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Case Report - Veterinary Medicine

Hydrocephalus in *Amazona aestiva* – case report

[*Hidrocefalia em* Amazona aestiva – *relato de caso*]

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ABSTRACT

Hydrocephalus is an important neurological disorder rarely described in birds. The present report aims to describe a natural case of hydrocephalus in a blue-fronted parrot (Amazona aestiva) from a conservationist breeding (Minas Gerais, Brazil), clinically affected with neurological signs. The post-mortem lesions noted were megalencephaly, distension and thinning of the skullcap, underdeveloped brain, thickening of the meninges and the presence of a large volume of approximately 10mL of clear cerebrospinal fluid (CSF). The histopathological evaluations demonstrated discrete multifocal malacia, associated with gliosis and neuronal satellitosis in the central nervous system. The screening for viruses revealed the absence of several common etiologies, including Alphavirus (genus universal primers), avian Bornavirus, Alphaherpesvirinae (subfamily universal primers), Paramyxovirus (family universal primers), Newcastle's disease virus (avian Orthoavulavirus 1), influenza A virus (Alphainfluenzavirus), Marek's disease virus (Mardivirus, Gallid herpesvirus 2), West Nile virus, Flavivirus (Orthoflavivirus universal primers), Apicomplexa (universal primers) and Toxoplasma gondii, all tested negative by PCR or RT-PCR. The aerobic bacteriology and mycology of the CSF did not reveal growth. The results suggest that the chronic accumulation of CSF with increased cranial pressure resulted in distension of the skull, possibly due to congenital causes of increased production, drainage or circulation.

Keywords: Psittaciformes, Minas Gerais, neurological disease, congenital alteration

RESUMO

Os distúrbios neurológicos em aves incluem vários agentes infecciosos ou não infecciosos, alguns envolvidos em malformações embrionárias. A hidrocefalia é uma importante desordem neurológica a ser pesquisada em aves. O presente relato tem como objetivo investigar um caso de hidrocefalia em um papagaio-verdadeiro (Amazona aestiva) *de um criadouro conservacionista, localizado no estado de Minas Gerais, Brasil, clinicamente acometido por sinais neurológicos. As lesões* post mortem *observadas foram megaloencefalia, distensão e adelgaçamento da calota craniana, cérebro subdesenvolvido, espessamento das meninges e presença de grande volume (aproximadamente 10mL) de líquido cefalorraquidiano (LCR) claro. As avaliações histopatológicas demonstraram discreta malácia multifocal, associada à gliose e satelitose neuronal no sistema nervoso central. A triagem molecular para vírus e parasitos revelou a ausência de várias etiologias comuns, incluindo* Alphavirus *(protocolo universal do gênero),* Bornavirus *aviário, Alphaherpesvirinae (*primers *universais para subfamília),* Paramyxovirus *(*primers *universais da família), vírus da doença de Newcastle (*Orthoavulavirus *aviário 1), vírus influenza A* (Alphainfluenzavirus), *vírus da doença de Marek* (Mardivirus, *Gallid herpesvirus 2), vírus do Nilo Ocidental,* Flavivirus (Orthoflavivirus primers *universais), Apicomplexa (*primers *universais para a ordem) e* Toxoplasma gondii, *todos testados negativos por PCR ou RT-PCR. A bacteriologia e a micologia aeróbias do líquido cefalorraquidiano não revelaram crescimento. Os resultados sugerem que a hidrocefalia resultou do acúmulo crônico craniano de LCR, com aumento da pressão craniana e distensão do crânio antes da ossificação. A acumulação de LCR possivelmente foi devido a causas congênitas de malformação na produção, na drenagem ou na circulação.*

Palavras-chave: Psittaciformes, Minas Gerais, doenças neurológicas, alteração congênita, zoonoses

