

BRIEF COMMUNICATION

Idiopathic Unilateral Transient Miosis in Dogs

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ABSTRACT

Purpose: To characterize the clinical features of idiopathic unilateral transient miosis in dogs.**Methods:** Owner- and veterinarian-completed questionnaires were analyzed to identify and characterize dogs with idiopathic unilateral transient miosis.**Results:** Seventy-eight dogs met the inclusion criteria, showing no additional ocular or systemic abnormalities. Australian Shepherds (ASH), ASH mixes ($n=41$), and Miniature Australian Shepherds (MASH) ($n=15$) were markedly overrepresented, while other breeds were only sporadically affected. The merle phenotype was documented in 70% of cases, and 61% of affected dogs were female. The mean age at onset of the first episode was 6.5 months. Miosis was reported after sleep in 74% of cases and following physical activity in 22%. Blue eyes were affected in 76% of dogs, light-colored irises in 6%, heterochromatic eyes in 8%, and brown eyes in 13%. The median episode duration was 30 min. Recurrent episodes were reported in 71% of dogs (median: 2 episodes), with side alternation occurring in 62% of these cases. No significant interbreed differences were observed.**Conclusions:** Idiopathic unilateral transient miosis, in the absence of other ocular or systemic disease, occurs predominantly in young dogs and is seen across various breeds and eye colors. Australian Shepherds, Miniature Australian Shepherds, the merle phenotype, blue eyes, and females were disproportionately represented in this cohort. Episodes were most commonly observed after sleep, typically lasted around 30 min, and recurred in more than half of affected dogs. To the authors' knowledge, this is the first report of idiopathic unilateral transient miosis in dogs.

1 | Introduction

Transient unilateral miosis in the absence of ocular, neurological, or systemic abnormalities has been observed in dogs and occasionally reported anecdotally by owners, breeders, and veterinarians. To date, however, the phenomenon has not been systematically described in the scientific literature. In online discussion forums and mailing lists, it has frequently been referred to as intermittent anisocoria and anecdotally associated with blue-eyed dogs. The objective of the present study was to

characterize the clinical features of idiopathic unilateral transient miosis in dogs.

2 | Materials and Methods

Dog owners who had observed distinct episodes of unilateral transient miosis and/or marked anisocoria in their dogs, without concurrent ocular or systemic abnormalities, were invited to complete a standardized questionnaire. For the purpose of this

study, miosis was defined as excessive constriction or abnormal narrowing of the pupil [1].

2.1 | Data Collection

The questionnaire collected information on breed, age at onset, merle phenotype, sex, laterality of the affected eye, and iris color of the affected eye(s). Additional items addressed potential triggering events (e.g., vigorous play, prolonged walks, agility training, or sleep), duration of episodes, side switching in recurrent cases, total number of episodes, results of ophthalmological and/or general clinical examinations, concurrent physical findings, and pre-existing medical conditions. Owners were asked to provide photographic documentation of affected eyes during miosis episodes. Written informed consent for the use of data and photographs for research and publication was obtained from all participants. This study was conducted in accordance with GERVO guidelines and was approved by the competent authority (Landesamt für Gesundheit und Soziales, Berlin, Germany; registration number: StN° 0042-2024).

2.2 | Inclusion and Exclusion Criteria

Only fully completed questionnaires were included in the analysis. Exclusion criteria were incomplete responses, any ocular or systemic abnormalities occurring before, during, or after the episodes, or the administration of medication prior to or during an episode. Questionnaire data were analyzed descriptively.

2.3 | Statistical Analysis

Statistical analyses were performed using SPSS version 29. Breed differences in categorical variables were assessed using chi-square tests. Breed differences in continuous variables were analyzed using the Wilcoxon rank-sum test (Mann–Whitney *U* test). A comparison of means was not considered appropriate

due to the presence of strong outliers. Confidence intervals (95%) were calculated to indicate the plausible range of population values. Analyses were conducted by Novostat GmbH (Wollerau, Switzerland).

