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Acute blindness in a dog with *Acinetobacter*-associated postencephalitic hydrocephalus

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ABSTRACT. A 10-month-old male Welsh Corgi with a history of acute blindness underwent neuro-ophthalmological testing and magnetic resonance imaging (MRI). Vision testing revealed complete visual deficits but the electroretinograph and pupillary light reflex were normal in both eyes. The motor and sensory functions of the eyelids and eyes were also normal. The MRI revealed compression of the optic chiasm caused by severe ventriculomegaly in the lateral and third ventricles. Such lesions are associated with inflammatory stenotic lesions in the mesencephalic aqueduct. Moderate neutrophilic pleocytosis was observed during cerebrospinal fluid analysis and *Acinetobacter lwoffii* was isolated, leading to a diagnosis of *Acinetobacter*-positive obstructive hydrocephalus. This is the first reported case of culture-proven *Acinetobacter*-associated postencephalitic hydrocephalus with acute blindness in a dog.

KEY WORDS: bacterial infection, canine, MRI, ventriculomegaly, visual deficit

Hydrocephalus is the abnormal accumulation of cerebrospinal fluid (CSF) in the brain or cranial cavity [17]. Based on the nature of the causative disease, hydrocephalus is classified as either compensatory or obstructive [14]. In compensatory hydrocephalus, a disease process destroys the brain parenchyma and the CSF volume increases to compensate for this loss [17]. In obstructive hydrocephalus, a disease causes obstruction in the flow of the CSF or absorption of the CSF, causing the CSF pressure to increase and the ventricular system to expand. This type of hydrocephalus can be sub-classified as either congenital or acquired [14, 17]. The congenital form is a condition in which the animal is born with typical clinical signs. On the other hand, the onset of the acquired form may not always be clear, but it usually has obvious causes, such as neoplasia, vitamin A deficiency, occipital bone hypoplasia, or inflammation leading to an obstruction [10, 14].

Inflammatory diseases that involve the ependyma at the narrower components of the CSF flow pathway may commonly cause mesencephalic aqueductal obstruction and result in enlarged third and lateral ventricles [13]. In relation to this, feline infectious peritonitis viral infection in cats and bacterial meningoencephalitis in young farm animals had been reported [17]. Although periventricular, choroidal, and suppurative meningeal inflammation suspected to be of bacterial origin have been associated with hydrocephalus in 6- to 8-week-old dogs, this association has never been proven [11, 17]. This article is the first to describe the clinical findings, CSF analysis, and MRI results of culture-proven *Acinetobacter*-associated postencephalitic hydrocephalus and acute blindness in a dog.

A 10-month-old castrated Welsh Corgi was referred to the Konkuk University Veterinary Medical Teaching Hospital (KU-VMTH) for evaluation of acute blindness, anorexia, ataxia, and depression lasting two weeks. The patient had been diagnosed with a recurrent bacterial dermatitis and recently started a regimen of antibiotics (amoxicillin and clavulanate 12.5 mg/kg PO twice daily, Amocla®; Gunil Pharm, Seoul, Korea) prescribed by the referring veterinarian, but had otherwise not received any medications.

Upon arrival to the hospital, the dog had normal body temperature (38.3°C) and heart rates (120 beats/min), but mild tachypnea (40 breaths/min). On physical examination, the dog's calvarium appeared to be normal in shape and the fontanelles were closed on palpation. There were no remarkable findings except decreased hearing and blindness. An ophthalmic examination revealed a normal pupillary light reflex. The motor and sensory functions in the eyelids and eyes were also normal. The electroretinography (Retiport®; Roland Consult, Havel, Germany) results were normal in both eyes. However, vision testing revealed complete visual

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deficits. The results of the ophthalmologic examination are normal except mild lens subluxation and mild retinal vessel distension. These two symptoms were considered very mild and unrelated to visual acuity. The neurological examination revealed decreased hearing on the left side and bilateral absence of vision. The menace response was absent in both eyes. Spinal reflexes and pain perception were normal. The complete blood cell counts were within the normal reference ranges (white blood cell 10.8 × 10^9/*l*, reference interval 5.05–16.76 × 10^9/*l*; hematocrit 41.7%, reference interval 40–55%; platelet count 204 × 10^3/μ*l*, reference interval 200–500 × 10^3/μ*l*,). The serum biochemical profile showed no remarkable findings including normal C-reactive protein (CRP 21 mg/*l*, reference interval 0–35 mg/*l*). On the basis of the history, clinical signs, and neuro-ophthalmological examinations, a bilateral lesion of the upper visual pathway was suspected.

