Acute postretinal blindness: ophthalmologic, neurologic, and magnetic resonance imaging findings in dogs and cats (seven cases)

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Abstract

Objective To describe the ophthalmologic, neurologic, and magnetic resonance imaging (MRI) findings of seven animals with acute postretinal blindness as sole neurologic deficit.

Methods Medical records were reviewed to identify dogs and cats with postretinal blindness of acute presentation, that had a cranial MRI performed as part of the diagnostic workup. Only animals lacking other neurologic signs at presentation were included. Complete physical, ophthalmic, and neurologic examinations, routine laboratory evaluations, thoracic radiographs, abdominal ultrasound, electroretinography, and brain MRI were performed in all animals. Cerebrospinal fluid analysis and postmortem histopathologic results were recorded when available.

Results Four dogs and three cats met the inclusion criteria. Lesions affecting the visual pathways were observed on magnetic resonance (MR) images in six cases. Location, extension, and MRI features were described. Neuroanatomic localization included: olfactory region with involvement of the optic chiasm (n = 4), pituitary fossa with involvement of the optic chiasm and optic tracts (n = 1), and optic nerves (n = 1). Of all lesions detected, five were consistent with intracranial tumors (two meningiomas, one pituitary tumor, two nasal tumors with intracranial extension), and one with bilateral optic neuritis that was confirmed by cerebrospinal fluid analysis. Histologic diagnosis was obtained in four cases and included one meningioma, one pituitary carcinoma, one nasal osteosarcoma, and one nasal carcinoma.

Conclusions Central nervous system (CNS) disease should be considered in dogs and cats with acute blindness, even when other neurologic deficits are absent. This study emphasizes the relevance of MRI as a diagnostic tool for detection and characterization of CNS lesions affecting the visual pathways.

Key Words: central nervous system, MRI, vision loss, visual pathways

INTRODUCTION

Blindness can be caused by bilateral lesions in four different locations and by different mechanisms: lesions that produce opacification of the clear ocular media, lesions that cause failure of the retina to process the image, lesions that impede transmission or relay of the message through the visual pathways, or lesions that cause failure of the final processing of the image in the visual cortex.1,2

A systematic clinical approach should be performed in cases of blindness. A thorough medical history and complete physical and ophthalmic examinations are usually the first steps. In addition, electroretinography (ERG) is frequently used to assess retinal function. In cases of lack of significant ocular lesions and normal ERG, blindness should be related to a process located anywhere along the afferent visual pathway (postretinal blindness).1,2 Although routine diagnostic procedures such as neurologic examination, routine laboratory examinations, and cerebrospinal fluid (CSF) analysis can help to establish the most likely location and etiology of CNS lesions in these cases, advanced imaging techniques are necessary to confirm the exact location and reach a prompt
preliminary diagnosis. In human beings, magnetic resonance imaging (MRI) is the current imaging modality of choice for detection and characterization of most CNS lesions affecting the visual pathways. However, little information is available regarding MRI features of visual pathway pathology in domestic animals.

As a result of the intimate association of the visual pathways with other intracranial structures, concurrent neurologic deficits have been typically described in dogs and cats with CNS lesions causing vision loss. To the best of our knowledge, postretinal blindness as the only neurologic deficit in domestic animals has been reported infrequently.

The purpose of this study is to describe the ophthalmologic, neurologic and MRI findings of seven domestic animals (dogs and cats) with acute postretinal blindness as sole neurologic deficit.

MATERIALS AND METHODS

Medical records from animals seen between January 2006 and December 2008 at the Veterinary Teaching Hospital of the Autonomous University of Barcelona (VTH-UAB) were reviewed. Dogs and cats with postretinal blindness of acute presentation, and an MRI performed as part of the diagnostic workup were identified. Among these, only animals lacking other neurologic signs at the time of initial evaluation were included in the study.

Each animal had complete physical, ophthalmic, and neurologic examinations. Ophthalmic examination included assessment of the palpebral reflex, tests of vision (menace response, cotton ball test, visual placement response, and maze test), pupillary light and dazzle reflexes, Schirmer tear test I, slit-lamp biomicroscopy, applanation tonometry, and indirect ophthalmoscopy.

