Diagnostic Values of Clinical and Magnetic Resonance Findings in Presumptive Trigeminal Neuropathy: 49 Dogs

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ABSTRACT

The goal of this retrospective, cross-sectional study was to describe the different etiologies of trigeminal neuropathy based on clinical and MRI findings and to evaluate the significance of associated concomitant disorders. MRI studies of 49 dogs with trigeminal neuropathy were blindly reviewed and were classified into the following three groups: neoplasia, neuritis, or idiopathic trigeminal neuropathy (ITN). Thirty-one percent were suspected to have neoplasia (all unilateral), 16% to have neuritis (1 bilateral and 7 unilateral), and 53% to have ITN (4 unilateral and 22 bilateral). Dogs with clinical bilateral trigeminal dysfunction were most likely to have a diagnosis of ITN (predicted probability 95.7%). Unilateral clinical signs were significantly associated with neoplasia or neuritis compared with ITN ($P < .001$ and $P = .002$, respectively). Even with marked brainstem neoplastic involvement, central neurological deficits may be absent. Sensory impairment was significantly associated with either neoplasia or neuritis compared with ITN ($P = .007$ and $P = .03$, respectively). Ipsilateral noninfectious middle ear effusion was only seen in dogs with neoplasia (33%). Horner's syndrome was present in 12% of all dogs (2 dogs in each group). Dogs with neoplasia were significantly older than dogs with neuritis ($P = .02$) and ITN ($P = .002$). JAAHA-MS-6997 (*J Am Anim Hosp Assoc* 2020; 56:106–113. DOI 10.5326/JAAHA-MS-6997)

Introduction

The trigeminal nerve is the dog's largest cranial nerve and is composed of motor and sensory fibers. Its nucleus is located in the pons, and the nerve trunk emerges from the skull through the trigeminal canal; it divides into the following three branches: the mandibular branch (motor and sensory), the maxillary branch (sensory), and the ophthalmic branch (sensory).

Clinically, disorders of the trigeminal nerves can result in unilateral or bilateral masticatory muscle atrophy if one or both mandibular branches are affected. In some cases of bilateral paralysis, dogs may present with an inability to close the mouth (dropped jaw).[1] Reduced or absent sensation may be detected in areas innervated by one or more sensory branches of the trigeminal nerve.

Nontraumatic disorders resulting in trigeminal neuropathy are multiple and include neoplasia such as peripheral nerve sheath tumor (PNST)[2–8] or lymphoma,[9] neuritis,[4] and idiopathic trigeminal neuropathy (ITN).[1,10–12] Extracranial tumors or perineural inflammatory processes such as abscess or cellulitis causing a mass effect or infiltration of the trigeminal nerve may induce similar clinical signs.

Normal trigeminal nerves and nuclei typically have mild to moderate contrast enhancement on T1-weighted MRI in 90% of dogs. The intensity of enhancement is subjectively less than or equal to the pituitary gland enhancement.[13]

Trigeminal neoplasia such as PNST often are characterized by enlargement of the nerve and marked homogenous contrast enhancement, usually with a compressive mass effect on the brainstem.[2–5,7,14–16] Trigeminal neoplasms can also be associated with enlargement of the trigeminal canal, the orbital fissure, and the round and oval foramina. MRI findings consistent with neuritis

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CSF (cerebrospinal fluid); ITN (idiopathic trigeminal neuropathy); MEE (middle ear effusion); MUO (meningoencephalitis of unknown origin); PNST (peripheral nerve sheath tumor); TVP (tensor veli palatini)

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include nerve enlargement and/or heterogeneous or homogeneous contrast enhancement but without mass effect on the brainstem.[4] ITN is not associated with detectable MRI abnormalities of the trigeminal nerve. Except for bilateral acute neuropathies, marked atrophy of the masticatory muscles with hyperintense signal on T1- and T2-weighted images and mild to moderate contrast enhancement is present on MRI, regardless of the etiology.

