THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
March 2024 Vol.4(3),1193-1209
Popular Article

Horner's Syndrome in Companion Animals: An Update

***Sonali Mahore[1], Shashi Pradhan[2], Ranbir Jatav[3], Devendra Gupta[4], Anju Nayak[5], Jolly Thomas[6], Radha[7]**
Research scholar[1,6&7], Assistant Professor[2&3], Professor and Head[4&5](Departement of Veterinary Medicine and Departement of Veterinary Microbiology)
Department Of Veterinary Medicine
College of Veterinary Science and A. H., Ndvsu, Jabalpur
https://doi.org/10.5281/zenodo.10896261

Introduction

Horner's syndrome affecting the face and eye, unilaterally or bilaterally, is named after the Swiss ophthalmologist J. F. Horner in recognition of his valuable contribution: complete description of this malady and the pathobiomechnism: disruption of a nerve pathway originating from the brain leading to oculosympathetic paresis in 1869. Penderis (2015) documented the pioneering research contribution of F.P. du Petit, who in as early as 1727 investigated the basics of this biomedical anomaly through creation of surgical cervical dysplasia and intercostal lesions in dogs. Notably, whereas CNS neuronal lesions are rarely reported, pre-ganglionic and post-ganglionic neuronal lesions are predominant (Van den Broek, 1987). Meticulously scanned published literature on the malady afflicting the companion animals, namely cats and dogs around the globe revealed a paucity of evidence-based clinical reports. Evidently, Horner's syndrome is not a separate disease, but represents a secondary clinical manifestation of a diverse group of diseases, targering the head and neck region. In perspective, this presentation aims to furnish updated information on this unique pathobiological episode in cats and dogs.

Pathophysiological Aspects

Miosis was established as the cardinal diagnostic sign of Horner's syndrome. Ptosis, third eyelid prolapse, and conspicuous enophthalmos are common clinical manifestations, other symptoms like facial or aural vasodilatation are less frequently observed. Horner's

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

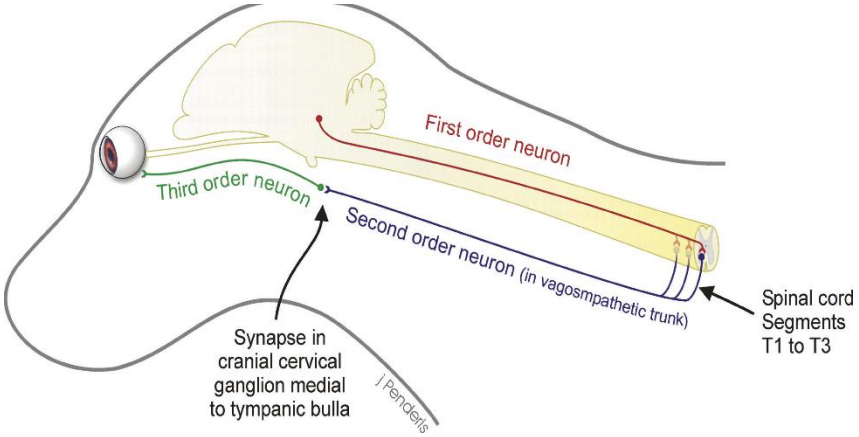
syndrome may also be related to a plethora of medical problems such as a stroke, spinal cord injury,inter-vertebral disc displacement, traumatic insult like a bite or blunt wound, tumor, and middle or inner ear involvement (otitis media or otitis interna). Disruption of the oculo-sympathetic pathway by a variety of lesions in different sites appears to be the path biomechanism. In this context, it is pertinent to put on record that the precise etiology of Horner' syndrome could not be determined in 50% of dog and 42.3% of cat patients (Kern *et al*., 1989). This underscores the need for result- oriented research in the higher centres of learning in collaboration with the field veterinarians.

Currently, diagnosis of Horner's syndrome is based on anamnesis, clinical signs, pharmacological testing through topical application 1% percent solution of phenylephrine in both eyes, magnetic resonance imaging (MRI) and radiographic examinations. The optimized treatment of depends on proper evaluation of the underlying etiology, Symptomatic therapy α-1 blocker such as 1% or 10% solution of phenylephrine is stated to be effective (Boydell, 1995).

The autonomic nervous system comprises the sympathetic parasympathetic components. In the eye ball, the functional sympathetic nerve fibres dilate the pupil, widen the eye lids, drop the third eyelid, and keep the eye in the normal forward position within the socket. On the other hand, the parasympathetic nerves constrict the pupil,raise the third eyelid and retract the eye ball for protection from any injury. Notably, if the sympathetic nerve fibres controlling one of the eyes are damaged from any traumatic insult, only the parasympathetic nerves function uncontrolled, and Horner's syndrome is created.

Optic Sympathetic Pathway

Sympathetic innervations of the eye exhibit a characteristic three-tier hierarchy.



First order upper motor neurons

In the hypothalamus and rostral midbrain, axons from the cell bodies travel via the brain

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

stem and cervical spinal cord to connect with second order neurons.

