., 2009). However despite the absence of a similar correlation in dogs, VEGF was correlated with a shorter survival time (Platt et al., 2006) which suggests that angiogenesis is an independent predictor of meningioma’s behavior in dogs (Motta et al., 2012) and an attainable target for novel therapies including metronomic chemotherapy and inhibition of VEGF (London, 2009; Borrego, 2014).

Further markers related to this tumour’s genesis (cyclooxygenase-2) and malignant behavior (telomerase activity, metalloproteinases 2 and 9) have been used for human meningiomas, but their significance in veterinary medicine is still unknown (Motta et al., 2012).

Treatment

Most human meningiomas are treated surgically or in combination with radiotherapy (Platt et al., 2003). Meningiomas in cats are often fibrotic and usually do not infiltrate the brain tissue, thus surgery is recognized as the primary therapy for these patients and most of them can be fully excised. Postoperative mortality associated with craniotomy for meningioma in cats is reported to be approximately 19% (Gordon et al., 1994), and postoperative complications include central blindness, acute renal failure and anemia (Motta et al., 2012). Nevertheless, surgery alone provides survival rates of 71% at six months, 66% at one year and 50% at two years in this species and radiation therapy may not be necessary in cats after complete gross tumour resection (Sessums and Mariani, 2009). However a completely different picture is seen in dogs, in which most meningiomas cannot be easily and completely excised. Surgery with no further treatments results in a median survival of seven months (36%), but adjuvant radiotherapy can prolong survival times to 16.5 months (Axlund et al., 2002). Post-surgical complications appear sporadically and include intraventricular tensions, pneumocephalus and cervical subarachnoid pneumorrhachis (Cavanaugh et al., 2008).

An ultrasonic aspirator allows the surgeon to perform a better excision of the tumour while reducing hemorrhage and it is highly recommended in dogs, in which it can provide a median survival time of 42 months (Peterson et al., 2008).

Spinal meningiomas are usually more adhered to the underlying neuroparenchyma than intracranial meningiomas, which can result in a more problematic excision. In contrast to brain tissue which can be gently retracted with little deleterious effect, the spinal cord is much more sensitive to iatrogenic trauma. As a single treatment, surgery may offer a survival time of approximately 17 months for cats (Rossmeisl et al., 2006) and 19 months for dogs, but 80% of them will eventually face regrowth (Peterson et al., 2008).

Standard megavoltage external beam radiation therapy may be used as a first line treatment for intracranial meningiomas and it is considered a valid procedure for non-resectable presumptive intracranial meningiomas in dogs and also for recurrent meningiomas in cats (Spugnini et al., 2000). Radiotherapy used as an adjunctive treatment is highly recommended in dogs since it may dramatically improve survival times. Collateral damage to the brain tissue includes acute and late side effects. Acute side effects are represented by cerebral edema due to transient demyelination and may result in increased seizure activity. Late side effects, represented by brain necrosis, may occur several months after the course of radiation. They lead to clinical signs similar to those caused by the tumour itself, but they cannot be effectively treated (Sessums and Mariani, 2009). As a result, hyperfractionated protocols with smaller doses per fraction must always be chosen instead of hypofractionated protocols where larger doses per fraction are used. In one report, dogs with intracranial masses were treated with hypofractionated radiation protocol to a total dose of 38 Gray (Gy) given in five escalating doses (5, 7, 8, 9 and 9 Gy) once a week, with 4-MeV X-rays. This resulted in a mean survival time of 12 months for extra-axial tumours, but 14.5% (12/83) of dogs died or were euthanized due to suspected delayed radiation side effects related to delivery of a high dose per fraction (Brealey et al., 1999). A wide variety of hyperfractionated schemes have been used in veterinary medicine, but no study has compared those. Effective brain tumour radiation in dogs usually involves a total dose of 38 to 50 Gy divided in fractions of 2.5 to 5 Gy/fraction, given in a daily (Monday to Friday), Monday-Wednesday-Friday or Monday-Tuesday-Thursday-Friday schedule (three to five weeks of treatment), with 4 to 6-MeV photons delivered from a linear accelerator (Bley et al., 2005; Yoshikawa and Mayer, 2009; Uriarte et al., 2011). Combination of surgery and hyperfractionated radiotherapy may also increase survival times in dogs with intraspinal meningiomas (Petersen et al., 2008), but limited information is available for cats. As a single treatment modality it may offer survival times up to 569 days in dogs, comparable to those obtained after adjuvant radiotherapy (Bley et al., 2005).
A more precise delivery of radiation may be obtained through proper 3D planning or usage of stereotactic radiosurgery, capable of giving a total dose of 10 to 15 Gy within the tumour in one or few fractions. Although limited information is available in veterinary medicine, a recent preliminary study with stereotactic radiosurgery included 20 dogs with presumed meningioma, resulting in a mean survival time of 594 days and no relevant side effect (Lester et al., 2001).