Few concomitant disorders have been described in association with trigeminal neuropathy. The presence of an ipsilateral middle ear effusion (MEE) has been previously reported in cases of trigeminal neoplasia; however, the presence of this finding has not been reported for other trigeminal neuropathies.[5,15,17] Horner's syndrome also has been reported occasionally with various trigeminal diseases such as neoplasia,[18] ITN,[10] and polyradiculoneuritis.[19]

The neurological examination should readily localize the lesion to the trigeminal nerve(s). MRI of the pontine brain and skull region is highly sensitive in the detection of lesions involving the trigeminal nerve of dogs; however, to the authors' knowledge, there have been no studies assessing the characterization of these magnetic resonance lesions and their diagnostic value in a group of dogs with trigeminal neuropathies. Furthermore, epidemiological factors and concomitant disorders have not been evaluated in cases of unilateral or bilateral trigeminal neuropathy. The goals of this study were to describe the prevalence of the three main etiologies of trigeminal neuropathy based on clinical and MRI findings, to evaluate concomitant disorders associated with trigeminal neuropathies, and to determine if any clinical and imaging findings are predictive for specific causes of trigeminal neuropathy.

Materials and Methods

In this cross-sectional study, medical records of dogs presented to the CHV Frégis between January 2010 and March 2018 with signs of trigeminal dysfunction were retrospectively reviewed. Dogs were included if they had a neurological examination consistent with trigeminal neuropathy and had an MRI examination performed. Neurological deficits associated with trigeminal involvement included unilateral (**Figure 1**) or bilateral masticatory muscle atrophy, dropped jaw, absent or diminished palpebral and/or corneal reflex(es), absent or diminished response to nasal septum stimulation, and decreased facial sensation. As steroids can cause secondary temporal muscle atrophy and reduce contrast enhancement on MRI, dogs receiving prednisolone for >5 days prior to presentation were excluded from the study.

Information extracted from the medical records included signalment, history, time of onset, neurological examination findings, MRI findings, and cerebrospinal fluid (CSF) and blood test results (including T4, thyroid-stimulating hormone, and cholesterol values) when available and follow-up.

All MRI studies were retrospectively and blindly reviewed by three observers, a board-certified neurologist (L.C.), a board-certified radiologist (E.G.), and a senior Neurology resident (C.M.). MRI evaluations were performed using a 0.20 MRI unit[a] before October 2015, and with a 0.25 MRI unit[b] after October 2015. All dogs were imaged under general anesthesia in a prone position. All MRI examinations included T1-, T2- and postcontrast T1–weighted images in the transverse plane of the head. Other sequences, although not required for inclusion, such as fluid-attenuated inversion recovery–weighted images and very thin postcontrast T1-like sequences[c], were also reviewed when available.

MRI presumptive diagnosis was based on the following criteria, determined a priori by consensus among the three reviewers. For a diagnosis of ITN, the MRI findings were considered normal, meaning mild to moderate contrast enhancement of both trigeminal nerves, with less intensity compared with the pituitary gland (**Figure 2A–C**). For a diagnosis of neoplasia, the trigeminal nerves must be enlarged with marked homogeneous contrast enhancement and a mass effect on the brainstem or outside the cranial cavity along the nerve (Figures 2D–I). Enlargement of the nerve and/or abnormal contrast enhancement without mass effect on the brainstem was considered consistent with neuritis (Figures 2J–L). In the case of doubt between neoplasia and neuritis, the dog was classified into the neoplasia group if neurological deterioration consistent with ipsilateral

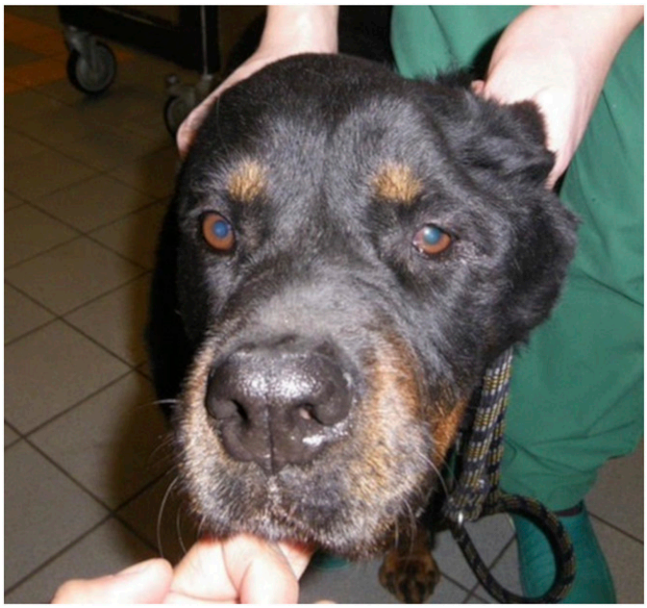

FIGURE 1 *A 7 yr old rottweiler presented with left unilateral masticatory muscle atrophy.*

