

Scientific Report

Pupillotonia in a Spitz dog: a case report

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(Received 21 Jan 2006; revised version 15 Mar 2006; accepted 31 May 2006)

Summary

An 8-year-old female Japanese Spitz dog was referred to Shiraz University Veterinary Clinic, with sign of anisocoria. Clinical examinations revealed mydriasis in the right eye. Ocular examination revealed a dilated and unresponsive right pupil to focal illumination. By testing with topical 2% pilocarpine, the tentative diagnosis was parasympathetic denervation of the right iris sphincter muscle—pupillotonia.

Key words: Anisocoria, Dog, Eye, Japanese Spitz, Pupillotonia

Introduction

Pupil abnormalities are a common finding in man and other animals. A miotic pupil may be the result of a miotic drug such as pilocarpine, uveitis, synechia, or loss of sympathetic tone (Horner's syndrome). Conversely, a dilated pupil may be associated with mydriatic drugs (*e.g.*, atropine), loss of retinal or optic nerve function, glaucoma, or loss of parasympathetic tone. The loss of parasympathetic tone in human is termed Adie's tonic pupil (Lahunta, 1995; Kawana *et al.*, 2003).

Although there are references in veterinary medical literature with respect to pupillary abnormalities (Scagliotti, 1980), canine cases of parasympathetic denervations of the pupil were found only in a German shepherd dog (Gerding *et al.*, 1986).

Case history

An 8-year-old female Japanese Spitz dog was referred to the Veterinary Clinic of the School of Veterinary Medicine, University of Shiraz, Shiraz, Iran, because of anisocoria (Fig. 1). The general conditions, appetite, cardiovascular and respiratory systems, and body temperature were normal. Ocular examination revealed a dilated right pupil.

The left pupil had a normal diameter for the ambient illumination. By covering of either eye separately, the ability of animal to negotiate doorways and stairs was detected which revealed that the vision of animal is normal.

Fig. 1: Anisocoria in a Japanese Spitz dog. Right pupil is dilated and left pupil is normal

In pupillary light reflex testing, the right pupil had no response to strong light directed to either eye. The left pupil had a positive direct and indirect response to strong light.

Ophthalmoscopy revealed normal lens, vitreous, retinas and optic nerves. The eyelids, corneas and irides were also normal. Intraocular pressures, measured by palpation with fingers, were normal.

The tentative diagnosis was parasympathetic denervation of the right iris sphincter muscle—pupillonia. To confirm the diagnosis, a drop of 2% pilocarpine was instilled into the right eye which resulted in constriction of the pupillary diameter to the same size as in the left eye. This test confirmed that the lesion was neurologic and that there were no primary or secondary iris diseases or pharmacologic blockade due to atropine or atropine-like drugs. The animal was treated for two weeks by 2% pilocarpine. At this time, the animal's eye responded to therapy but by withdrawing the drug, pupil was dilated again. On the basis of the right pupil's response to pilocarpine and absence of any other signs in this case, a diagnosis of parasympathetic denervation (pupillonia) of the right pupil was made.

Discussion

The final common pathway to the iris sphincter muscle (the efferent arm of the light reflex) is the parasympathetic lower motor neuron located in the oculomotor nerve (cranial nerve III). The preganglionic parasympathetic fibers arise from cell bodies located in the parasympathetic nucleus of the third cranial nerve, which is in the rostral end of the oculomotor nucleus of the mesencephalon. These fibers exit the mesencephalon in intimate association with the motor efferent fibers of the oculomotor nerve but depart from these fibers within the orbital cone, just proximal to their synapse in the ciliary ganglion. The ciliary ganglion contains the cell bodies of postganglionic axons of parasympathetic pathway. These axons course to the eyeball in ciliary nerves, penetrate the sclera and follow the choroid to the iris and the constrictor muscle of the pupil (Scagliotti, 1991; Lahunta, 1995).

Anisocoria can result from lesions of the sympathetic nerves of eyes, retina, optic nerve, optic chiasma, visual cortex and parasympathetic nerves of eyes. Thus, in a case of anisocoria, differentiating of these conditions is necessary. Sympathetic denervation of pupil causes miosis in the affected eye in ambient light. In darkness, this pupil dilates due to the inactivity of oculomotor neurons. In addition to miosis, there is also a protrusion of the third eyelid

and slight narrowing of the palpebral fissure (ptosis) (Lahunta, 1995). In the present case, the left pupil was constricted in ambient light but was completely dilated in dark area which indicated that the sympathetic innervation was normal.