Second order pre-ganglionic neurons

In the lateral horn of the grey matter of the spinal cord, cell bodies exist in the T1 -T3 segment. The axons emerge to merge with the thoracic sympathetic trunk and travel through the middle cervical ganglion and the cervico-thoracic ganglion. In the thoracic inlet shared epineurium the sympathetic trunk and vagus nerve combine inside a shared epineurium. The sympathetic trunk diverges and terminates in the cranial cervical ganglion, located ventromedial to the tympanic bulla.

Third order post-ganglionic neurons

The axons leave the cranial cervical ganglion to form a plexus around the internal carotid artery. Some of the fibres penetrate the tympanic bulla in the ventral surface. Post-ganglionic fibres then join the internal carotid artery and travel through the tympano-occipital fissure and carotid canal to enter inside the cranial cavity. The post-ganglionic fibres travel ventrally towards the trigeminal ganglion before emerging through the occipital fissure along with the ophthalmic branch of trigeminal nerve. The fibres constitute the long ciliary and nasociliary nerves, which supply the periorbital and eyelid muscles concurrent with the iris dilator (Sumanth and Pathak, 2022).

Gross pathological features

Table 01: Location of morbidity, types of lesions and neurological deficit

Targeted organ	Lesion	Neurological Deficit
Cervical spinal cord	Focal myelopathy External injury Fibrocartilage emboli Intervertebral disc extrision	Spastic tetraplegia, Dyspnea Spastic Hemoplegia: ipsilateral
T1-T3 spinal cord	Focal myelopathy External injury Fibrocartilage emboli Neoplasm Diffuse myelomalacia	Tetraparesis and ataxia or tetraplegia with LMN deficit in thoracic limbs, and UMN and GP deficit in pelvic limbs Diffuse LMN signs and loss of nociception with diffuse myelomalcia
T1-T3 ventral r oots, proximal spinal nerves	Avulsion of roots of brachial plexus lymphoma	LMN paresis or paralysis of the ipsilateral thoracic limb with variable loss of nociception
Cranial thoracic	Lymphoma	None, if confined to the trunk

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

sympathetic trunk, cervicothoracic ganglion, middle cervical ganglion	Nerve sheath neoplasm	or ganglia
Cervical sympathetic trunk	Injury by surgery, jugular venipuncture, dog bite Neoplasm: nerve sheath, lymphoma, thyroid adenocarcinoma	None, if unilateral: bilateral lesion interfere with laryngeal and esophageal functions because of associated vagal nerve involvement
Tympanc (middle ear) cavity in small animals	Otitis media Neoplasm	Clinical signs of peripheral vestibular system dysfunction: ipsilateral ataxia, head tilt, abnormal nystagmus, facial paresis or paralysis, facial tetanus
Retrobulbar	Injury, abscess Neoplasm	Varies with degree of involvement of optic and oculomotor nerves, which influence pupillary size and vision (optic).

(De Lahunta *et al*., 2020)

Table 02: Etiologies on the basis of ganglionic involvement

Location	Etiology
Preganglionic	Idiopathic, Iatrogenic- Brachial plexus block, Epidural, ropivacaine vagus nerve stimulator placement, Thoracic surgery, Traumatic-During birth, Brachial plexus avulsions, Neoplastic- Mediastinal lymphoma, PNST of vagus nerve, Infection -Tick paralysis (*Ixodes holocyclus*)
Postganglionic	Neoplasia, Neuroblastoma, Carotid body paraganglioma,Idiopathic , Iatrogenic- Post-operative TECA-LBO, infectious- Otitis media/interna
Central	Traumatic- Air pellet (spinal cord), Infectious- NeosporaOther- Fibrocartilagenous embolism (cervical)
Unspecified	Diabetic polyneuropathy

(Zwuetse *et al*., 2019)

Idiopathic Horner's Syndrome

Representing nearly 50% of canine presentations, idiopathic Horner's syndrome deserves special attention of the international veterinary fraternity. Though family pets ranging in age from 4 to 13 years are known to be afflicted, the age group of 5 to 8 year is most vulnerable in the carefully scanned literature. About 40% of the reported cases of Horner's syndrome in cats are idiopathic (Kern *et al*., 1989). Clinical symptoms: unilateral or bilateral,

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

acute in onset, and caused by both pre- and post-ganglionic lesions.

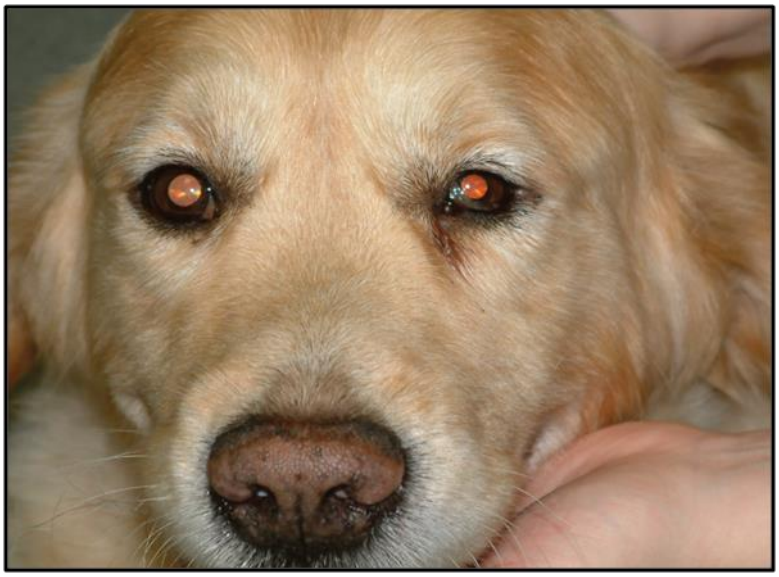


Fig 01: Idiopathic horner's syndrome in a golden retriever.

