First case of presumed trigemino-oculomotor synkinesis in a dog

Silvia Pompilio, Michela Scuttari, Katia Zerbetto, Maria Elena Andreis, Federica Tirrito

First published: 08 February 2024

https://doi.org/10.1111/vop.13191

Abstract

An 11-year old, intact male Border Collie was referred with a history of subacute and progressive left eye exophthalmos and mydriasis associated with reduced pupillary light reflex, ventrolateral strabismus, and absence of physiologic nystagmus in the left eye. Neuroanatomical localization was consistent with a left oculomotor neuropathy, involving the general somatic and visceral parasympathetic efferent components. Computed tomography and magnetic resonance imaging of the head were performed. Imaging findings were consistent with an infectious-inflammatory process involving the left retrobulbar space and regional muscles, extending intracranially through the left orbital fissure. Cerebrospinal fluid (CSF) was collected from the cerebellomedullary cistern, and the analysis revealed albuminocytologic dissociation. The dog was treated with amoxicillin and clavulanic acid and prednisolone at anti-inflammatory dose; a significant improvement of neurologic status was observed afterward. However, 4 weeks after the initial presentation, the dog showed an abnormal, bilateral adduction of both eyes and third eyelid protrusion of the left eye while chewing the leash; the dog's mental status was normal, and the patient did not appear to be in discomfort during these episodes. A presumptive diagnosis of acquired trigemino-oculomotor synkinesis, induced by the intracranial inflammation was made. To the authors' best knowledge, this is the first case of presumed trigemino-oculomotor synkinesis reported in veterinary medicine.

1 INTRODUCTION

Synkinesis is widely reported in the human literature but is relatively rare in veterinary medicine.[1-3] It is defined as an unintentional movement of one part of the body when a voluntary movement is performed by another body part.[1] This condition is known to have a variety of patterns and cause involuntary stimulation and contraction of muscles that are not normally supplied by those nerves.[4] As a result, the facial movements become uncoordinated leading to functional disabilities while drinking and eating and it may impact nonverbal communication and facial expressions.[5]

The different patterns of synkinesis are named by the motor groups that are abnormally connected: firstly, naming the voluntary motor group, followed by the associated involuntary muscles movement.[6]

In human medicine, the synkinesis is described as being either congenital or acquired.[7] The congenital form may be due to familiar predisposition, abnormal migration of nerve fibers in utero, aberrant nerve regeneration following congenital disease, or fetal exposure to isoretinoin A.[7, 8] Different variants of congenital synkinesis are described, collectively known as congenital cranial dysinnervation disorders (CCDDs), which are a group of congenital, nonprogressive disorders that are associated with anomalous ocular, eyelid, and facial movements.[9] This group of

disorders includes congenital fibrosis of extraocular muscles (CFEOM), congenital ptosis, Duane syndrome (DS), horizontal gaze palsy with progressive scoliosis (HGPPS), congenital facial palsy, Möbius' syndrome, the Bosley-Salih-Alorainy/Athabaskan brainstem dysgenesis syndrome (BSAS), congenital third and fourth nerve palsies, and Marcus Gunn jaw winking syndrome (MGJWS).[2, 10, 11] The most commonly described forms of CCDDs are the DS and the MGJWS.[2, 10, 12] The Marcus Gunn jaw winking syndrome involves the oculomotor and the trigeminal nerves, and is a form of trigemino-oculomotor synkinesis.[8, 12] The trigemino-oculomotor synkinesis consists of an abnormal connection between the mandibular branch of the trigeminal nerve, controlling the muscles of mastication (masseter, temporal, pterygoid, rostral digastricus, and mylohyoideus) and the oculomotor nerve which innervates some of the rectus muscles (medial, dorsal, and ventral), the ventral oblique muscle, and levator palpebrae superioris muscle.[12-14] As a result, mastication leads to coordinated activation of the associated muscles, causing synkinetic movements of the upper eyelids and of the eyes.[8, 12, 15] The abnormal movement can be seen during smiling, chewing, respiration, Valsalva maneuver, contraction of the sternocleidomastoid muscle, suction, and even tongue protrusion.[15] The acquired synkinesis may develop secondary to trauma, neoplasms, infectious diseases, autoimmune disorders, cerebrovascular accidents, or iatrogenic causes such as orbital surgeries.[1, 5, 16, 17] In veterinary medicine, a case of acquired trigemino-abducens synkinesis has been reported in a Rhodesian Ridgeback.[1] Additionally, a case of suspected congenital cranial dysinnervation disorder resembling the DS in humans, has been documented in a young French Bulldog.[2] To the authors' knowledge, trigemino-oculomotor synkinesis has only been described in human literature with no reports in veterinary medicine.[6, 11] The aim of this study was to describe the clinical presentation, diagnostic findings, treatment, and outcome of a dog affected by presumed, acquired trigemino-oculomotor synkinesis induced by retrobulbar infectious-inflammatory disease.