brainstem involvement occurred within 2 yr after diagnosis (based on lifetime survival established in previous articles).[3,5]

All other MRI abnormalities, including MEE, were also reported. All cases with extracranial tumor or inflammatory process involving the emergences or along the trigeminal nerve were excluded.

Statistical analysis was performed in a hypothesis-generation context using commercially available software[d]. For descriptive statistical purposes, continuous data were assessed for Gaussian distribution by histogram evaluation and the Shapiro-Wilk test (Gaussian if $P > .05$). Gaussian data were presented using mean (SD) and non-Gaussian data using median (minimum–maximum). Categorical data were presented as number of dogs (percentage).

Associations between signalment, duration of clinical signs, presence of sensory impairment, presence of Horner's syndrome, lateralization of deficits, presence of other clinical signs, presence of MEE, and other MRI findings, and trigeminal disease category were assessed by univariable multinomial logistic regression. Independence of irrelevant alternatives assumption was assessed via Hausman, Suest-based Hausman, and Small-Hsiao tests, with a $P > .05$ indicating that the odds of each outcome were independent of other alternatives. In order to identify potential confounding effects among variables, all variables significantly associated with a trigeminal disease category on univariable analysis were entered into a multivariable analysis, which consisted of a multivariable multinomial logistic regression. For all statistical analyses, significance was set at $P < .05$.

Results

Forty-nine dogs met the inclusion criteria. Twenty-six (53%) with ITN, 15 (31%) were presumptively diagnosed with neoplasia, and 8 (16%) with neuritis.

