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PARTICULARITIES OF HORNER'S SYNDROME IN DOGS AND CATS: A LITERATURE REVIEW

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ABSTRACT

Horner's syndrome (HS) is a neurological alteration resulting from the interruption of the sympathetic innervation of the eyeball, characterizing the ophthalmic clinical signs; miosis, enophthalmos, eyelid ptosis, and prolapse of the third eyelid. The sympathetic nervous tract of the eye is extensive, dividing into central, pre- and post-ganglionic neurons, or first, second, and third-order neurons, as mentioned in older literature. From this, HS should be classified according to the neurolocation of the lesion, there are other possibilities that are not well elucidated, such as hypothyroidism and idiopathic presentation. The gold standard definitive diagnosis is made through the instillation of 5 to 10% cocaine-based eye drops, however, due to the difficult access, the 1 to 10% phenylephrine solution can be a substitute, in both cases the animals positive for HS continued with miosis and the other signs already presented. Because it is a disease secondary to an injury during the nervous tract, a systematic investigation using imaging tests is necessary, since treating the underlying cause is the beginning of treatment. This, as already mentioned, will depend on the location of the lesion, for symptomatic treatment phenylephrine 1 to 10% is used, which temporarily relieves parasympathetic signs. Thus, this study aims to perform a literature review on the particularities of Horner's syndrome in dogs and cats.

Keywords: Phenylephrine. Sympathetic disorder. Ophthalmology.

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Revista ARACÊ
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LITERATURE REVIEW

Horner's syndrome (HS) is a set of neuro-ophthalmic disorders resulting from the disruption of the sympathetic innervation of the eye. This alteration can compromise several ocular and periocular functions, leading to characteristic clinical signs, such as miosis, which results in anisocoria, eyelid ptosis, enophthalmos, and prolapse of the third eyelid. In most cases, HS occurs as a condition secondary to diseases that affect the sympathetic nervous tract, including trauma, inflammatory processes, neoplasms, or vascular alterations. Therefore, a detailed diagnostic approach is essential to determine the location of the lesion and identify the underlying cause, enabling appropriate and effective treatment (NELSON; COUTO, 2015).

The first description was in 1727, when changes were observed in the face and eye of animals submitted to resection of the intercostal nerves. In the following years, the neuro-ophthalmic disorder was reported more often, but without a theoretical basis. It was only in 1852 that the French physiologist Claude Bernard analyzed the disorder in detail with other doctors and professionals in the area (KHAN; BOLLU, 2018). However, it was only in 1869 that the Swiss ophthalmologist Friedrich Horner formally described it, reporting a case in a woman, in which he substantiated the characteristic ophthalmic manifestations, from then on the disorder was named Claude Bernard-Horner syndrome or just Horner's syndrome (MAZZA; OLIVEIRA, 2022).

Horner's syndrome is a relatively common condition in clinical veterinary medicine, being reported mainly in dogs and cats. In dogs, idiopathic HS is the most prevalent form, accounting for about 40-50% of diagnosed cases. Breeds such as Golden Retriever and Collie demonstrate a greater predisposition to the development of the syndrome without an apparent cause (ORLANDI et al., 2020). In cats, HS is often associated with otitis media, cervical trauma, and neoplastic processes, with a lower incidence of the idiopathic form compared to dogs (ZWUESTE; GRAHN, 2019).

The age distribution of HS is not well defined, since the syndrome can occur in young and old animals. However, the idiopathic form tends to be more common in middle-aged to elderly dogs. In felines, the prevalence of HS is lower than in dogs, and its incidence may be underestimated due to diagnostic difficulty in some cases (MARTINS et al., 2020).

Studies indicate that the location of the lesion in the sympathetic pathway also influences the epidemiology of HS. In dogs, most lesions are classified as postganglionic,

Revista ARACÊ
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usually associated with otitis media and orbital diseases. In cats, preganglionic lesions are more frequent, mainly due to cervical trauma and mediastinal neoplastic diseases (VIANA et al., 2022).