3 | Results

3.1 | Study Population

A total of 78 dogs met the inclusion criteria. Dogs with incomplete questionnaires or evidence of ocular or systemic abnormalities before, during, or after episodes of miosis were excluded. Breeds represented included Australian Shepherds (ASH; $n=37$), ASH mixes ($n=4$), and Miniature Australian Shepherds (MASH; $n=15$). Additional breeds were Siberian Huskies (SH; $n=5$) and SH mixes ($n=2$), Labrador Retrievers (LR; $n=1$) and LR mixes ($n=3$), Border Collies (BC; $n=2$) and a BC mix ($n=1$), as well as Catahoula mixes ($n=2$). Single cases included French Bulldog, Greek Shepherd, German Shepherd, Golden Retriever, Australian Cattle Dog, and Bernedoodle ($n=1$ each) (Figures 1 and 2).

3.2 | Demographics

Of the affected dogs, 62% (48/78) were female (95% CI: 50.5%–71.8%), comprising 40 intact and 8 spayed females. Males accounted for 38% (30/78), of which 26 were intact and 4 neutered. The mean age at first episode was 6.5 months (range: 2.5–108 months) (Figure 3). Most dogs (81%, 63/78) experienced their first episode before 2 years of age, whereas only four dogs (5%) were older than 4 years at onset.

3.3 | Phenotypic Characteristics

The merle phenotype was observed in 71% (55/78) of dogs. Among non-Australian Shepherd breeds, 9/22 dogs expressed

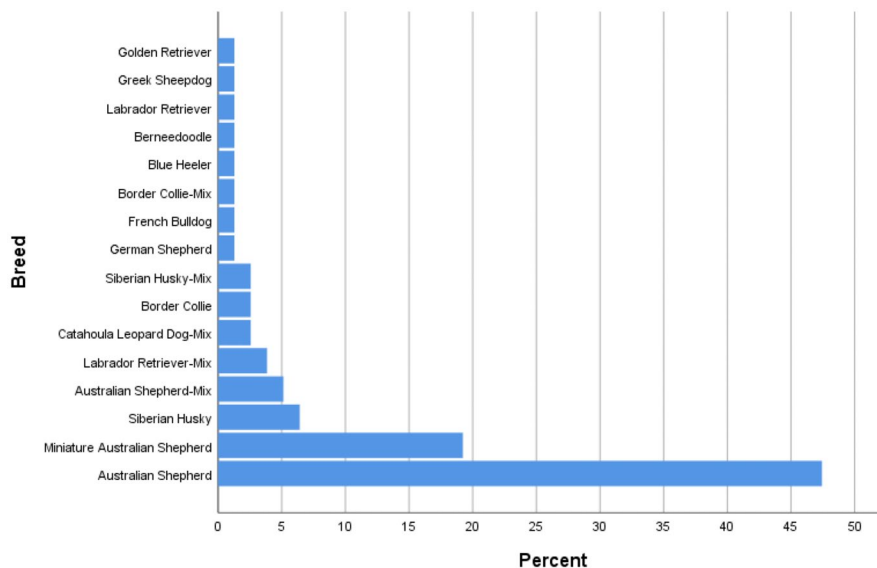


FIGURE 1 | Dog breeds in which transient unilateral idiopathic miosis was observed.



FIGURE 2 | Distribution of transient unilateral idiopathic miosis among different breeds.

