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What Is Your Neurologic Diagnosis?

A 1.5-year-old 3.4-kg (7.5-lb) neutered male domestic shorthair cat was evaluated because of sudden onset of bilateral blindness. Four days earlier, the cat had become lethargic and was examined by the referring veterinarian. At that time, the cat was febrile; performance of a diagnostic evaluation was offered but declined by the owners. Treatment was initiated with amoxicillin-clavulanic acid, which the owners discontinued 2 days later. The cat had been found as a stray and was kept indoors. The owners had recently moved to Florida from Illinois. The cat had no notable medical problems previously and no known exposure to toxins. Another cat in the household was apparently healthy. Prior to the evaluation at the referral hospital, the patient had been anorectic for 2 days and was examined because of worsening lethargy, signs of depression, and blindness in both eyes.

Physical examination revealed that the cat had depressed mentation and was dehydrated (approx 5% to 7%); rectal temperature was 38.8°C (101.9°F). The cat had signs of mild depression and was disoriented. Body condition score was 4/9. Bilaterally, the cat had elevation of the third eyelids, no menace responses, mydriasis, and decreased pupillary light reflexes (left eye more severely affected). Mild decreased facial sensation on the right side, horizontal inducible nystagmus with the fast phase to the left, right-sided head tilt, and mild decrease in conscious proprioception on the right side were also noted. Uveitis was evident bilaterally. Thoracic and cardiac auscultation and abdominal and lymph node palpation revealed no additional abnormalities.

Neurologic examination

Observation

Mental	Alert		Depressed	X	Disoriented	X	Stupor		Coma		
Posture	Normal		Head tilt	X	Tremor		Falling				
Gait	Normal		Ataxia	X	Pelvic limbs		All 4		Circling		
Paresis	Pelvic limbs		Tetra		Hemi	X	Mono				
Other											

Key: 4 = exaggerated, clonus; 3 = exaggerated; 2 = normal; 1 = diminished; 0 = none; NE = not evaluated

Postural reactions

	LF	RF	LR	RR
Wheelbarrow	NE	NE		
Hopping	2	1	2	1
Ext postural thrust			2	1
Proprioceptive pos	2	1	2	1
Hemistand/walk	NE	NE	NE	NE
Placing–tactile	NE	NE		
Placing–visual	NE	NE		

Spinal reflexes

	LF	RF	LR	RR
Quadriceps			2	2
Extensor carpi	2	2		
Flexion	2	2	2	2
Crossed extensor	NE	NE	NE	NE
Perineal			2	2

Cranial nerves

	L	R		L	R
II, VII–Vision menace	0	0	VIII–Nystagmus, resting	0	0
II, III–Pupils resting	3	3	VIII–Nystagmus, change	3	3
Stim L	1	1	V–Sensation	2	1
Stim R	1	1	VII–Facial mm	2	2
II–Fundus	NE	NE	V, VII–Palpebral flex	2	2
III, IV, VI–Strabismus, resting	0	0	IX, X–Gag	NE	NE
III, IV, VI, VIII–Strabismus, position	0	0	XII–Tongue	2	2

Comments CN

Mydriasis was present bilaterally. The fundus of each eye could not be evaluated because of uveitis.

Sensation (Locate and describe abnormal)

Hyperesthesia	3	Reluctance to manipulate neck
Superficial pain	2	
Cutaneous reflex	2	
Deep pain	2	

What is the problem? Where is the lesion? What are the most probable causes of this problem? What is your plan to establish a diagnosis? Please turn the page.

Assessment

Anatomic diagnosis

Problem	Rule out location
Altered level of consciousness	Cerebrum, thalamus, or brainstem
Partial blindness with intact pupillary light reflex	Thalamus or cerebrum
Decreased sensation in the right maxilla	Right maxillary branch of trigeminal nerve or left side of cerebrum
Head tilt	Brainstem, cerebellum, or cranial nerve VIII
Bilateral elevation of third eyelid	Spinal cord (T1-T3 segment), midbrain, and vagosympathetic trunk (Horner's syndrome) or other body location (ie, systemic illness [Haw's syndrome])
Positional nystagmus	Brainstem, cerebellum, cranial nerve VIII, or cerebrum with secondary pressure on central vestibular apparatus
Mild right-sided postural reaction deficits	Left side of cerebrum, right side of brainstem, or right side of cervical spinal cord

Likely location of one lesion

Multifocal intracranial location

Etiologic diagnosis—Rule out disease processes included infectious CNS disease (feline infectious peritonitis or infection with *Toxoplasma* spp, *Cryptococcus* spp, or other fungal organisms), neoplasia (juvenile lymphoma), inflammatory CNS disease, nutritional deficiency (thiamine), or metabolic disease (liver failure). The diagnostic plan included performing a CBC and serum biochemical analyses (to assess WBC count and metabolic changes), evaluation of a CSF sample (to detect inflammatory disease, neoplastic cells, or infectious organisms), analysis of a CSF sample via PCR assay (to detect coronavirus indicative of possible feline infectious peritonitis), assessment of titers of serum antibodies against *Toxoplasma* or *Cryptococcus* spp, measurement of serum creatine kinase activity (to detect muscle inflammation), and magnetic resonance imaging of the head (to identify structural intracranial abnormalities).

