

Hydrocephalus with Visual Deficits in a Cat

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ABSTRACT. A 22-month-old male Japanese mongrel cat with a history of dysuria and recurrent generalised tonic-clonic seizure was examined by neuro-ophthalmological testing and computed tomography (CT). Vision testing revealed narrowing of the visual field in the right eye, and complete visual deficits in the left eye. Pupillary reactions, and motor and sensory function in the eyelids and the eyes were normal. The cat was diagnosed as hydrocephalus by CT examination, because dilation of the right lateral ventricle, and compression of the right temporal and occipital cortices was shown. The etiology of the hydrocephalus was unclear. Although a unilateral lesion of the upper visual pathway was suspected, a complete homonymous hemianopsia was not shown.

KEY WORDS: computed tomography, feline, hydrocephalus with visual deficits.

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Hydrocephalus is the abnormal accumulation of cerebrospinal fluid (CSF) within the ventricular system, and may be subclassified into acquired or congenital forms based on the nature of the causative disease [14]. The clinical signs of hydrocephalus, irrespective of the cause, reflect the anatomical level of the lesions in the brain. For example, if the forebrain is involved, decorticate activities such as pacing, aimless walking, purposeless activity, restlessness, head pressing, marked behavioural changes, and seizures can occur [5, 14]. If the brainstem or cerebellar structures are compromised, gait may be affected [5, 14]. Abnormalities of the eyes and vision, such as strabismus or blindness, may be evident. Strabismus is a clinical sign of not only vestibular disease in animals but also hydrocephalus in humans and animals [9]. Blindness can also be a clinical symptom of hydrocephalus or hydranencephaly in animals [4, 9]. Although visual field testing may be important for lesion localization [10], assessment of the visual field in animals is difficult. This article describes the clinical findings in a cat with hydrocephalus together with details of visual testing.

A 22-month-old male Japanese mongrel cat weighing 4.0 kg was referred to Kagoshima University Animal Hospital with a history of dysuria, and generalised tonic-clonic seizure. The first seizure was observed at 7 months of age, and thereafter recurred every two or three months. The duration of the ictal period was only a few minutes, and no post-ictal period was observed. The client mentioned that the cat showed a visual deficit when a hand was placed in front of the cat's left eye. Routine hematological parameters including a complete blood count and serum chemistry were found to be within the normal ranges. Serum samples were negative for feline infectious peritonitis (FIP), feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), and toxoplasmosis.

A detailed neuro-ophthalmological examination was carried out. Vision testing included the ability to negotiate an

obstacle course, the cotton wool ball test, and the menace response [10]. Prior to the obstacle course test, the cat was sufficiently acclimated to the laboratory room and a custom-made eye bandage. When the right eye was covered with the bandage, the cat did not walk spontaneously and often crouched. On the other hand, without the eye bandage of the right eye, and with or without coverage of the left eye, the cat walked around the laboratory room spontaneously. The menace test was performed for each eye while the contralateral eye was covered with the eye bandage. According to Beaver [2], the visual field in cats is 155–208.5 degrees for each eye, and 90–130 degrees for binocular vision. Therefore, the lateral visual field was tested around the ipsilateral auricle, and the medial field was tested around the medial angle of the ipsilateral eye separately while the contralateral eye was covered. The cotton wool ball test was also carried out at the same positions. Table 1 shows the results of vision testing. Both the menace and cotton wool ball tests revealed that all visual fields in the left eye were totally deficient. However, the result for the right eye was not simple, the lateral visual field was normal, but the medial field was complicated. Blink reflex was often absent in the medial field of the right eye, and the cat often did not follow the cotton wool ball when it was dropped in front of the medial visual field. Thus, narrowing of the visual field in the right eye was suggested. No other abnormal findings were observed for either pupillary reactions (pupil size, pupillary light reflex (PLR), the swinging flashlight test, cover test) or

Table 1. Results of vision testing and PLR

	Left eye	Right eye
Medial visual field	defect	occasional defect
Lateral visual field	defect	normal
Obstacle course*	frequent crouching	normal
PLR	normal	normal

* when the contralateral eye was covered by an eye bandage.

** PLR : pupillary light reflex (both direct and indirect).



Fig. 1. Precontrast (a) and postcontrast (b) coronal images at the level of upper line of the zygomatic arch. a, b: The right lateral ventricle was dilated, and the right temporal and occipital cortices were compressed (arrow). b: A slight contrast enhancement was seen in the dilated right lateral ventricle (arrow head), but the enhancement was not homogenous and well-demarcated.

motor and sensory function in the eyelids and eyes.