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INTRODUCTION

Diseases that cause neurological disorders in birds may originate from zoonotic diseases or embryonic disorders that are responsible for population declines. However, clinicopathological similarities make ante-mortem diagnosis a challenge and require a more detailed and definitive laboratory diagnosis (Bennett, 1994; Jones and Orosz, 1996; Hedley and Kubiak, 2015). Congenital changes associated with malformation of the central nervous system in these animals are possible, although rarely reported. Hydrocephalus is one of these changes and is characterized by an exaggerated accumulation of cerebrospinal fluid in the ventricular system of the brain, been reported in Cockatoos (*Cacatua* sp.), African gray parrots (*Psittacus erithacus*), turkeys and chickens. Among the causes of hydrocephalus, it is important to report early malformation and bifurcation of the neural tube during embryonic development, intoxication with cyclophosphamide, cyanocobalamin (B12), pyridoxine (B6) and zinc or excess vitamin A. Clinical signs observed in animals with hydrocephalus include tremors, motor incoordination, ataxia, lethargy, head pressing and blindness. Occasionally, there may be cerebellar hypoplasia, skull enlargement, expansion of the ventricles, compression, and atrophy of the parenchyma and hypomyelination (Bennett, 1994; Jones and Orosz, 1996; Ecco *et al*., 2016; Schmidt and Ondreka, 2019).

Among the infectious diseases that can lead to neurological conditions in birds are West Nile virus, Venezuelan equine encephalitis, avian influenza and Newcastle's disease, which require immediate notification of any suspected case (Brasil, 2013; Costa *et al*., 2020; Hedley and Kubiak., 2015). Also important are infections by avian bornavirus (Kistler *et al*., 2008), the herpesviruses of Marek's disease and Pacheco's disease (Vandevanter *et al*., 1996; Chang *et al*., 2002; Tomaszewski *et al*., 2006; Barbosa *et al*. 2022), avian adenovirus (Meulemans *et al*., 2001), *Sarcocystis* sp. (Alves *et al*., 2023), *Toxoplasma gondii* (Dini *et al*., 2023), *Aspergillus* sp., *Listeria monocytogenes*, *Chlamydia psittaci*, *Salmonella* Typhimurium (Bennett, 1994; Jones and Orosz, 1996; Hedley and Kubiak, 2015). Nervous signs generally include head tremors, torticollis, opisthotonos, motor incoordination, facial and head edema, ataxia, culminating in death. Macroscopically, there is cerebral hemorrhage, edema, and microscopically it is possible to notice inflammatory infiltration (Bennett, 1994; Costa *et al*., 2020; Dini *et al*., 2023).

To correctly diagnose the cause of hydrocephalus, it is necessary to carry out a clinical neurological examination and the evaluation of the animal clinical history. The progressive neurological impairment may result in the reduction of food and water consumption, which may thereafter result in damage to the kidneys, pancreas, liver, other parts of the digestive system, and other organs. Necropsy is essential for describing the macroscopic lesions, will enable the submission to histopathological analysis, and other laboratory tests such as PCR, RT-PCR, and bacteriological, mycological and parasitological examinations of organ samples (Bennett, 1994; Schmidt and Ondreka, 2019; Costa *et al*., 2020; Silva *et al*., 2021).

The scientific reports on hydrocephalus in birds are scarce (Keller *et al*., 2011; Thurber *et al*., 2015; Fleming *et al*., 2003; Johnston *et al*., 2006), and did not describe the disorder in blue-fronted amazon. The aim of this paper is to describe a case of hydrocephalus in a 1,5-year-old blue-fronted amazon (*Amazona aestiva*) and to investigate the nervous tissues for the most common viral etiologies.

CASUISTRY

In 2022, a 1 year 7 months old birdf of the species *Amazona aestiva* was received at Avian Diseases Laboratory, Veterinary School, UFMG for clinical evaluation and sampling (Fig. 1), weighing 330 g, original of Ribeirão das Neves, Minas Gerais, Brazil.

