**TITLE PAGE**

**Monocular nystagmus representing Heimann - Bielschowsky phenomenon in a dog with ipsilateral vision loss**

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**ABSTRACT**

A 6-year-old female-neutered vaccinated Cocker Spaniel presented for pre-adoption neurological evaluation due to abnormal left-eye movements that had been noticed since young age. Clinical examination revealed left-eye cataract with a non-visible left and a normal right retina on ophthalmoscopy. Neurological examination revealed absent left-sided menace response and cataract-related ipsilateral visual impairment, and intermittent left-sided abnormal eye movements consisted of intermittent, slow, coarse, variable amplitude, vertical movements of the eye that they were giving the impression of random movements of the eye within the globe as it was floating (“wandering” eye) interchangeable with periods of rest. Blood and infectious diseases tests were unremarkable. Magnetic resonance imaging of the brain was unremarkable, whilst cerebrospinal fluid analysis revealed mildly inflammatory in the light of blood contamination. A presumptive diagnosis of meningoencephalitis of unknown origin was established and trial with dexamethasone was performed, however nystagmus remained unchanged two weeks post-treatment. Therefore, based on the fact that monocular nystagmus existed since youth and remained static , and the dog was otherwise neurological-sign-free regardless the discontinuation of steroids, the diagnosis of meningoencephalitis was considered as unlikely and a presumptive diagnosis of *Heimann - Bielschowsky phenomenon* of the left eye due to cataract-related ipsilateral visual impairment was established. This vergence eye movement abnormality also known as *searching*, *wandering* or *amaurotic nystagmus* is a constant or intermittent benign eye movement abnormality mostly related with vision impairment due to ophthalmological or neurological disease. *Heimann - Bielschowsky phenomenon* is an underreported eye abnormality in veterinary medicine. Although the most common type of eye movement abnormalities seen in veterinary practice is the bilateral conjugate jerk nystagmus, monocular nystagmus representing *Heimann - Bielschowsky* phenomenon exists in animals, it has been related with partial or complete vision impairment and it should be recognised by the clinicians.

**KEYWORDS**

amaurotic nystagmus; dissociated nystagmus; searching nystagmus; unilateral nystagmus; uniocular nystagmus; wandering nystagmus

**ABBREVIATIONS**

CSF Cerebrospinal fluid

HBP Heimann - Bielschowsky phenomenon

MRI Magnetic resonance imaging

MUO Meningoencephalitis of unknown origin

RI Reference intervals

**CONFLICT OF INTEREST**

The authors declare that there is no conflict of interest.

**AUTHOR’S CONTRIBUTIONS**

TL was the primary clinician of the case and wrote the article. GBC was the clinical supervisor of the case and reviewed the article.

**MANUSCRIPT**

**Introduction**

Eye movement abnormalities reported in veterinary practice include pathological nystagmus such as binocular conjugate (rarely dysconjugate) jerk nystagmus (vestibular system lesion; most common eye movement abnormality in veterinary practice),1 conjugate pendular nystagmus (congenital abnormality in the visual pathway of cats, occasionally seen with cerebellar disease and visual deficits),2 and saccadic intrusions such as opsoclonus and macrosaccadic oscillations (cerebellar lesions),3 and convergence-retraction nystagmus (dorsal midbrain lesions).4,5

*Heimann - Bielschowsky phenomenon* (HBP) is a constant or intermittent, monocular or binocular, benign eye movement abnormality characterized by slow, coarse, pendular, variable amplitude movements in an eye with long-term partial or complete blindness.6,7 The term “nystagmus” has been erroneously used for HBP, as it represents apparently a vergence eye movement abnormality.7 HBP can be secondary to a congenital or an acquired cause of blindness.7

HBP has been vaguely reported in veterinary literature, with different nomenclature (*amaurotic*, *wandering* or *searching* nystagmus), as an incidental finding within clinical, ophthalmological or neurological examination of dogs,8-18 horses19,20 and goats21 with congenital ophthalmological or neurological diseases. Nevertheless, HBP has never been discussed and demonstrated as a clinical sign within veterinary literature. This case report aims to highlight HBP as an uncommon type of monocular nystagmus in a dog, to describe and demonstrate it as a clinical sign through a video recording and discuss current veterinary and human medicine literature, resulting in a better understanding and recognition of the sign by veterinary practitioners, as well as enrichment of the current literature.