Pathogenesis

Otic neurological disorders in Horner's syndrome

- **Facial nerve paralysis**

Closely related facial nerve paralysis and Horner's syndrome bioepisode is accentuated by the middle and inner ear disorders (otitis media, otitis interna), more often facial nerve paralysis. The facial canal, connected to the tympanic cavity, exists without a protective bony sheath for a short distance in the petrosal region of the temporal bone. Because of geometric configuration, the facial nerve remains vulnerable to otitis media. Depending on severity of the lesion, total facial paresis, or paralysis ensues in individual canine and feline patients. Widening of the palpebral fissure, absence of spontaneous movement, and ipsilateral drooping and immobility of the ear and lip constitute the wide spectrum symptoms of CN VII motor dysfunction. The clinical profile includes blinking, absence of nostril abduction during inspiration, and tilting of the nose towards the unaffected side because of the marked muscular imbalance (Garosi *et al*., 2012).

- **Vestibular Syndrome**

Because of biodegradation of the vestibular receptors present in the inner ear and vestibulocochlear nerve, otitis media and otitis interna may promote peripheral vestibular syndrome. Because the facial and the oculosympathetic nerves are juxtaposed inside the petrous temporal bone, facial nerve paralysis and/or Horner' syndrome can be detected from the vestibular symptoms**.** Wide ranging clinical symptoms: conspicuous head tilt, ataxia,

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

toppling down suddenly, leaning, rolling, circling, spontaneous or positional nystagmus, and positional strabismus point to vestibular illness (Rossmeisl, 2010).

Because of the close geometric proximity of the facial nerve and the ocular sympathetic nerve, and location of vestibular nerve in the petrous temporal bone, in an animal suffering from otitis facial nerve paralysis and/ or Horner's syndrome may be detected alongwith the vestibular symptoms (Garosi *et al*., 2012).

In peripheral vestibular dysfunction in dogs and cats, the well-established primary cause is otitis media and/or otitis interna (Schunk and Averill, 1983). Additionally, hypothyroidism, auditory neoplasia, nasopharyngeal and otopharyngeal polyps, iatrogenic ototoxicity (especially arising from the injudicious use of aminoglycoside antibiotics, topical iodophors, or chlorhexidine), and acute idiopathic peripheral vestibular disease may be involved (Garosi *et al*., 2012).

The method of treating vestibular disorder is addressing the underlying cause. Meclizine (12.5 mg orally every 12 hours for dogs and 6.25 mg orally every 12 hours for cats), diazepam (0.1-0.5 mg orally every 8 hours for dogs and 1-2 mg orally every 12 hours for cats), and/or maropitant (1 mg orally or subcutaneously or 2 mg orally every 24 hours for dogs) may be used to lessen the symptoms of an acute vestibular disorder (Garosi *et al*., 2012).

Fig 02: Left brachial plexus mass lesion in a cat with impaired sympathetic innervation to ipsilateral side of head, resulting in left horner's syndrome.

Breed predilection

The breeds such as doberman, pinscher, collie, golden retrievers, labrador retrievers, shetland sheepdogs, and weimaraners could be predisposed (Herrera and Suranit, 1998). Compared to other breeds, golden retrievers exhibited a significantly greater incidence of idiopathic horner syndrome in two reports (Simpson *et al*., 2015).

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

Pathoclinical profile

The degree of malfunction in the oculosympathetic circuit determines how horner's syndrome is classed as central, preganglionic, or postganglionic, but the symptoms are the same in small animals regardless of the lesion's location. The lack of sympathetic innervation to the eye results in miosis, ptosis, enopthalmos and third eyelid protrusion.

Miosis

The most prevalent manifestation of horner's syndrome is miosis of the diseased eye, which happens as a result of the iris dilator muscle losing its innervations. The parasympathetic portion of cranial nerve III innervates the iris sphincter muscle, which is subsequently permitted to work unchecked and causes pupillary constriction (Collins and O'Brien, 1990). Another distinctive quality of the iris dilator muscle is that it receives dual innervation from the sympathetic and parasympathetic nervous systems. The iris dilator muscles and pupil dilation are made possible by the sympathetic component. Contrarily, the parasympathetic innervation stops this muscle from contracting. The parasympathetic innervation's inhibiting effect further limits pupil dilation and worsens the miosis when the sympathetic route is impaired (Zwueste and Grahn, 2019). Because the damaged eye cannot dilate to the same extent under scotopic conditions, anisocoria will occur with unilateral lesions and will be most evident like the natural eye (Penderis, 2015). In the damaged eye, pupillary light reflexes and vision will be unaffected (Collins and O'Brien,1990).

Fig 03: Clinical manifestation of unilateral horner's syndrome in right eye of a cat with miosis.