The patient was anesthetized and brain MRI scans were performed using a 1.5 T scanner (Magnetom Essenza®; Siemens Medical Solutions, Erlangen, Germany). Extensive fluid-filled, bilateral, lateral and third ventricular ventriculomegaly lesions were identified. These lesions were hypointense on T1- and hyperintense on T2-weighted images (Fig. 1A and 1B). Dilation of the lateral and third ventricles induced marked compression of the optic chiasm and the interthalamic region was observed to be irregular and atrophied (Fig. 1C, 1D and 1E). In the MRI images of the dorsal plane, mesencephalic aqueduct stenosis was identified (Fig. 1); it was isointense but slightly hyperintense in the T2-weighted images and fluid-attenuated inversion recovery (FLAIR) images (Fig. 1G and 1H). However, intravenous (iv) administration of gadolinium (0.1 mmol/kg iv, Omniscan®; GE Healthcare, Princeton, NJ, U.S.A.) did not enhance any lesions on the T1-weighted images. A CSF sample obtained from the cerebellomedullary cistern revealed moderate neutrophilic pleocytosis (increased total nucleated cell counts, 70 cells/μ*l*; reference range, less than 5 cells/μ*l*), high normal total protein (23 mg/d*l*; reference range, less than 25 mg/d*l*), and no red blood cells. A sample of the CSF was submitted for culture and antibiotic sensitivity testing, and *Acinetobacter lwoffii* was isolated. Additionally, the results of the CSF polymerase chain reactions for toxoplasma, neospora, and canine distemper virus were negative.

Based on the case history, CSF analysis, and imaging findings, a diagnosis of *Acinetobacter*-associated postencephalitic hydrocephalus with visual defect was made. Treatment was initiated with furosemide (1 mg/kg PO twice daily, Lasix®; Handok Pharm, Seoul, Korea), omeprazole (0.7 mg/kg PO daily, Prasec®; Hana Pharm, Seoul, Korea), and a combination of antibiotics determined by the results of the antibiotic sensitivity testing (Table 1); these included amoxicillin and clavulanate (12.5 mg/kg PO twice daily, Amocla®; Gunil Pharm, Seoul, Korea) and enrofloxacin (5 mg/kg PO twice daily, Baytril®; Bayer Korea Corp., Seoul, Korea), each administered for four weeks. After the initiation of treatment, the patient's activity and anorexia gradually improved, but his vision status did not return to normal. Two months later, the patient had a seizure episode and died at home. Unfortunately, the owner refused to allow a necropsy to be performed.

We describe the case of a 10-month-old castrated dog that had acute blindness, ataxia, and depression. The MRI showed inflammatory stenotic lesions in the mesencephalic aqueduct associated with ventriculomegaly in the lateral and third ventricles. Concerning the visual defect in this dog, dilation of the ventricles induced marked compression of the optic chiasm. Additionally, it could compromise the optic radiation in the internal capsule, in which it forms the lateral wall of the dilated lateral ventricle [19]. Bilateral visual deficits and ataxia as shown in this case are common signs, reflecting attenuation of the cerebral white matter, optic radiations, and visual cortex [19].

The cerebrospinal fluid analysis revealed moderate neutrophilic pleocytosis and the presence of *Acinetobacter lwoffii*. Given the finding of moderate pleocytosis and the result of the CSF bacterial culture, we believe that a bacterial infection may have been the cause of the inflammatory lesions, which in turn led to hydrocephalus. A similar condition has been previously reported in the pathogenesis of spontaneously occurring acquired canine hydrocephalus, but no viral or bacterial causes have been investigated to date [11, 17].

Bacterial infection of the central nervous system (CNS) is may develop by direct access, local spread, or hematogenous route; the hematogenous route is usually incriminated in animals and humans [12]. In the case described here, the route of infection was unknown, but the recurrent bacterial dermatitis would suggest that the skin infection may have been the original source of hematogenous spread, although bacteriologic examination of dermatitis or blood culture was not performed. Additionally, insufficient nutrition, concurrent systemic disease, and an immature immune system predispose dogs to CNS infections [6]. In human, being a pediatric patient is a common risk factor for bacterial infections of the CNS [6]; the age of the present dog was less than 1 year.

Acinetobacter is an aerobic, non-fermentative gram-negative bacillus that is widespread in nature [2]. *Acinetobacter lwoffii* is less frequently isolated than other species and used to be considered a contaminant derived from the environment. In immuno-compromised patients, however, *Acinetobacter* has been recognized as a pathogen that plays a significant role in colonization and infection, and it can cause septicemia and meningitis [9, 16, 23]. In a retrospective study of a human population, *Acinetobacter meningitis* accounted for 10% of the bacterial meningitis infections [5]. A final diagnosis of *Acinetobacter* meningoencephalitis can only be confirmed by CSF cultures in human medicine [5].