The bird was kept in the laboratory for observation, with clinical and neurological examinations conducted, enabling the observation of and enlarged skull (Fig. 1), sensitivity to abdominal palpation, head pressing, head tilted to the left side, and uncoordinated dislocation and flight. Flight attempts were uneven, short and resulted in collision Also, the left side of the body was unresponsive, the left eye iris did not respond to light stimuli, absence of pupillary and eyelid reflex, absence of

response to touch, absence of superficial and deep sensitivity in the left pelvic limb, the absence of proprioception, and left leg incorrectly positioned (Fig. 2). Excreta was adhered to the cloaca (Fig. 3)

Figure 1. Blue-fronted amazon (*Amazona aestiva*), 1 year and 7 months old, with head tilted to the left side and enlarged cranial circumference.

Figure 2. *Amazona aestiva*. Incorrect positioning of the left leg.

Figure 3. *Amazona aestiva*. Excreta adhered to the pericloacal region.

The bird was tube fed and kept in a cage with other animals, not treated with antiviral (Acyclovir), expressed progressive worsening, with an unfavorable prognosis. In 2021, the animal was negative for the RT-PCR test to bornavirus in a cloacal swab sample. The conservationist breeding contained a total of 400 birds of various orders and families, where several animals presented low egg laying rate, apathy and high mortality, being suspected of avian Bornavirus infection, a disease that causes problems neurological disorders in birds. Due to the worsening clinical signs and prognosis, and at the owner's request, the animal was euthanized at the place of origin on May 24th, 2022. Euthanasia was carried out by chemical restraint followed by inhalation anesthesia by carbon dioxide (CO_2) according to "*Resolução nº 1.000/2012 of Conselho Federal de Medicina Veterinária"* (Federal Council of the Veterinary Medicine - CFMV) (Guia…, 2013).

A necropsy was performed following the standard technique for the species, in which a macroscopic evaluation of the organs was made, with a detailed description of the lesions, and the collection of samples for histopathology and microbiology. During the post-mortem examination, macrocephaly with distension and thinning of the skullcap (Fig. 4A and 4B), underdeveloped brain, thickening of the meninges (*dura mater*) (Fig. 5A) and the presence of approximately 10 mL of clear cerebrospinal fluid (CSF) (Fig. 5B) were observed. The CSF was collected for bacteriological (BHI broth and agar) and mycological (Sabouraud dextrose agar) evaluations (37°C, aerobically) and did not reveal growth within 72 hours of evaluation.

The duodenum was hyperemic, with well-defined and congested vessels, gizzard without food and adenomegaly approximately 6 mm in length.

Samples of the contents of the crop, proventriculus and duodenum were also collected, during the necropsy, to perform the flotation technique (Willis-Mollay Method, Willis, 1921). In the intestinal mucosal scrapings, structures compatible with *Blastocystis* sp. were observed in samples of digestive contents in the small intestine.

