

HORNER`S SYNDROME- EYE OR NEUROLOGICAL DISEASE?

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Abstract

The interruption of sympathetic innervations at the head level is the main cause that produces the Horner`s syndrome. The damage of the nerve fibres may occur: central, preganglionic or postganglionic.

Most patients were sent for an ophthalmologic examination as a result of a sudden attack, often described by owners as “closed, painful eye”.

The purpose of this paper is to establish an etiologic differential diagnosis protocol in Horner`s syndrome.

The patients examined in the Surgery Clinics of the Faculty of Veterinary Medicine Bucharest presented enophthalmos, upper eyelid ptosis, palpebral slid reduction, third eyelid protrusion and miosis. The ophthalmologic examination was performed by direct methods and indirect methods, as Schirmer test, fluorescein test and the pupil size in the darkness. For the most patients, the disease started suddenly, with epiphora and very painful eye. Only for few of them, the onset was sudden and no ocular pain or epiphora were mentioned.

The results of the tests showed normal values for the Schirmer test, miosis, with negative response of the pupil in the darkness. The fluorescein test was negative and the internal face of the third eyelid presented no foreign bodies. In this cases, the etiology of the syndrome is idiopathic or secondary to media otitis, frequently subclinical.

It was achieved a diferential diagnosis between Horner`s syndrome and the superficial or deep corneal wounds, when the fluorescein test is positive and there were highlighter foreign bodies at the internal third eyelid.

Key words: *Horner`s syndrome, miosis, protrusion, otitis, foreign bodies.*

INTRODUCTIONS

Horner`s syndrome is characterized by unilateral protrusion of the nictitants, ptosis, anisocoria and miosis.

The most common causes of this syndrome include otitis media, preganglionic injuries as trauma or neoplasia, and idiopathic.

Horner`s syndrome results from impairment of ocular sympathetic innervations. The sympathetic innervations of the eye consists of a long, three neurone, pathway, extending from the diencephalon, through the timpanic bula, to the eye.

“Loss of sympathetic innervations causes a lack of tone in the orbital smooth muscle, with the result that the eye retracts slightly, producing enopthalmos. Loss of innervations to the muscle of the upper eyelid (Muller`s muscle) and sympathetically innervated tissue in the lower eyelid results in narrowing of the papebral fissure and incomplete elevation of the upper eyelid or ptosis. Lack of sympathetic tone and enophtamos result in protrusion of nictitans. Reduction of normal sympathetic tone to the iris dilator muscle result in the anisocoria and miosis in the affected eye.”

Protrusion of the third eyelid and miosis are usually the most evident and bring the patient to medical attention.

Many patients present, besides these two symptoms, blepharospasm, epiphora, photophobia and intense pain. The examination of the affected eye can be very difficult and the lesion is most often unilateral.

MATERIALS AND METHODES

Ophthalmologic examination of the patients who came in the Surgery Clinics of the Faculty of Veterinary Medicine Bucharest, during January 2010 - October 2012, presenting enopthalmos, third eyelid protrusion, miosis, palpebral slit reduction and signs of ocular pain performed by direct methods, as inspection, and indirect methods.

We examined 3 cats and 4 dogs.

In most patients, clinical signs appeared spontaneously, after a long walk outside. They presented pronounced miosis, third eyelid protrusion, with redness and edema, epiphora, photophobia and blepharospam (Figure 1). Because of the intense pain, the eye examination was done only after local anesthesia with benoxicaine drops.

The indirect methods performed are: Schirmer test, the examination of the internal face of the third eyelid and the fluorescein test.

In patients with no pain, to whom, the clinical signs occurred gradually, the eye examination was made easily. It was performed the test for a papillary near response (Figure 1). Schirmer test and the fluorescein test were also made. We examined the ear with the otoscope.

RESULTS AND DISCUSSIONS

Table 1. Indirect methods results

	Schirmer test	Fluorescein test	Observations	Diagnosis
Eye in pain	>20mm/min	positive	Internal face of the nictitant: foreign body	Superficial erosions
Eye without pain	<20mm/min	negative	Otoscope: otitis media	Horner syndrome

In patients with pain, fluorescein test is positive and revealed superficial lesions of the cornea at the inner canthus. The aspect is similar to the mark left by the windshield. (Figure 2)

The Schirmer test values were bigger than 20mm/min. On the internal face on the nictitant membrane we found foreign bodies. (table 1, figure 2)

In Horner`s syndrome, otoscopy showed the presence of otitis media with mucosa inflammation . The patients kept the head tilt on the same part as the affected ear (figure 3). Miosis persisted in dark (figure 4).



Figure 1.

Foreign body at the internal surface of the third eyelid (see the arrow)



Figure 2.

Left eye: examination of the internal face of the nictitant.
Left eye: corneal superficial wound. Fluorescein test positive



Figure 3.

Left eye: Horner's syndrome, secondary to chronic otitis. (note the enophthalmia, miosis, protrusion of the third eyelid and anisocoria)



Figure 4.

Left eye: Horner`s syndrome, secondary to subclinical otitis, in a Husky, 11 years old (note the anisocoria, enophthalmia)



Figure 5.

Right eye: Idiopathic Horner`s syndrome in a Golden Retriever, 3 years old (note the miosis and the protrusion of the third eyelid)

CONCLUSIONS

Miosis and protrusion of the nictitant membrane can be a common sign for many ocular diseases.

Horner's syndrome often appears as a secondary manifestation of subclinical otitis media.

Incomplete examination of the eye may mask the presence of foreign bodies on the internal face of the third eyelid.

Treating the otitis is the most important thing, Horner's syndrome being a neurological disease appears like ophthalmological disease with full recovery in postganglionic lesions.

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