CSF analysis is the single most useful diagnostic test for intracranial inflammatory conditions. Increased CSF proteins or white blood cells are found in approximately 90% of affected dogs [18]. In a recent retrospective study of 54 human patients whose CSF cultures were positive for *Acinetobacter*, neutrophilic pleocytosis and elevated protein levels were noted in the CSF [4]. In that study, a fever was the only significant clinical symptom [4]. In the present case, the CSF analysis revealed moderate neutrophilic pleocytosis, high-normal protein, and the presence of *Acinetobacter lwoffii*, as shown in the human study. In our case, although the patient had a visual defect and finally developed a seizure, he was afebrile, possibly due to the previous history of antibiotics administered at the local animal hospital.

MRI is also a sensitive diagnostic tool for lesions associated with intracranial inflammation in dogs; however, mild encephalitis

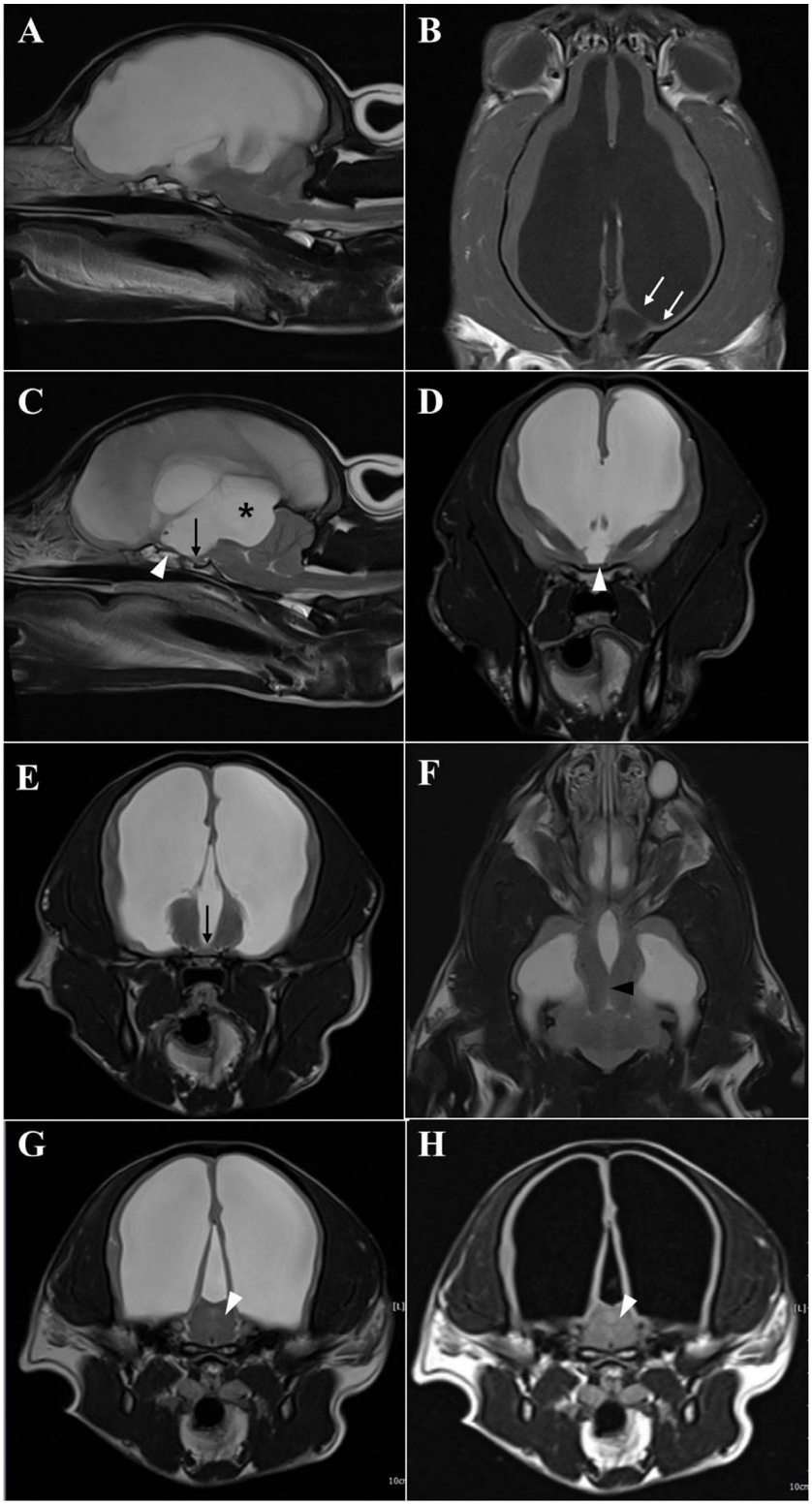

Fig. 1. The magnetic resonance image (MRI) of a 10-month-old Welsh Corgi presenting with acute blindness. (A) The sagittal T2-weighted MRI image demonstrates extensive dilatation of the dorsal aspect of the lateral ventricle and third ventricle. (B) On the T1-weighted MRI of the dorsal plane, a thin strand of white matter (white arrows) traversing the distended lateral ventricle is noted. (C) The midsagittal T2-weighted MRI reveals that dilation of the third ventricle (asterisk) impinges on the interthalamic adhesion ventrally and the cerebellum caudally. The interthalamic adhesion is irregular and atrophied (black arrow) and the optic chiasm is compressed (white arrowhead). (D, E) On the transverse T2-weighted MRI through the frontal lobe, dilation of the lateral ventricle and marked compression of the optic chiasm (white arrowhead) and the interthalamic region (black arrow) can be seen. (F) On the T2-weighted MRI of the dorsal plane, the stenosis of the mesencephalic aqueduct (black arrowhead) is identified. (G) On the transverse T2-weighted image and (H) the fluid-attenuated inversion recovery (FLAIR) image, there is a hyperintense lesion affecting the mesencephalic aqueduct (white arrowhead).

may not be evident on an MRI [18, 22]. Use of contrast such as gadolinium increases the sensitivity of the MRI for intracranial inflammatory lesions [20]. However, in a retrospective study that described the MRIs of 25 dogs with inflammatory CSF, meningeal enhancement after a contrast injection was identified in only 28% of the dogs, suggesting that it is an insensitive marker [18]. Furthermore, meningeal enhancement may also be a relatively nonspecific sign, as it has also been described in various intracranial neoplasia in dogs [15, 21]. In studies of large numbers of human patients, FLAIR images have been demonstrated to be more sensitive for examining a wide variety of brain conditions than T2-weighted images or post-gadolinium T1-weighted images [1, 18]. In our case, mesencephalic