Antemortem diagnosis of hydrocephalus in two Congo African grey parrots (Psittacus erithacus erithacus) by means of computed tomography

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Case Description—A 7-year-old and a 10-year-old Congo African grey parrot (Psittacus erithacus erithacus; parrots 1 and 2, respectively) were evaluated because of neurologic deficits.

Clinical Findings—Parrot 1 had an 8- to 9-month history of lethargy and anorexia, with a recent history of a suspected seizure. Parrot 2 had a 6-month history of decreased activity and vocalizing, with an extended history of excessive water intake; a water deprivation test ruled out diabetes insipidus, and psychogenic polydipsia was suspected. Both birds had ophthalmologic asymmetry, with anisocoria detected in parrot 1 and unilateral blindness in parrot 2. Metal gastrointestinal foreign bodies were observed on whole-body radiographs of both birds, but blood lead concentrations were below the range indicated for lead toxicity. Findings were consistent with hydrocephalus in both cases.

Treatment and Outcome—Parrot 1 received supportive care and died 3 months after the diagnosis of hydrocephalus. Parrot 2 was treated with omeprazole and prednisolone for 10 days without any improvement in neurologic deficits; euthanasia was elected, and hydrocephalus was confirmed on necropsy. No underlying or concurrent disease was identified.

Clinical Relevance—Hydrocephalus should be considered a differential diagnosis for parrots evaluated because of CNS signs. Computed tomography was an excellent screening tool to diagnose hydrocephalus in these patients. Compared with MRI, CT is more frequently available and offers reduced scanning times, reduced cost, and less concern for interference from metallic foreign bodies. (J Am Vet Med Assoc 2015;246:770–776)

An approximately 7-year-old male Congo African grey parrot (Psittacus erithacus erithacus; parrot 1) was referred to the University of Wisconsin School of Veterinary Medicine for evaluation because of an 8- to 9-month history of abnormal behavior and a recent single episode of seizure-like activity. The bird was maintained in a private household with no other pets. The bird was acquired from a pet store as a juvenile and fed a diet of seeds, fruit, vegetables, peanuts, and sunflower seeds, with an unspecified vitamin supplement.

The owner had noticed abnormal behavior, including improper placement of the right foot, difficulty walking, flying into obstacles, progressive anorexia, decreased vocalization, and lethargy, for the past 8 to 9 months. Approximately 10 days prior to the referral visit, the bird had a suspected generalized seizure that was treated with diazepam by the referring veterinarian. On the day before the referral examination, the referring veterinarian identified a metallic foreign body in the gastrointestinal tract on whole-body radiographs of parrot 1 and ultrasound images of parrot 2. On initial evaluation, the bird was quiet and obtunded and had fluffed feathers. Its pectoral musculature was thin, and body condition score was assessed as 1.5 of 5; body weight was 402 g (0.88 lb). The bird was unable to perch for extended periods of time.

On neurologic examination, the bird was obtunded, which was more obvious when it was left alone in the cage. The bird was unable to balance; it had a vestibular ataxia: when standing, it leaned to the left and the body swayed; it had difficulty balancing, maintained a broad-based stance, and occasionally fell backward. On cranial nerve examination, anisocoria was evident, with mydriasis and reduced pupillary light reflex in the left eye (Figure 1). The menace response was present bilaterally, and there was a normal response to touching of the face. The neuroanatomic localization was multifocal, including the left oculomotor nerve and the left central vestibular system.

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was evident, with mydriasis and reduced pupillary light reflex in the left eye. A CBC revealed a PCV of 41% (reference range, 42% to 50%), mild leukocytosis (19.6 × 10^3 leukocytes/µL; reference range, 5 × 10^3 leukocytes/µL to 11 × 10^3 leukocytes/µL), and mild lymphocytosis (51%; reference range, 25% to 45%). The blood lead concentration, measured with a portable unit, was 0.1 µg/dL (reference range, 0.5 to 20 µg/dL). A small (< 0.5 cm) metallic focus consisting of wire-like and ovoid portions was seen within the ventriculus on whole-body survey radiographs. The proventriculus contained homogeneous material of soft tissue opacity and was of normal size (proventriculus-to-keel ratio, 0.37; reference range, < 0.48). A moderate amount of gas was seen within the intestines. The lungs and air sacs were radiographically normal.