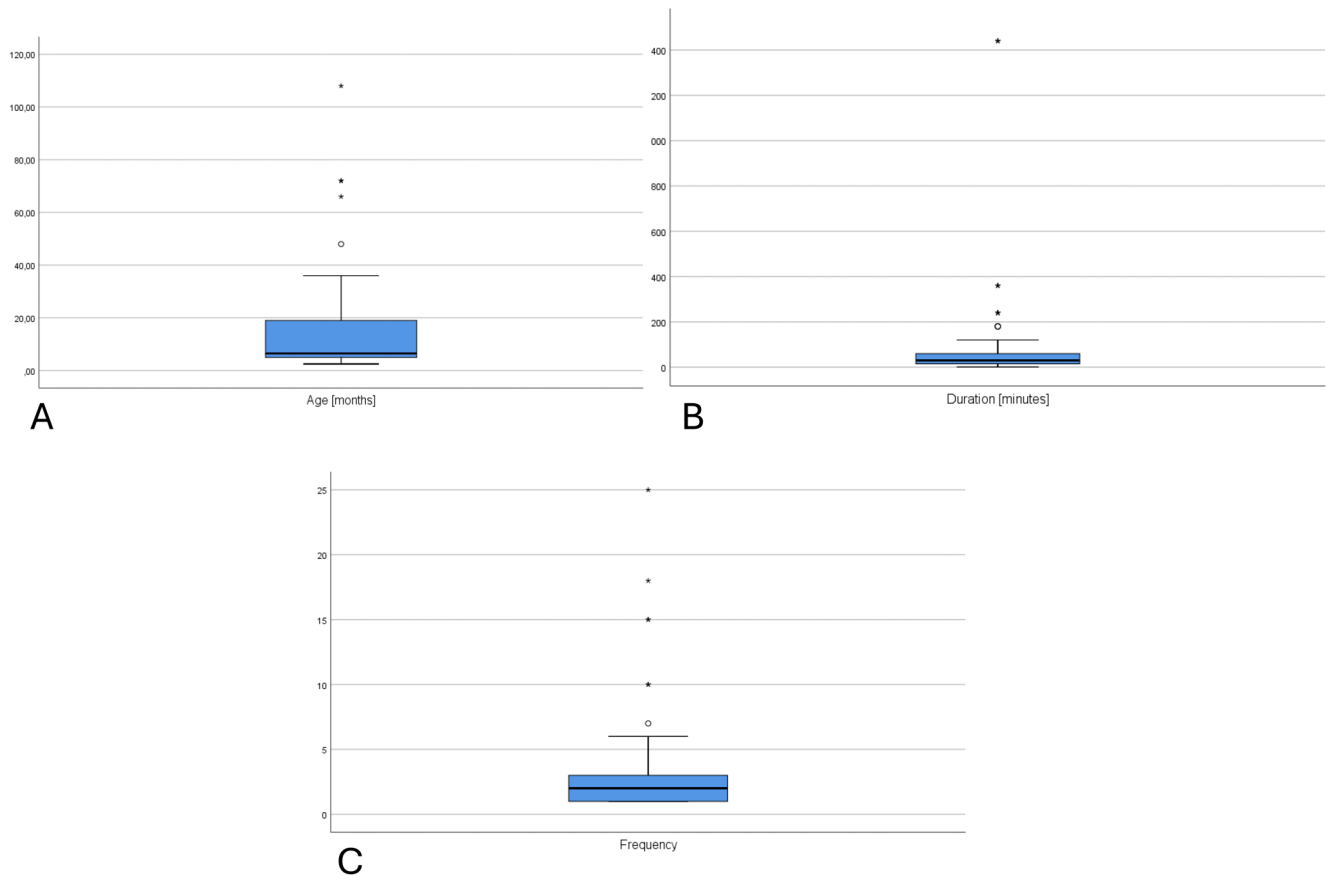


FIGURE 3 | (A) Age at first episode, (B) mean episode duration, and (C) mean episode frequency in dogs with transient unilateral idiopathic miosis.



FIGURE 4 | Close-up images of affected eyes, illustrating variation in iris color in dogs with transient unilateral idiopathic miosis.



FIGURE 5 | Representative cases of dogs experiencing multiple episodes and alternating laterality.

the merle phenotype; the remaining were Siberian Huskies or mixed breeds. Three sibling pairs were reported, with the first episodes occurring at 2.5 months (two dogs) and 6 months (four dogs). Despite their relatedness, siblings differed in sex, merle status, eye color, episode duration, and frequency.