Diagnostic test findings—Abnormalities revealed by a CBC and serum biochemical analyses included high concentrations of total protein (8.6 mg/dL; reference range, 6 to 8 mg/dL), albumin (3.6 mg/dL; reference range, 2.3 to 3.5 mg/dL), and globulin (5.0 mg/dL; reference range, 2.8 to 4.8 mg/dL). Results of an analysis of a sample of CSF were within reference limits.

Serologic testing indicated that the cat had no circulating anti-coronavirus antibodies; the cat was seronegative for anti-*Toxoplasma* IgM but had anti-*Toxoplasma* IgG (titer, 1:5,120). Results of a coronavirus-specific PCR assay were negative. Serum creatine kinase activity was 345 U/L (reference range, 50 to 310 U/L). Magnetic resonance imaging was declined by the owners because of financial constraints.

Comments

On the basis of the cat's serum antibody titers and clinical signs, a presumptive diagnosis of *Toxoplasma* encephalitis was made. Cats are the definitive host of *T gondii*. Seroprevalence is high in homeless cats and is estimated to be approximately 30% in cats in the United States.[3] Although many cats are positive for antibodies against *T gondii*, it is unknown why most of these infected cats remain clinically unaffected. However, immunosuppression can lead to reactivation of encysted *T gondii* bradyzoites.[3] Common neurologic signs of infection include ataxia, circling, behavioral changes, seizures, twitching, and tremors. Neurologic and ocular disease without other systemic involvement is more common with reactivated infection.[3] Given the lack of serum anti-*Toxoplasma* IgM and the high anti-*Toxoplasma* IgG titer detected in the cat of this report, an acute exposure to *T gondii* was ruled out and reactivation of encysted bradyzoites was suspected; however, serial titer assessments would have to have been performed to confirm that assumption. Although many cats with toxoplasmosis have serum biochemical changes, the only detected abnormality in the cat of this report was hyperglobulinemia, which can be an indicator of chronic infection. In addition, most animals with *Toxoplasma* encephalitis have increased protein concentration and detectable pleocytosis in CSF samples; these findings were not evident in this patient. A tentative diagnosis is made on the basis of increasing serial anti-*Toxoplasma* IgG titers, exclusion of other causes of encephalitis, and response to treatment. Definitive diagnosis requires detection of the organism in body tissue or fluid.[3] With regard to prognosis, clinical signs usually begin to abate within 24 to 48 hours after treatment with clindamycin is initated.[3] Full recovery may take weeks, but signs may never fully resolve. Some animals require lifelong treatment with antimicrobials to prevent reinfection.

The cat of this report was hospitalized, and fluid therapy with a balanced electrolyte solution (2× maintenance rate) was initiated. Clindamycin (12.5 mg/kg [5.68 mg/lb], PO, q 12 h) and prednisolone (1 mg/kg

[0.45 mg/lb], PO, q 12 h) for 3 days were administered. The cat's appetite and mentation improved during hospitalization. The head tilt remained, but the menace responses, pupillary light reflexes, and mydriasis improved in both eyes. The cat was discharged from the hospital, and the owners were instructed to administer clindamycin (12.5 mg/kg, PO, q 12 h) for 28 days. Two days after discharge from the hospital, the owners reported that the cat had become lethargic and its appetite had decreased. Treatment with prednisolone was reinstituted (0.5 mg/kg [0.23 mg/lb], PO, q 12 h). Two days later, the cat's condition had not improved and vestibular signs were observed by the owners. The owners again declined magnetic resonance imaging and further hospitalization; administration of meclizine hydrochloride (3.7 mg/kg [1.7 mg/lb], PO, q 24 h) was instituted. Three days later, the cat was returned to the hospital in severe respiratory distress; the patient was then euthanatized at the owners' request.

A necropsy was performed, and examination of the brain revealed a soft, white-tan discolored area in the right parietal lobe of the cerebral hemisphere. Histologic examination revealed severe pyogranulomatous inflammation in this area with necrosis and the presence of a thick-walled yeast. Malacia was present within the lesion and elsewhere in the right parietal lobe. No protozoal cysts were identified. Periodic acid–Schiff staining of sections of affected tissue revealed that the yeast had double cell walls and underwent broad-based budding. The histopathologic findings were consistent with *Blastomyces dermatitidis* encephalitis. *Blastomyces* infections in cats are rare.[4] Involvement of the CNS occurs with disseminated disease.[4] In the cat of this report, no other signs of systemic involvement were detected. Systemic disease may also be diagnosed if fungal hyphae are detected in urine samples. For this cat, urinalysis or other antemortem testing that may have identified a mycotic infection was not performed.[4]

References

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