Idiopathic Horner’s syndrome in collie dogs

Héctor Daniel Herrera,*† Adriana Patricia Suraniti* and Nuria Fernanda Kojusner‡
*Veterinary Medicine Teaching Hospital, School of Veterinary Sciences, University of Buenos Aires, Argentina, †Private practice, Buenos Aires, Argentina

Abstract

Seven cases of idiopathic Horner’s Syndrome in the Collie are described. Five males and two females presented with unilateral miosis, ptosis of the upper eyelid, enophthalmos and protrusion of the third eyelid. Thorough examination, pharmacological testing with phenylephrine, complete blood counts and radiography of the tympanic bullae and thorax were performed. The etiology was not identified in any of the cases. Clinical signs improved with pharmacologic testing within 20–40 min. In five dogs, total resolution of clinical signs was observed at 4 and 16 weeks after their initial appearance. Pharmacological testing suggested that the deficit could be at the preganglionic neuron.

Key Words: Horner’s syndrome, dog, collie

INTRODUCTION

Horner’s syndrome (HS) is a group of clinical signs that result from loss or interruption of sympathetic innervation to the globe and adnexa. These signs include miosis, ptosis of the upper eyelid, enophthalmos and protrusion of the third eyelid.

The sympathetic innervation of the eye and adnexa may be divided into three neuroanatomic parts: central, preganglionic and postganglionic. The central component consists of fibers descending from the higher centers of the autonomic nervous system in the brain stem. They pass from the hypothalamus, tectum and tegmentum down the tegmentospinal tract to synapse with preganglionic cell bodies in the intermediate grey column of the cranial thoracic segments of the spinal cord. Pain, fear or emotional responses may cause pupillary dilatation by activating this pathway. The preganglionic cell bodies are located in the intermediate grey column of the first three or four segments of the thoracic spinal cord. The axons leave the spinal cord with the ventral roots of the spinal nerves to join the thoracic sympathetic trunk, and then pass through the cervicothoracic ganglion without synapsing, very close to the cranial lung lobe. These preganglionic axons travel up the neck in the vagosympathetic trunk ending in the cranial cervical ganglion, located caudomedial to the tympanic bulla, where they synapse with the postganglionic cell bodies. The postganglionic sympathetic axons travel forward, pass through the middle ear adjacent to the facial nerve and join the ophthalmic branch of the trigeminal nerve. The ophthalmic nerve enters the periorbita through the orbital fissure and distributes to the smooth muscles of the periorbita, eyelids and the dilator muscle of the iris.

Several causes of HS have been proposed including congenital anomalies, severe head, neck and chest trauma following road accidents, otitis media/interna, avulsion of the roots of the brachial plexus, thoracic masses, central nervous system infection or neoplasia, ischaemic myelopathy, intervertebral disc disease, and thyroid neoplasia. However, in approximately half of affected dogs the cause cannot be determined but these cases are recognized as idiopathic HS. There are no reports of any breed predisposition to the disease but a high incidence of idiopathic HS has been described in the Golden Retriever. The purpose of this paper is to describe the findings of seven cases of idiopathic HS in Collies in a total of eight dogs with HS presented to the authors between October 1994 and December 1996.

MATERIALS AND METHODS

Eight dogs (seven Collies and a Great Dane) with idiopathic HS were studied between October 1994 and December 1996. The medical records of the seven Collies were reviewed in the present study. Five males and two females (Table 1). All of them presented with acute unilateral miosis, ptosis of the upper eyelid, enophthalmos and protrusion of the third eyelid. Clinical examination included a complete ophthalmic examination (seven cases), thorough ear examination (seven cases), complete neurologic examination (seven cases), pharmacological testing with topical 10% phenylephrine (seven cases), complete blood counts and blood chemistry parameters (four cases), and radiographs of the thorax (three cases) and the tympanic bullae (two cases).

In order to localize the site of the lesion, pharmacological testing was performed through assessment of ocular response to the topical administration of 10% phenylephrine
after its appearance. Signs was observed between 4 and 16 weeks (mean 9.2 weeks) in dogs that could be followed up, total resolution of clinical control eyes at the same time as the affected eyes. In the five of the affected eyes (Table 1). Mydriasis occurred in all the except the pupil, which had an incomplete response in four 20±40 min after application of the topical phenylephrine, All the clinical signs showed a complete improvement within dogs responded to the pharmacological testing (Figs 1 and 2).

**Table 1** Distribution by sex, age and affected eye, response to pharmacologic testing and recovery times in the seven studied Collies

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Affected eye</th>
<th>Pupillary response with phenylephrine</th>
<th>Recovery time (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>9</td>
<td>OD</td>
<td>mydriasis</td>
<td>12</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>6</td>
<td>OS</td>
<td>incomplete</td>
<td>16</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>5</td>
<td>OD</td>
<td>incomplete</td>
<td>–</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>8</td>
<td>OS</td>
<td>incomplete</td>
<td>–</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>11</td>
<td>OS</td>
<td>mydriasis</td>
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<td>6</td>
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<td>4</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>5</td>
<td>OD</td>
<td>incomplete</td>
<td>6</td>
</tr>
</tbody>
</table>

M, male; F, female; OD, right eye; OS, left eye.