3.4 | Triggers and Eye Color

Episodes occurred after sleep in 77% (58/75) of cases and following physical activity in 23% (17/75). Three dogs with recurrent episodes experienced miosis in both contexts. The affected

eye displayed blue irides in 76% (59/78), light-colored irides in 6% (5/78), heterochromia in 8% (6/78), and brown irides in 13% (10/78) (Figure 4). Of the brown-eyed dogs, 8/10 were Australian Shepherds, with the remainder being a German Shepherd and a Golden Retriever.

3.5 | Episode Characteristics

The median duration of miosis was 30 min (range: 5–1440 min) (Figure 3). Prolonged episodes (> 1 h) occurred in 18% (14/78), with 9% (7/78) lasting more than 2 h. Recurrent episodes were reported in 71% (55/78), with a median of two episodes (range: 1–25). Only six dogs experienced ≥ 10 episodes. In recurrent cases ($n = 45$), laterality shifted in 62% (48/78) (Figure 5). Some owners reported constriction developing within less than 5 min. In one case, a right eye episode lasted 3–5 min, followed within 3 h by a left eye episode lasting approximately 2 h. No significant breed differences were detected for age at first episode ($U = 272$, $p = 0.509$), duration ($U = 263$, $p = 0.406$), or frequency ($U = 251$, $p = 0.271$).

3.6 | Clinical Examination

Comprehensive examinations (general, ophthalmological, and neurological) were performed in 62% (48/78) of dogs by ECVO- or ACVO-certified ophthalmologists. Apart from miosis, no ocular abnormalities were identified. Neuro-ophthalmological assessments were not available. Advanced diagnostics (MRI, cerebrospinal fluid analysis) performed in one dog yielded unremarkable results. No complications associated with transient miosis were reported.

4 | Discussion

4.1 | Interpretation of Findings

This study provides the first formal description of a clinical phenomenon we have termed *idiopathic unilateral transient miosis*. Until now, it had been reported only anecdotally by owners, breeders, and veterinarians. In our cohort, Australian Shepherds, Australian Shepherd mixes, and Miniature Australian Shepherds were markedly overrepresented (72%, 56/78). Most affected dogs not only belonged to the Australian Shepherd group but also exhibited the merle phenotype. Episodes were, however, also observed in unrelated breeds such as Golden Retrievers and German Shepherds, which lack both color dilution and the merle phenotype. Genetic testing was not performed, and thus the role of the merle gene in affected individuals remains undetermined.

Dogs with blue eyes, light-colored irises, or heterochromia iridis comprised the majority of cases, although 13% of affected eyes were brown. Of these brown-eyed dogs, 8/10 were Australian Shepherds, while two (a German Shepherd and a Golden Retriever) belonged to other breeds. These findings demonstrate that the previously used term *intermittent miosis in blue-eyed dogs* is inaccurate. The apparent predominance of blue-eyed dogs may reflect increased detectability of miosis in lighter eyes,

as episodes in brown-eyed dogs could be overlooked. Given the short duration and subtle presentation, the true incidence is likely underestimated.

In this series, 62% of affected dogs were female (95% CI: 50.5%–71.8%). At the first documented episode, 81% were younger than 2 years. It is plausible that some dogs experienced unrecognized episodes earlier in life.

Previous anecdotal reports linked transient unilateral miosis to vigorous activity or rough play. In our series, however, 77% of episodes occurred after sleep, and only 23% followed physical activity. Three dogs with recurrent episodes exhibited miosis after both conditions. Episode duration was short in nearly all dogs, with a median of 30 min (range: 5–1440 min; see outliers in Figure 3). Prolonged episodes (> 1 h) were rare. Recurrent episodes occurred in 71% of cases, usually twice, although up to 25 episodes were documented in individual dogs.