Disorders involving HS denounce some lesion in the sympathetic segment, being classified based on the level of dysfunction within the occulosyptic pathway (RAMKUMAR *et al.*, 2023). Its anatomy is broad and extensive, joining other nerve fibers, such as the vagus-sympathetic trunk. The lesions can be classified based on location, and may be a dysfunction of the central sympathetic neuron, pre- and post-ganglionic, in older literature they can be called first, second and third order neurons (ZWUESTE; GRAHN, 2019).

The causes of central HS include a variety of neurological conditions, such as cervical myelopathies, intracranial neoplasms, traumatic brain injury (TBI), and infectious processes affecting the central nervous system (ZWUESTE; GRAHN, 2019). The syndrome can manifest both unilaterally and bilaterally, depending on the extent of the lesion. When severe damage to the cervical cord occurs, particularly in the C1-C5 regions, there are reports of bilateral HS associated with ventilatory impairment, since this area also houses the centers responsible for respiratory control.

Murthy et al. (2023) analyzed the relationship between Horner's syndrome and cervical myelopathies and concluded that, despite the correlation between both conditions, the presence of HS should not be used alone as a physiological biomarker predictive of myelopathies. This observation reinforces the need for a comprehensive diagnostic approach, including imaging, neuro-ophthalmic testing, and thorough clinical evaluation for accurate identification of the underlying cause.

In addition, central lesions of the sympathetic pathway may be associated with degenerative processes, such as cervical spondylomyelopathy (Wobbler's syndrome) in large dogs, inflammatory diseases such as meningoencephalomyelitis of unknown origin (MUO) and cerebrovascular accidents (ETTINGER; FELDMAN, 2022).

Revista ARACÊ
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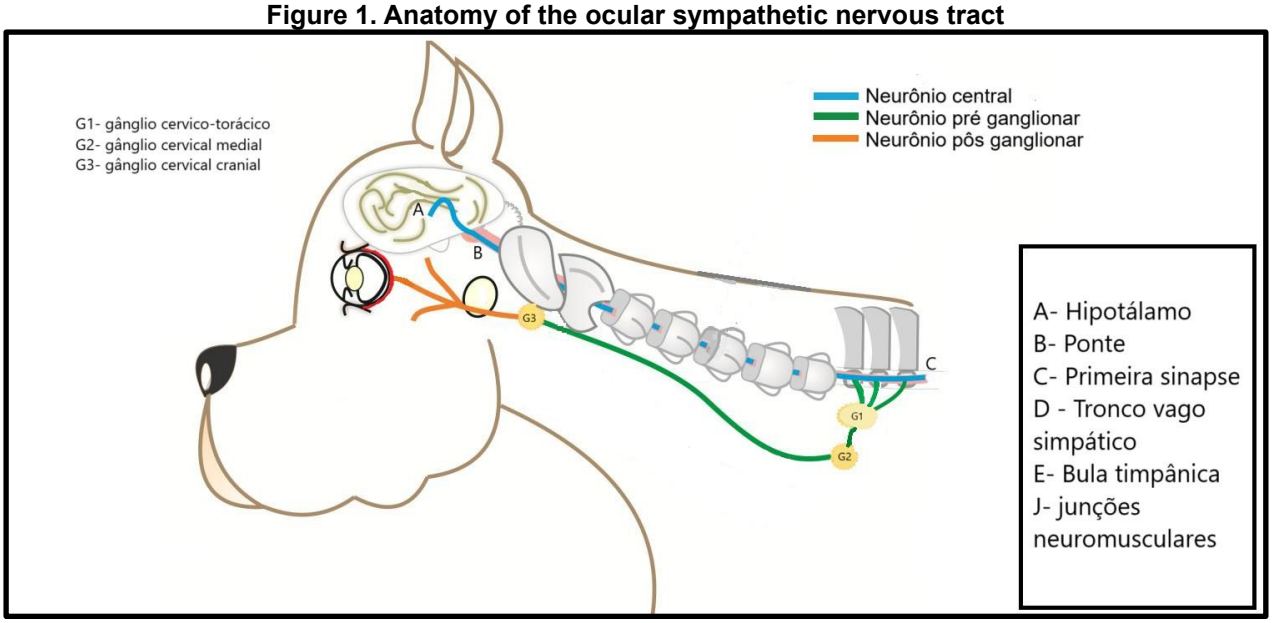
Figure 1. Anatomy of the ocular sympathetic nervous tract



Source: Adapted from Antunes, 2011.