Historically, chemotherapy was shown to be of limited value for intraspinal and intracranial neoplasms due to difficulty on penetrating the blood-brain barrier (BBB), heterogeneity of cell types in tumours and the need for extremely toxic doses to reach the tumour (McEntee and Dewey, 2013). To overcome drug delivery limitations imposed by BBB, direct infusion of high-molecular-weight substances into the brain parenchyma through small diameter cannula was developed with a technique called convection-enhanced delivery (CED). CED can be performed safely throughout the central nervous system and can be performed as a single therapeutic modality as well as prior to and immediately after surgical resection (Rossmeisl, 2014). Mannitol administered prior to chemotherapy, can modify the permeability of BBB and increase penetration of chemotherapeutic agents into CNS (Kroll and Neuwelt, 1998). Alkylating cytotoxic agents of the nitroso urea class- carmustine and or lomustine, have high lipid solubility, with reasonable penetration of BBB (McEntee and Dewey, 2013). They cause direct damage to DNA and RNA molecules and therefore are considered non-specific cell cycle-drugs (Gustafon and Page, 2013). Both drugs resulted in a reduction of tumour size and clinical signs in dogs with inoperable gliomas (McEntee and Dewey, 2013). However, alkylating agents are not usually indicated as first line treatment of these tumours (Coates and Johnson, 2010; McEntee and Dewey, 2013). A report has shown a 13-month survival in a Schnauzer dog treated with lomustine (60 mg / m² orally every 6 weeks) in combination with prednisolone (1mg/kg, VO, q 12 horas) (Jung et al., 2006).

Hydroxyurea is an antimetabolite that specifically affects S-phase due to inhibition of DNA synthesis. It is known to arrest growth of tumours with low mitotic index (Gustafon and Page, 2013). Its benefits are well established in human patients with meningiomas (Schrell et al., 1997) and despite the absence of controlled studies in veterinary medicine it has been often used as a noninvasive and less expensive treatment. In one study, hydroxyurea (20mg/kg/Day) provided survival times of 7 to 8 months in dogs with intracranial meningiomas, while those treated palliatively achieved a survival time of only three months (Sessums and Mariani, 2009). As an adjuvant treatment hydroxyurea resulted in improved survival times in two case reports from one single study (Greco et al., 2006). A similar dose was used in six cats with no drug-associated complications (Forterre et al., 2006).

Palliative treatment is focused on seizure control, edema and ICP reduction. Anti-epileptic drugs, especially phenobarbital (3-5mg /kg twice daily) and potassium bromide (30-40 mg/kg once daily) should be used but seizures in these patients tend to be less responsive to anticonvulsant therapy (McEntee and Dewey, 2013). Glucocorticoids, (i.e., prednisone, prednisolone and dexamethasone) may be used in order to reduce vasogenic edema, and may result in a significant improvement in clinical signs (Coates and Johnson, 2010; McEntee and Dewey, 2013). Dose should be adjusted according to clinical response, which can last for several weeks (Coates and Johnson, 2010). It is indicated to start treatment with an immunosuppressive dose of 2 mg/kg (prednisone or prednisolone) or 0.25 mg/kg IV (dexamethasone) twice daily (BID) tapering it down. Sudden increase in ICP, associated with brain herniation, may respond, in a short term, to administration of mannitol (2 g/kg given over 15 minutes), hypertonic saline (3%) or furosemide (1 mg/ kg BID), with a quick reduction of vasogenic oedema (Coates and Johnson, 2010). The reported median survival time in dogs treated palliatively with corticosteroids and antiepileptic drugs, is 2.5 months (range: 1 day to 13 months) (Foster et al., 1988) and 3.9 months (range: 2, 8 to 6 months) (Platt et al., 2003).

Emergency treatment should be carried out for animals that are uncompensated, probably due to the excessive increase in ICP, as detailed in Figure 2.

Conclusion

Although meningiomas represents the most common intracranial primary neoplasm, they can be widely variable in biological behavior and clinical signs (Louis et al., 2007; Peterson et al., 2008; Motta et al., 2012). A presumptive diagnosis may be obtained through MRI or, if not available, with CT. Definitive diagnosis may be obtained through histopathology. Stereotactic or excisional biopsy are highly recommended, but they are rarely performed due to high cost and morbidity, respectively (McEntee and Dewey, 2013). Surgery is considered the first line therapy for feline solitary meningiomas, however it is usually performed as a debulking procedure in dogs due to invasion of Virchow-Robin spaces (Axlund et al., 2002). For spinal meningiomas, surgery can be harder because the spinal cord is more sensitive to iatrogenic trauma than the brain tissue (Rossmeisl et al., 2006). In dogs, radiotherapy should be performed as an adjunctive procedure or single therapy modality, where it can achieve comparable survival times as if applied after surgical resection. Hyperfractionated protocols must always be chosen to reduce incidence of acute and late side effects (Bley et al., 2005). Stereotactic radiotherapy is a good treatment choice, but it is not always available in veterinary medical centers (Lester et al., 2001). Chemotherapy is not particularly effective but it may be used at a relatively low-costs. Palliative care consists in ICP control and use of anti-epileptic drugs (McEntee and Dewey, 2013).
Figure 2 – Schematic drawing of emergency treatment for patients with intracranial tumours (adapted from Rossmeisl and Pancotto, 2012).
Bibliography