Ptosis

A narrower palpebral fissure results from ptosis, or drooping of the upper eyelid, in the eye that is affected (Van den Broek, 1987). It is believed that the delicate muscles that make up the eyelids- referred to as Müller's muscle-lose sympathetic input with the onset of ptosis (Mughal and Longmuir, 2009).

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

Fig 04: Drooping of left upper eyelid

Enopthalmos

In the orbit, the globe is maintained in the normal anterior configuration with the help of circularly arranged orbital smooth muscles. The antagonistic retractor bulbi muscles, relaxing as a bioresponse to deranged sympathetic input pull the globe back into the orbit to cause enophthalmos (Collins and O'Brien, 1990).

Fig 04: Enophthalmos and upper eyelid ptosis in the right eye

Third eyelid protrusion

The third eyelid protrusion of varying magnitude in individual patients is the second most commonly recorded clinical symptom (Kern *et al*., 1989). In dogs, this passive bioepisode follows enophthalmos (Van den Broek, 1987). In cats, an additional active component is sympathetically-mediated smooth muscle in the third eyelid. Horner's lesion impairs the muscle's tonicity to keep the eyelid retracted (Murphy *et al*., 2013).

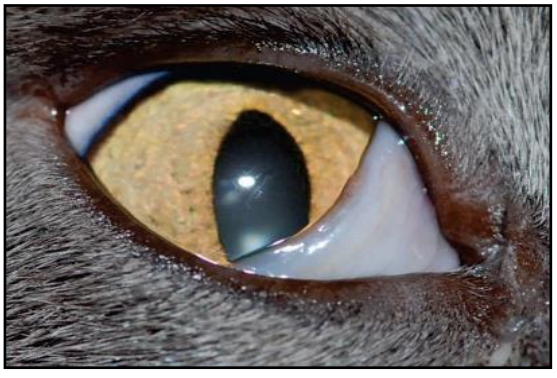


Fig 05: Protrusion of third eyelid in a cat.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

Partial horner's syndrome

Occurrence of miosis and ptosis (without concurrent facial anhidrosis) representing partial horner's syndrome is often reported in humans (Flaherty and Flynn, 2011). Partial horner's syndrome with only miosis in dogs with brachial plexus lesion is on record (Penderis, 2015).

Diagnostic protocol

Pharmacological testing

The onset of denervation hypersensitivity in the patient's eye serves as the foundation for pharmacological testing in Horner's syndrome. It's crucial to follow a consistent procedure when doing pharmacological tests on the eye (Antonio-Santos, 2005). Before administering any medications to the eyes, the following factors should be taken into account.

- It is always best to test both eyes while comparing them to the healthy one.
- The pharmacological substance should be injected into the conjunctival sac of each eye in exactly the same amounts and concentrations. Any topical ocular medication that is applied will trigger reflex tear production; as a result, a second dose should be given in both eyes a minute or two later, just in case the first dose was eliminated by the induced tear production.
- During the pharmacological testing, contact with or manipulation of the eye should be avoided as this could impact drug absorption or cause tear formation, which could dilute the pharmacological agent.
- The animal should be subjected to the same amount of stimulus and light throughout the test because both of these factors have a significant impact on the pupil's resting sympathetic and parasympathetic tone.

Table 03: Pharmacological tests and localization of horner's syndrome lesions

Drug	Mechanism of action	Use	Effect
Cocaine (5% or 10%)	Prevents norepinephrine reuptake	Confirm Horner's syndrome	Dilates Horner's pupil No effect on normal pupil
Apraclonidine (0.5% or 1%)	Weak α-1 adrenergic agonist	Confirm Horner's syndrome	Dilates Horner's pupil (not validated in veterinary patients)
Phenylephrine (0.1% or 1%)	Direct sympathomimetic	Localize Horner's syndrome	Dilates with postganglionic lesion , 20 min No effect on preganglionic, central lesions or normal

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

			eye
Hydroxyamphetamine (1%)	Indirect sympathomimetic	Localize Horner's syndrome	Dilates with preganglionic or central lesion, normal eye , 45 min No effect on postganglionic lesion

(Zwueste and Grahn, 2019)

Cocaine

The application of 1 drop of a 5% or 10% solution of cocaine topically is the gold standard test for detecting horner's syndrome in all animals (Kangalingam and Miller, 2015). Cocaine causes pupillary dilatation by preventing norepinephrine from being reabsorbed by the presynaptic membrane of the postganglionic neuron (Gross *et al*., 2016). Even in the presence of cocaine, there is not enough norepinephrine accumulating at the synapses to alter pupil size since any injury affecting the oculosympathetic pathway will block the normal release of norepinephrine. Topical cocaine will not cause either pupil in a bilateral horner's case to dilate significantly, and unilateral horner's instances will worsen anisocoria since the affected pupil will only slightly dilate while the unaffected pupil will dilate fully (Smit, 2010)

Apraclonidine

A normal pupil will be only mildly affected when applied topically because of its modest α-1 adrenergic activity, while a horner's syndrome patient will experience dilated pupils (Koc *et al*., 2005). Both pre- and postganglionic lesions exhibit this dilatory impact (Morales, 2000). A receptor on the postsynaptic membrane of the iris dilator muscle is upregulated in response to the reduction or absence of norepinephrine release (Koc *et al*., 2005). In general, 0.5% to 1% apraclonidine reduces anisocoria within 30 to 45 minutes (Morales *et al*., 2000).

