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CASE REPORT

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Transitional Meningioma in a Dog

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ABSTRACT

Background: Dogs and cats are the most common domestic animal species diagnosed with primary neoplasms of the central nervous system. Of these, meningioma is the brain tumor most commonly reported in dogs and is associated with several neurological changes, such as seizures. The *ante mortem* diagnosis is difficult to make because the use of computerized tomography, the gold standard for the presumptive diagnosis of brain tumors, is still a limited and expensive exam in veterinary medicine. The aim of this study is to report the case of a grade II transitional meningioma in a female dog attended and necropsied at the Veterinary Hospital of the Universidade Estadual de Santa Cruz (HV-UESC).

Case: A 13-year-old female Poodle was examined at HV-UESC with a clinical history of prostration, apathy, anorexia, partial vision loss, and neurological abnormalities, including seizures. The dog was being treated with phenobarbital (2 mg/ kg three times a day) for seizures and doxycycline (5 mg/kg twice a day) for anaplasmosis diagnosed prior to clinical care at HV-UESC. On physical examination, the dog was observed to be mentally altered, with increased aggression, walking in large circles to the left, tremors and spasticity of the thoracic limbs. Laboratory analysis revealed a normal complete blood count. Biochemical analysis revealed an increase in alanine aminotransferase (296 U/mL), alkaline phosphatase (286 U/ mL) and urea (121 mg/dL) levels. Based on the clinical and laboratory abnormalities, the suspicion of cerebral degenerative neuropathy or neoplasia was established. The dog was examined again approximately two months after initial presentation and no improvement in the dog's neurological condition was noted; in fact, the dog's condition had deteriorated, which was evidenced by worsening seizures, tetraparesis and bedsores. Due to the severity of the neurological changes, euthanasia was performed. After authorization from the owner, the dog was referred to the HV-UESC Necropsy Department, where the dog was necropsied. The primary macroscopic finding was a well-defined mass measuring approximately 2.5x1.5x1.5 cm in the frontal cortex of the left cerebral hemisphere. The lesion was firm and white with an irregular surface and was adhered to the dura mater. The mass compressed the adjacent brain tissue. Histopathological analysis demonstrated a nonencapsuled, expansive, and partially delimited neoplasm characterized by sheets of meningoendothelial cells arranged primarily in mantles, though sometimes in lobes, in two distinct patterns: meningoendothelial and fibrous. In addition, a moderate amount of psammomatous bodies were observed. These microscopic characteristics led to the diagnosis of a grade II transitional meningioma.

Discussion: This report describes a case of meningioma in a female dog, which is the most common type of brain tumor diagnosed in dogs according to the literature. The affected animal was advanced in age, a factor that is associated with an increased risk of developing this neoplasm. The neurological alterations noted in this report were associated with the neoplastic process. The macroscopic finding of a cerebral neoformation was suggestive of neoplasm; this was confirmed by microscopic evaluation of the mass. As has been previously reported, the clinical neurological signs of most cases of suspected brain tumors occur late in the disease process, at which point the neoplasm is already in an advanced stage of development. Thus, the prognosis of the affected animal is poor, as it was in this case, which leads to euthanasia. In summary, this is a report of a case of intracranial transitional meningioma accompanied by progressive neurological alterations, in which an *ante mortem* diagnosis is difficult due to the limited use of computerized tomography in routine veterinary practice.

Keywords: canine, meninges, tumor mass.

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INTRODUCTION

CASE

Meningioma is the most common primary brain tumor in dogs and originates from the abnormal proliferation of meningoendothelial cells. The primary sites of development are the olfactory bulb and the frontal lobes, although any intracranial or extracranial region can be affected [2,3,7].

There are variations in clinical manifestations, depending on the anatomical location of the tumor, which may be mild early in the development of the neoplasia. Typically, progressively worsening neuro-logical manifestations such as alterations of consciousness, seizures, and vestibular dysfunctions occur as the tumor grows [11,15].

Given that the clinical manifestations are not pathognomonic, the diagnosis of this disease requires the use of specialized imaging techniques such as computed tomography and magnetic resonance imaging [6,8,9], which are not yet available in most veterinary centers in Brazil. Such tests might accurately diagnose canine intracranial meningiomas, however, they do not allow for evaluation of the specific neoplastic subtype or gradation. Therefore, histopathological evaluation is necessary [11], and the use of immunohistochemistry techniques is also sometimes required [4,10].