After synapses in the thoracic segments of the spinal cord (T1, T2 and T3), the sympathetic neuron is called preganglionic neuron. This neuron leaves the spinal cord and follows its path through the cervico-thoracic and cervico-medial ganglia, projecting cranially through the cervical region, where it synapses with the cranial cervical ganglion. During this path, the vagosympathetic trunk and the brachial plexus are formed, structures of great anatomical relevance. Injuries along this path can result in HS of preganglionic origin (ETTINGER; FELDMAN, 2022).

Several conditions can lead to these lesions, including fibrocartilaginous embolism, spinal cord trauma, thoracic and cervical neoplasms, with mediastinal lymphoma in FeLV positive felines being an example, although rare (SCHIMANSKI, 2023). In addition, iatrogenic causes have been reported, such as injuries secondary to surgical procedures, including compression or avulsion of the brachial plexus during orthopedic surgeries of thoracic limbs, as well as surgical manipulations in the cervical region, especially in procedures such as esophagostomy (SUGUINO, 2022). There are also descriptions of HS secondary to venous punctures in the jugular region, due to the anatomical proximity of the vagus-sympathetic trunk, which highlights the importance of precise and careful techniques in these procedures (ETTINGER; FELDMAN, 2022).

After the synapse in the cranial cervical ganglion, the sympathetic neuron is called the postganglionic neuron. This neuron follows a delicate path, crossing the tympanic bulla in the middle ear and joining the fibers of the trigeminal nerve before entering the orbit.

Revista ARACÊ
ISSN: 2358-2472

Upon reaching its final destination, it establishes neuromuscular junctions in the iris dilator muscle and periorbital smooth muscle. The lack of sympathetic stimulation in these structures results in the characteristic clinical signs of HS, such as miosis, enophthalmos, eyelid ptosis, and prolapse of the third eyelid (ZWUESTE; GRAHN, 2019).

Postganglionic neuron lesions are the most frequently observed in veterinary medicine. The main cause is otitis media or internal otitis, often associated with peripheral vestibular syndrome and facial nerve palsy (VIANA et al., 2022). In addition, other conditions can result in postganglionic HS, including head trauma, surgical procedures in the orbital region, and retrobulbar neoplasms. Although less common, retrobulbar abscesses, trigeminal nerve neuritis, and facial neuritis can also trigger the syndrome, although its incidence may be underestimated due to the diagnostic difficulty and underdiagnosis of some of these conditions (ANTUNES; BORGES, 2011).

Idiopathic HS is considered the most common among the possible origins, with the Golden dog breed as the most predisposed (ORLANDI *et al*., 2020). This disorder is self-limiting, with a tendency to resolve with treatment with 1% phenylephrine in 15 to 30 days (ZWUESTE; GRAHN, 2019). In cases of hypothyroidism in dogs, the most common neurological findings are facial nerve palsy and Horner's syndrome, but its pathophysiology is not well elucidated (ANOOP *et al.*, 2020).

Horner's syndrome is characterized by a series of clinical signs that result from dysfunction of the sympathetic pathway that innervates the eye. One of the most common signs is miosis, which occurs due to the loss of innervation of the iris dilator muscle, leading to anisocoria in unilateral cases. The absence of sympathetic stimulus causes an imbalance in the action of the oculomotor nerve, which performs the parasympathetic innervation of the eye, without opposition from sympathetic control (ETTINGER; FELDMAN, 2022).

Another important sign of Horner's syndrome is enophthalmos, which occurs due to the loss of sympathetic stimulus in the retractor muscles of the eye bulb. With the relaxation of these muscles, the eye is retracted into the orbit, resulting in a decrease in eye projection. Eyelid ptosis, in turn, is an indirect consequence of enophthalmos, being associated with loss of muscle tone and displacement of the upper eyelid. A similar phenomenon is observed in third eyelid prolapse in dogs (ZWUESTE; GRAHN, 2019).