Pharmacological testing to predict the site of the lesion in horner's syndrome

Denervation hypersensitivity is a phenomenon peculiar to smooth muscle innervated by the autonomic nervous system. Following denervation there is increased sensitivity of the muscles to neurotransmitters. The sympathetic denervation that causes denervation hypersensitivity allows pharmacological tests to be done to identify the site of the lesion based on enhanced sensitivity to topical phenylephrine when horner's syndrome has been present for some time (usually seven to 14 days or more) (Bistner *et al*., 1970). According to the level of the lesion in the sympathetic system, horner's syndrome is typically categorised as first order, second order (preganglionic), or third order (postganglionic). Following topical application of 1% phenylephrine to both eyes, the time until pupillary dilatation is measured. Basically, the closer the lesion is to the iris, the quicker the time to pupillary dilatation is:

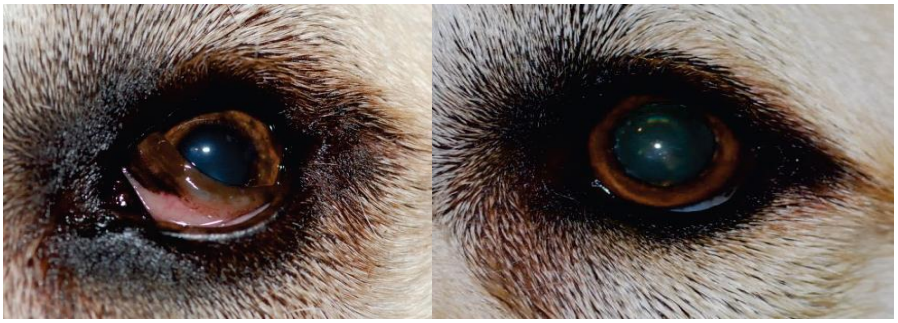
- A third order horner's syndrome is possible with less than 20 minutes.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

- Horner's syndrome of second order may appear between 20 and 45 minutes.
- A first order horner's syndrome or no sympathetic denervation of the eye is suggested after 60 to 90 minutes.
- In postganglionic (third order neuron) lesions, mydriasis happens in five to eight minutes if 10% phenylephrine is applied.

The use of a diluted direct sympathomimetic (phenylephrine) is the most effective way to localise a lesion with third order horner's syndrome, which is by far the most frequent presentation. With a postganglionic lesion, pupil dilation is observed more quickly and at a lower phenylephrine concentration. The highest sensitivity of the post-synaptic membrane to exogenous adrenergics results from the total depletion of norepinephrine within the synapse when the postganglionic neuron is damaged, which is one explanation for this. However, the postganglionic neuron that is still viable continues to emit minute amounts of norepinephrine when the lesion is preganglionic. Although, there is still some degree of denervation hypersensitivity, it is not as complete, and as a result, the reaction to topical adrenergics is not as strong (Simpson *et al*., 2015).

Another way to identify a third order lesion from a first and second order lesion is to use hydroxyamphetamine (1%) in the sample (Collins and O'Brien, 1990). Within 45 minutes, the dilatation should be noticed. However, a third order lesion on a postganglionic neuron causes a decreased or nonexistent norepinephrine supply, preventing the pupil from dilating (Zwueste and Grahn, 2019).



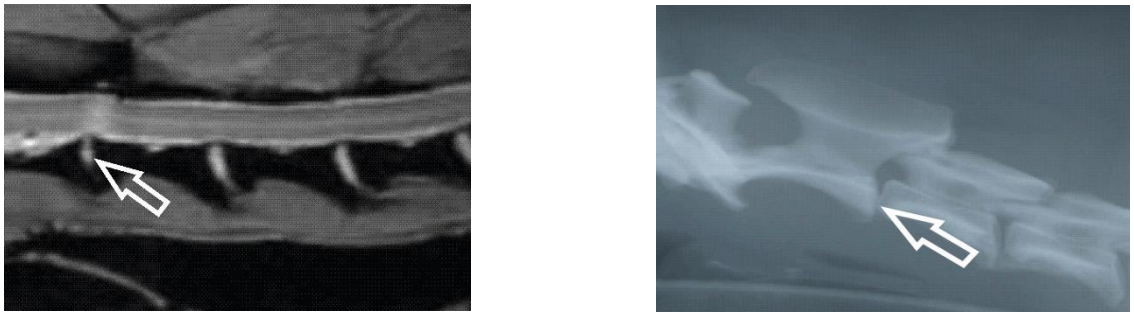
(a) Affected eye in a dog (b) 10 minutes after phenylephrine instillation

Ancillary diagnostics

- A thorough otoscopic examination should be performed in cases with post-ganglionic lesions to check for any signs of otitis (Gotthelf, 2004).