The aim of this study was to report the case of a transitional grade II meningioma in a Poodle dog, aged 13 years, which was attended at the UESC Veterinary Hospital. It will discuss the progressive clinical findings of the disease and the pathological alterations observed in the postmortem examination.



Figure 1. A 13-year-old female Poodle, was attended at the Veterinary Hospital of UESC, Ilhéus, Bahia.

A 13-year-old, 3.5 kg Poodle dog was referred to the UESC Veterinary Hospital (Figure 1) with a history of prostration, seizures, partial loss of vision, walking in circles, and aggression for approximately 20 days. According to the owner, the dog was being treated with phenobarbital¹ (2 mg/kg three times daily) to control seizures and doxycycline² (5 mg/kg twice daily) for anaplasmosis diagnosed prior to presenting to HV-UESC. During the clinical examination, moderate dehydration, sinus arrhythmia, hepatomegaly, tremors, spastic anterior limbs, and pacing in wide left circles were observed. During the exam, behavioral alterations were noticed, which varied from stupor to intense aggression independent of stimulus.

Blood samples were collected for a complete blood count (CBC) and biochemical tests. The hematological test showed no abnormalities. Biochemical analysis revealed increased serum levels of alanine aminotransferase (296 U/mL), alkaline phosphatase (286 U/mL), and urea (121 mg/dL).

Based on the clinical and laboratory abnormalities, a diagnosis of neurological disease with a cerebral localization compatible with neoplasia or degenerative disease was established. Nephropathy and liver disease were also considered as concomitant diseases. It was recommended that doxycycline² be discontinued, phenobarbital be continued, and treatment with silymarin³ (50 mg/kg once daily) initiated in association with propolis extract⁴. After two months, the dog returned for reevaluation. According to the owner, there had been significant progression of the dog's symptoms, with worsening seizures despite the use of phenobarbital, as well as the development of tetraparesis and bedsores. Due to the severity of the neurological decline, euthanasia was chosen, which was performed by anesthetic deepening with propofol⁵.

After authorization from the owner, the dog was referred to the Necropsy Sector of the HV-UESC, where the post-mortem examination of the animal was performed. The primary macroscopic finding was a fairly well-delimited mass in the frontal lobe of the left cerebral hemisphere, measuring approximately 2.5x1.5x1.5 cm, which was firm, white in color with an irregular surface, and which was adhered to the dura mater. The mass was compressing adjacent brain tissue (Figure 2).

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DISCUSSION



Figure 2. Left cerebral hemisphere of a 13-year-old female Poodle. A white mass is located in the cortex, measuring approximately 1.5 cm (arrows).

Samples of the mass were collected, preserved in 10% formalin, and sent to the HV-UESC Histopathology Laboratory, where they were processed using the conventional paraffin inclusion technique followed by hematoxylin-eosin (HE) staining. Microscopic evaluation revealed a non-encapsulated neoplasm which was partially delimited and expansive, and which was characterized by the proliferation of meningoendothelial, polygonal, and stellate cells arranged in mantles or lobes and presenting in two distinct patterns: meningoendothelial and fibrous (Figures 3A and 3B). The first consisted of moderately abundant cells without defined limits and with eosinophilic cytoplasm, which was sometimes vacuolated, with round oval nuclei, loose chromatin, and multiple prominent nucleoli (up to three per nucleus). The second pattern consisted of spindle-shaped cells, which were arranged in bundles interspersed by meningoendothelial cells and which had cytoplasm with imprecise boundaries and elongated nuclei. In addition, a moderate number of psammomatous bodies were observed. These characteristics led to the diagnosis of a grade II transitional meningioma.

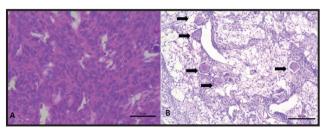


Figure 3. A- Transitional grade II meningioma in a dog. Note groups of fusiform cells, arranged in bundles, with cytoplasm with imprecise boundaries and elongated nuclei [HE]. B- Transitional grade II meningioma in a dog. Note meningoendothelial cells with no defined limits, and with eosinophilic and moderately abundant cytoplasm, which is sometimes vacuolated, and round to oval nuclei and occasional psammomatous bodies (arrows) [HE].