2 CASE PRESENTATION

2.1 History, clinical evaluation, and diagnostic findings

An 11-year old, intact male Border Collie was referred to the ophthalmology department of the AniCura Istituto Veterinario di Novara (Italy) with a 3-week history of subacute and progressive left eye (OS) exophthalmos. The eye was previously topically treated by the referring veterinarian with netilmicin 0.3% (Xanternet; SIFI S.p.A., Catania, Italy) and tobramycin 0.3% (S.A. Alcon-Couvreur; Puurs, Belgium). Aside from the OS exophthalmos, the owner reported the dog was otherwise normal.

A complete physical examination was unremarkable. The ophthalmic examination revealed exophthalmos, ventrolateral strabismus, and mild third eyelid protrusion OS; pupils were asymmetrical, with the OS mydriatic compared to OD (right eye). Direct and consensual (OD to OS) pupillary light reflex was incomplete OS and normal OD. The menace reaction, dazzle reflex, as well as corneal and palpebral reflexes were normal in both eyes. Examination OD was unremarkable. Periorbital palpation and globe ocular retropulsion were bilaterally unremarkable, without any pain upon opening the jaw. Intraocular pressure assessed by rebound tonometry (Tonovet, Helsinki Icare, Oy, Finland) was 14 mmHg and 16 mmHg in the OD and OS, respectively. Schirmer tear test 1 was 22 mm/min in the OD and 21 mm/min in the OS. Slit-lamp biomicroscope (Kowa SL-15 Ltd., Tokyo, Japan) and indirect ophthalmoscopy (Heine Omega 500; Heine Instruments, Herrsching, Germany) excluded other intraocular abnormalities. Fluorescein stain test was bilaterally negative.

Based on the ophthalmic examination, retrobulbar disease with concurrent involvement of the left oculomotor nerve was suspected. Primary differential diagnoses included a foreign body migration, an infectious-inflammatory process, or neoplasia.

Hematology, serum biochemistry, and computed tomography (CT) of the head were performed to better characterize the potential retrobulbar lesion.

The blood cell count and serum biochemistry profile showed no significant abnormalities.

Pre- and post-contrast CT (64 slices, Optima 660, General Electric, Milan, Italy) was conducted under general anesthesia. A left-sided retrobulbar mass effect was noted, with retrobulbar soft tissue swelling and enhancement effacing retrobulbar fat, adjacent left extraocular muscles, left ophthalmic plexus, left optic nerve, and left maxillary artery, resulting in exophthalmos. Partial intracranial extension of the pathologic process through a mildly widened left orbital fissure and alar foramen was observed, with evidence of a sub-centimetric nodular soft tissue attenuating and homogeneously contrast-enhancing expansile lesion, visible in the left parasellar region (Figure 1). Differential diagnoses for these changes included inflammatory and neoplastic diseases. A urinalysis, including bacterial and mycotic culture was performed with no abnormal findings.

Treatment with orally administered amoxicillin and clavulanic acid (Synulox, Zoetis S.r.l., Rome, Italy) 20 mg/kg q12h and prednisolone (Prednicortone, Dechra Veterinary Products S.r.l., Turin, Italy) 0.5 mg/kg q24h, as well as topical therapy with ophthalmic hyaluronic acid-based solution (Theraoftal Ialu, Bioforlife S.r.l., Milan, Italy) q8h was instigated. The dog was reexamined 10 days after starting treatment. The OS exophthalmos was improved. However, the third eyelid protrusion, mydriasis, and strabismus of OS were not notably changed. Due to the incomplete remission of the clinical signs, a neurologic examination was recommended. Treatment with amoxicillin and clavulanic acid was continued, while the dose of prednisolone was reduced to 0.25 mg/kg q24h for 4 days before being directly interrupted, considering the low current dose and the possibility of an inflammatory-infectious disease.

