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CASE REPORT
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Ischemic Stroke Involving the Right Perforating Artery in a Bitch

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ABSTRACT

***Background*:** Cerebrovascular accidents arise from an interruption in cerebral blood flow and lead to neurological deficits associated with either infarction or hemorrhage. In dogs, ischemic infarcts result from obstructive events that impair cerebral perfusion and may appear as territorial or lacunar lesions. These infarcts are typically linked to embolic processes of diverse origins and concomitant diseases that influence their pathogenesis. This study aims to correlate clinical manifestations with the pathological lesions identified in a bitch diagnosed with ischemic stroke of the right perforating artery, with consequent encephalomalacia.

***Case*:** A 5-year-old Shih Tzu bitch presented with hyperacute neurological signs that began 6 days earlier, initially characterized by nystagmus and sialorrhea, and later progressing to epileptic seizures. Physical and neurological examinations revealed obtundation, non-ambulatory tetraparesis, right-sided head turn and pleurothotonus, unresponsive anisocoria, deficits in menace response and nasal sensation, and absence of proprioception in all limbs, indicating a lesion involving the forebrain and brainstem. Clinicopathological evaluation demonstrated normocytic normochromic anemia, neutrophilia, and mild hyperproteinemia. Magnetic resonance imaging identified an amorphous intra-axial hyperintense area on T2-weighted and FLAIR sequences involving the left thalamus and midbrain, with mild mass effect, consistent with infarction. Due to the guarded prognosis and lack of clinical improvement, euthanasia was elected. Cerebrospinal fluid (CSF) analysis was within normal limits. Gross neuropathology revealed thalamic-mesencephalic areas of malacia, and histopathology confirmed liquefactive necrosis with vacuolization, tissue rarefaction, gitter cell infiltration, and hemorrhage. Renal degeneration and necrosis were also observed. The definitive diagnosis was ischemic cerebrovascular accident due to occlusion of the right perforating artery.

***Discussion*:** The patient was younger than the age range typically reported for canine cerebrovascular accidents, which are more commonly observed in older dogs. The membranous glomerulonephritis identified suggested a protein-losing nephropathy, a condition associated with hypercoagulability and microthrombus formation, which may have contributed to ischemic infarction through occlusion of the right perforating artery. The elevated creatine kinase level was attributed to prolonged recumbency. The hyperacute neurological signs, including seizures, depressed mentation, postural reaction deficits, anisocoria, head turn, and pleurothotonus, were consistent with lesions affecting the thalamus, diencephalon, and midbrain. Magnetic resonance imaging revealed T1-weighted hypo-intensity and T2-weighted and FLAIR hyperintensity, corresponding to previously described features of ischemic infarction. The hyperacute onset and non-progressive course supported the diagnosis of ischemic stroke involving the right perforating artery. Gross neuropathological examination revealed areas of malacia with pronounced gitter cell infiltration, findings consistent with cerebral ischemia. This case demonstrates that ischemic stroke, although less common in young animals, should remain a differential diagnosis in patients presenting with hyperacute neurological deficits and highlights the value of targeted diagnostic investigations for etiologic determination.

Keywords: cerebrovascular accident; dog; encephalomalacia; midbrain.

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INTRODUCTION

Cerebrovascular accidents (CVA) result from temporary or permanent interruption of cerebral blood flow, leading to hyperacute neurological dysfunction caused by infarction or local hemorrhage [10]. Ischemic CVA may arise from disturbances in arterial or venous circulation due to vascular obstruction or abnormalities that induce hypoperfusion [5].

Cerebral perfusion is maintained by the circle of Willis, which includes the left and right internal carotid arteries (ICA) and their branches, such as the rostral, middle, and caudal cerebral arteries; the proximal, caudal, and distal perforating arteries; and the rostral and caudal cerebellar arteries supplied by the basilar artery [7]. The perforating arteries supply the thalamus, subthalamic nucleus, ventromedial midbrain, and medial pons [4]. Ischemia in dogs can therefore be classified as territorial infarcts, involving large-caliber vessels, or lacunar infarcts, affecting perforating cerebral arteries [1]. The involvement of these vessels is often associated with embolic phenomena, including thrombotic, septic, fatty, parasitic, or neoplastic emboli, indicating that underlying systemic diseases may significantly influence stroke pathogenesis [1].