(Poenefrina: Poen Lab., Buenos Aires). Two drops were placed in each eye using the control eye for comparison. Ocular examination was repeated every 10 min for 50 min; the time when the affected eye resumed to normality and mydriasis occurred in both the control and affected eye was noted. Two cases had a history of otitis externa; radiographs of the tympanic bullae were performed in these two patients, in addition to the otoscopic examination. Follow-up could be carried out by telephone contact with the owners in five of the cases.

**RESULTS**

Four dogs were affected on the left side and three dogs were affected on the right side. The cause could not be determined in any of the cases. There was no history of trauma, infectious or non infectious disease of the central nervous system, or ear disease except in two cases with previous mild otitis externa. Ophthamologic and otoscopic examination, and hematological and serum biochemical analysis were normal as well as radiographic examination, except in one case which showed spondylodisc deformans from C₆ to T₂ with no peripheral neurologic signs. All the dogs responded to the pharmacological testing (Figs 1 and 2). All the clinical signs showed a complete improvement within 20–40 min after application of the topical phenylephrine, except the pupil, which had an incomplete response in four of the affected eyes (Table 1). Mydriasis occurred in all the control eyes at the same time as the affected eyes. In the five dogs that could be followed up, total resolution of clinical signs was observed between 4 and 16 weeks (mean 9.2 weeks) after its appearance.

**DISCUSSION**

The sympathetic supply of the eye is responsible for maintaining normal tone of the smooth muscles of the periorbita, eyelids and dilator muscle of the iris. It keeps the globe positioned normally, the third eyelid retracted, the pupil partially dilated, and modulates palpebral fissure width. Horner’s syndrome (HS) is the name given to the group of clinical signs which results from interruption or loss of sympathetic innervation. These signs include miosis (actually the affected pupil dilating incompletely in low light conditions), ptosis of the upper eyelid, enophthalmos, and protrusion of the third eyelid.

HS is not an uncommon finding in dogs. Previous reviews of cases of HS have shown a relatively high incidence of the condition. One author diagnosed 74 cases in 10 years. In another report, 33 dogs with HS were diagnosed over a period of six years, while only two cases were diagnosed in 4 years by another author. In our series, eight cases of idiopathic HS were diagnosed in the last two years and seven of these cases were in Collies.

Collies are not a common breed in the Argentinian dog population. In fact, only 12 Collies with ophthamlogic disease were presented to one of the authors (H.D.H.) in the review period and seven of them had HS. Thorough examination and pharmacological testing with phenylephrine were performed in seven Collies; complete blood counts and radiographs of the tympanic bullae and thorax were performed in some of them but the cause could not be determined in any case. Boydell has reported a high incidence of idiopathic HS in Golden Retrievers; however, there are no reports of any breed predisposition in other studies.

There is no general agreement about the utility of pharmacologic testing for HS. Use of 0.001% epinephrine and 2 or 4% cocaine, hydroxyamphetamine, hydroxyamphetamine and 10% phenylephrine, epinephrine, and phenylephrine have been utilized in both humans and dogs in order to localize the site of the lesion. The combined use of an indirect and a direct-acting sympathomimetic agent appears to be the most useful method. Instillation of an indirect-acting sympathomimetic such as 1% hydroxyamphetamine results in normal mydriasis of the miotic pup if the lesion is preganglionic. This drug acts by releasing endogenous norepinephrine from adrenergic nerve endings. When the lesion is postganglionic, the stores of norepinephrine contained in the nerve terminals are depleted and the pupil remains miotic. A direct-acting sympathomimetic such as 10% phenylephrine or 0.001% epinephrine can be used to confirm a postganglionic lesion in cases of incomplete or absent mydriasis after instillation of hydroxyamphetamine. In these cases of sympathetic denervation, adrenergic nerve endings become supersensitive to direct-acting sympathomimetics and mydriasis quickly occurs in postganglionic lesions.

Boydell refers to the criteria developed by Kay (1981) of noting the time when mydriasis occurs after instillation of 10% phenylephrine. If mydriasis occurs within 20 min of the instillation, a postganglionic lesion is suggested. If dilatation occurs between 20 and 45 min, a preganglionic lesion is diagnosed. Finally, if mydriasis takes longer than 45 min, a central lesion may exist. Different authors have reported, however, that 10% phenylephrine does not consistently produce mydriasis in postganglionic lesions. In our cases all pharmacological testing was performed with 10% phenylephrine. Clinical signs improved within 20 and 40 min.
after application in all cases except for the pupils demonstrating incomplete recovery in four of the affected eyes. Control eyes dilated at the same time as the affected eyes.

Regarding recovery times, Morgan mentioned that 25 out of 33 dogs showed complete reversal of clinical signs in a time ranging from 24 h to 30 weeks. In our study, five out of seven patients demonstrated total spontaneous resolution between 4 and 16 weeks.

According to our results, Collies may have a breed predisposition to idiopathic Horner’s syndrome. Pharmacological testing performed in these cases suggested that the lesion could be affecting the preganglionic neuron, but the correct site is not able to be confirmed using only phenylephrine.

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REFERENCES