The neurologic examination confirmed exophthalmos, mydriasis, and reduced direct and consensual (OD to OS) pupillary light reflex OS; direct and consensual (OS to OD) pupillary light reflex was normal OD (Figure 2). Therefore, a dysfunction of the efferent parasympathetic component of the left oculomotor nerve was considered.[13] Furthermore, the OS presented ventrolateral strabismus and did not adduct normally on testing physiologic nystagmus (Figure 2) suggesting an involvement of the general somatic efferent component of the third cranial nerve.[13] Vision appeared unaffected with a normal bilateral menace reaction and dazzle reflex. No other abnormalities were detected during the neurologic examination. Based on the abovementioned findings, the neuroanatomical localization was consistent with a left oculomotor neuropathy, involving the visceral parasympathetic and the general somatic efferent components.

To further evaluate the case, magnetic resonance imaging (MRI) of the head, and cerebrospinal fluid (CSF) analysis were recommended. The MRI study (1.5 Tesla, SIGNA Creator, General Electric, Milan, Italy) was performed 1 week after the neurologic examination. The MRI protocol included transverse and sagittal T2-weighted images (T2-WI), transverse T1-weighted images (T1-WI), dorsal short tau inversion recovery (STIR) sequences, and transverse fluid attenuated inversion recovery (FLAIR) images. Post-contrast transverse T1-WI were acquired after intravenous administration of 0.2 mL/kg of gadolinium (gadoteric acid, claricyclic, 0.5 mmol/mL, GE Healthcare S.r.l., Milan, Italy).

The MRI study demonstrated improvement but not a complete resolution of the left retrobulbar abnormalities previously detected on CT images. Specifically, T2- and STIR-hyperintense left-sided retrobulbar soft tissue swelling was observed with mild heterogeneous enhancement on post-contrast T1-WI. The most relevant muscle changes were identified in the left extraocular muscles and left temporalis muscle (Figure 3). The extraocular muscles effected by the pathologic process were the portion of the left dorsal oblique, medial and ventral rectus muscles closer to the eyeball, as well as the deep segments of the extraocular muscles (dorsal rectus, ventral rectus, medial rectus, lateral rectus, dorsal oblique, and retractor bulbi) near to the optic canal and orbital fissure, where they are closely situated. However, it was not possible to clearly distinguish them due to the retrobulbar soft tissue swelling and the ill-defined enhancement. Enhancement of the left ventral oblique muscle was also detected. Partial intracranial extension through the left orbital fissure was also confirmed; however, the left-sided intracranial parasellar space-occupying lesion previously identified on CT images was no longer visible on MRI. As a result of the improvement of the lesions previously identified on the CT scan, an infectious-inflammatory process was judged as the main differential diagnosis.

Cerebrospinal fluid was collected from the cerebellomedullary cistern, and an albuminocytologic dissociation was identified. The protein concentration was 48 mg/dL (normal value: <25 mg/dL); nucleated cell count was 1/μL (normal value: <5 cells/μL). No bacterial growth was observed in CSF culture.

Due to the partial improvement of clinical signs and the diagnostic findings, antibiotic therapy with amoxicillin and clavulanic acid was continued.

2.2 Follow-up

Two weeks after the MRI study, the dog was reexamined, and further improvement in clinical signs was observed. Mild exophthalmos, mydriasis, and ventrolateral strabismus OS were still present; the physiologic nystagmus and the direct and consensual pupillary light reflex were present OU. In addition, the owner reported that the dog showed an abnormal eye movement when chewing the leash. This movement consisted of a bilateral adduction of the eyes, third eyelid protrusion OS, and abnormal, bilateral, upper eyelid movements (Video S1). During these episodes, the dog's mental status was completely normal and there were no signs of pain or discomfort.

These clinical signs were considered compatible with a form of synkinesis, involving the trigeminal and oculomotor nerves.

The antibiotic treatment with amoxicillin and clavulanic acid was continued, due to the partial improvement but not full resolution of the neuro-ophthalmic signs.