The dog in this case report had a neoformation of the central nervous system that was diagnosed as a meningioma, which, according to the literature, is the most commonly diagnosed primary neoplasm of the central nervous system [6,7], representing about 41% [13] to 51.5% of intracranial tumors [14].

The female dog was a 13-year-old Poodle. There is disagreement among studies regarding which breeds are most affected. Some studies report that dogs of the Boxer [12,15] and Poodle [12] breeds are predisposed to the development of this type of neoplasia, while others report a greater predisposition in Golden Retrievers, miniature Schnauzers, and mixed breeds [14]. There is also some evidence that dogs of dolichocephalic breeds, i.e. breeds with long snouts, are more affected [5,9].

The age of the dog presented in this case is consistent with the literature, which describes a linear relationship between longevity and the incidence of meningiomas, with the highest incidence in animals aged 12-14 years [7,9,11,12,14].

The localization of the neoplasia in the frontal lobe of the left cerebral hemisphere accounts for the neurological manifestations in this dog. The frontal lobe is the area of the central nervous system that regulates behavior. Injuries at this location result in behavioral changes, such as excessive aggression which was noted in this case. The frontal and parietal lobes are also responsible for the control of upper motor neurons, or central motor control, which accounts for the spasticity. Patients with frontal and parietal lobe lesions may also display compulsive behaviors such as walking in broad circles, usually on the same side of the lesion. In this case, both the lesion and the direction of circle walking were the left-sided [1].

Tumors located in the cerebral cortex may also lead to seizures [9,12,15]. Studies show that convulsion is the neurological alteration most frequently associated with intracranial tumors, occurring in about 62% of dogs with this type of neoplasia. This manifestation is not necessarily associated with the malignancy of the neoplasia, but rather with the localization [13]. Finally, the visual deficit most likely occurred due to continuity or compressive injury in the occipital lobe [1].

In most cases, suspicion for a cerebral neoplasia only occurs late in the disease process when the neurological signs are already evident, at which point the neoplasia and its sequelae are already advanced and the possibility of intervention is low. Furthermore, imaging tests such as magnetic resonance imaging and computed tomography, though they have good sensitivity for the diagnosis of meningioma [6,8,15], still have limited use in veterinary medicine. In the geographic region where this dog was treated, this type of examination is not performed.

The definitive treatment for meningiomas is surgical excision of the mass. For this, an experienced veterinary neurosurgeon, anesthesia, and appropriate intensive care are required [9]. Furthermore, the risks and benefits of surgery and anesthesia should be considered. In the case of a possible surgical procedure, associated radiotherapy is recommended [11]. In this case, neither surgical excision nor radiotherapy were feasible.

Chemotherapy has a low efficacy for the treatment of canine meningiomas and is not recommended [11]. Thus, only palliative treatment for convulsive seizures with phenobarbital, in addition to silymarin and propolis extract as antioxidant and hepato-protective agents, were chosen. According to other studies, palliative therapy can be achieved with phenobarbital (3-5 mg/kg twice daily) and potassium bromide (30-40 mg/ kg once daily), potentially extending life expectancy by two to four months [5,9].

Despite palliative care, the patient's neurological condition deteriorated and the convulsive crises became refractory to therapy, worsening the patient's quality of life. At this point, euthanasia was chosen. As in this case, the majority of animals affected by meningiomas have a poor prognosis and euthanasia is often indicated [7].

In the postmortem evaluation, the macroscopic presentation of the neoplasia was consistent with characteristics described in the literature [3]. The literature mentions that meningiomas in dogs develop more commonly in the thalamus region (the diencephalon portion) [12], though in this case the neoplasia was located in the cortical region. The diagnosis was confirmed by microscopic evaluation of the lesion, and the cellular characteristics were also consistent with those described in the literature for transitional meningiomas [2,3]. The lesion was histopathologically classified as transitional, which is the most commonly reported subtype [6,7]. Although transitional meningiomas are graded microscopically as grade I lesions, which have the best prognosis, according to the literature any subtype can be classified as a grade II lesion when presenting with aggressive behavior [3]. In this case, there were characteristics of malignancy, such as local invasion, which led to a grade II classification, which has a more severe prognosis.