**Case presentation**

A 6-year-old female-neutered vaccinated Cocker Spaniel presented to a referral hospital for pre-adoption neurological evaluation due to abnormal left-eye movements that had been noticed since a puppy.

**Clinical Findings**

Clinical examination was unremarkable. Ophthalmological examination revealed left-eye cataract, whilst ophthalmoscopy revealed a non-visible left and an unremarkable right retina. Neurological examination revealed absent left-sided menace response, cataract-related ipsilateral visual impairment (Figure 1), and intermittent left-sided abnormal eye movements (Video 1). The latter consisted of intermittent, slow, coarse, variable amplitude, vertical movements of the eye that they were giving the impression of random movements of the eye within the globe as it was floating (“wandering” eye) interchangeable with periods of rest.

**Differential Diagnoses**

Differential diagnoses for monocular nystagmus in dogs are mostly limited to HBP secondary to visual impairment (pre-retinal, retinal, post-retinal) based on the sparse reports in the veterinary literature. Other causes of monocular nystagmus in dogs should include cerebellar cortical dysplasia and hereditary hemichiasma/achiasma. Therefore, diagnostic tests including diagnostic imaging should pay attention to visual pathway and cerebral cortex (occipital lobe), as well as the vestibular system, dorsal midbrain and cerebellum so as to rule out other causes of pathological nystagmus or saccadic intrusions.

**Investigations**

Complete blood count, serum biochemistry, bile acid stimulation test and thyroid profile (including total thyroxine) were within normal limits. Infectious diseases tests, including serology (indirect immunofluorescence) for *Toxoplasma gondii* (IgM/IgG) and *Neospora caninum* (IgG) were negative.

A low-field magnetic resonance imaging (MRI) (Hitachi Lucente 0.4 T) of the head a was performed. The study included T2-weighted images in sagittal, transverse and dorsal plane, T2\*-weighted and T2 - Fluid attenuation inversion recovery (FLAIR) images in transverse plane, and T1-weighted pre-and-post contrast (gadolinium) images. MRI of the brain and optic nerves was unremarkable. Cerebellomedullary cisternal cerebrospinal fluid (CSF) analysis revealed mononuclear pleocytosis (total nucleated cell count 11/μL, reference intervals (RI): 0-6) and normal proteins (total proteins 0.22 g/l, RI: 0.14 - 0.30) in light of blood contamination (red blood cell count 954/μl, RI: 0). Cytology of the CSF revealed 55% monocytes, 39% small lymphocytes and 6% non-degenerate neutrophils.

**Treatment and outcome**

A presumptive diagnosis of meningoencephalitis of unknown origin (MUO) was made based on the CSF analysis, and therapeutic trial with dexamethasone (0.2 mg/kg PO q24h) was performed. Nevertheless, nystagmus remained unchanged two weeks post-treatment and therefore a possible relation between a presumptive MUO and nystagmus was considered unlikely in the light of presence of nystagmus since early youth. Then, dexamethasone was discontinued, and a presumptive diagnosis of left-sided monocular nystagmus representing HBP secondary to the ipsilateral cataract and vision impairment was made. The dog was adopted, and it remained static with present HBP and medication-free at a two-year follow-up phone call. Based on the absence of neurological signs two years later on no treatment, the previously diagnosed presumptive MUO was considered unlikely either attributed to a missed diagnosis of another neurological disease that could cause mixed cell pleocytosis or to a false-pleocytosis in CSF analysis due to the blood contamination.

**Discussion**

To the authors’ knowledge, this is the first case report to discuss HBP as a distinct clinical entity in dogs and provide a video recording of it.