Although the vascular territory of perforating arteries and their magnetic resonance imaging characteristics have been described, clinical neurological manifestations remain poorly documented. In this sense, this report aims to describe clinical and pathological findings in a Shih Tzu bitch with ischemic stroke involving the perforating artery.

CASE

A 5-year-old intact Shih Tzu bitch weighing 5.3 kg was examined at the Veterinary Hospital of the School of Veterinary Medicine and Animal Science of the Federal University of Goiás (Goiânia, Goiás, Brazil). The patient had a 6-day history of hyperacute neurological signs initially characterized by nystagmus and moderate-to-severe sialorrhea, later progressing to epileptic seizures. Real-time PCR for canine distemper virus was negative.

On physical examination, the dog showed obtundation, non-ambulatory tetraparesis, persistent sternal recumbency, and right head turn and pleurothotonus (Figure 1A). Vital parameters were within normal limits. Neurological assessment revealed anisocoria unresponsive to light (Figure 1B), reduced menace reflex and decreased nasal sensation on the left and absent on the right, mild generalized hypertonia, and absence of proprioceptive responses in all limbs. Neurolocalization suggested a lesion involving the forebrain and brainstem.

Given the hyperacute presentation, ischemic stroke, trauma, intoxication, and acute inflammatory conditions such as meningoencephalitis were included as differential diagnoses. The patient was hospitalized and received supportive care. Treatment included prednisolone[1] [Prediderm® - 1 mg/kg PO q12h], B-complex vitamins[2] [Citoneurin® 5000 - 2500 IU PO q24h], phenobarbital[3] [Fenocris® - 5 mg/kg IV q12h], and mannitol[4] [1,400 mg/kg IV q8h for 3 days]. Fluid therapy with lactated Ringer's solution[4] [3 mL/kg/h], and assisted feeding via syringe were instituted, with monitoring of urinary output and regular repositioning.

During hospitalization, the patient remained clinically stable with adequate food and water intake and normal urination and defecation; however, the neurological deficits persisted without improvement.

Clinicopathological evaluation included hematology, which showed normocytic normochromic anemia (hematocrit 31%; reference: 37-55%), neutrophilia without left shift, and mild hyperproteinemia (total plasma protein 8.4 g/dL; reference: 6.0-8.0 g/dL). Serum alanine aminotransferase (ALT) and creatinine concentrations were within reference intervals. Serum creatine kinase (CK) was markedly increased (532 U/L; reference: 1.15-28.40 U/L).

Brain magnetic resonance imaging was performed using a low-field 0.2 Tesla scanner[5] with and without gadolinium contrast, including T1-, T2-, FLAIR-, and T2*-weighted sequences (3-4 mm

Figure 1. Clinical presentation in a dog with ischemic stroke. A- Sternal recumbency with right-sided pleurothotonus and head turn. B- Anisocoria unresponsive to light stimulation.

slices). The diencephalon and brainstem contained an amorphous intra-axial lesion with regular margins and well-defined borders, showing moderately hyperintense signal on T2-weighted and FLAIR images and slight hypo-intensity on T1-weighted images (Figure 2A). The lesion measured 1.6 × 1.2 cm and extended from the left thalamus into the left midbrain, causing mild mass effect with slight compression of the left lateral ventricle.

Due to the extensive focal neurolocalization and guarded-to-poor prognosis, the owners elected euthanasia. Following confirmation of death, cerebrospinal fluid (CSF) was collected for cytological evaluation, which yielded normal biochemical and cytometric results for the species, with no evidence of cellular atypia.