Classical models proposed exclusive parasympathetic innervation of the sphincter and sympathetic innervation of the dilator [2]. More recent studies in dogs demonstrate dual reciprocal innervation of both muscles by cholinergic and adrenergic systems [3]. Functionally, activation of one muscle is reciprocally inhibited by its antagonist. Thus, sphincter contraction results from cholinergic excitation, while concurrent cholinergic inhibition of the dilator augments miosis. Conversely, mydriasis involves adrenergic stimulation of the dilator, adrenergic inhibition of the sphincter, and central suppression of parasympathetic output [3]. Cholinergic inhibition of the dilator has been demonstrated in both dogs and cats, while alpha-adrenergic inhibition of the sphincter enhances contraction and miosis via alpha-1-adrenergic receptors [3].

4.2 | Pupil Size Control

The efferent limb of the pupillary light reflex (PLR) comprises two parasympathetic neurons: preganglionic parasympathetic pupillomotor fibers travel superficially and medially along CN III to the ciliary ganglion, where they synapse [4]. Postganglionic fibers of the short ciliary nerve then join sympathetic fibers of the long ciliary nerve to innervate both sphincter and dilator muscles, reflecting the dual reciprocal innervation [4].

The dilator muscle is controlled by a three-neuron sympathetic pathway originating in the hypothalamus. Fibers descend to T1–T4, synapse in the spinal cord, and exit as preganglionic fibers via the vagosympathetic trunk to the cranial cervical ganglion. Postganglionic fibers then traverse the middle ear and orbit to reach the iris [5].

Pupil size and PLR function are further modulated by the state of consciousness. During arousal, sympathetic activation enhances dilator activity while simultaneously inhibiting the sphincter, leading to rapid dilation. In contrast, sleep reduces supranuclear inhibition, thereby increasing parasympathetic tone and producing miosis [5].

In dogs, anisocoria commonly results from afferent or efferent pupillary pathway dysfunction, iris muscle myopathy, or

mechanical restriction (e.g., synechia). Idiopathic unilateral transient miosis reflects such pathology. The partial decussation of canine optic nerves allows differentiation of afferent and efferent PLR lesions via dark adaptation [4].

Our dataset lacks results from dark adaptation, as this critical assessment was not performed. The procedure requires placing the patient in complete darkness for 3–5 min—for example, by coverage of both patient and examiner with a blanket. With the light source extinguished, a direct ophthalmoscope is positioned between the eyes, while the patient's muzzle is aligned with the optics at arm's length. After the prescribed dark adaptation period, the light source is abruptly activated, and the pupils are observed through the ophthalmoscope [5].

Afferent lesions (retina, optic nerve, visual pathway) induce maximal, symmetric dilation due to absent sphincter stimulation and ongoing sympathetic input to the dilator, distinguishing them from mechanical causes [5]. Retinal or prechiasmal optic nerve lesions typically cause contralateral anisocoria or miosis, a positive swinging-flashlight test, and a negative dazzle reflex. Isolated optic nerve lesions produce subtle ipsilateral miosis with a negative swinging-flashlight test but a positive dazzle reflex [5].

Mechanical restriction can usually be excluded clinically [5]. While iris smooth muscle myopathy has been reported in humans (e.g., MSMDs) [6], it has not yet been documented in dogs.

Cortical influences on pupillary control remain incompletely understood [7–9]. While experimental studies have demonstrated cortical areas capable of eliciting pupillary constriction [10–13], the cortico-subcortical networks that regulate autonomic pupil function are not yet fully elucidated [8, 9, 14]. Ictal miosis has been reported as a rare manifestation of focal epilepsy without impairment of consciousness in humans [8, 14–17]. Although not described in dogs, ictal miosis should be considered a differential diagnosis; electroencephalograms (EEGs) during or between episodes may provide confirmation.

Physiological anisocoria, common in humans (10%–20%) [18], has not been described in dogs. The phenomenon reported here differs in that it is transient and resolves spontaneously.

The transient, unilateral nature of this condition in the absence of additional neurological deficits argues against a cortical origin. A peripheral mechanism affecting efferent pupillomotor fibers appears more plausible. Severe lesions in these pathways typically produce persistent anisocoria, whereas transient irritation of pre- or postganglionic fibers could account for brief miotic episodes.