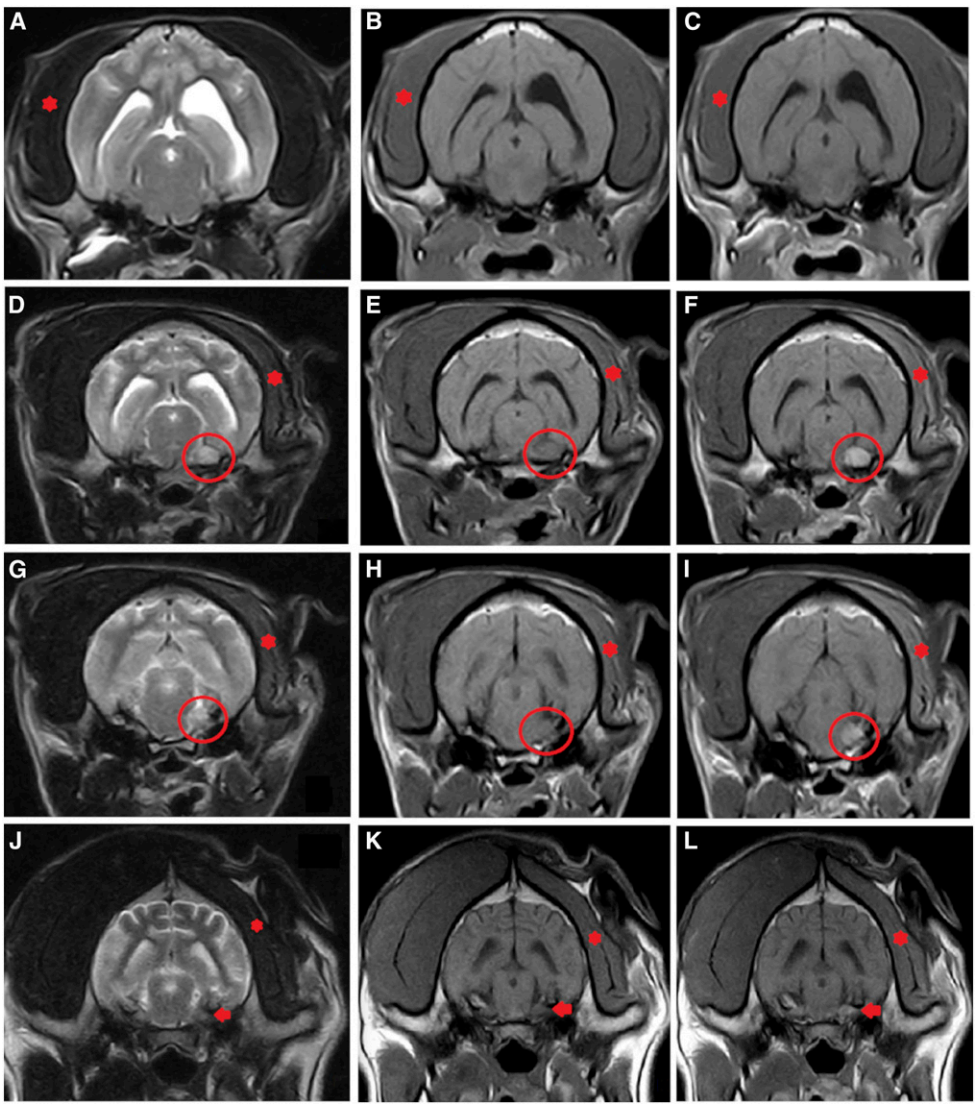

FIGURE 2 *Example of MRI. T2W (*A, D, G, and J*), T1W (*B, E, H, and K*), and T1W after gadolinium injection (*C, F, I, and L*). (*A–C*) A dog with suspected right idiopathic trigeminal neuropathy—presence of a right moderate masticatory muscle atrophy (*star*) without any lesion in the trigeminal nerve. (*D–I*) A dog with suspected left trigeminal neoplasia. The emergence of the trigeminal nerve shows an enlargement with intense contrast enhancement and moderate compressive mass effect on the brainstem (*circle*). Notice the presence of a severe atrophy of all masticatory muscles ipsilaterally to the lesion (*star*). (*J–L*) A dog with suspected left trigeminal neuritis. The emergence of the trigeminal nerve shows an enlargement with T2W heterogeneity and heterogenous contrast enhancement and without mass effect on the brainstem (*arrow*). Notice the presence of a severe atrophy of all masticatory muscles ipsilaterally to the lesion (*star*).*

ITN Group

Twenty-six dogs (53%) were presumptively diagnosed with ITN. The mean age at presentation was 6.1 yr (SD: 3.1). Eight dogs (31%) were females (6 neutered) and 18 (69%) were males (3 neutered). There were 3 golden retrievers, 3 American Staffordshire terriers, 3 mixed-breed dogs, 2 basset hounds, 2 cocker spaniels, and 1 each of the following breeds: beagle, Beauceron, German shepherd dog, Australian shepherd dog, Bernese mountain dog, Doberman pinscher, Brittany spaniel, French bulldog, German shorthaired pointer, Groenendael, Irish wolfhound, Jack Russell terrier, and shih tzu. The median duration of clinical signs was 8 days (1–365 days). Twenty-two dogs (85%) exhibited bilateral signs, whereas only 4 (15%) dogs had unilateral signs. The most common findings were dropped jaw in 18 dogs (70%), bilateral masticatory muscles atrophy in 9 dogs (35%; 5 with dropped jaw), unilateral masticatory muscle atrophy in 4 dogs (15%), and bilateral facial sensory deficits in 1 dog (4%; with dropped jaw).

Nontrigeminal-related neurological deficits included ipsilateral Horner's syndrome in two dogs (7.5%), head tilt and peripheral vestibular syndrome in one dog (4%), paradoxical vestibular syndrome in one dog (4%) as a result of cerebellar infarction 4 mo prior, and bilateral facial paresis in one dog (4%).

MRI nontrigeminal abnormalities included unilateral MEE in one dog with bilateral ITN (4%), bilateral otitis media in one dog (4%), and cerebellar geometrical nonenhancing lesion (suspicion of gliosis) in one dog (4%). These three dogs were diagnosed with bilateral ITN and all had a dropped jaw.