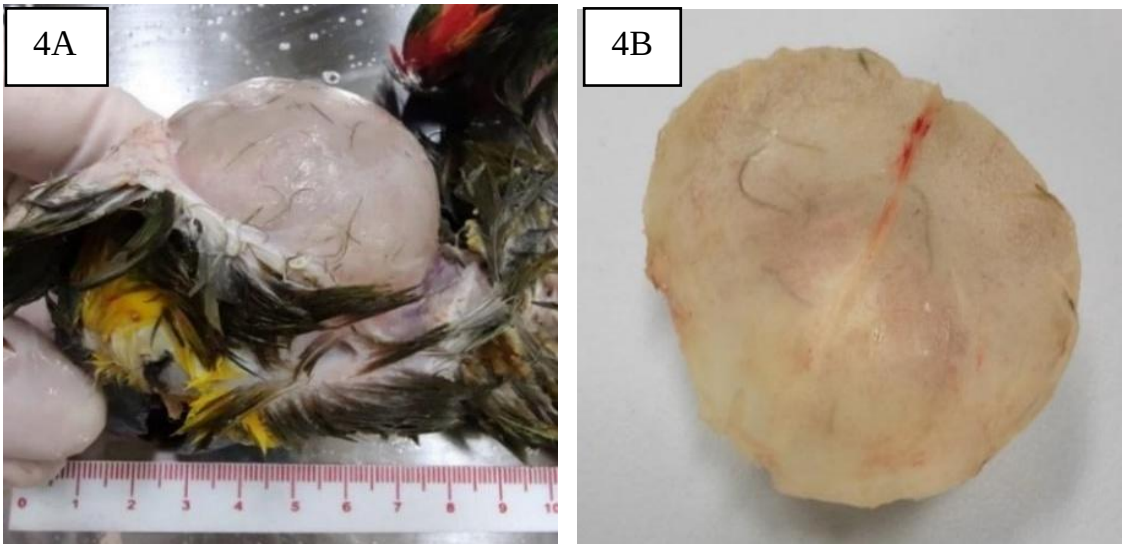

Figure 4. *Amazona aestiva*. Head. A. Distension of the skull. B. Thinning of the skullcap wall.

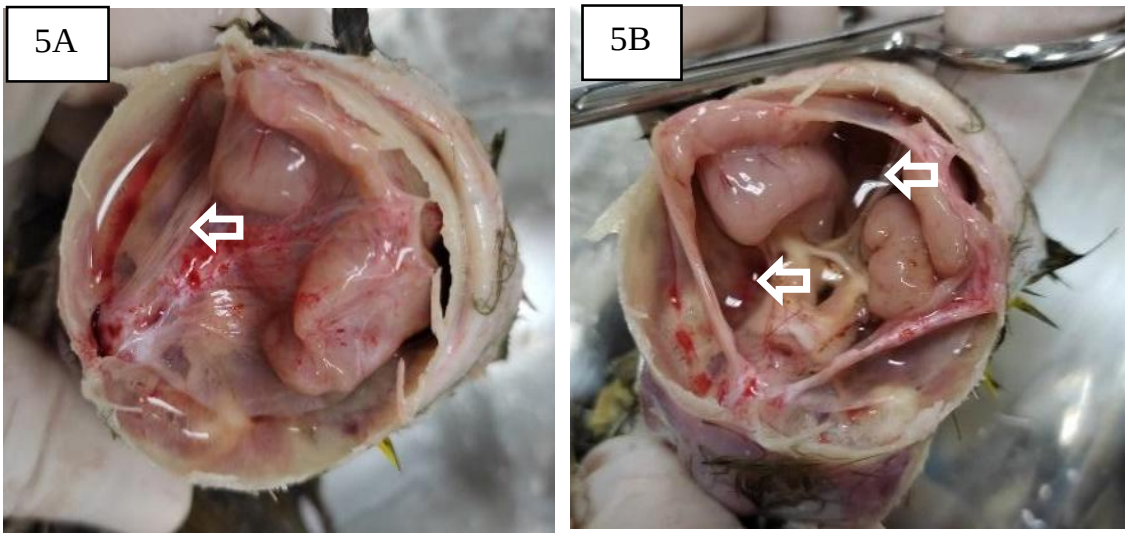

Figure 5. *Amazona aestiva*. Central nervous system. A- Meningeal (*dura mater*) thickening. B- Microencephaly and presence of clear cerebrospinal fluid within the lateral ventricules.

Fragments of brain, cerebellum, meninges, lungs, trachea, liver, spleen, crop, proventriculus, gizzard, adrenal, duodenum, jejunum, ileum were collected. These tissues were fixed in 10% buffered formalin, subjected to the histological processing technique (Prophet *et al*., 1992), processed according to the Hematoxylin and Eosin (H&E) Protocol (Luna, 1968) and examined under a specific standard. On histopathological examination of the brain, discrete multifocal malacia associated with gliosis (Fig. 6A) and normal glial-neuron satellitosis (6B). It was possible to observe atrophy of the white and gray matter and a disordered distribution of neurons, astrocytes, oligodendrocytes and gitter (microglial) cells. A very discrete deposition of golden to brownish content inside neurons was noted (Fig. 7), compatible with lipofuscin, and moderate multifocal vacuolation. Multifocal areas of white matter atrophy were observed (Fig. 8). Vacuolation was also observed in the medulla, which could be due to insufficient fixation. Finally, moderately diffuse glycogen hepatocellular degeneration was detected in the liver (Fig. 9).