Lesions involving the pupillomotor fibers of either the efferent arm of the pupillary light reflex (PLR) or the sympathetic pathway generally result in persistent anisocoria. Severe damage to the efferent arm produces a fixed, dilated pupil, whereas interruption of sympathetic conduction causes miosis, as in Horner's syndrome. In both conditions, the changes are typically neither transient nor recurrent.

For diagnostic evaluation, assessment of the pupils under dark adaptation is essential. A lack of pupillary change, or only

minimal change in miosis during dark adaptation, indicates involvement of the preganglionic parasympathetic fibers of the oculomotor nerve (cranial nerve III). These fibers are located superficially and are therefore particularly susceptible to inflammatory or irritative processes. If the fibers remain intact—whose disruption would otherwise result in fixed mydriasis—irritation may instead manifest as intermittent miosis.

The fluctuating degree of miosis observed in this study supports a mechanism of irritation within the efferent pupillomotor pathway leading to constriction. The orbit represents a likely site of involvement, particularly in the presence of penetrating foreign bodies or extension of odontogenic abscesses. Given the cranial course of these fibers into the orbit, both preganglionic and postganglionic segments may be affected. However, such involvement would not typically manifest as transient or intermittent miosis.

Sympathetic pathway lesions may arise from middle ear disease, orbital pathology, or disorders involving the vagosympathetic trunk. Severe interruption of this pathway to the iris dilator muscle produces the classic miosis of Horner's syndrome, which is likewise characteristically persistent rather than transient or recurrent.

5 | Summary and Conclusion

Dogs presenting with idiopathic unilateral transient miosis should undergo a complete evaluation, including dark adaptation, swinging flashlight, and dazzle reflex testing, as well as EEG to exclude focal epilepsy. Behavioral or dietary changes should also be documented, and all medications recorded. Differential diagnoses include congenital pupil anomalies, uveitis, synechia, and Horner's syndrome. Potential mechanisms involve irritation of peripheral pupillomotor fibers or disturbances in reciprocal iris innervation. Trauma, inflammation, and medication effects remain possible but unlikely causes given the brief, self-limiting nature of episodes.

We propose the term *idiopathic unilateral transient miosis*, preferring *transient* to *intermittent*, as 40% of dogs in this series exhibited a single episode only.

The principal limitation of this study is its dependence on retrospective owner questionnaires and subjective photographic documentation, without standardized clinical testing. Additional limitations include variability in lighting conditions, subjective evaluation of miosis by owners, lack of standardized pupillometry, and inconsistent monitoring of progression.

Digital photography has been investigated as an alternative to standardized clinical techniques for pupillary assessment of pupil size under varying illumination [18]. While the sensitivity of routine clinical examination for detecting early anisocoria remains limited, digital image-based methods demonstrate superior reproducibility and accuracy when compared with conventional clinical methods [19], and allow archiving for independent classification. Digital photography permits permanent archiving and provides an independent classification approach that is well-suited for research purposes [18]. Accordingly, the

submitted digital photographs of the dogs were deemed sufficient to establish anisocoria in this context. Despite observer variability, consistent findings across cases highlight the clinical relevance of this observation.

6 | Conclusion

Idiopathic unilateral transient miosis, in the absence of other ocular or systemic disease, occurs in young dogs across various breeds and eye colors. Australian Shepherds, Miniature Australian Shepherds, the merle phenotype, blue eyes, and females were overrepresented. Episodes occurred predominantly after sleep, lasted ≤ 30 min, and recurred in more than half of cases. To our knowledge, this is the first formal description of this phenomenon in dogs.

Author Contributions

Ingrid Allgoewer: conceptualization, investigation, funding acquisition, writing – original draft, methodology, validation, visualization, writing – review and editing, project administration, data curation, resources, formal analysis, supervision. **Randall H. Scagliotti:** writing – original draft, writing – review and editing.

Acknowledgments

We thank the owners and veterinarians who reported cases of transient miosis and submitted completed questionnaires with photographs.

Disclosure

Artificial intelligence generated content: The authors have not used AI to generate any part of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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