The bird was kept in an incubator and was treated with calcium disodium EDTA (40.0 mg/kg [18.2 mg/lb], IM, q 12 h) for suspected non–lead-related heavy metal intoxication, terbinafine hydrochloride (20.0 mg/kg [9.1 mg/lb], PO, q 24 h) as antifungal prophylaxis, fluid therapy (30.0 mL/kg [13.6 mL/lb], SC, q 12 h), and gavage feeding (15 mL of a hand-feeding formula q 12 h) for 3 days. The bird's condition was stable, and advanced diagnostic imaging of the brain was recommended but declined by the client.

The bird was discharged from the hospital and prescribed continued treatment with terbinafine and calcium disodium EDTA of the same dose and frequency for 14 days. The bird initially appeared to improve with treatment and syringe feeding of a hand-feeding formula; however, 3 weeks later, with little to no further progress in resolution of clinical signs or increase in appetite, the bird was reevaluated for endoscopic removal of the metallic gastric foreign body and CT scan of the head. On second evaluation, the bird's weight remained stable at approximately 440 g (0.97 lb). The bird was mildly obtunded, and its feathers were unkempt; the pupils remained anisocoric. Problems walking and maintaining balance persisted, with the bird leaning to its left. Its grip was weak, and there was a loss of proprioception in both feet. Repeated blood work revealed no remarkable abnormalities. Recheck radiography confirmed persistence of the gastric metallic foreign material. The bird was anesthetized for CT of the head. Following premedication with midazolam (2.0 mg/kg [0.91 mg/lb]) and butorphanol (2.0 mg/kg) IM, a 26-gauge IV catheter was placed in the right ulnar vein for administration of fluids and contrast medium. The bird was intubated, and anesthesia was maintained with isoflurane in oxygen throughout the procedure.

Transverse plane CT images collimated to 0.625 cm were obtained before and after iodinated contrast medium administration (1 mL of iohexol/kg; 300 mg of I/mL, IV). Images were reformatted by use of a high-frequency bone algorithm (precontrast only) and detail soft tissue algorithm. Sagital and dorsal plane reconstructions were made from the detail soft tissue volumes. A large volume of non–contrast-enhancing, fluid-attenuating (6 HU) material was seen in the region of the transverse fissure between the cerebral hemispheres (Figure 2). Moderate diffuse accumulation of similar non–contrast-enhancing, fluid-attenuating material was seen superficial to both the cerebrum and cerebellum. Both cerebral hemispheres were subjectively small and of homogenous soft tissue attenuation and mild uniform contrast enhancement. A poorly defined central focus of hypodensity with subtle nonuniform contrast enhancement was detected within the cerebellum. Differential diagnoses included hydrocephalus of congenital or acquired etiologies. The cerebral and cerebellar atrophy were thought to be secondary to hydrocephalus; infectious, toxic, or metabolic disorders; or age-related changes. Differential diagnoses for the cerebellar lesion included an intra-axial lesion (eg, neoplasia, granuloma, or vascular incident) or hydrocephalus with or without atrophy. Diagnostic interpretation was hydrocephalus with suspected secondary brain atrophy.

With the bird under general anesthesia, gastroscopy was performed with a rigid endoscope via a standard antrum. A small, (< 0.5 cm) metallic focus consisting of wire-like and ovoid portions was seen within the ventriculus on whole-body survey radiographs. The proventriculus contained homogeneous material of soft tissue opacity and was of normal size (proventriculus-to-keel ratio, 0.37; reference range, < 0.48). A moderate amount of gas was seen within the intestines. The lungs and air sacs were radiographically normal.

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With the bird under general anesthesia, gastroscopy was performed with a rigid endoscope via a standard antrum.
The metal gastric foreign body was removed with a grasping forceps. Recovery from anesthesia was uncomplicated.

The bird was prescribed amoxicillin (125 mg/kg [56.8 mg/lb], PO, q 8 h for 5 days), and terbinafine was continued as previously prescribed for another 21 days. The bird was brought to the referring veterinarian for suture removal 34 days after discharge from the hospital. Three months after the diagnosis of hydrocephalus, the bird died and no postmortem examination was performed.