In felines, Horner's syndrome may have a distinctive feature: direct sympathetic innervation on the nictanning membrane, which makes prolapse of this membrane a more frequent clinical sign compared to dogs. This change is most noticeable in cats (Figure 2)

Revista ARACÊ
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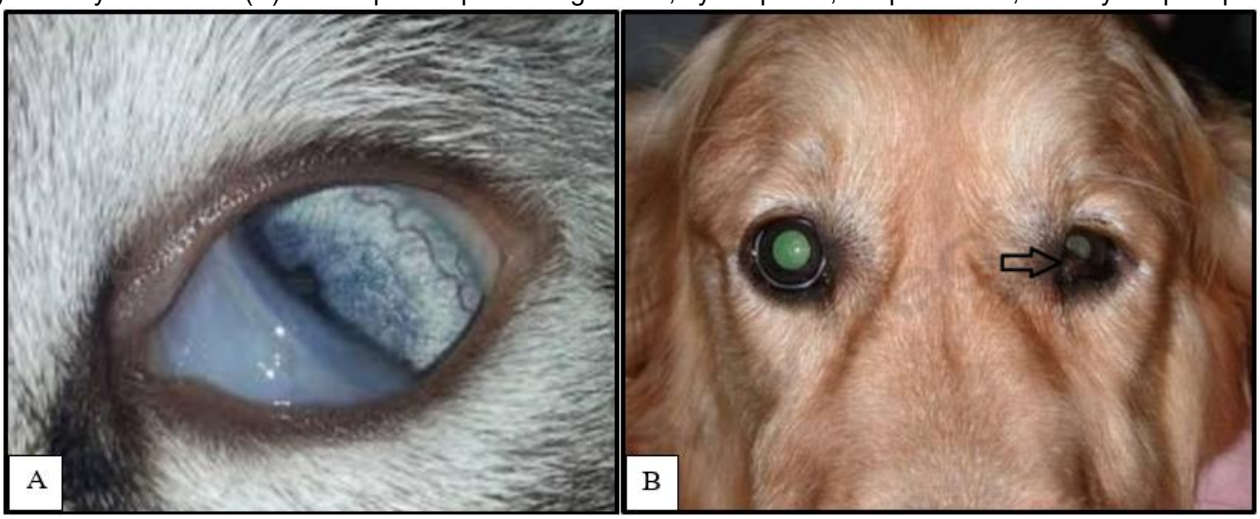
and must be carefully differentiated from other conditions, such as Haw syndrome, which can also cause similar symptoms. Haw's syndrome is an important differential diagnosis to be considered in the diagnosis of Horner's syndrome in cats (MARTINS et al., 2020).

Figure 2. Presentation of clinical signs of HS. In (A) feline patient presenting miosis and prolapse of the third eyelid very evident. In (B) canine patient presenting miosis, eyelid ptosis, enophthalmos, third eyelid prolapse



Source: Adapted from Guimarães (2018).

The gold standard diagnostic test to identify Horner's syndrome is the instillation of a cocaine eye drop (5-10%) over the affected eye. Cocaine works by blocking the reuptake of norepinephrine at the neuromuscular junction, which increases the availability of norepinephrine and generates an intense adrenergic response. This results in mydriasis (pupil dilation) and reversal of the other signs of the syndrome in healthy eyes. In eyes with Horner's syndrome, however, clinical signs, such as miosis, persist, indicating the presence of the lesion (ZWUESTE; GRAHN, 2019). This test is highly effective, but the limited availability of cocaine in many locations makes using alternatives a viable option.

Phenylephrine (1-10%) is a viable alternative for diagnosis, treatment, and localization of postganglionic lesions. This drug is a direct sympathomimetic agonist, which induces mydriasis in the affected eye. In cases of postganglionic Horner's syndrome, mydriasis occurs about 10 to 20 minutes after eye drop instillation (LEE; HORIKAWA, 2021). Phenylephrine is effective in identifying lesions that occur after the upper cervical ganglion, and is useful in determining the location of the lesion (RODRIGUES et al, 2019)

Another testing option is the use of hydroxyamphetamine (1%), an indirect sympathomimetic. This drug is capable of releasing norepinephrine in nerve endings, producing mydriasis under normal conditions. However, in the case of Horner's syndrome,

Revista ARACÊ
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the response to the drug depends on the location of the lesion. Hydroxyamphetamine is useful for differentiating between preganglionic and central lesions, although it is not able to accurately identify the lesion between these two locations. Bilateral mydriasis occurs within 45 minutes of use, and is indicative of sympathetic nervous system dysfunction (ZWUESTE; GRAHN, 2019).