A survey radiograph of the skull showed no abnormal findings. Computed tomography (CT) examination was performed under general anesthesia, and continuous 3-mm-thick coronal noncontrast scans were obtained (HITACHI 950SR). Following bolus intravenous injection of an iodinated contrast material, Iopamidol (Iopamiron 150, Nihon Schering, Japan, 2 ml/kg), the scan procedure was repeated immediately. By both noncontrast and contrast scanning, the right lateral ventricle appeared dilated, and the right temporal and occipital cortices were compressed (Fig. 1a and 1b). No mass effect such as a midline shift was identified.

A slight contrast enhancement was seen in the dilated right lateral ventricle, indicating the probable site of the choroid plexus (Fig. 1b).

Hydrocephalus may be subclassified as congenital or acquired [14]. The former is a condition in which the animal is born with typical clinical signs. On the other hand, the acquired form usually has an obvious cause, and its onset may not always be clear [9, 14]. Most cases of hydrocephalus seen in veterinary practice are congenital, and both genetic and environmental factors are involved [9]. Hereditary hydrocephalus in Siamese cats is transmitted as an autosomal recessive trait [13]. Environmental factors include

brain damage at birth, in utero or perinatal infection with a variety of viruses, toxoplasma and possibly mycoplasma [14]. In cats, infection with the FIP and FPL viruses has been associated with the development of hydrocephalus or hydranencephaly in the neonate [4, 8, 16]. Exposure to a number of potentially teratogenic drugs or chemicals at particular stages of gestation also results in congenital hydrocephalus [6, 14]. The diagnosis of hydrocephalus in young animals is relatively certain if the characteristic clinical signs such as large and rounded head, persistent fontanella, obvious neurologic abnormalities (seizure or episodic behavioral changes) are evident [9]. In cats, congenital hydrocephalus is associated with abnormal skull shape [1]. On the contrary, hydrocephalus in the adult animal with less severe involvement may produce a more subtle clinical signs, and ancillary tests are necessary to confirm the diagnosis [9]. The electroencephalograph (EEG) is one of the non-invasive and specific ancillary diagnostic test in dogs less than 18 months of age, but not as specific in older or mildly affected dogs [14]. Recently CT and ultrasonography through the open fontanelles, are the most accurate, least invasive and the best diagnostic tests [9, 14]. Although EEG was not recorded in this case, the dilation of the right lateral ventricle was evident by the CT examination. This asymmetrical dilation indicates an increase in the volume of CSF in the right lateral ventricle, which imply hydrocephalus as tentative diagnosis.

The asymmetrical dilation of the lateral ventricles is suggestive of intraventricular obstruction at the level of the ipsilateral interventricular foramen in dogs [12]. Obstruction can result from tumor, granuloma, hemorrhage or inflammation [5]. In dogs, choroid plexus tumors were well-demarcated, hyperdense masses with well-defined margins and marked, homogenous contrast enhancement [17]. Plummer *et al.* have indicated that asymmetry of the ventricles without falx deviation was shown in a cat with non-suppurative meningoencephalitis [11]. Feline cerebral vascular disease may cause unilateral lesions in optic radiation and visual cortex [15], and ischaemic encephalopathy also causes hydrocephalus in cats by a compensatory increase of the CSF volume which fills the space resulting from tissue destruction [1]. In the present study, a mass effect, homogenous contrast enhancement and inflammatory or infarction findings were not apparent on CT scans. However, a neonatal event could not be excluded, because a neonatal event, presumably intraventricular hemorrhage, may cause progressive dilation of the ventricles for about two years in Maltese dogs [14].

Serum testing for FIP, FIV, and FeLV infections and toxoplasmosis was also negative in this case. However, such testing does not exclude an infectious cause of hydrocephalus in cats, especially the neurologic form of FIP. Kline *et al.* evaluated 24 cats with histopathologically confirmed FIP with neurologic involvement in a retrospective study [7]. They rechecked the serum titers for FIP in 11 cats, and four showed negative titers (less than 1:200). Thus the value of the titer has been questioned as a diagnostic tool for the

detection of neurologic FIP [7].