The MRI study was repeated 3 months after the first one and revealed further improvement of the retrobulbar changes. A faint STIR-hyperintense and mildly contrast-enhancing area was still visible, mainly affecting the same left extraocular muscles and temporalis muscle.

For this reason, antibiotic treatment was continued, and another follow-up MRI scan was planned.

The dog's clinical conditions continued to improve and 7 months after the initial presentation, the neurologic and ocular examination were unremarkable. The OS abnormalities were completely resolved but the clinical signs compatible with a form of trigemino-oculomotor synkinesis persisted.

Full hematologic tests and the MRI scan of the head were repeated. The hematology was normal and the previously described MRI changes were almost completely resolved. An electromyographic study (Micromed, Mogliano Veneto, Treviso, Italy) of the head was conducted under general anesthesia to investigate the presence of spontaneous pathologic muscle activity. Muscle activity of masticatory

(temporal, masseter, and digastricus) and extraocular muscles of the left and right side were recorded; no spontaneous, pathologic muscle activity was identified.

The antibiotic treatment was discontinued, the dog's conditions remained stable, and no abnormalities were detected on hematologic examination at 1 month follow-up.

The trigemino-oculomotor synkinesis persisted and was exclusively evident during the mastication of the leash; no treatment was advised because it was not associated with pain, discomfort during eating or dysphagia, and according to the owner, did not seem to affect the dog's quality of life.

3 DISCUSSION

Synkinesis is a syndrome characterized by aberrant innervation, resulting in involuntary stimulation of a muscle that is not normally innervated by that specific nerve.[4]

Two cases of cranial dysinnervation disorders have been previously described in veterinary literature.[1,2] A 10-year-old neutered Rhodesian Ridgeback was presented for abnormal bilateral protrusion of the third eyelid and globe retraction during mastication; the neuro-ophthalmologic examination, MRI of the head and CSF analysis did not reveal significant abnormalities.[1] A presumptive diagnosis of trigemino-abducens synkinesis was suspected and the exact etiology was not determined.[1] In the second case, a 9-month-old French Bulldog showed mild ptosis of the left upper eyelid, mild lateral strabismus OS, and left external ophthalmoparesis during neurologic and ophthalmologic evaluations. In addition, the dog exhibited abnormal movements on attempting voluntary adduction of the left eye.[2] Diagnostics were unremarkable, and a diagnosis of congenital cranial dysinnervation resembling DS in humans was proposed.[2]

In this case, the dog exhibited abnormal and involuntary eye movements during leash mastication, characterized by bilateral adduction of the eyes, movement of the upper eyelids, and third eyelid protrusion. These clinical signs were compatible with a form of trigemino-oculomotor synkinesis; they involved both eyes but were more evident OD than OS. There are several proposed mechanisms for the development of synkinesis.[3,5] One hypothesis suggests that synkinesis may occur as a result of nerve axon damage.[3] After a nerve injury, the damaged axons may send sprouts to muscle fibers of two different muscles, resulting in synkinetic movements.[3,5] Another theory proposes that synkinesis may be due to the unmasking of a primitive pathway; a previous study demonstrated co-firing of the extraocular and masticatory muscles in normal subjects when the motor branch of the trigeminal nerve was stimulated.[12] Additionally, it has been shown that proprioceptors of the pterygoid muscle can send anomalous impulses to the midbrain in the region of the oculomotor nucleus, resulting in a synkinesis between the pterygoid muscle and muscles innervated by the oculomotor nerve.[12] In our specific case, the CT and MRI studies revealed the inflammatory process was affecting the left masticatory muscle. This finding would support the hypothesis that the proprioceptors of the inflamed masticatory muscle transmitted abnormal signals in the midbrain region involving the oculomotor nucleus bilaterally, leading the ocular synkinesis. The incomplete resolution of the left ophthalmoparesis may be the reason for the less evident synkinetic movements OS compared to OD.