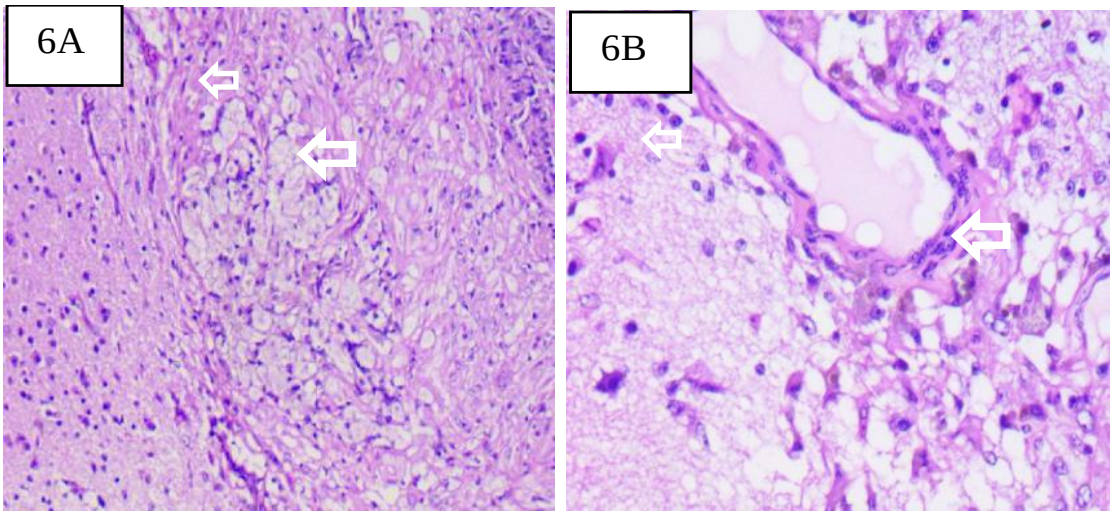

Figure 6. *Amazona aestiva*. Brain. A. Discrete multifocal malacia (arrow) associated with swollen astrocytes (gemistocytes). 100x. B. Normal multifocal glial-neuronal satellitosis. Hematoxylin and Eosin stain (H&E). 400x.

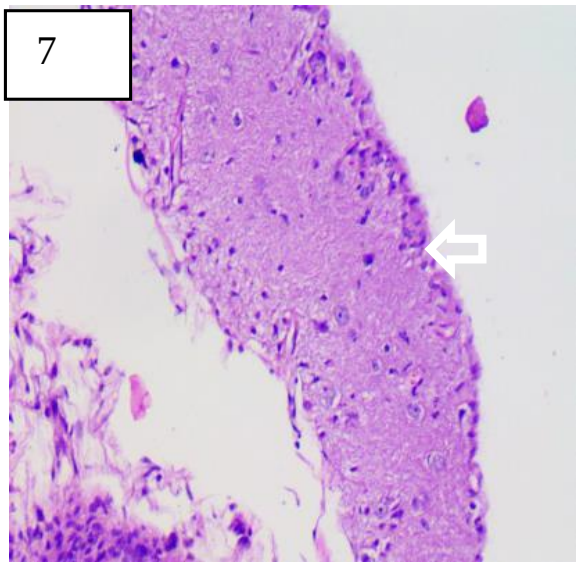

Figure 7. *Amazona aestiva*. Brain, Marked cortical atrophy (arrow) associated with cortical vacuolation and disorganization and deposition of golden to brownish content inside neurons. H&E. 100x.