All animals had a complete blood cell count (CBC), complete serum biochemistry, urinalysis, thoracic radiographs, abdominal ultrasound, short protocol ERG, and cranial MRI performed. MR images of the brain were obtained with a 0.2 T permanent magnet (Esaote Vet-MR unit, Esaote Biomedica, Genova, Italy). The MRI protocol consisted of sagittal, transverse and dorsal T2-weighted images (T2WI), and precontrast and postcontrast T1-weighted images (T1WI). Postcontrast T1WI were obtained immediately after intravenous administration of gadolinium dimeglumine (Dotarem; Guerbet Sa, Madrid, Spain) at 0.2 mmol/kg. Short tau inversion recovery (STIR) and fluid-attenuated inversion recovery (FLAIR) images were obtained as dictated by the case. The acquisition parameters were the following: T1WI spin echo (SE), repetition time (TR) = 500 ms, and echo time (TE) = 20 ms; T2WI SE, TR/TE = 2800/80 ms. Slice thickness ranged from 3 to 4 mm.

The following MRI characteristics were assessed: topographic location, axial origin (intra-axial, of nervous tissue origin; extra-axial, originating in non-nervous tissue), appearance (focal or multifocal), shape, signal intensity and homogeneity, degree of contrast enhancement, presence of mass effect, and miscellaneous features such as presence of peritumoral edema, cyst formation, dilation of ventricles, and presence of extracranial lesions.

Results of CSF analysis, serological tests, and polymerase chain reaction (PCR) for several infectious diseases in blood and CSF, and histopathologic studies were recorded when available. CSF was collected from the cerebellomedullary cistern. Total nucleated cell count (TNCC), total red blood cell count (TRCC), differential nucleated cell count, and total protein concentration were determined in all CSF samples. Histologic specimens were obtained postmortem, fixed in 10% neutral-buffered formalin, embedded in paraffin, sectioned at 5 μm, and stained with hematoxylin and eosin. Histochemical and immunohistochemical stainings were performed as dictated by the case. In these animals, presumptive and definitive diagnoses (histologically confirmed) were correlated.

RESULTS

Seven animals (four dogs and three cats) of different breeds and genders met the inclusion criteria (Table 1). The median age was 7.5 years for dogs (range 2–12 years) and 7.3 years for cats (range 3–10 years).

In all cases, the owners reported an acute onset of vision deficiency with rapid progression to complete and permanent blindness. In no case, the initial vision deficiency was related to scotopic/photopic conditions or to unfamiliar surroundings. Although blindness was the only sign reported in all animals at presentation, two cases had a previous history of systemic illness (Table 1; cat 1 and dog 5). Cat 1 had a history of sneezing and nasal discharge of several months duration that had improved with oral enrofloxacin (5 mg/kg SID) and metronidazole (15 mg/kg BID). Dog 5 was diagnosed with leishmaniasis 1 year prior to blindness onset and was currently on treatment with allopurinol (10 mg/kg BID PO). However, the patient was sero-negative to Leishmania infantum at presentation for blindness. Apart from these two patients, none of the other animals was receiving any topical or systemic medication.

Physical examination was unremarkable in all animals except for cat 3 (Table 1), that had upper respiratory noise and reduced bilateral nostril airflow.

Complete blindness was confirmed in all patients by ophthalmic examination. Anterior and posterior segment evaluation failed to reveal any cause for the vision loss in 5/7 animals. In two patients (dogs 6 and 7), both optic nerve heads appeared swollen, hyperemic, and edematous on funduscopic examination. All animals had absent dazzle reflex, complete or nearly complete afferent pupillary light reflex deficits, and normal ERG (peak amplitude and latency values for a and b waves within the reference range for our laboratory). Based on the neuro-ophtalmic examination and results of ERG, lesions were localized bilaterally in the optic nerve, optic chiasm, or rostral part of the optic tracts.
Table 1. Signalment, MRI features, presumptive diagnosis, and histopathologic diagnosis in seven patients with acute postretinal blindness