Neoplastic Group

Fifteen dogs (31%) were presumptively diagnosed with trigeminal neoplasia. The mean age at presentation was 10.5 yr (SD 2.6). Nine dogs (60%) were females (4 neutered) and 6 (40%) were males (3 neutered). There were 5 mixed-breed dogs (33%), and 1 each of the following breeds (6.7%): Australian shepherd dog, Maltese, German shepherd dog, French bulldog, golden retriever, Jagd-terrier, Labrador retriever, miniature schnauzer, rottweiler, and West Highland white terrier. The median duration of clinical signs was 63 days (4–245 days). The most common neurological abnormalities on examination included masticatory muscle atrophy (100%) affecting the left side in 9 dogs (60%) and the right side in 6 dogs (40%) as well as sensory deficits in 7 dogs (47%).

Nontrigeminal-related neurological abnormalities, all ipsilateral to the neoplasia, included Horner's syndrome in two dogs (13%), head tilt in two dogs (13%), thoracic and pelvic limb proprioceptive deficits in two dogs (13%), external ophthalmoplegia in two dogs (13%), and nonresponsive mydriasis in one dog (7%).

On MRI, all dogs had unilateral lesions of the trigeminal nerve ipsilateral to the amyotrophic side. Other MRI abnormalities, all ipsilateral to the neoplasia, included MEE in five dogs (33%), retrobulbar cellulitis in one dog (7%), and a suprasellar enhancing mass not continuous with trigeminal nerve neoplasia in one dog (7%).

The median survival time was 6 mo (2–24 mo). Three dogs (20%) lived for >1 yr after diagnosis. Most of the dogs (11/15, 73%) were euthanized because of neurological decompensation and progression of the neurological deficits, mainly ipsilateral to the tumor, and tetraparesis.

Neuritis Group

Eight dogs (16%) were presumptively diagnosed with neuritis. The mean age at presentation was 6.8 yr (SD: 1.6 yr). Two dogs (25%) were females (2 neutered) and six (75%) were males (1 neutered). There were two Jack Russell terriers (25%), and one each of the following breeds (12.5%): American Staffordshire terrier, mixed-breed dog, Labrador retriever, Afghan hound, rottweiler, and Newfoundland retriever. The median duration of clinical signs was 31 days (5–119 days). The most common neurological findings abnormalities were unilateral masticatory muscles atrophy in seven dogs (87.5%), ipsilateral trigeminal sensory deficits in three dogs (37.5%), ipsilateral facial hyperalgesia in one dog (12.5%), and dropped jaw in one dog (12.5%).

Nontrigeminal-related neurological abnormalities included proprioceptive deficits in three dogs (37.5%) with meningoencephalitis of unknown origin (MUO) affecting the brainstem, ipsilateral Horner's syndrome in two dogs (25%), lack of menace response in two dogs (25%, 1 ipsilateral and 1 contralateral), ipsilateral nonresponsive mydriasis in one dog (12.5%), neck pain, and bilateral pelvic limbs proprioceptive ataxia in one dog (12.5%).

Of the eight dogs with neuritis, seven dogs (87.5%) had unilateral trigeminal lesion on MRI, whereas only one dog (12.5%) had bilateral involvement.

CSF tap was performed on four dogs. Results were normal in two dogs, but in two dogs with multifocal MUO on MRI, results of CSF analysis revealed mild pleocytosis and increased total protein. Polymerase chain reaction in CSF was negative for distemper, *Neospora caninum*, and *Toxoplasma gondii*.

Other concomitant MRI abnormalities included MUO in three dogs (37.5%, including the dog with bilateral trigeminal disorder) and contralateral MEE in one dog (12.5%).

The median survival time was 12.5 mo (0.2–60 mo). Five dogs (62.5%) lived for <2 yr. The dogs with MUO involving the brainstem were treated with prednisolone and cytarabine. These three dogs were euthanized because of progressive neurological deterioration during treatment. All the remaining dogs died of unrelated causes without neurological signs.