The dog displayed abnormal eye movements exclusively during leash chewing and not during eating. We speculated that the firmer texture of the leash, compared to food, and its deep chewing might contribute to initiating these abnormal movements, potentially by stimulating proprioceptors of the masticatory muscle. In this case, an electrophysiologic test might have helped investigate any aberrant connection between the trigeminal and oculomotor nerves by simultaneously recording from masseter and rectus muscles during the voluntary contraction of masseter muscle or by stimulating the mandibular branch of the trigeminal nerve.[1,18] In this case, however, a complete electrophysiologic evaluation was not conducted, but the EMG performed during the follow-up recorded no abnormal, spontaneous muscles activities. In this case, trigemino-oculomotor synkinesis is assumed to be acquired and secondary to

retrobulbar infectious-inflammatory disease. This hypothesis was supported from the initial improvement of clinical signs with systemic antibiotics but low dose steroids, followed by complete resolution with no recurrence when steroids were stopped. It is noteworthy that the bacteriologic analysis of the CSF may have yielded negative results due to concurrent antibiotic treatment or inadequate sampling; however, the only mild abnormalities in the CSF analysis (i.e., nucleated cells count 1/μL) may suggest that it was not involved in the pathologic process.

Gram-negative bacilli, *Corynebacterium* spp., *α-hemolytic Streptococci* spp., *Escherichia* spp., *Pasteurella* spp., *Enterobacter* spp., *Micrococcus*, and *Bacillus* spp. have been reported as infectious agents associated with orbital inflammation.[19] In this case, therapy with amoxicillin and clavulanic acid and prednisolone was prescribed to treat the suspected infectious-inflammatory process.[19] To the authors' best knowledge, there are no established guidelines for the duration of antibiotic treatment in case of orbital inflammation. Therefore, the treatment duration was based on clinical progress and repeat MRI findings. After 7 months of treatment, the dog showed complete resolution of previously reported neuro-ophthalmic abnormalities (aside from trigemino-oculomotor synkinesis) and the MRI pathologic findings were almost completely resolved. Therefore, antibiotic treatment was terminated, and no clinical sings recurred.

In human medicine, synkinesis is a condition that significantly impacts a patient's quality of life due to functional disabilities and social impairment.[3, 6] There is currently no standard treatment for ocular synkinesis, and in some cases, spontaneous recovery has been reported.[1] Various treatment strategies have been proposed, including rehabilitation therapy such as soft tissue massage, meditation or relaxation, and neuromuscular retraining.[3, 5, 6] In addition, chemodenervation with botulinum toxin A injections has been shown to improve facial symmetry.[3, 5, 6] In severe cases of synkinesis, surgical intervention may be required, including selective neurectomy, nerve grafting, selective myectomy, and muscle transfer.[5, 6]

In this case, the presumed form of trigemino-oculomotor synkinesis did not cause discomfort and did not impact the dog's quality of life. Therefore, treatment was not indicated. Similarly, in the other case of trigemino-abducens synkinesis reported in veterinary medicine, no treatment was attempted.[1]

In conclusion, we have presented a case of presumed, bilateral, and acquired trigemino-oculomotor synkinesis in a dog with a recent history of retrobulbar inflammation. Although the exact mechanism of trigemino-oculomotor synkinesis was not clarified, this condition did not impact the dog's quality of life and did not progress with time. It is important for veterinary neurologists and ophthalmologists to consider ocular synkinesis as a potential complication of retrobulbar inflammation when encountering dogs with similar clinical signs.

AUTHOR CONTRIBUTIONS

Silvia Pompilio: Conceptualization; data curation; investigation; writing – original draft; writing – review and editing. **Michela Scuttari:** Conceptualization; data curation; writing – original draft. **Katia Zerbetto:** Conceptualization; data curation; investigation; writing – original draft. **Maria Elena Andreis:** Conceptualization; data curation; investigation; writing – original draft. **Federica Tirrito:** Conceptualization; data curation; investigation; methodology; project administration; supervision; writing – original draft; writing – review and editing.

ACKNOWLEDGMENTS

The authors thank Dr. Valentina Barbieri for providing videos.

CONFLICT OF INTEREST STATEMENT

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

ETHICS STATEMENT

This article describes the findings of a clinical and diagnostic investigation of a clinical case with no element of experimental treatment with fully informed written consent from the owner. Therefore, separate ethical approval was not required.