<table>
<thead>
<tr>
<th>Patient</th>
<th>Specie</th>
<th>Breed</th>
<th>Age (year)</th>
<th>Sex</th>
<th>Lesion localization</th>
<th>Axial origen</th>
<th>Shape</th>
<th>Signal intensity and regularity</th>
<th>Contrast uptake</th>
<th>Mass effect</th>
<th>Miscellaneous features</th>
<th>Presumptive diagnosis</th>
<th>Histopathologic diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Feline</td>
<td>DSH</td>
<td>3</td>
<td>FS</td>
<td>Nasal cavity with intracranial extension</td>
<td>Extra-axial</td>
<td>Irregular/Plaquelike</td>
<td>T1: Isointense T2: Iso/Hyperintense Heterogeneous</td>
<td>Moderate Homogeneous</td>
<td>No</td>
<td>Nonuniform hyperintensity within both tympanic bullae</td>
<td>Nasal tumor</td>
<td>Nasal carcinoma</td>
</tr>
<tr>
<td>2</td>
<td>Feline</td>
<td>DSH</td>
<td>9</td>
<td>FS</td>
<td>Pituitary fossa</td>
<td>Extra-axial</td>
<td>Regular ovoid</td>
<td>T1: Isointense T2: Iso/Hyperintense Heterogeneous</td>
<td>Mild Homogeneous</td>
<td>Mild</td>
<td>Mild peritumoral edema</td>
<td>Pituitary tumor</td>
<td>Pituitary carcinoma</td>
</tr>
<tr>
<td>3</td>
<td>Feline</td>
<td>Persian</td>
<td>10</td>
<td>MN</td>
<td>Nasal cavity with intracranial extension</td>
<td>Extra-axial</td>
<td>Irregular</td>
<td>T1: Isointense T2: Hyperintense Heterogeneous</td>
<td>Marked Homogeneous</td>
<td>Marked</td>
<td>Marked peritumoral edema</td>
<td>Nasal tumor</td>
<td>Nasal osteosarcoma</td>
</tr>
<tr>
<td>4</td>
<td>Canine</td>
<td>German Shepherd</td>
<td>8</td>
<td>M</td>
<td>Frontal lobe</td>
<td>Extra-axial</td>
<td>Regular</td>
<td>T1: Isointense with hypointense area (cyst) T2: Hyperintense</td>
<td>Marked Homogeneous</td>
<td>Marked</td>
<td>Cystic</td>
<td>Meningioma</td>
<td>Not performed</td>
</tr>
<tr>
<td>5</td>
<td>Canine</td>
<td>Mixed breed</td>
<td>12</td>
<td>M</td>
<td>Olfactory region (olfactory peduncle)</td>
<td>Extra-axial</td>
<td>Irregular</td>
<td>T1: Isointense T2: Hyperintense/Isointense Heterogeneous</td>
<td>Marked Homogeneous</td>
<td>No</td>
<td>Mild peritumoral edema</td>
<td>Meningioma</td>
<td>Meningioma</td>
</tr>
<tr>
<td>6</td>
<td>Canine</td>
<td>Mixed breed</td>
<td>8</td>
<td>M</td>
<td>Optic nerves (intraorbital and intracanalicular part)</td>
<td>Extra-axial</td>
<td>–</td>
<td>T1: Isointense T2: Hyperintense STIR: Hyperintense</td>
<td>Moderate Homogeneous</td>
<td>–</td>
<td>–</td>
<td>Optic neuritis (presumptive GME)</td>
<td>Not performed</td>
</tr>
<tr>
<td>7</td>
<td>Canine</td>
<td>French Bulldog</td>
<td>2</td>
<td>M</td>
<td>1st presentation: unremarkable 2nd presentation: Multifocal; Gray matter and subcortical white matter of both cerebral hemispheres.</td>
<td>Intra-axial</td>
<td>Irregular</td>
<td>T1: Hypo/Isointense T2: Hyperintense</td>
<td>Mild Heterogeneous</td>
<td>No</td>
<td>Mild peritumoral edema</td>
<td>Multifocal brain disease (presumptive GME/EME)</td>
<td>Not performed</td>
</tr>
</tbody>
</table>

FS = spayed female; MN = neutered male; M = intact male; DSH = Domestic Shorthair; MRI = magnetic resonance imaging.
Complete neurologic examination at the time of initial evaluation failed to reveal any other neurologic deficits in all cases.

The results of CBC, serum biochemistry profile, and urinalysis were within reference ranges in all animals, with the exception of two cats, that had a moderate increase in serum protein concentration. Cat 1 had a monoclonal gammopathy (α2 globulin 2.11 g/dL, reference range [ref.] 0.4–0.9 g/dL), and cat 3 had a polyclonal gammopathy (α2 globulin 2.70 g/dL; β globulin 2.67 g/dL, ref. 0.9–1.9 g/dL). Negative enzyme-linked immunosorbent assay results for feline immunodeficiency and feline leukemia virus were obtained in all cats. Thoracic radiographs and abdominal ultrasound were unremarkable in all cases.