A gross anatomopathological examination was performed to elucidate the patient's clinical condition. Upon opening the cranial vault, the meninges were intact, and the brain exhibited typical size and conformation. A transverse section revealed an area of malacia involving the thalamus region, characterized by surface depression, brownish coloration, poorly defined margins, and softened consistency (Figure 2B). The remaining brain regions showed typical coloration, consistency, morphology, and topography. In the abdominal cavity, the kidneys displayed moderate congestion. Samples of the brain and kidneys were collected for histopathological evaluation, fixed in 10% buffered formalin for 48 h, processed routinely, embedded in paraffin, and stained with hematoxylin and eosin (HE).

In the kidneys, there was tubular degeneration and necrosis, as well as membranous glomerulonephritis with mesangial thickening, in addition to marked accumulation of eosinophilic material in Bowman's space and within the tubular lumina (Figure 2D). Histopathological examination of the brain revealed, in the thalamus and midbrain, an extensive area of malacia characterized by liquefactive necrosis, loss of neural

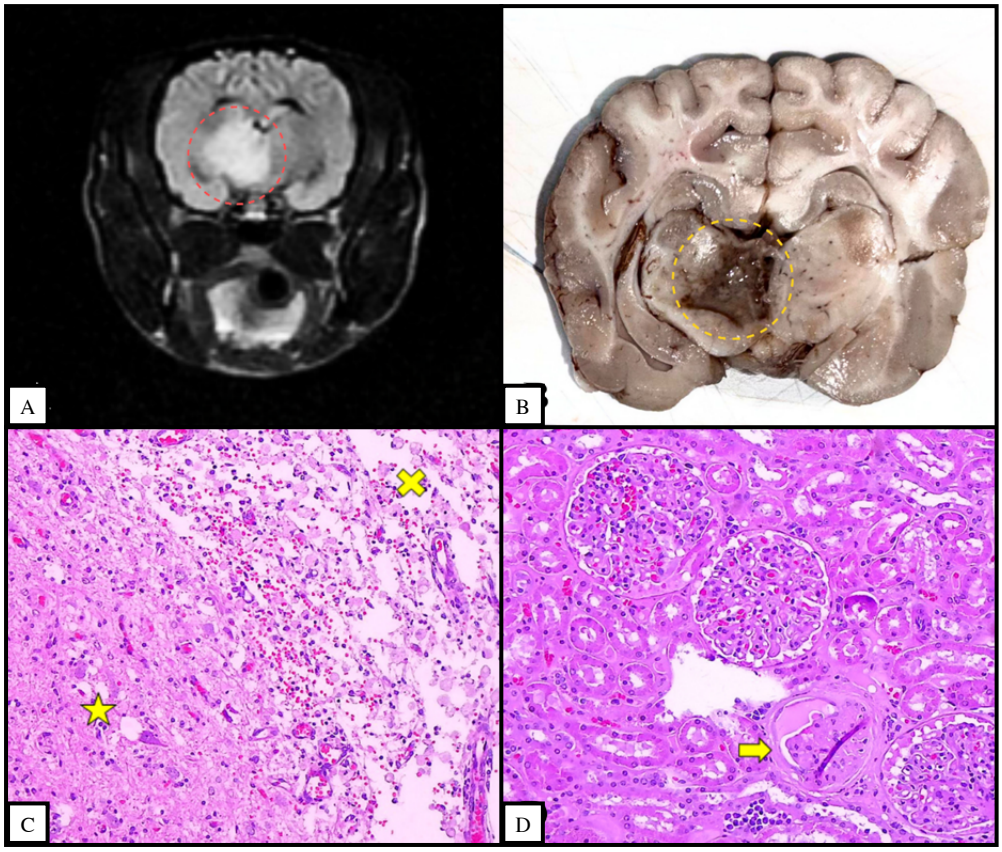

Figure 2. Imaging, gross, and microscopic alterations in a dog with ischemic stroke. A-Amorphous intra-axial lesion, moderately hyperintense, with regular margins and well-defined borders (dotted outline). B- Area of malacia in the thalamus region, characterized by a depressed surface, brown coloration, and irregular contours (dotted outline). C- Transition zone between normal neural tissue (asterisk) and malacia, characterized by liquefactive necrosis, neuropil vacuolization, tissue rarefaction, and gliosis, including marked infiltration of gitter cells (X). D- Accumulation of amorphous eosinophilic material in Bowman's space and glomerular sclerosis (arrow), as well as membranous glomerulonephritis characterized by thickening of the mesangial membrane [HE; 10×].