A 10-year-old male Congo African grey parrot (parrot 2) was evaluated because of abnormal behavior and left leg weakness of approximately 1 month’s duration. The bird had been treated 2 years previously for a fracture of the lower beak, which healed without complications. The bird lived in a large wire cage in a private household. The bird had been fed a commercial pelleted parrot diet for the past 2 years, with peanuts, carrots, and grapes as snacks. The bird had an approximately 5-year history of excessive water consumption (approx 440 to 500 mL of water/d). Its behavior had been abnormal for ≥1 month prior to the examination; reported signs included lack of vocalization, poor balance, and apparent weakness of the left leg during perching as well as decreased vision, especially in the dark, and apparent trouble finding food. Three days before the evaluation, the left limb appeared very weak and the bird was found with its head in the food dish, unable to perch or hold itself up.

The bird was quiet and lethargic during examination. It was well hydrated and weighed 425 g (0.94 lb). Weakness, lack of balance, and proprioceptive deficits in the left foot were evident (Figure 3). For diagnostic procedures and physical examination, the bird was sedated with butorphanol (2 mg/kg, IN) and midazolam (2 mg/kg, IN); sedation was reversed with flumazenil (0.05 mg/kg, IN) after all procedures were completed. Complete ophthalmologic examination performed by a board-certified veterinary ophthalmologist revealed lack of pupillary light reflex and menace response of the left eye but no anatomic or facial nerve abnormalities. On the basis of examination findings, the neuroanatomic localization was multifocal within the CNS, including the right prosencephalon, left side of the brainstem, and left oculomotor nerve.

Blood was drawn for a CBC and plasma biochemical analysis, results of which were within respective reference ranges. Examination of whole-body radiographs revealed a rectangular metallic gastrointestinal foreign body, presumed to be within the ventriculus, and ventricular dilatation with gas (proventriculus-to-keel ratio, 0.62). The bird’s blood lead concentration was 4.6 µg/dL, below the reported lower limit for toxicosis but above values commonly found in psittacine species.

Figure 2—Postcontrast CT images of the head of a healthy adult Congo African grey parrot (A) and of parrot 1 (B). Both transverse plane CT images (window level, 50 HU; window width, 100 HU) were obtained at the level of the thalamus. In panel B, notice the large volume of fluid-attenuating material within the lateral and third ventricles and superficial to the cerebrum, indicative of hydrocephalus. Notice the small size of the cerebral hemispheres and thalamus in parrot 1.

Figure 3—Photograph of a 10-year-old male Congo African grey parrot (parrot 2) showing proprioceptive deficits affecting the left foot and evidence of urination in excessive amounts.
birds at the authors' clinic. A CT scan of the brain was declined by the owner at this time.

The bird was hospitalized for 4 days and treated with calcium disodium EDTA (40 mg/kg, IM, q 12 h) and terbinafine (20 mg/kg, PO, q 24 h) for antifungal prophylaxis. The blood lead concentration decreased to 1.3 µg/dL after 4 days of chelation treatment, with no notable change in behavior or neurologic status.

During hospitalization, the bird was observed to consume large volumes of water (approx 450 mL/kg) daily; therefore, a water deprivation test was performed. This test had previously been used to distinguish central diabetes insipidus from psychogenic polydipsia in African grey parrots.5,6 Food and water were withheld 8 hours, and the bird's body weight was measured every hour from 8 am to 4 pm. Urine specific gravity was measured by refractometry during this time period. During the 8-hour period, the bird had a 3% reduction in body weight and was able to concentrate urine: the urine specific gravity was initially 1.005 and increased to 1.020 by 7 hours after test initiation. These results suggested the bird had psychogenic polydipsia with secondary polyuria.