The agonist muscles of each eye are usually coordinated (yoked) in either physiological or pathological eye movements, but may function independently if the task dictates or if binocularity did not develop (e.g. achiasma).22 Independent control of each eye and its associated brainstem architecture involved in eye movements, implies that each individual muscle is driven by independent populations of neurons (burst cells, neural integrator cells, etc.).22 Therefore, monocular nystagmus as a clinical sign can occur. Specifically for HBP, although its pathogenetic mechanism is not completely known, it has been hypothesized that long-term vision impairment and subsequent sensory deprivation can result in central fusion disruption (vergence eye movement abnormality, that is the affected eye is unable to fix gaze at an object once it is not visual) and thus result in HBP of the affected eye.7,23,24

HBP has been reported vaguely and scarcely within the veterinary literature, referring as *searching* or *wandering* nystagmus. It is probably a reasonably common and very underrecognized condition, mainly reported by veterinary ophthalmologists, as happens in human medicine.25 HBP has been reported in dogs, horses and goats with mainly congenital ophthalmological disease, but also with neurological disease in dogs and horses. Other reports for monocular nystagmus in animals are limited to a cat with suspected brainstem anaesthesia as a complication of retrobulbar block,26 and Belgian sheepdogs with hereditary hemichiasma/achiasma and uniocular saccades.27,28

HBP is mainly seen in ophthalmological disease affecting directly or indirectly the vision. Dogs with retinal dysplasia,13,16 as well as dogs with congenital blindness14 have been reported to manifest HBP. Congenital cataract is one of the most commonly reported HBP cause,15 and particular in Cocker Spaniels.10 Familial cataract in Chow chow has manifested HBP along with other ocular abnormalities such as entropion, microphthalmia, persistent pupillary membrane remnants, and multifocal retinal folds.14 Persistent pupillary membranes were accompanied by HBP in Basenji dogs.8 In Great Danes with inherited bilateral persistent hyperplastic primary vitreous and persistent hyperplastic *tunica vasculosa lenti* and secondary blindness, HBP was observed.17 HBP has been recently reported in a 5-month-old Northern Inuit dog diagnosed with inherited oculosceletal dysplasia.18 This dog had congenital microphakia, vitreopathy, retinal detachment, immature cataract resulting in complete bilateral blindness and thus HBP, described as binocular and vertical.18 Although neither described in detail nor referred as monocular or binocular, nystagmus that might represent HBP has been described in young Whippet dogs with progressive retinal atrophy,29 Briard dogs with inherited retinopathy,30 Miniature Schnauzer dogs with congenital cataract and microphthalmos,31 wirehaired Dachshunds with hemeralopia,32 but also in cats with hereditary retinal dystrophy33 and a maned wolf puppy with nutritional cataract,34 all with visual impairment. In horses, HBP has been described as vertical, but with wandering features and it has been associated with congenital peripheral blindness,35 such as in foals with suspected deprivation amblyopia.36 HBP has been also described in Toggenburg goats with retinal degeneration.37

Only few cases of dogs have been reported to manifest HBP due to a neurological disease. The first case report describes a litter of St Bernard puppies with cerebellar cortical dysplasia of unknown aetiology, who were unable to stand, unable to fix the gaze, lacked of pupillary light reflexes and had bilateral ventrolateral strabismus and excursive movements of the head. All dogs were euthanized due to deterioration and histopathology revealed cortical dysplasia of the cerebellum.12 In the previous case, it is not clear if the eye movement abnormality is a true HBP, or a saccadic intrusion related with the cerebellar disease. At that time, saccadic intrusion had not been described broadly, and thus we suspect that a possible saccadic intrusion such as opsoclonus or macrosaccadic oscillations could be in the differential diagnoses list for this dog along with HBP. Nevertheless, due to the lack of a video recording and the nomenclature used within the manuscript, the authors consider it as an HBP case. The second case described a miniature poodle with congenital cortical blindness and associated HBP.9 At last, horses with congenital optic nerve hypoplasia and visual impairment have been reported to manifest HBP.36

It is important not to confuse HBP with congenital nystagmus. The latter might be recognized in association with ocular abnormalities and congenital visual deficits (e.g. Belgian Sheepdogs with achiasma),27 but it can be also idiopathic presented as binocular conjugate jerk or pendular nystagmus in the absence of other ocular abnormalities (e.g. cats with congenital pendular nystagmus).38 Congenital nystagmus, also, can coexist with other type of nystagmus such as HBP and see-saw nystagmus.27