In the case reported here, a grade II intracranial meningioma with associated progressive neurological changes was confirmed based on clinical, macroscopic, and histopathological findings.

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Declaration of interest. The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

REFERENCES

- 1 De Lahunta A., Glass E.N. & Kent M. 2014. Veterinary Neuroanatomy and Clinical Neurology-E-Book. St. Louis: Elsevier Saunders, pp.1-587.
- 2 Fauchon E., Husson J.C., Hernandez J. & Gomes E. 2018. Primary extracranial nasopharyngeal meningioma in a dog. *Veterinary Radiology and Ultrasound*. doi: 10.1111/vru.12617. 1-5.
- **3 Higgins R.J., Bollen A.W., Dickinson P.J. & Sisó-Llonch S. 2017.** Tumors of the Nervous System. In: Meuten T.J. (Eds). *Tumor in Domestic Animals*. New Jersey: Willey BlackWell, pp.834-891.
- 4 Johnson G.C., Coates J.R. & Wininger F. 2014. Diagnostic Immunohistochemistry of Canine and Feline Intracalvarial Tumors in the Age of Brain Biopsies. *Veterinary Pathology*. 51(1): 146-160.
- **5 Horta R.S., Martins B.C., Lavalle G.E., Costa M.P. & Araújo M.B. 2013.** Neoplasias Intracranianas em Pequenos Animais Revisão de Literatura. *Acta Veterinária Brasilica*. 7(4): 272-281.
- 6 Kraft S.L., Gavin P.R., Dehaan C., Moore M., Wendling L.R. & Leathers C.W. 1997. Retrospective review of 50 canine intracranial tumors evaluated by magnetic resonance imaging. *Journal of Veterinary Internal Medicine*. 11: 218-225.

- 7 Marcasso R.A., Moreira J.R., Valentim L.G., Arias M.V.B. & Bracarense A.P.F.R.L. 2015. Meningiomas em cães: aspectos clínicos, histopatológicos e imuno-histoquímicos. *Pesquisa Veterinária Brasileira*. 35(10): 844-852.
- 8 Masciarelli A.E., Griffin I.V., Fosgate G.T., Hecht S. Mankin J.M., Holmes S.P., Platt S.R., Kent M., Pancotto T.E., Chen A.V. & Levine J.M. 2017. Evaluation of magnetic resonance imaging for the differentiation of inflammatory, neoplastic, and vascular intradural spinal cord diseases in the dog. *Veterinary Radiology and Ultrasound*. 58: 444-453.
- 9 McEntee M.C. & Dewey C.W. 2013. Tumors of the nervous system. In: Withrow S.J., Vail D.M. & Page R.L. (Eds). *Withrow & MacEwen's Small Animal Clinical Oncology*. Philadelphia: Saunders, pp.583-596.
- 10 Montoliu P., Añor S., Vidal E. & Pumarola M. 2006. Histological and Immunohistochemical Study of 30 Cases of Canine Meningioma. *Journal of Comparative Pathology*. 135(4): 200-207.
- 11 Motta L., Mandara M.T. & Skerritt G.C. 2012. Canine and feline intracranial meningiomas: An updated review. *The Veterinary Journal*. 109: 153-165.
- 12 Santos R.P., Figuera R.A., Beckmann D.V., Brum J.S., Ripplinger A., Neto D.P., Baumhardt R. & Mazzanti A.
 2012. Neoplasmas envolvendo o sistema nervoso central de cães: 26 casos (2003-2011). *Pesquisa Veterinária Brasileira*.
 32(2): 153-158.
- 13 Schwartz M., Lamb C.R., Brodbelt D.C. & Volk H.A. 2011. Canine intracranial neoplasia: clinical risk factor for development of epileptic seizures. *Journal of Small Animal Practice*. 52(12): 632-637.
- 14 Song R., Vite C., Bradley C. & Cross J. 2013. Postmortem Evaluation of 435 Cases of Intracranial Neoplasia in Dogs and Relationship of Neoplasm with Breed, Age, and Body Weight. *Journal of Veterinary Internal Medicine*. 27: 1143-1152.
- **15** Snyder J.M., Shofer F.S., Van Winkle T.J. & Massicotte C. 2006. Canine intracranial primary neoplasia: 173 cases (1986-2003). *Journal of Veterinary Internal Medicine*. 20: 669-675.