Thyroid function tests (T4/thyroid-stimulating hormone, Cholesterol) were performed in 19 out of 26 dogs (73%), and the results were normal in all dogs.

Median survival time could not be calculated because most of the dogs (15/26, 58%) were still alive at the time of writing, and 4 out of 26 were lost to follow-up. Of the 7 dogs who died, death was unrelated to the trigeminal disease in all dogs.

Statistical Analysis

On univariable analysis, age at diagnosis, presence of sensory impairment on neurological examination, clinical sign lateralization, and presence of MEE on MRI were positively associated with trigeminal disease category.

Dogs with neoplasia were significantly older than dogs with neuritis ($P = .02$) and ITN ($P = .002$). Predicted probabilities for each disease category depending on age at diagnosis are presented in **Figure 3**.

Presence of sensory impairment was significantly associated with neoplasia ($P = .007$) and neuritis ($P = .03$), as opposed to ITN. Predicted probabilities for each disease category depending on presence or absence of sensory impairment are presented in **Table 1**.

Unilateral clinical signs were significantly associated with neoplasia ($P < .001$) and neuritis ($P = .002$), as opposed to ITN. Predicted probabilities for each disease category depending on presence or absence of lateralized clinical signs are presented in **Table 2**.

Presence of MEE on MRI was significantly associated with neoplasia ($P = .003$), as opposed to ITN. The predicted probability of neoplasia was 100% (95% confidence interval: 99–100%) if ipsilateral MEE was detected on MRI, as opposed to 0% and 0% for ITN and neuritis, respectively.

Finally, age at diagnosis ($P = .02$), lateralized clinical signs ($P < .001$) and presence of MEE on MRI ($P = .04$) remained statistically significant on multivariable analysis. Presence of sensory impairment was no longer significant ($P = .62$) and was likely confounded by clinical sign lateralization.

Discussion

The goal of this study was to describe the prevalence of the three main etiologies of trigeminal neuropathy based on clinical and MRI findings, as well as the concomitant disorders associated with them, and the accuracy of clinical signs and MRI findings to predict these diagnoses.

Bilateral signs of trigeminal neuropathy, including dropped jaw and bilateral masticatory muscle atrophy, were found in 23 dogs and were very suggestive of ITN (22/23 dogs, 95.7%). One case of neuritis had bilateral trigeminal nerve involvement but also had a diagnosis of MUO mainly affecting the brainstem. In our study, no dogs with bilateral trigeminal neuropathy were classified as having neoplasia. Five dogs have been previously reported with nonidiopathic bilateral trigeminal disorders.[9,10,18] Four of them had other cranial nerve involvement. A previous study revealed that in 26 dogs with suspected bilateral ITN, 1 dog had also facial paralysis, 1 had peripheral vestibular deficits, and 2 had Horner's syndrome; no MRI was performed to investigate possible lesions associated with ITN.[10] In our study, 1 dog had bilateral facial paralysis, 2 dogs had vestibular dysfunction (1 paradoxical vestibular syndrome and 1 peripheral idiopathic vestibular syndrome), and 2 had Horner's syndromes. Our results reinforce the conclusions of Mayhew's and Robins' studies that dogs with clinical bilateral trigeminal dysfunction without other cranial nerve involvement—except for facial, vestibulocochlear, or Horner's syndrome—or without central nervous deficit are most likely affected by ITN (predicted probability 95.7%).[1,10]

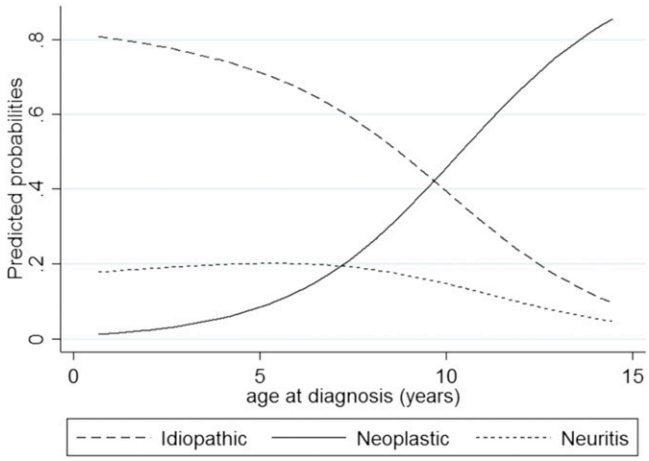

FIGURE 3 *Graph illustrating the predicted probabilities depending on age at presentation for the different groups of trigeminal neuropathies.*