aqueduct stenosis was identified on the MRI. There was no enhancement on the T1-weighted images after intravenous administration of gadolinium, but slightly hyperintense T2-weighted images and FLAIR images were observed. In the end, this patient was diagnosed with *Acinetobacter*-positive inflammation on CSF analysis. In the case under study, we excluded the possibility that the hydrocephalus was congenital, owing to the absence of an increased volume, reduced thickness, or other deformation of the calvarium. We further excluded canine distemper virus, toxoplasmosis, and neosporosis as possible causes, given negative test results.

Table 1. Results of antibiotic sensitivity testing

Antibiotic	Result
Amikacin	S
Ampicillin	R
Amoxicillin/Clavulanic acid	S
Cefaclor	R
Ceftriaxone	S
Cefixime	R
Cephalothin	R
Clindamycin	R
Chloramphenicol	S
Ciprofloxacin	R
Enrofloxacin	S
Erythromycin	R
Gentamicin	S
Tetracycline	S

S=susceptible, R=resistant.

A rare form of meningoencephalitis, termed hydrocephalus with periventricular encephalitis (HPE), has been described in puppies 2–3 months of age [7]. This idiopathic disease is characterized by hydrocephalus and intense inflammatory and hemorrhagic encephalomalacia and appears to be rapidly progressive and usually fatal [3, 9, 12]. Although the etiology of this syndrome is unknown, a bacterial cause is suspected; however, causative bacteria have never been isolated in the ante-mortem bacterial culture of CSF [8, 11]. In these puppies, CSF analysis revealed a mixed-cell pleocytosis, increased proteins, and xanthochromia [7]. However, a definitive diagnosis of HPE is based upon gross and histopathological examination of the brain at the necropsy [3, 11]. In the present case, there was a possibility of HPE because the patient had hydrocephalus and inflammatory CSF and the clinical condition progressed to spontaneous death within two months. However, our patient was relatively older and the progress was slower than in other documented cases of HPE. There was also no evidence of acute or chronic hemorrhage in the CSF.

In conclusion, this report details the findings of visual defect-associated postencephalitic hydrocephalus in a dog. In addition, MRI and CSF analysis was helpful in establishing an etiology of the postencephalitic hydrocephalus. We believe that this case is important because of the extraordinary clinical features, neuro-ophthalmological testing, CSF analysis, and MRI findings of *Acinetobacter*-associated postencephalitic hydrocephalus in a dog with acute blindness. Furthermore, veterinarians should recognize *Acinetobacter lwoffii* as one of the causative pathogens of central nervous system inflammation, which is associated with acquired obstructive hydrocephalus. Additionally, *Acinetobacter*-associated encephalitis in dogs can be fatal when severe post-inflammatory hydrocephalus occurs concurrently, despite prompt initiation of antimicrobial therapy.

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