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

- As metabolic conditions like diabetes mellitus have been linked to horner's syndrome in dogs, a complete blood (cell) count and serum biochemistry are advised (De la Fuente *et al*., 2013).
- In situations of preganglionic lesions, cervical and thoracic radiographs are recommended.
- A complete evaluation of the oculosympathetic pathway as it passes through the brain, spinal cord, mediastinum, neck, middle ear, and orbit is possible with advanced imaging, such as computed tomography (CT) or magnetic resonance imaging (MRI), which should also be considered when preliminary diagnostics fail to identify a cause in pre- and post-ganglionic cases.
- Invasive diagnostic procedures like myringotomy/bulla osteotomy with cerebrospinal fluid analysis or mass biopsy may be necessary to identify the precise etiological agent if a structural lesion can be diagnosed **(**Zwueste and Grahn, 2019).



(A) **(B)**

Fig 06: Acute asymmetrical cervical or cranial thoracic spinal cord trauma may present with horner's syndrome (A) showing magneticresonance imaging (B) demonstrate collapse of the C2 to C3 intervertebral disc space and spinal cord trauma

(Penderis, 2015)

Differential diagnosis

- Middle cranial fossa pathologic abnormalities (neoplasm, vascular anomalies, infection).
- Retrobulbar pathologic abnormalities (contusion, abscess, or neoplasm).
- Idiopathic HS are further causes of third order HS that should be distinguished from HS secondary to middle ear disease. The latter is third order HS's most frequent cause (Boydell, 1995).

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

- HS can also be a side effect of middle ear surgery, particularly ventral bulla osteotomy in cats. In one study, it has been documented in up to 53% of cases, with the majority of those cases (46%) being transient (Bacon *et al*., 2003).

Table 04: Diagnostic procedures

Diagnostic Procedures	Results
Radiography of thorax	Mediastinal mass Mediastinum widened Pulmonary mass
Radiography of head/skull	Osseous (tympanic) bulla fluid density Osseous (tympanic) bulla opacified Osseous (tympanic) bulla osteolysis Osseous (tympanic) bulla sclerosis
Radiography of spine	Vertebral bony lesion
Ocular examination	Third eyelid, nictitating membrane protruded
Otoscopy	Fluid behind tympanum Tympanic membrane ruptured
Computed tomography (CT) or MRI of tissues affected	Avulsion/damaged nerve roots/nerves Characterization and extent of the lesion

(Penderis, 2015)

Treatment of horner's syndrome

Finding the location of the lesion in an animal with horner's syndrome is the most crucial task. Generally, post-ganglionic lesions have better prognosis than preganglionic lesions. When there is no known aetiology for horners syndrome, phenylephrine drops (0.125% or 10%) may be used as a symptomatic therapy to alleviate the clinical symptoms (Van Hagen *et al*., 1999). According to one study, 74% of instances with postganglionic horner's syndrome resolve on their own and within 7.7 weeks. (Morgan and Zanotti, 1989).

Acupuncture in horner's syndrome

Acupuncture is a viable approach for the treatment of idiopathic horner's syndrome, according to several case reports.

Acupuncture treatment of idiopathic horner's syndrome in dog

A one-year-old female english cocker spaniel dog was presented with the main complaint of protrusion of the third eyelid and drooping of the left upper eyelid. Physical examination revealed only ipsilateral left side ptosis, miosis, enophthalmos, and prolapsed

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

nictitans as clinical symptoms. The tentative diagnosis: idiopathic horner's syndrome was based on the case history, physical examination, neurological, and radiological profiles.

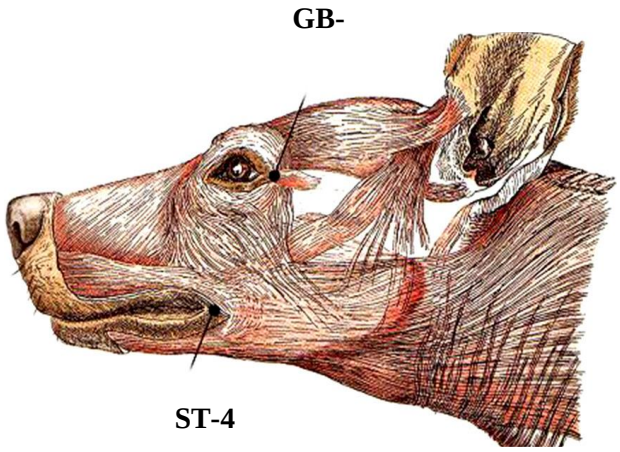


Fig 07: Acupoints GB-1 and ST-4 used for idiopathic horner's syndrome in the dog.

The Foot-Yang meridian's ST-4 and GB-1 acupoints were chosen for this study. The fourth acupoint on the stomach meridian is called ST-4. The lateral corner of the mouth is where ST-4 is located. The Gallbladder Meridian's first acupoint is designated as GB-1. The lateral canthus's corner is where GB-1 is situated. At each of these acupoints, a filiform stainless-steel needle (AP needle No. 263; Dong Bang, Korea) was used to administer bilateral treatment. The needle was inserted horizontally at an angle of 10 to 20 degrees. Twenty minutes were spent keeping the needles.

Ptosis, enophthalmos, and miosis were considerably improved the day following the first AP therapy. The prolapsed nictitans had additionally fully healed. The same methods were used for a second AP treatment. All of the clinical indications vanished by the third examination day (Cho and Kim, 2008).