Because medical management failed to improve the bird's neurologic signs, a whole-body CT scan was performed with the patient under general anesthesia to evaluate for intracranial and vascular abnormalities. The bird was premedicated with midazolam (2.5 mg/kg [1.14 mg/lb]) and butorphanol (1 mg/kg) IM, and a 26-gauge catheter was placed in the right ulnar vein for administration of fluids and contrast media. Following intubation, anesthesia was maintained with isoflurane in oxygen throughout the procedure. Transverse plane CT images collimated to 0.625 cm were obtained before and after iodinated contrast medium administration (1 mL of iohexol/kg, IV). Images were reformatted with a high-frequency bone algorithm (precontrast only) and detail soft tissue algorithm. Sagittal and dorsal plane reconstructions were made from the detail soft tissue volumes. A large volume of non–contrast-enhancing, fluid-attenuating (7 HU) material was seen in the cranial vault, present at midline, and surrounded by both cerebral hemispheres, presumably within the lateral ventricles; this was more severe on the right side than on the left (Figure 4). The third ventricle was mildly dilated with similar non–contrast-enhancing, fluid-attenuating material. The cerebral hemispheres were small in size, with the right smaller than the left. Homogeneous, symmetric contrast enhancement was seen in both cerebral hemispheres. The diagnostic interpretation was hydrocephalus, most likely acquired secondary to obstruction or overproduction; however, congenital hydrocephalus could not be ruled out. Cerebral atrophy was most likely secondary to the hydrocephalus or secondary to inflammatory (infectious or noninfectious), toxic, or metabolic processes.

Parrot 2 was treated with prednisolone* (0.5 mg/kg [0.23 mg/lb], PO, q 12 h for 14 days) and omeprazole* (2 mg/kg, PO, q 24 h for 14 days) to decrease the suspected high intracranial pressure and decrease CSF production. Administration of terbinafine was continued to prevent secondary fungal infections, and the bird was discharged from the hospital. After 10 days of treatment, the bird was returned to the hospital because of worsening clinical signs and lack of response to medical treatment. Euthanasia was elected, and a complete necropsy was performed.

On necropsy, there was moderate whole-body emaciation and marked, bilaterally symmetric hydrocephalus. Grossly and histologically, the ventricles were bilaterally and symmetrically mildly to markedly dilated with clear contents, including all horns of the lateral ventricles (markedly dilated), the third ventricle (moderately dilated), and the mesencephalic tectal ventricles (mildly dilated) within the optic lobes. There was moderate to marked atrophy of the medial wall of the lateral ventricles, including the septum pellucidum and medial pallium (avian equivalent to the hippocampus).7 The fourth ventricle was not dilated.

Figure 4—A—Postcontrast, transverse-plane CT image of the head of parrot 2 (A), obtained at the level of the thalamus (window level, 50 HU; window width, 100 HU) and a photograph of a cross section of the brain at the level of the thalamus of parrot 2 at necropsy (B). In panel A, notice the large volume of fluid-attenuating material within the lateral and third ventricles and superficial to the cerebrum and the small size of the cerebral hemispheres and thalamus, which correspond to the necropsy findings in panel B and the diagnosis of hydrocephalus.
Histologically, the dilated ventricles were lined by a single layer of cuboidal to attenuated epithelial cells, which were multifocally ciliated. In the cerebral cortices, there was bilaterally symmetric neuronal necrosis within medial nuclei adjacent to the lateral ventricles with mineral encrustation, along with moderate gliosis. The histopathologic diagnosis was marked bilaterally symmetric hydrocephalus with cerebral atrophy and bilaterally symmetric neuronal necrosis with mineralization and gliosis. There was no evidence of infectious, inflammatory, or neoplastic disease. A stenosis in the mesencephalic aqueduct was suspected but could not be identified. There was no evidence of underlying vascular disease such as atherosclerosis, although there were some mild vascular changes (mild medial hypertrophy) consistent with increased intracranial pressure.