The oculomotor nerve lesions result in external as well as internal ophthalmoplegia. As a result of the close proximity of the motor efferent nuclei in the oculomotor complex to the Edinger-Westphal nuclei, and the fact that the axons of all these nuclei travel together in the trunk of the oculomotor nerve as it exists the mesencephalon, it is not uncommon for oculomotor nerve damage to result in external as well as internal ophthalmoplegia. The clinical features of external ophthalmoplegia are ptosis, lateral strabismus and that of internal ophthalmoplegia (unilateral paralysis of the efferent pupillary pathway is a widely dilated pupil).

Pharmacologic testing can be used to determine whether the lesion in the parasympathetic innervation of the eye is in the first (preganglionic) or second (postganglionic) neuron of the lower motor neuron system. Lesions of the second neuron that denervate the pupil constrictor make it hypersensitive to low concentrations of the direct-acting drug pilocarpine. A 0.1% solution causes contraction of the pupil of the denervated iris but not a normal or an iris in which the lesion involves the first neuron. Instilling a 2% solution of pilocarpine is not specific in its localizing effect, but confirms that the lesion is neurologic, since the affected pupil constricts sooner, to a great extent, and for a longer time than the control eye. However, a negative response indicates the presence of primary or secondary iris disease or pharmacologic blockade (Scagliotti, 1991; Lahunta, 1995).

Unilateral oculomotor nerve lesion has been reported in a 15-year-old Belgian sheepdog. The dog presented with ventrolateral strabismus of the left eye, ptosis of the left upper eyelid, and anisocoria with the left pupil fixed and dilated (Larocca, 2000). Further evaluation via magnetic resonance imaging (MRI) revealed a well-defined mass to the left of midline and lateral to the sella turcica. Necropsy and immunohistochemistry studies revealed that the mass was

a meningioma.

Compression of oculomotor nerve has been reported in cavernous sinus syndrome in four dogs (Theisen *et al.*, 1996). Cavernous sinus syndrome is characterized by deficits in more than one of the cranial nerves that traverse the cavernous sinus at the base of the cranial vault: cranial nerve (CN) III (oculomotor), IV (trochlear), VI (abducent), and the first two branches of CN V (trigeminal). The most common clinical signs were ophthalmoparesis or ophthalmoplegia, mydriasis with no direct or consensual pupillary light reflexes, ptosis, decreased corneal sensation and decreased retractor oculi reflex. In all dogs (n = 4) the lesions were localized to the right cavernous sinus. A definitive pathological diagnosis had been obtained in two dogs—a primary intracranial neoplasm and a metastatic intracranial neoplasm.

In the present case, since there were no signs of cranial dysfunction except right eye mydriasis, it seems that the etiology of anisocoria is extracranial. The mydriasis may be due to a lesion of the only parasympathetic part of the oculomotor nerve, but the exact cause of this condition is not clear.

Pupillotonia has also been reported in a German shepherd dog (Gerding *et al.*, 1986). In that case, in addition to anisocoria, other clinical signs such as oral ulcerations, occasional vomiting, difficulty in exhaling through the nares, decreased broncho-alveolar sounds, suppurative inflammation of the oral mucosa, and enlarged submandibular lymph nodes were also seen. They reported that the exact cause of pupillotonia is obscure, but the pathogenesis seems to consist of a degeneration of the ciliary ganglion or postganglionic parasympathetic fibers to the affected eye (Alder and Scheie, 1940; Gerding *et al.*, 1986). The process of degeneration generally is considered to be progressive and may be due to an immune mechanism or a slow-acting virus. They treated the patient by prednisolone and cimetidine. The dog's overall condition had improved considerably and the pupil had constricted slightly (Gerding *et al.*, 1986). The decrease in pupil size is a common finding in follow-ups of human patients with pupillotonia (Loewenfeld and Thompson,

1967).

A retrobulbar or intracranial lesion affects both optic nerves and parasympathetic part of the oculomotor nerve on the same side and causes a widely dilated pupil in the ipsilateral eye at rest. There is no menace response from this affected eye. Light directed into the affected eye elicits no response in either eye. Light directed into the unaffected eye causes pupillary constriction only on that eye (Lahunta, 1995). In the present case, when light directed to the affected eye the normal eye was constricted. On the other hand, in ophthalmoscopic examination of the eyes, no abnormality was detected. Thus, the retina, optic nerve and optic chiasma were considered to be normal.

The finding of this case is similar to Gerding *et al.* (1986). But this patient had no signs other than anisocoria and thus the cause of dilated pupil remains obscure. In human patients, pupillotonia due to sarcoidosis in ciliary ganglion has been reported (Bowie and Givre, 2003).

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