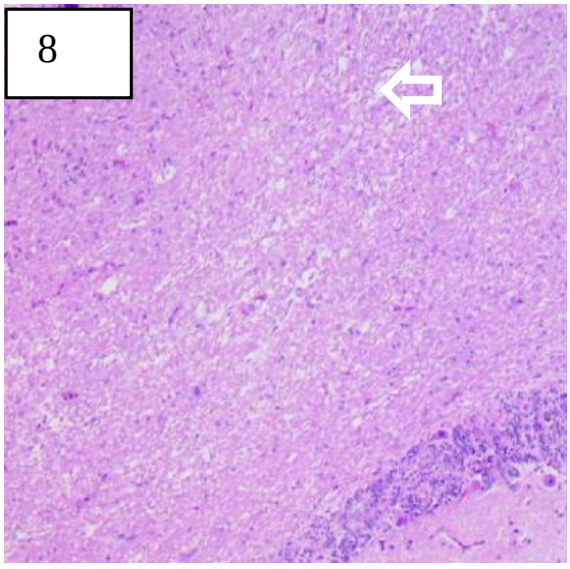

Figure 8. *Amazona aestiva*. Cerebellum. Multifocal vacuolization (arrow). H&E. 40x.

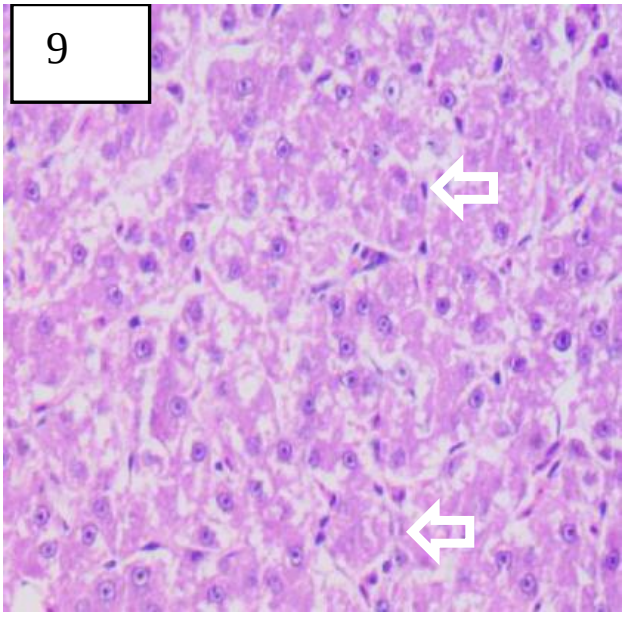

Figure 9. *Amazona aestiva*. Liver stained with Hematoxylin and Eosin showing moderate hepatocellular glycogen degeneration (arrow). H&E. 400x.

The crop with moderate serosal edema; moderately diffuse autolysis in ileum and jejunum; discrete multifocal tubular dilation in the kidneys; moderate multifocal inflammatory infiltrate of lymphocytes, plasma cells and heterophils in the duodenum (not shown).

Fragments of the organs were frozen in microtubes at -80 ℃ for the molecular detection techniques. Due to the neurological signs, molecular analyzes were performed using PCR on nervous tissue to examine any association between the presence of viruses and/or parasites

and hydrocephalus lesions (Table 1). CNS fragments were macerated and homogenized in sterile 1X PBS at a 1:5 dilution using a vortex and centrifuged at 5000 xg, at 4ºC, for 10 minutes. The supernatant was aliquoted into 1.5µL RNA-DNAse free microcentrifuge tubes, with RNA and DNA extraction performed using the QIAamp® MinElute® Virus Spin DNA/RNA kit (QIAGEN), according to the manufacturer's protocol. The extracted material was tested for viral and parasitic agents using a conventional PCR technique and the use of appropriate primers in accordance with the protocols defined by each author as mentioned below. The DNA obtained was read in agarose gel electrophoresis [1.5% agarose, 100V in TBE buffer (890mM Tris-borate, 890mM boric acid, 20mM EDTA)] using GelRed (Biotium, USA) under Ultraviolet light (280nm), there was no DNA revelation.

The molecular evaluation of the central nervous system for known etiologies (Table 1) revealed all negative.