TABLE 1

Predicted Probabilities of Idiopathic, Neoplastic, and Inflammatory Trigeminal Disease Based on Presence or Absence of Sensory Impairment on Neurological Examination

	Predicted Probabilities, % (95% CI)		
Sensory Impairment	Idiopathic	Neoplasia	Neuritis
No	65.8 (50.7–80.9)	21.0 (8.1–34)	13.2 (2.4–23.9)
Yes	9.1 (0a26.1)	63.6 (35.2–92)	27.3 (1–53.6)

The percentages correspond to the probability of having idiopathic, neoplastic, and inflammatory trigeminal disease when no sensory impairment is detected on physical examination on one hand, and when sensory impairment is detected on physical examination on the other hand. CI, conference interval.

TABLE 2

Predicted Probabilities of Idiopathic, Neoplastic, and Inflammatory Trigeminal Disease Based on Presence or Absence of Unilateral Clinical Signs on Neurological Examination

	Predicted Probabilities, % (95% CI)		
Unilateral Clinical Signs	Idiopathic	Neoplasia	Neuritis
No	95.7 (87.3–100)	0 (0–0)	4.3 (0–12.7)
Yes	15.4 (1.5–29.3)	57.7 (38.7–76.7)	26.9 (9.9–44)

The percentages correspond to the probability of having idiopathic, neoplastic, and inflammatory trigeminal disease when bilateral clinical signs are detected on physical examination on one hand, and when unilateral clinical signs are detected on physical examination on the other hand. CI, confidence interval.

Unilateral trigeminal disorders were found in 26 dogs. Unilateral dysfunctions were more often secondary to an infiltrative process (22/26, 84.6%), either neoplastic (15/26, 57.7%) or inflammatory (7/26, 26,9%). Idiopathic neuropathy was rarely unilateral (4/26, 15.4%). PNST is usually a focal neoplasm affecting only one nerve or few adjacent nerves.[2–4,7,8,15] Two cases have been previously reported with nerve sheath fibrosarcoma affecting the trigeminal nerve and other cranial nerves.[20] In our study, all 15 dogs with suspected neoplasia were affected unilaterally, and none of these dogs had signs of extension of neoplastic tissue to adjacent cranial nerves.

Although most neoplastic lesions had some degree of brainstem compression, only three dogs (20%) had neurological signs relative to the mass effect at time of diagnosis. Two dogs had ipsilateral central vestibular deficits and one had ipsilateral proprioceptive deficits. Our findings suggest that a compressive brainstem lesion associated with trigeminal neoplasia is often present in dogs despite the absence of proprioceptive or other long-tract deficits.

In the neuritis group, seven out of eight dogs (88%) had unilateral signs of trigeminal involvement. There are a limited number of previous reports in the literature that have described only four cases of trigeminal neuritis in dogs, with two dogs having unilateral involvement[4] and two dogs having bilateral[10] involvements. One case of bilateral inflammation also had a nonsuppurative encephalitis secondary to *N caninum* infection. The previously reported two cases of bilateral neuritis also had MRI features of MUO in which multiple other cranial nerves (III, V, VII, XII, and vagosympathetic trunk in one dog and III, V, IX, X, and XI in the other) were involved at postmortem examination. In our study, CSF analysis was performed in four cases of suspected neuritis; the results of two CSF specimens were normal and two had mild monocytic pleocytosis but were negative for distemper, *N caninum*, and *T gondii.* Additionally, in our study population, only sympathetic innervation of the eye was affected in two dogs with no associated MUO. Our results confirm the previous published statement that trigeminal neuritis may be unilateral or bilateral and should, therefore, not be excluded even if CSF analysis is normal.