Congenital cataract accompanied by other ocular abnormalities and ocular nystagmus are frequently seen in Cocker Spaniels.39 In our case, we could not safely conclude that the cataract was congenital, however historically the dog had nystagmus since a puppy and in the light of the nature of the nystagmus (HBP), we can presume that congenital cataract accompanied by HBP on the left eye could be high likely. The CSF mixed cell pleocytosis in our case initially was associated with presumptive MUO regardless of the unremarkable MRI findings. Nevertheless, the dog did not respond to immunosuppressive steroid treatment and the HBP continued until the two-year follow-up. Additionally, the dog manifested HBP since early youth, and thus an ongoing MUO without other neurological signs was considered unlikely. Mild-to-moderate CSF mixed cell pleocytosis might result from necrosis or inflammation secondary to a variety of diseases such as intervertebral disc disease, infection, haemorrhagic myelomalacia, ischaemia, or infarction.40 Therefore, this pleocytosis was attributed to either a cause that we missed (e.g. the whole central nervous system was not scanned and thus a potential age-related degenerative mildly compressive intervertebral disc protrusion cannot be ruled out) or to the blood contamination of CSF. The latter might be less strong scenario, based on the fact that a CSF blood contamination is characterized predominantly by neutrophils,40 as well as that the previously published correction formulas suggest that CSF blood contamination increases the total nucleated cell count by 1 leukocyte and protein concentration by 0.5 mg/dL for every 500 RBC/μL.41 In our case this formula would result in a corrected total nucleated cell count of 9 cells/μL, which would be again compatible with pleocytosis. Additionally, recent studies support that there is lack of correlation between RBC counts and total nucleated cell counts, and therefore these formulas overcorrect for total nucleated cell counts and therefore can obscure possible disease processes.42 As a result, the scenario of having missed a disease which could have caused a true mixed cell pleocytosis in the CSF (e.g. intervertebral disc disease) is most likely.

HBP is a benign clinical sign.7 For an accurate clinical recognition of HBP, it is recommended that the affected eye must be observed for at least one minute to clearly demonstrate the tell-tale slow-low frequency vertical oscillation.25,43 Then, both two eyes should be observed simultaneously, so as any unilateral eye movements to be easily picked up. Differential diagnoses for monocular nystagmus in humans, though, include alternating hemiplegia of childhood, amblyopia, strabismus, monocular blindness, internuclear ophthalmoplegia, monocular nystagmus of childhood (associated with longstanding monocular vision impairment due to amblyopia, optic neuropathy, or dense cataract), acquired pendular nystagmus, ocular neuromyotonia, oculopalatal myoclonus, see-saw nystagmus, latent nystagmus, spasmus mutans, multiple sclerosis, optic chiasm neoplasia (e.g. glioma) or epilepsy.6,7,44,45 There is not established medical treatment for HBP,7 however gabapentin has been reported to suppress HBP in humans.46 In human patients with HBP associated to strabismus, strabismus surgery resulted sporadically in reduction of HBP, however some of them did not respond at all.7 Although vision improving surgery has not consistently shown to improve the nystagmus,47 studies revealed improvement of HBP after vision impairment repair.48

**Conclusions**

Although the most common type of eye movement abnormality seen in veterinary practice is the bilateral conjugate jerk nystagmus, monocular nystagmus exists in dogs and it should be recognised. HBP is a benign phenomenon of monocular nystagmus associated with partial or complete vision impairment, which should raise mostly a suspicion of mostly ophthalmological, but also neurological pathology affecting the vision. The authors felt it was important to raise awareness of clinicians, so as to recognise the HBP in the frame of clinical reasoning.

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**Figure captions**

**Figure 1**

A 6-year old Cocker Spaniel with a left-sided cataract, ipsilateral visual impairment and left monocular eye movement abnormality (*Heimann - Bielschowsky phenomenon*) which gives to the examiner the impression of a ‘floating’ eye within the globe.