The clinically suspected lesions affecting the visual pathways were confirmed by MRI in 6/7 cases. The MRI study of dog 7 was unremarkable at first, but showed multifocal brain lesions not affecting the visual pathways at a second presentation 2 years later. The MRI characteristics of the different lesions are summarized in Table 1. The median time from onset of blindness to performance of the first MRI study was 1.3 days (range 0.5–3 days). Neuroanatomic localization of the lesions included: frontal/olfactory lobe region with involvement of the optic chiasm (4/7), pituitary fossa with involvement of the optic chiasm and optic tracts (1/7), optic nerves (1/7), and cortical gray matter and subcortical white matter of both cerebral hemispheres (1/7). Of all these lesions, five were consistent with intracranial tumors (two meningiomas, one pituitary tumor, and two nasal tumors with intracranial extension), one with acute bilateral optic neuritis, and one with multifocal brain disease.

The two presumptive meningiomas were located on the base of the skull, one affecting the frontal lobe (Fig. 1), and the other the olfactory peduncle (dogs 4 and 5, respectively). Involvement of the optic chiasm was observed in both of them.

Two cats (cats 1 and 3) had irregular masses that originated in the nasal cavity and extended to the rostral brain, affecting the optic chiasm (Fig. 2), and one cat (cat 2) had a pituitary mass involving the optic chiasm and optic tracts (Fig. 3).

Of the two dogs with optic nerve head inflammation on funduscopic examination, only dog 6 had evident lesions affecting the visual pathways in the MRI study. In this case, a hyperintense signal in the intraorbital portion of both optic nerves was observed on T2WI and STIR images. After contrast administration, both optic nerves enhanced moderately, but homogeneously (Fig. 4). These findings were suggestive of acute bilateral optic neuritis.

Cerebrospinal fluid analysis was performed in five animals, and was unremarkable in two. One cat with a pituitary tumor (cat 2) had albuminocytologic dissociation (mild increase in protein concentration [44 mg/dL; ref. <25 mg/dL] with normal TNCC), and one dog with optic neuritis (dog 6) had an elevated TNCC (24 TNCC/µL; ref. <5 TNCC/µL), which consisted of a predominantly lymphocytic population, and a mild increase in protein concentration (43.2 mg/dL). In this dog, results for real-time PCR and reverse transcriptase PCR (RT-PCR) tests in blood and CSF for canine distemper virus, *Toxoplasma gondii*, *Neospora caninum*, and *Ehrlichia canis*, were negative. On the basis of these findings, a presumptive diagnosis of the ocular form of granulomatous meningoencephalitis (GME) was made. CSF analysis of dog 7 at the second presentation...
revealed an elevated TNCC (39 TNCC/µL), consisting of a predominantly eosinophilic cell population and a mild increased in protein concentration (45.5 mg/dL). A serum latex agglutination test for *Cryptococcus neoformans*, and real-time PCR and RT-PCR tests in blood and CSF for canine distemper virus, *Toxoplasma gondii*, *Neospora caninum* and *Ehrlichia canis* were performed with negative results. A fecal flotation and a Baermann test performed to rule out intestinal parasitic diseases were also negative. Differential diagnoses at this time were idiopathic eosinophilic meningoencephalitis (EME) or GME.

In dog 7, although the results of MRI and CSF analysis were unremarkable at first presentation, bilateral optic neuritis was considered based on ophthalmic examination. Serologic tests for *Ehrlichia canis*, *Leishmania infantum*, and *Toxoplasma gondii* were performed by the referring veterinarian with negative results. Empiric treatment with prednisone (1 mg/kg BID PO) was prescribed and visual improvement was observed. Two years later, the dog re-presented for a new episode of acute blindness. Ophthalmic examination

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**Figure 2.** Magnetic resonance (MR) images of a cat with a nasal carcinoma extending into the cranial cavity (cat 1). The lesion is isointense and ill defined on transverse T1WI (a). The transverse postcontrast T1WI (b) shows a contrast-enhancing mass at the level of the optic chiasm (arrows). The sagittal postcontrast T1WI (c) shows an irregular, contrast-enhancing mass originating in the nasal cavity and extending to the rostral brain through the cribriform plate in a plaque-like grow pattern (arrows).