The visual pathways include the optic nerve, optic chiasm, optic tract, lateral geniculate nucleus, optic radiation and occipital visual cortex [3, 10]. The degree of vision loss depends on the extent of the lesion in these visual pathways. A prechiasmal lesion can cause vision loss in the ipsilateral eye, and direct PLR and the consensual PLR from the affected eye is absent in humans as well as in domestic animals [10]. However, the higher region of the visual pathway from the optic chiasm may give rise to differences in clinical signs between humans and domestic animals. Hemianopsia is caused by lesions in this area in humans [18]. Heteronymous hemianopsia is caused by a lesion in the optic chiasm, and homonymous hemianopsia by hemilateral lesions in the optic tract, lateral geniculate nucleus, optic radiation and the occipital cortex. In domestic animals, however, visual field deficits associated with optic tract or higher visual pathway lesions are observed mainly in the contralateral eye, because the degree of decussation of optic nerve fibers at the optic chiasm is higher in animals [3, 10]. This is referred to as a hemianopsia in animals because there is a visual deficit in 50 percent of the total visual field [3]. The present case showed complete visual deficit in the left eye and occasional defect of the visual field in the right eye, but normal pupillary right reflex in both eyes. Normal pupillary responses are common with hydrocephalus because of damage to the optic radiation and the occipital cortex [9]. On the contrary, the extent of the visual deficits was complicated. The examiner applied the cotton wool ball test and the menace response when the optic axis in the cat was fixed close to a line parallel to the sagittal plane. Furthermore the test was applied repeatedly until reproducibility was confirmed; nevertheless the deficit of all visual field in the left eye, and occasional deficit of the medial visual field in the right eye was observed. These findings indicate the possibility that hemilateral lesion(s) at the lateral geniculate nucleus, optic radiation or occipital cortex in cats may cause visual deficits over 50% of the total visual fields. Although the degree of decussation in optic nerve fibers in cats is 65% [3], which may correspond to the same extent as the visual deficits, quantitative assessment of the visual field was difficult. To verify this assumption, improvement of the vision test, for both visual acuity and motion vision, may be necessary to obtain reliable and detailed visual field data in animals with lesions at various visual pathways.

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REFERENCES

1. Barnett, K.C. and Crispin, S.M. 1998. pp. 169–183. *In: Feline Ophthalmology.*
2. Beaver, B.V. 2003. pp. 15–62. *In: Feline Behavior: A Guide for Veterinarians.* WB Saunders Co., Philadelphia, U.S.A.
3. De Lahunta, A. 1983. Visual system—special somatic afferent system. pp. 279–303. *Veterinary Neuroanatomy and Clinical Neurology.* 2nd ed., WB Saunders Co., Philadelphia, U.S.A.

4. Greene, C.E., Gorgacz, E.J. and Martin, C.L. 1982. *J. Am. Vet. Med. Assoc.* **180**: 767–768.
5. Harrington, M.L., Bagley, R.S. and Moore, M.P. 1996. *Vet. Clin. North Am.* **26**: 843–856.
6. Hopkins, A.L. 1995. pp. 219–232. *In*: Manual of small animal neurology. 2nd ed. (Wheeler, S.J. ed.). British Small Animal Veterinary Association, Gloucestershire, UK.
7. Kline, K.L., Joseph, R.J. and Averill, D.R. 1994. *J. Am. Anim. Hosp. Assoc.* **30**: 111–118.
8. Krum, S., Johnson, K. and Wilson, J. 1975. *J. Am. Vet. Med. Assoc.* **167**: 746–748.
9. Oliver, Jr, J.E., Lorenz, M.D. and Kornegay, J.N. 1997. pp. 274–312. *In*: Handbook of Veterinary Neurology. WB Saunders Co., Philadelphia, U.S.A.
10. Petersen Jones, S.M. 1995. pp. 125–142. *In*: Manual of small animal neurology. 2nd ed. (Wheeler, S.J. ed.). British Small Animal Veterinary Association, Gloucestershire, UK.
11. Plummer, S.B., Wheeler, S.J., Thrall, D.E. and Kornegay, J.N. 1992. *Vet. Radiol. Ultrasound* **33**: 307–312.
12. Rivers, W.J. and Walter, P.A. 1992. *J. Am. Anim. Hosp. Assoc.* **28**: 333–343.
13. Silson, M. and Robinson, R. 1969. *Vet. Rec.* **84**: 477.
14. Simpson, S.T. 1989. pp. 842–846. *In*: Current Veterinary Therapy X. (Kirk, R.W. ed.). WB Saunders Co., Philadelphia, U.S.A.
15. Slatter, D. 1990. Neuro-ophthalmology. pp. 437–477. *In*: Fundamentals of Veterinary Ophthalmology. WB Saunders Co., Philadelphia, U.S.A.
16. Tani, K., Taga, A., Itamoto, K., Iwanaga, T., Une, S., Nakaichi, M. and Taura, Y. 2001. *J. Vet. Med. Sci.* **63**: 1331–1334.
17. Turrel, J.M., Fike, J.R., LeCouteur, R.A. and Higgins, R.J. 1986. *J. Am. Vet. Med. Assoc.* **188**: 851–856.
18. Van Allen, M. and Rodnitzky, R. 1981. pp. 92–95. *In*: Pictorial Manual of Neurologic Tests. 2nd ed, Chicago, U.S.A.