It is important to remember that a central lesion is usually associated with additional neurological deficits, such as signs of brainstem impairment, thalamus, or cervical myelopathy, which can be identified on a detailed physical examination. Imaging tests, such as magnetic resonance imaging (MRI) and computed tomography (CT) scans, may be useful to visualize central nervous system structures and identify lesions involving the brainstem or spinal cord, confirming the central origin of Horner's syndrome. (ZWUESTE; GRAHN, 2019).

Although the diagnosis of Horner's syndrome relies heavily on pharmacological tests, imaging tests play an important role in investigating the underlying cause of sympathetic dysfunction. Magnetic resonance imaging (MRI) is particularly useful for detecting central lesions, such as tumors, infarctions, or vascular malformations affecting the brainstem or thalamus. When cervical or preganglionic injury is suspected, cervical spinal cord MRI can also help identify compressions or other pathologies that affect the sympathetic pathways (RODRIGUES et al, 2019).

In addition, laboratory tests may be performed to investigate possible systemic causes, such as autoimmune diseases, infections, or tumors, which can lead to secondary Horner's syndrome. Blood tests, such as complete blood count, liver and kidney function, and the search for specific antibodies, may be indicated depending on the clinical context (THRALL, 2023).

The first step in the treatment of Horner's syndrome is the identification and treatment of the primary cause. This may involve treating conditions such as ear infections, tumors, or infections. When the syndrome is caused by a secondary pathology, such as otitis media, treatment usually includes ear cleaning and the use of specific antibiotics or anti-inflammatory drugs (BARKYN, 2023). In cases where the cause is a tumor or other neurological condition, treatment may involve surgery, radiotherapy, or chemotherapy (RODRIGUES et al, 2019).

In addition, some treatments aim to relieve the clinical signs of the syndrome. The instillation of mydriatic eye drops, such as phenylephrine (10%) is often used to treat

Revista ARACÊ
ISSN: 2358-2472

miosis, one of the most evident symptoms. However, it is worth noting that correcting miosis with eye drops does not resolve the underlying dysfunction of the sympathetic nervous system, but only temporarily controls the signals (ZWUESTE; GRAHN, 2019).

In addition to treating the underlying cause and using eye drops, some complementary therapies, such as acupuncture, have been explored as a way to aid recovery, especially when the syndrome is not associated with a treatable cause or irreversible injuries (MARTINS et al., 2020). Although acupuncture is not a conventional treatment, there are reports of improvement in some cases, especially in pain management and stimulation of the peripheral nervous system (TAFFAREL; FREITAS, 2009).

The prognosis of Horner's syndrome depends largely on the nature of the injury and the effectiveness of treatment of the primary cause. In many cases, when the injury is not severe, clinical signs may improve or disappear spontaneously after a period that can vary from 2 to 8 weeks. However, if the cause is more severe or if there is central nervous system involvement, the prognosis may be more poor, and recovery may be partial or non-existent (BARKYN, 2023).

FINAL CONCLUSIONS

Horner's syndrome is a significant neuro-ophthalmic disorder, which can be indicative of several underlying conditions, from benign diseases to serious processes, such as neoplasms or trauma. Its early identification is essential, as the presence of clinical signs such as miosis, eyelid ptosis, enophthalmos, and prolapse of the third eyelid can direct the diagnosis of systemic or local pathologies, allowing the adoption of appropriate therapeutic interventions. Although the treatment of the syndrome depends on the primary cause, the correct clinical evaluation and the use of diagnostic tests, such as phenylephrine and hydroxyamphetamine, are essential for the precise location of the lesion. In addition, HS can be self-limiting in some forms, such as idiopathic, which provides a favorable prognosis in specific cases.

In this way, Horner's syndrome represents not only a diagnostic challenge, but also an opportunity to identify serious underlying diseases, requiring a careful, diagnostic and therapeutic approach to ensure the patient's recovery and well-being.

Revista ARACÊ
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