**Figure 3.** Magnetic resonance (MR) transverse images at the level of the optic chiasm in a cat with a pituitary carcinoma (cat 2). The lesion is isointense and ill defined on T1WI (a). Postcontrast T1WI (b) shows a homogeneous, contrast-enhancing mass in the pituitary fossa (arrow). The lesion (arrow) is hyperintense and heterogeneous on T2WI (c). Mild peritumoral edema is present.
confirmed complete blindness and funduscopic evaluation revealed moderate papillitis in both eyes. The results of ERG were within the reference range. Complete neurologic examination revealed this time behavioral abnormalities, and signs of depression and disorientation.

Therapeutic options offered to patients with intracranial tumors included surgical intervention (complete excision, partial debulking or biopsy), radiation therapy, chemotherapy, and palliative therapy with glucocorticoids. Among these patients, two cats were euthanized immediately after diagnosis, and two dogs and one cat received palliative treatment with prednisone (0.5–1 mg/kg BID PO). Both dogs with meningoencephalitis received immunosuppressive glucocorticoid therapy (1 mg/kg BID PO) for 2 months, with subsequent tapering of the dose along time according to clinical signs and results of CSF analysis.

A good response was observed in one case of meningoencephalitis (dog 7). At the time of publication, 3 years after the first presentation, the dog had decreased vision in both eyes and no other neurologic signs. The remaining four animals developed progressive neurologic signs with time. Of these, three were euthanized and one died.

Definitive diagnoses were obtained by postmortem histopathologic examination in four cases (Table 1). Anatomic location and diagnosis matched grossly with the ones performed during the animals’ life. Neoplasia was diagnosed in all of them, and final diagnoses included one meningioma, one pituitary carcinoma, one nasal osteosarcoma, and one nasal carcinoma.

**DISCUSSION**

Vision loss with lack of significant ocular lesions and normal ERG is indicative of a process located anywhere along the afferent visual pathway (postretinal blindness). Although routine diagnostic procedures, such as neurologic examination, routine laboratory evaluations, and CSF analysis might be of help establishing the most likely localization and etiology of CNS lesions, advanced imaging techniques are necessary to confirm the exact location and achieve a presumptive diagnosis.1,2 MRI is the diagnostic imaging modality of choice in human medicine for most CNS diseases affecting the visual pathways. MRI provides superior resolution of retrobulbar and intracranial visual pathway lesions compared to computed tomography (CT), and enables a focused examination of the entire visual pathway along its course, from the inner aperture of the optic canal to the occipital lobe.3,4 Several reports describe the findings on standard and advanced MRI images of diseases affecting the globe and visual pathways in humans, such as neoplasms, inflammatory/infectious conditions, vascular, congenital and degenerative disorders. Based upon their location, shape, invasiveness, signal intensity and homogeneity, and enhancement properties, it has often been possible to predict the etiology or malignancy of certain CNS lesions that cause visual deficits or complete blindness as primary clinical sign.3,4,17–24 In the veterinary literature, little information is available regarding the MRI features of visual pathway lesions.5–11

In this study, lesions affecting the visual pathways were confirmed at presentation by MRI in six of the seven reported patients. In all of them, MRI enabled an accurate and detailed description of the morphology of the lesions. Lesion localization and extension was in accordance with the neuro-ophthalmic dysfunction in all animals, and with the anatopathological results in patients in which a postmortem study was performed. In all these cases, the MRI features of the lesions allowed differentiation between neoplastic and inflammatory disorders, and identified the tumoral and inflammatory secondary effects.

Based on similarities with the MR images of previously reported cases,5,9,25–28 two masses involving the optic chiasm were considered meningiomas, and one of them was histopathologically confirmed. These tumors have been previously described as extra-axial lesions, isointense on T1WI and hyperintense or isointense on T2WI, which enhance markedly and homogeneously after contrast administration. Mass effect is usually present and edema varies widely from absent to extensive.29 A previous report of an intracranial meningioma causing blindness as sole neurologic deficit was described in a cat.9 The tumor was located in the rostral thalamus and extended to compress the optic nerves and chiasm. MRI findings in this cat were similar to the ones of the two cases here described.