Table 1. Sequence of oligonucleotides used

Agent	Primers	Sequence (5'-3')	Product size (pb)	Reference
Flavivirus	NS5 (+)	TCAAGGAACTCCACACATGAGATGTACT	256	Fulop *et al*., 1993
	NS5 (−)	GTGTCCCATCCTGCTGTGTCATCAGCATACA		
Alphavirus	M2W	YAGAGCDTTTTCGCAYSTRGCHW	434	Pfeffer *et al*., 1997
	cM3W	ACATRAANKGNGTNGTRTCRAANCCDAYCC		
Influenza A	M52C	CTTCTAACCGAGGTCGAAACG	244	Fouchier *et al*,. 2000
	M253R	AGGGCATTTTGGACAAAGTCGTCTA		
Paramyxovirus	PAR-F1	GAAGGITATTGTCAIAARNTNTGGAC	200 a 500	Tong *et al*., 2008
	PAR-F2	GTTGCTTCAATGGTTCARGGNGAYAA		
	PAR-R	GCTGAAGTTACIGGITCICCDATRTTNC		
Newcastle disease vírus	NDVF	CCTTGGTGAITCTATCCGIAG	254	Seal *et al*., 1995
	NDVR	CTGCCACTGCTAGTTGIGATAATCC		
Avian *Bornavirus*	ABV_MF	GGRCAAGGTAATYGTYCCTGGATGGCC	360	Kistler *et al*., 2008
	ABV_PR	CCAACACCAATGTTCCGAAGMCG		
Pan-herpesvirus	DFA	GAYTTYGCNAGYYTNTAYCC	210 a 315	Vandevanter *et al*., 1996
	ILK	TCCTGGACAAGCAGCARNYSGCNMTNAA		
	KG1	GTCTTGCTCACCAGNTCNACNCCYTT		
	TGV	TGTAACTCGGTGTAYGGNTTYACNGGNGT		
	IYG	CACAGAGTCCGTRTCNCCRTADAT		
Marek's disease vírus	MR-S	TGTTCGGGATCCTCGGTAAGA	583 a 763	Chang *et al*., 2002
	MR-AS	AGTTGGCTTGTCATGAGCCAG		
PsHV-1	PsHV UL16/17F	TGCGTGGGGTTAAACTCGGAAC	667	Tomaszewski *et al*., 2006
	PsHV UL16/17R	CGACTACACGAGCCTAACATC		
	PsHV UL16/17 Internal	CGACTTCTCAACGACGTC	418	
Aviadenovirus	Hexon AF	CAARTTCAGRCAGACGGT	897	Meulemans *et al*., 2001
	Hexon BR	TAGTGATGMCGSGACATCAT		
Apicomplexa	RIB-19	CGGGATCCAACCTGGTTGATCCTGC	1700	Zahler *et al*., 2000
	RIB-20	CCGAATTCCTTGTTACGACTTCTC		
	BabRumF	ACCTCACCAGGTCCAGACAG	420	Silveira *et al*., 2011
	BabRumR	GTACAAAGGGCAGGGACGTA		
Toxoplasma gondii	TOX 4	CGCTGCAGGGAGGAAGACGAAAGTTG	529	Homan *et al*., 2000
	TOX 5	CGCTGCAGACACAGTGCATCTGGATT		

DISCUSSION

The clinical and pathological aspects of hydrocephalus in a fronted-head amazon (*Amazona aestiva*), are described, with no inflammatory changes in the central nervous system. This report took part of a larger survey project of encephalitic diseases in avian species. As the finding of hydrocephalus was incidental, we discuss the non-infectious character and the apparent absence of a role for major avian pathogens.

The clinical diagnosis, post-mortem findings and histopathological analysis were consistent with the diagnosis of hydrocephalus, a syndrome with different causes. No evidence was found for bacterial, fungal, parasitic, or viral infection. An infective or parasitic cause was discarded after culture for bacteria or fungi, or the molecular investigation of etiologies, such as *Alphavirus*, avian *Bornavirus*, herpesvirus, *Paramyxovirus*, Newcastle's disease virus, influenza A virus, Marek's disease virus, West Nile virus *Flavivirus*, Apicomplexa (universal primers) and *Toxoplasma gondii.*