Acupuncture treatment of feline horner's syndrome

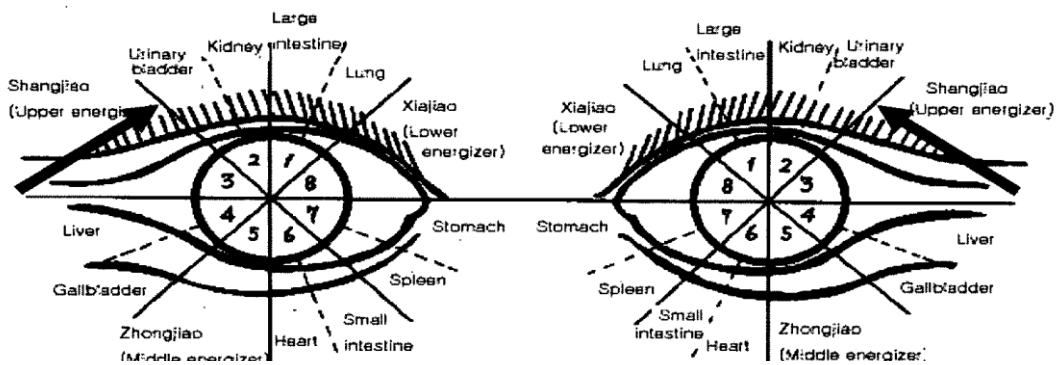


Fig 08: Oculo-acupuncture at Shiang Jiao region and the acupoints used in injection-acupuncture in the presented patient.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

A mixed-breed female cat that was about two months old appeared with minor miosis in the left eye as well as miosis, ptosis, and protrusion of the nictitating membrane in the right eye. Based only on clinical signs, the patient in this case was identified as having feline HS.

AP Treatment

The patient received Dexamethasone injection. Oculo-AP was applied for 10 minutes on the Shang Jiao areas of both eyes. Dexamethasone injection-AP (4 mg/ml:0.2 ml/acupoint) at ST-01 was also performed (Hsu *et al*., 2007).

Preventive measures

If the tympanum is ruptured, ear flushes and irritative topical treatments should be scrupulously avoided. Catherinization and jugular venipuncture, and surgical approaches to the thorax, osseous bulla, and neck area need to be performed carefully.

Recent updates

There are numerous causes of horner's syndrome, and these reasons can lead to lesions anywhere along the oculo-sympathetic pathway. These include nerve tissue degradation, neoplasia, cervical disc protrusion and trauma. Carotid catheterization, jugular venepuncture, otitis media, and animal bites. The results of a research done in dogs between 2000 and 2018 that is indicative of early imaging findings in all cases of horner's syndrome that have been reported are as follows.

Table 05: Summary of imaging findings in 86/88 cases of horner's syndrome

Imaging finding	No. of cases
Mass lesions of the cerebrum, cerebellum and brainstem	22
Cranial mediastinal, brachial plexus or thoracic soft tissue tumors	13
Fibrocartilaginous emboli	12
Nerve root /spinal cord tumors	12
Otitis media	12

(Lockhart *et al*., 2022)

Conclusions

Horner’s syndrome in the companion animals affects the face and eye on one side of the body with sporadic incidence. Occulosympathtic nerve deficit is recognised as one of major causes of the syndrome wuth miosis, ptosis, enopthalmos and prolapse of third eyelid as the main clinical signs. Based on the location of lesions in the sympathetic nerve pathway the First, Second and Third order neurons are targeted. The aetiology consortium in horner’s syndrome includes neoplasm, lymphoma, trauma, otitis, injuries bite wound. Diagnosis of horner’s syndrome involves pharmacological testing with alpha-blockers, MRI and CT scan are other advanced diagnostic techniques for localisation of lesions. There is no specific treatment for

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

horner's syndrome, resolution of the primary cause and symptomatic treatment with phenylehrine drops are recommended. Acupuncture emerging as a viable treatment needs to be authenticated with evidence-based research and extensive field trials by the field veterinarians.

References

Antonio-Santos, A.A., Santo, R.N. and Eggenberger, E.R. (2005). Pharmacological testing of anisocoria. *Expert Opinion on Pharmacotherapy*, **6**(12): 2007-2013.

Bacon, N. J., Gilbert, R. L., Bostock, D. E., & White, R. A. S. (2003). Total ear canal ablation in the cat: indications, morbidity and long-term survival. *Journal of small animal practice*, *44*(10), 430-434.

Bistner, S., Rubin, L., Cox, T.A. and Condon, W.E. (1970). Pharmacological diagnosis of horner's syndrome in the dog. *Journal of the American Veterinary Medical Association*, *157*: 1220-1224.

Boydell, P. (1995). Idiopathic Horner's syndrome in the golden retriever. *Journal of Small Animal Practice*, **36**(9): 382-384.

Cho, S.J. and Kim, O. (2008). Acupuncture treatment for idiopathic Horner's syndrome in a dog. *Journal of Veterinary Science*, **9**(1): 117-119.

Collins, B.K. and O'Brien, D. (1990). Autonomic dysfunction of the eye. *In*: Seminars in Veterinary Medicine and Surgery (Small Animal), Missouri, 2, November 1990, Department of Veterinary Medicine and Surgery, pp 24-36.