The imaging findings of the pituitary tumor in this report were similar to those described in cats and dogs by others.26,27,29 These tumors usually present as focal masses in

the region of the pituitary gland, they are isointense on T1WI, and can present variable intensities on T2WI. Peritumoral edema is commonly absent to moderate. Contrast enhancement ranges from minimal to strong, but it is usually uniform, although heterogeneous enhancement can be found in some cases.

Even though neuro-ophthalmic signs are frequently seen in human beings with pituitary tumors, they are rare in animals.19–21 This difference has been attributed to the fact that the pituitary stalk of domestic animals is directed caudoventrally rather than rostroventrally, as in human beings.30

According to previous studies, cats with pituitary tumors had a significantly higher incidence of visual deficits than dogs (35.7% and <10%, respectively).33,34 Individual differences in the orientation of the pituitary neoplasm or subtle individual differences in hypothalamic/optic chiasm proximity have been proposed to explain the involvement of the visual pathways in animals with pituitary tumors,14 and may explain the visual deficits in the cat of this report.

The two nasal tumors of this report were irregular masses that invaded the rostral brain lobes. They originated in the caudal part of the nasal cavity, crossed the ethmoidal turbinates, and penetrated the cranial cavity, eventually reaching the optic chiasm. The signal intensity and contrast uptake of these tumors were similar to those previously described in cats and dogs.27,35–37 The fact that blindness was the only neurologic abnormality in these two cases is in contrast with the neurologic findings commonly reported in small animals with tumors of the nasal cavity that invade the brain.37,38 Although blindness as sole neurologic deficit was previously reported in a dog with a nasal tumor,5,14 to the author’s knowledge, these are the first reports of blindness as the only neurologic sign in cats with nasal tumors and intracranial extension.

Two cases of bilateral optic neuritis were seen in association with meningoencephalitis. For both of them, the diagnosis of optic neuritis was based on funduscopic examination findings. An MRI study of the brain was performed in each case to look for subclinical brain lesions, such as those related to meningoencephalitis or neoplasia. Hyperintensity on T2WI and STIR sequences and moderate contrast enhancement of both optic nerves were the only abnormalities detected in one case. These MRI changes were considered consistent with acute inflammation of the optic nerves. In veterinary medicine, little information is available about the MRI features of optic nerve pathology.6,9–11 In human beings, a hyperintense signal of the optic nerves on T2WI is a nonspecific sign, as several lesions such as edema, demyelination and axonal loss, as well as chronic gliosis can cause similar changes. However, contrast enhancement reflects breakdown of the blood–brain barrier, and therefore characterizes acute inflammation.4,17 In addition, analysis of the CSF in this patient revealed a lymphocytic pleocytosis. Potential causative infectious diseases were ruled out by determination of serum/CSF antibody titers or by means of infectious DNA detection by PCR. Based on negative results of all these tests, a presumptive diagnosis of ocular GME was made.

The MRI study and CSF analysis of the second optic neuritis case were unremarkable at first presentation. At that time, inflammatory/infectious diseases were considered the most likely cause of optic neuritis. Because further ancillary tests were declined by the owners, a definitive diagnosis could not be reached. However, based on the fact that the animal regained vision after receiving systemic corticosteroids, an inflammatory etiology for the optic neuritis was suspected. When the dog re-presented 2 years later, MRI of the brain revealed multifocal lesions unrelated to the visual pathways. These findings were thought to be consistent with a multifocal CNS inflammatory or neoplastic disease. CSF analysis revealed an eosinophilic pleocytosis. Although the cell population was typical of that seen in parasitic, protozoal, mycotic, and immune-mediated meningoencephalitis, other infectious agents were also investigated. After ruling out infectious diseases, the main differential diagnosis was idiopathic EME or GME. In this case, even though the MRI findings were not in accordance with the neuro-ophthalmic dysfunction, the imaging findings and the additional ancillary tests provided essential information to reach a diagnosis. Despite the fact that both presentations of optic neuritis could be caused by the same disease at different stages, the possibility of different diseases causing the same or similar clinical signs at different times should be also taken into account.

Central nervous system disease should be considered in dogs and cats with acute blindness, even when no other neurologic deficits are present. This study emphasizes the relevance of MRI as a diagnostic tool for detection and characterization of CNS lesions involving the visual pathways. MRI provided essential information to reach a definitive diagnosis, to treat and to give a prognosis in seven animals with acute postretinal blindness.

REFERENCES