Reports of hydrocephalus in parrots were previously reported as Keller *et al*. (2011) who reported the first case of ex-vacuum hydrocephalus, in a yellow-headed amazon (*Amazona ochrocephala oratrix*) with central nervous system atrophy. Thurber *et al.* (2015) diagnosed ante-mortem hydrocephalus using computed tomography in an African grey parrot (*Psittacus erithacus erithacus*), in addition to the description by Fleming *et al*. (2003) who also diagnosed hydrocephalus in gray parrots using magnetic resonance, finally, Johnston *et al*. (2006) who reported communicating hydrocephalus in a Goffin's cockatoo (*Cacatua goffini*). The circulation and absorption of cerebrospinal fluid in birds is similar to that in mammals, although birds have species-specific characteristics in their central nervous system, such as the difference between the bird control center for flight and bipedal walking. As some pathological processes are often more studied and understood in mammals, the diagnosis and investigation of changes in birds becomes a challenge and in chronic processes, determining the initial cause is not always possible (Fleming *et al*., 2003; Marusak *et al.*, 2010; Keller *et al*., 2011; Schmidt and Ondreka, 2019). Hydrocephalus had complex pathogenesis and multiple causes. In humans, four major types of hydrocephalus are known, namely, obstructive, communicating, hypersecretory, and NPH, all potentially classified as congenital or acquired, and most caused by tumors produce obstruction in the ventricular system and cause hydrocephalus, often associated to a genetic syndrome (Koleva and De Jesus, 2020).

The clinical signs observed of absence of stimulus reflex and loss of proprioception on the left side are in line with what was observed by Schmidt and Ondreka (2019) in lesions originating from hydrocephalus. Also, the atrophy of the central nervous system added to the expansion of the ventricles observed in the reported case are also in agreement with the descriptions by Johnston *et al*. (2006).

The histopathological results of accentuated cortical atrophy and mild malacia are compatible with a lesion arising from cortical compression due to hydrocephalus. It was not possible to determine the exact location of the blockage in the CSF flow that caused the hydrocephalus, however, considering the negative results for the different tested etiologies, a congenital cause was suggested.

Glycogen storage disease is an inherited condition in humans resulting of dysfunctional glycogen metabolism in the hepatocytes, enzymatic impairment which disturbs the glucose homeostasis, and could be associated to hydrocephalus in children (Muzetti *et al*., 2021), young age condition also found in the evaluated parrot.

In additional direct microscopical observations in intestinal mucosal scrapings, it was possible to observe the presence of a structure compatible with Blastocystis sp. in samples of the small intestine. The agent is a common Stramenopiles present in the digestive tract of healthy or unhealthy animals, including humans, found in turkeys, domestic ducks, geese, chickens, quails, and ostriches, and may be related to zoonotic infection (Abe *et al.*, 2003). As it is a commensal parasite, its pathogenesis may occur in cases of immunosuppression, poor nutrition, and infection by other agents, being, in most cases, an opportunistic agent. In these cases, it is common for humans to develop signs and symptoms of

diarrhea, abdominal discomfort, and anorexia (Boreham and Stenzel, 1993; Stenzel *et al.*, 1994). The changes in the consistency of the excreta may be associated to the *Blastocystis* sp. opportunism.

Although the animal in question was potentially susceptible to various pathogens, no pathogen could be associated to the central nervous system fluid dynamics.

CONCLUSIONS

The case reported was characterized as chronic hydrocephalus in young immature *Amazona aestiva*, determined by macro and microscopic description of the lesions in the central nervous system. The histopathological findings of malacia and cortical atrophy associated are compatible with a secondary lesion of cortical compression due to the accumulation of CRF mostly in lateral ventriculi and the formation of hydrocephalus. Negative results in bacteriology, mycology, parasitology and PCR techniques to pathogens of relevance, with the absence of inflammatory changes in the central nervous system, and the young age of the animal suggested a congenital cause. The lack of a complete dissection of the central nervous system made it impossible to determine the exact location of the eventual malformation and blockage in the flow of CRF.

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