tissue architecture, neuropil vacuolization, and tissue rarefaction, accompanied by marked infiltration of gitter cells and associated hemorrhage (Figure 2C). These findings supported the diagnosis of ischemic stroke of the right perforating artery.

DISCUSSION

The patient in this report was 5-years old, younger when compared to those typically presenting with stroke, which are usually patients over 8-years of age [1]. This distribution is related to comorbidities and predisposing conditions requiring investigation, including immune-mediated hemolytic anemia, protein-losing nephropathies, hyperadrenocorticism, hypothyroidism, hyperlipidemia, neoplasms, and sepsis [3,6]. In this case, the neurological presentation was considered secondary to renal alterations since membranous glomerulonephritis and eosinophilic material accumulated within Bowman's space and tubular lumina was observed. Protein-losing nephropathies in dogs, such as glomerulonephritis, result in extravasation of proteins into the glomerular ultrafiltrate and urine, including anticoagulant factors such as antithrombin, which favors hypercoagulability and thrombus formation [3]. The increased CK level was attributed to prolonged recumbency because its short half-life and high sensitivity to skeletal muscle injury result in rapid elevation during myopathic processes, consistent with this patient's condition [6].

The clinical signs observed were suggestive of stroke, based on the symptomatology characteristic of vascular changes, with hyperacute neurological signs. Thalamic involvement can explain the epileptic seizures, altered mental status, postural reaction deficits, and absence of ipsilateral menace response [8]. Head turn and pleurothotonus changes indicate right-sided diencephalic involvement, while the midbrain lesion supports the anisocoria with right-sided miosis due to oculomotor nerve nucleus lesion, and right nasal sensory loss due to interruption of pathways between the brainstem and forebrain, along with decreased consciousness and edema contributing to reduced contralateral nasal sensation [8].

The magnetic resonance imaging (MRI) findings were compatible with the clinical presentation and consistent with literature reports describing ischemic infarcts as hypointense on T1-weighted images and hyperintense on T2-weighted and FLAIR images [6]. However, similar alterations may also occur in meningoencephalitis due to increased fluid content in neural tissue [2]. In this case, the hyperacute and non-progressive clinical evolution, combined with focal involvement of the perforating artery territory, strongly supported the antemortem diagnosis of ischemic stroke affecting the right perforating artery, whereas meningoencephalitis typically presents with acute, progressive signs [2].

Gross and histopathological alterations in the brain correspond to ischemic injury resulting from interruption of blood flow in the vascular territory of the right perforating artery. Histologically, malacia was confirmed in the area supplied by this artery, associated with marked infiltration of gitter cells, findings compatible with cerebral ischemia [9]. In histomorphological terms, gitter cells indicate the chronicity of the lesion and act in the removal of necrotic tissue for subsequent tissue repair. In clinical terms, these alterations reflect the evolution time of the condition, considering that euthanasia was performed ten days after the onset of clinical signs [9].

Although uncommon at this age, cerebrovascular accidents should be considered in the differential diagnosis of hyperacute neurological conditions in adult animals. In addition, the clinical signs and imaging findings described contributed to the diagnosis of this type of stroke, with anatomopathological confirmation. As a limitation of this case, some complementary exams recommended for patients with suspected stroke [3] were not performed, including serum total proteins (albumin and globulins), urinalysis, urine protein/creatinine ratio, D-dimer, coagulation profile, and investigation of endocrine diseases.

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