**Discussion**

To our knowledge, this is the first report of ante-mortem diagnosis of hydrocephalus by use of CT in birds. Hydrocephalus (either congenital or acquired) is infrequently diagnosed ante-mortem in birds, with previous ante-mortem diagnoses relying exclusively on MRI.9,8

The circulation of CSF in birds is comparable to that in mammals. The CSF is produced by the choroid plexus of the lateral, third, and fourth ventricles. The fluid drains out of the ventricles through microscopic fenestrations within the fourth ventricle into the subarachnoid space and is absorbed into the venous system via arachnoid granulations.10,11 The ventricular system of birds consists of bilateral lateral ventricles and unpaired third and fourth ventricles. The mesencephalic tectal ventricles branch off the rostral portion of the mesencephalic aqueduct bilaterally and extend into the optic lobes.12

There are many forms of hydrocephalus. Internal hydrocephalus can be communicating or obstructive.12 Communicating hydrocephalus results in symmetric dilation of the ventricular system without any obvious macro- or microscopic lesion. Obstructive hydrocephalus can be either congenital, usually attributable to an anatomic stenosis within the ventricular system, or acquired, owing to stenosis and obstruction secondary to inflammation or a mass lesion. Potential loci of stenosis and obstruction are at the interventricular foramina, which connects the lateral ventricles to the third ventricle, and the mesencephalic aqueduct, which connects the third and fourth ventricles. A third form of hydrocephalus, hydrocephalus ex vacuo, occurs when the ventricles dilate secondary to atrophy of brain tissue.13

The cause of hydrocephalus in parrot 1 remained unknown because the patient died several weeks after discharge and did not undergo necropsy. A cause of obstruction was not identified on CT images, but owing to the relatively late onset of clinical signs, hydrocephalus was most likely acquired, rather than congenital, in this patient. In parrot 2, the lateral ventricles, third ventricle, and mesencephalic tectal ventricles were dilated but the fourth ventricle was unaffected, indicating a probable stenosis within the mesencephalic aqueduct; however, a cause or site of stenosis was not detected grossly or histologically. Although there was atrophy within the cerebral cortex, this was thought to be likely secondary to the hydrocephalus, considering the obstructive pattern of the hydrocephalus and the distribution of affected tissue adjacent to the most severely affected ventricles.

Several cases of hydrocephalus in psittacine birds have been reported in the literature.8,9,12,14 All birds had neurologic signs, predominantly seizures and ataxia, but a range of signs including weakness, weight loss, feather picking, lethargy, obtundation, incoordination, and delayed postural reactions have also been reported.9,9,12,14 Three of these birds were African grey parrots, ranging in age from 6 months to 10 years, and all 3 had a history of seizures.9,14

Seizures have been a common clinical sign in previously reported cases of hydrocephalus in psittacine birds.9,9,12,14 Differential diagnoses for seizures in a psittacine include trauma, hypocalcemia, viralencephalitis, epilepsy, and toxicosis.9,13 Parrot 2 of the present report had no history of seizure-like activity, and neither bird had seizure-like activity observed during hospitalization. Neither bird required seizure treatment during hospitalization at our facility or after discharge.

Both birds in this report had paresis, weakness, and unilateral limb weakness and proprioceptive deficits as well as obtundation, suggesting a lesion in the prosencephalon or brainstem. Paresis and weakness are nondiagnostic findings but help make a neuroanatomic diagnosis; these findings can be caused by a range of diseases, including peripheral neuropathy, lead intoxication, renal neoplasia, atherosclerosis, and Baylisascaris procyonis infection.13,15 Atherosclerosis can also cause acute death, blindness, ataxia, paresis, and seizures.15 Suspected ischemic stroke in an African grey parrot was associated with ataxia, pronation of the feet, and asymmetric leg weakness and hypertonicity.17

Absent or reduced pupillary light reflexes in both birds, presence of anisocoria in parrot 1, and unilateral blindness in parrot 2 were suggestive of intracranial lesions. In both cases of this report, hydrocephalus was the underlying cause for anisocoria and unilateral blindness; therefore, CT or MRI should be performed for evaluation of parrots with such signs.