De la Fuente, C., Ródenas, S., Pumarola, M. and Añor, S. (2013). Dural tear and myelomalacia caused by an airgun pellet in a cat. *The Canadian Veterinary Journal*, **54**(7): 679.

De Lahunta, A., Glass, E.N. and Kent, M. (2020). de Lahunta's Veterinary Neuroanatomy and Clinical Neurology, 5th Edn., St. Louis, Saunders Elsevier, Philadelphia, pp 106-246.

Flaherty, P.M. and Flynn, J.M. (2011). Horner syndrome due to carotid dissection. *The Journal of Emergency Medicine*, **41**(1): 43-46.

Garosi, L.S., Lowrie, M.L. and Swinbourne, N.F. (2012). Neurological manifestations of ear disease in dogs and cats. *Veterinary Clinics: Small Animal Practice*, **42**(6): 1143-1160.

Gotthelf, L.N. (2004). Diagnosis and treatment of otitis media in dogs and cats. *Veterinary Clinics: Small Animal Practice*, **34**(2): 469-487.

Gross, J.R., McClelland, C.M. and Lee, M.S. (2016). An approach to anisocoria. *Current Opinion in Ophthalmology*, **27**(6): 486-492.

Herrera, and Suranit (1998). Idiopathic Horner's syndrome in collie dogs. *Veterinary Ophthalmology*, **1**(1): 17-20.

Hsu, C.Y., Hsu, C.L., Hsu, C.H., Liao, J.C., Chueh, H.J., Cheng, H.W., Jun, H.K. and Kim, D.H. (2007). Acupuncture Treatment in Feline Horner's Syndrome: Case Report. *Journal of Veterinary Clinics*, **24**(2): 214-217.

Kanagalingam, S. and Miller, N. R. (2015). Horner syndrome: clinical perspectives. *Eye and Brain*, **7**: 35-46.

Kern, T.J., Aromando, M.C. and Erb, H.N. (1989). Horner's syndrome in dogs and cats: 100 cases (1975-1985). *Journal of the American Veterinary Medical Association*, **195**(3): 369-373.

Koc, F., Kavuncu, S., Kansu, T., Acaroglu, G. and Firat, E. (2005). The sensitivity and specificity of 0.5% apraclonidine in the diagnosis of oculosympathetic paresis. *British Journal of Ophthalmology*, **89**(11): 1442-1444.

Lockhart, R. L., Tzouganakis, I., Tsvetanova, A., Smith, K. M., and Smith, P. M. (2022). The diagnostic yield of advanced imaging in dogs with horner's syndrome presenting with and without additional clinical signs: a retrospective study of 120 cases (2000–2018). *Veterinary Ophthalmology*, **25**: 51-59.

Morales, J., Brown, S.M., Abdul-Rahim, A.S. and Crosson, C.E. (2000). Ocular effects of apraclonidine in horner syndrome. *Archives of Ophthalmology*, **118**(7): 951-954.

Morgan, R.V. and Zanotti, S.W. (1989). Horner's syndrome in dogs and cats: 49 cases (1980-1986). *Journal of the American Veterinary Medical Association*, **194**(8): 1096-1099.

Mughal, M. and Longmuir, R. (2009). Current pharmacologic testing for Horner syndrome. *Current*

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024

Neurology and Neuroscience Reports, **9**(5): 384-389.
Murphy C.J., Samuelson D.A., Pollock R.V.H. (2013). The eye. *In*: de Lahunta, A. and Evans, H.E. (ed.). Miller's Anatomy of the Dog. 4th Edn., St. Louis, Saunders Elsevier, Missouri, pp 746–783.
Penderis, J. (2015). Diagnosis of Horner's syndrome in dogs and cats. *In Practice*, **37**(3): 107-119.
Rossmeisl, J.H. (2010). Vestibular disease in dogs and cats. *Veterinary Clinics: Small Animal Practice*, **40**(1): 81-100.
Schunk, K.L. and Averill Jr, D. R. (1983). Peripheral vestibular syndrome in the dog: a review of 83 cases. *Journal of the American Veterinary Medical Association*, **182**(12): 1354-1357.
Simpson, K.M., Williams, D.L. and Cherubini, G.B. (2015). Neuropharmacological lesion localization in idiopathic Horner's syndrome in Golden Retrievers and dogs of other breeds. *Veterinary Ophthalmology*, **18**(1): 1-5.
Smit, D.P. (2010). Pharmacological testing in Horner's syndrome–a new paradigm. *South African Medical Journal*, **100**(11): 738-740.
Sumanth., B.M. and Pathak, P. (2022). A review of horner's syndrome in cat and dogs. *The Science World*, **2**(10): 1650-1653.
Van den Broek, A.H.M. (1987). Horner's syndrome in cats and dogs: a review. *Journal of Small Animal Practice*, **28**(10): 929-940.
Van Hagen, M.A., Kwakernaak, C.M., Boeve, M.H. and Stades, F.C. (1999). Horner's syndrome in the dog: a retrospective study. *Tijdschrift Voor Diergeneeskunde*, **124**(20): 600-602.
Zwueste, D.M. and Grahn, B.H. (2019). A review of Horner's syndrome in small animals. *The Canadian Veterinary Journal*, **60**(1): 81.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published 28.03.2024