REFERENCES

1Eminaga S, Williams D, Cherubini GB. Presumed canine trigemino-abducens synkinesis in a dog. *Vet Ophthalmol*. 2015; **18**(4): 341-344. doi:**10.1111/vop.12205**

2Mari L, Blacklock BT, Stavinohova R, De Risio L. Extraocular muscle dysinnervation disorder resembling Duane retraction syndrome in a 9-month-old French bulldog. *Vet Ophthalmol*. 2017; **20**(5): 472-476. doi:**10.1111/vop.12396**

3Guntinas-Lichius O, Prengel J, Cohen O, et al. Pathogenesis, diagnosis and therapy of facial synkinesis: a systematic review and clinical practice recommendations by the international head and neck scientific group. *Front Neurol*. 2022; **13**:1019554. doi:**10.3389/fneur.2022.1019554**

4Bursztyn LLCD, Makar I. Congenital Trochlear–Oculomotor Synkinesis. *J Neuroophthalmol*. 2014; **34**(1): 64-66. doi:**10.1097/WNO.0000000000000091**

5Krane NA, Loyo M. How best to manage facial Synkinesis? *Curr Otorhinolaryngol Rep*. 2018; **6**(2): 171-181. doi:**10.1007/s40136-018-0200-x**

6Husseman J, Mehta R. Management of Synkinesis. *Facial Plast Surg*. 2008; **24**(2): 242-249. doi:**10.1055/s-2008-1075840**

7Lemke C, Bably IE. Synkinesis between facial nerve and oculomotor nerve. A case report. *Ann Anat*. 1998; **180**(4): 339-342. doi:**10.1016/S0940-9602(98)80039-3**

8Kassem IS, Kodsi SR. Marcus Gunn jaw winking with trigemino-oculomotor synkinesis of the inferior division of the oculomotor nerve. *J Am Assoc Pediatr Ophthalmol Strabismus*. 2009; **13**(3): 315-316. doi:**10.1016/j.jaapos.2009.02.004**

9Gutowski NJ, Chilton JK. The congenital cranial dysinnervation disorders. *Arch Dis Child*. 2015; **100**(7): 678-681. doi:**10.1136/archdischild-2014-307035**

10Singh A, Pandey PK, Agrawal A, Mittal SK, Rana KM, Bahuguna C. Congenital cranial dysinnervation disorders. *Int Ophthalmol*. 2017; **37**(6): 1369-1381. doi:**10.1007/s10792-016-0388-z**

11Traboulsi EI. Congenital cranial dysinnervation disorders and more. *J AAPOS*. 2007; **11**(3): 215-217. doi:**10.1016/j.jaapos.2007.04.007**

12Pearce FC, McNab AA, Hardy TG. Marcus Gunn jaw-winking syndrome: a comprehensive review and report of four novel cases. *Ophthalmic Plast Reconstr Surg*. 2017; **33**(5): 325-328. doi:**10.1097/IOP.0000000000000780**

13De Lahunta A, Glass E. *De Lahunta's Veterinary Neuroanatomy and Clinical Neurology*. 5th ed. Elsevier; 2021.

14Joshi M, Tailor M, Ghugare B. Trigemino-oculomotor synkinesis: report of a rare case. *J Indian Acad Oral Med Radiol*. 2014; **26**(1): 92. doi:**10.4103/0972-1363.141870**

15Senthilkumar VA, Tripathy K. Marcus Gunn jaw winking syndrome. *StatPearls*. StatPearls Publishing; 2022.

16Nelson SK, Kline LB. Acquired trigemino-abducens synkinesis. *J Clin Neuroophthalmol*. 1990; **10**(2): 111-114.

17Park KA, Oh SY. Trigemino-abducens synkinesis after lateral orbitotomy. *Ophthalmic Plast Reconstr Surg*. 2013; **29**(2): e55-e56. doi:**10.1097/IOP.0b013e31826a5471**

18Cuddon PA. *Electrodiagnosis in Veterinary Neurology. Electromyography, Nerve Conduction Studies and Evoked Responses*. Veterinary Specialists of Northern Colorado; 2000.