Hydrocephalus can be managed medically in cats and dogs with acetazolamide or omeprazole to reduce CSF production.18 Prednisone or prednisolone is also commonly used in veterinary and human patients with hydrocephalus; however, whether prednisone reduces CSF production is debated.19 Anti-inflammatory prednisolone treatment was shown to be successful for the treatment of hydrocephalus in a yellow-headed Amazon parrot.8 Surgical management of hydrocephalus in mammals is performed with placement of intraventricular shunts that allow CSF to exit the skull and drain into the peritoneal space. Because of the small size of psittacines, this is not a viable treatment option and it has not been previously described.8

Previous reports8,9,20 have described the use of MRI to diagnose hydrocephalus in avian patients. The CSF-filled ventricles are T2-weighted hyperintense and T1-weighted hypointense, compared with brain parenchyma; do not contrast enhance; and suppress
on fluid-attenuation inversion recovery sequences. Although the use of CT has been previously described for evaluation of the head in psittacines and raptors, no comments were made in these reports regarding the internal structure of the brain and ventricles. Use of CT has been described for the diagnosis of hydrocephalus in canine and feline patients. Hydrocephalus in humans and small animals is identified on CT as enlargement of the ventricular system with non–contrast-enhancing, fluid-attenuating CSF. In evaluation of the birds described in the present report, CT scans from neurologically normal Congo African grey parrots in our facility’s medical records were used for relative size comparisons of ventricles in the brain because there are presently no published reference ranges for these variables in psittacines. In the parrots described here, the fluid-attenuating CSF-filled ventricles (0 to 10 HU) were readily distinguished from the soft tissue–attenuating brain parenchyma (30 to 40 HU) on CT. In parrot 2, the CT findings corresponded well with the gross pathological findings at necropsy performed 11 days after the CT scan. Obstructive foci leading to acquired hydrocephalus may be seen on CT as space-occupying or contrast-enhancing lesions, although no such lesion was identified in either patient. Magnetic resonance imaging and CT offer different strengths in diagnostic imaging of the brain; MRI provides greater sensitivity for identification of an obstructive focus or a small parenchymal lesion and periventricular edema, and it is the preferred imaging modality. However, MRI is not as widely available as CT in veterinary clinics. Magnetic resonance imaging has also been used to evaluate patients with enlarged ventricles for atrophy of brain tissues, and evidence of brain atrophy in dogs includes deepened tients with enlarged ventricles for atrophy of brain tissues, and evidence of brain atrophy in dogs includes deepened ventricles, so MRI cannot be used to evaluate avian patients for brain atrophy in this manner. The main advantages of CT are decreased scanning time and lower cost, compared with MRI. This reduces the anesthetic time or allows for the use of sedation only. Both patients in this report had gastrointestinal metallic foreign bodies. With MRI, unknown types of metal can pose a safety risk owing to heating and movement of the foreign object. Susceptibility artifact resulting from the metal can also interfere with image acquisition and could limit or prevent imaging of certain areas. Computed tomography is preferable in patients with unknown metallic foreign bodies for which MRI would be contraindicated.

References
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From this month’s AJVR

Renal, gastrointestinal, and hemostatic effects of oral administration of meloxicam to Hispaniolan Amazon parrots (Amazona ventralis)

Bas Dijkstra et al

Objective—To investigate renal, gastrointestinal, and hemostatic effects associated with oral administration of multiple doses of meloxicam to healthy Hispaniolan Amazon parrots (Amazona ventralis).

Animals—12 Hispaniolan Amazon parrots.

Procedures—Birds were assigned to receive meloxicam oral suspension (1.6 mg/kg, PO, q 12 h) and 2.5 mL of tap water inserted into the crop by use of a gavage tube (n = 8) or the equivalent volume of tap water only (control group; 4) for 15 days. Urine and feces were collected 2 hours after treatment administration each day. Feces were evaluated for occult blood. Results of a CBC and serum biochemical analysis and measured N-acetyl-β-D-glucosaminidase (NAG) activity and whole blood clotting time were evaluated before, during, and after completion of treatments. Results of urinalysis and measured urine NAG activity were also evaluated.

Results—Birds treated with meloxicam had a significant increase in number of WBCs and decrease in PCV after treatment. The PCV also decreased significantly, compared with results for the control group; however, WBC count and PCV for all birds remained within reference ranges throughout the study. One parrot treated with meloxicam had a single high value for urine NAG activity.

Conclusions and Clinical Relevance—Meloxicam administered orally at the dosage used in this study caused no apparent negative changes in several renal, gastrointestinal, or hemostatic variables in healthy Hispaniolan Amazon parrots. Additional studies to evaluate adverse effects of NSAIDs in birds will be needed. (Am J Vet Res 2015;76:308–317)