19Herrmann JW, Hamor RE, Plummer CE. Canine Retrobulbar cellulitis and Abscessation in the southeastern United States: a review of case management, diagnostic imaging, bacterial isolates, and susceptibility patterns. *Vet Ophthalmol*. 2021; **24**(4): 326-335. doi:**10.1111/vop.12882**

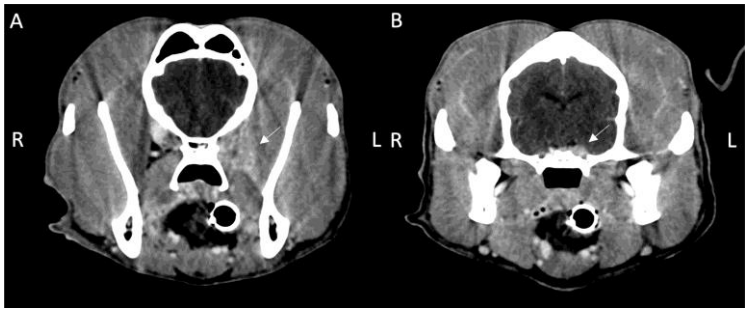

Figure 1

Post-contrast computed tomography images of the Border Collie's head demonstrating left-sided retrobulbar mass effect, with retrobulbar soft tissue swelling and enhancement effacing the retrobulbar fat, adjacent left extraocular muscles, left ophthalmic plexus, left optic nerve, and left maxillary artery (arrow) (A), with partial intracranial extension of the pathologic process (arrow) (B).

Figure 2

Picture of the head of the Border Collie demonstrating exophthalmos, mydriasis, and ventrolateral strabismus of the left eye.

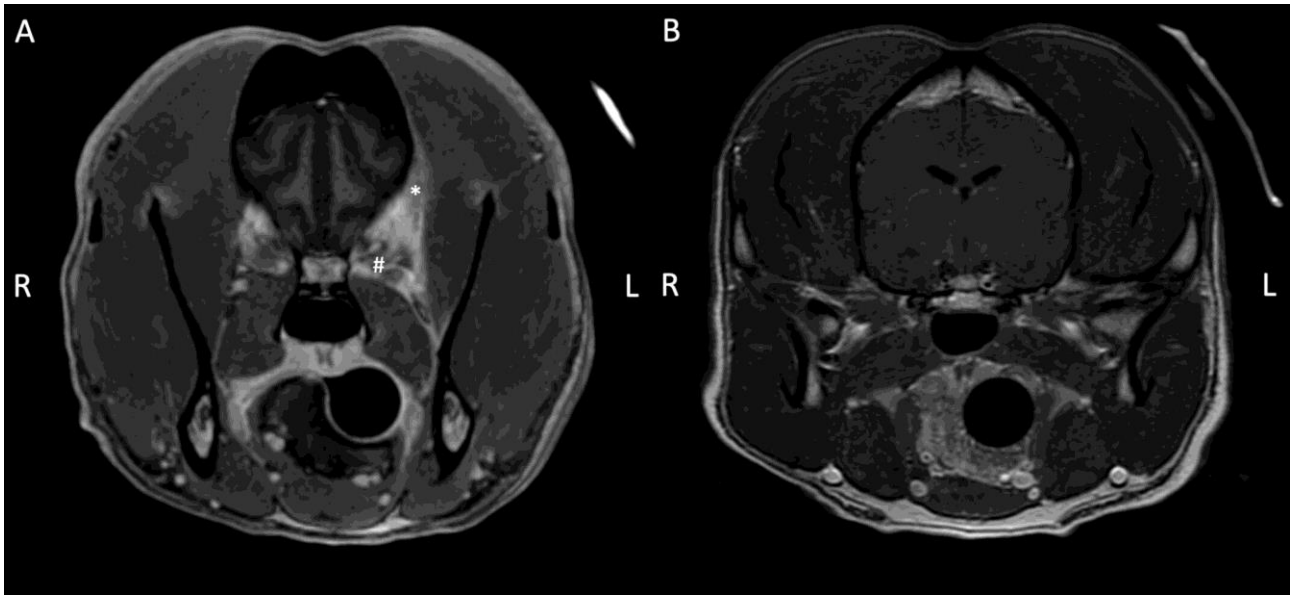

Figure 3

Post-contrast transverse T1-weighted magnetic resonance images of the Border Collie's head showing heterogeneous contrast enhancement of the left extraocular muscles (#) and left temporalis muscle (*) (A). No intracranial expansile lesion is visible (B).