Regarding age, 10 out of 15 dogs of the neoplastic group were >10 yr (67%), compared with only 4 out of 26 in the idiopathic group (15%) and 2 out of 8 in the neuritis group (25%). According to our results, older dogs, especially those >10 yr old, are significantly predisposed to neoplasia as opposed to ITN and neuritis**.** This is in accordance with previous reports.[3–5]

Several reports have described sensory impairments with trigeminal neuropathies in the dog.[7,9,10,14,19] A previous study on bilateral trigeminal neuropathies identified sensory deficits in 35% of dogs with ITN.[10]. In our population, only 11 dogs (22.4%) presented sensory trigeminal impairment, including 7 dogs with neoplasia (63.6%), 3 dogs with neuritis (27.3%), and 1 dog with bilateral ITN (9.1%). According to our results, sensory impairment was found less frequently than previously reported and was significantly associated with neoplasia or neuritis rather than ITN. In ITN, the motor branch (mandibular nerve) of trigeminal nerve seems to be more clinically affected than other branches of the nerve. The sensory branches may be affected as well but to a degree that cannot be identified clinically during routine neurological examination. Alternatively, it is possible that ITN preferentially involves the motor branches of the nerve, whereas neoplasms and inflammation of the nerve equally affect both motor and sensory branches resulting in the more commonly observed sensory deficits as well as motor deficits in these conditions. On the other hand, if the motor branches of the nerve are primarily involved in cases of tumors and inflammation, then sensory deficits may be due to local compression or contact with the sensory components of the nerve. In either case, if the sensory deficits are mild, then they could easily be undetected during clinical examination.

Ipsilateral MEE associated with trigeminal disease has been previously reported [14,15,21] in 33–63% of dogs with neoplasia.[14,15] In two of these reports, MEE was mainly associated with neoplasia involving the brainstem, and only one dog had MEE associated with a lesion in the trigeminal canal.[14,15] This phenomenon is likely due to dysfunction of the auditory tube (Eustachian tube) secondary to denervation of the tensor veli palatini (TVP) muscle, which is innervated by a branch of the mandibular branch of the trigeminal nerve.[17] In our study, the overall prevalence of ipsilateral MEE was 10%; however, when only the neoplastic group is considered, the prevalence of MEE increased to 33%. All dogs (n = 5) with MEE ipsilateral to the trigeminal disorder had a neoplasia. Theoretically, any severe disease affecting the motor function of the mandibular branch of the trigeminal nerve might cause paralysis of the TVP

muscle and possibly secondary MEE. It is possible that only severe neoplasia is able to cause notable denervation of the TVP muscle and completely abolish its ability to open the nasopharyngeal orifice.

Lesions involving the bullae and consistent with MEE appeared hyperintense on T1W and T2W series with no or mild peripheral enhancement. None of these dogs showed signs of external canal inflammation on MRI studies.

As previously reported, ipsilateral MEE can be associated with trigeminal neoplasia. Our study suggests that ipsilateral MEE is restricted to trigeminal neoplasia, as it was not encountered in other trigeminal disorders. Presence of ipsilateral MEE should, therefore, strongly suggest trigeminal neoplasia in ambiguous cases.

Horner's syndrome was identified only occasionally in the dogs report here (6/49, 12.2%, 2 dogs in each group). Middle ear pathology was not identified on MRI in any of the dogs with Horner's syndrome; therefore, otitis was excluded as a cause of Horner's syndrome. The sympathetic axons join the ophthalmic nerve as it branches from the trigeminal ganglion and continue to the orbital fissure to join the periorbital area.[22,23] Any inflammatory or infiltrative lesion affecting the trigeminal nerve at that level could cause a Horner's syndrome, especially if the lesion involves the ophthalmic branch. Cases of Horner's syndrome concomitant with trigeminal neuropathy have been reported in the veterinary literature, and the etiologies included ITN,[10] bilateral myelomonocytic neoplasia,[18] and polyradiculoneuritis.[19] Our study confirms that Horner's syndrome can be associated with trigeminal disorders but cannot be used to narrow the differential diagnosis.

Hypothyroidism has often been associated with idiopathic peripheral cranial nerve disorders, including the vestibulocochlear, facial, and vagal nerves.[24–27] This association may relate to the presence of xanthomata or myxedematous compression of the nerves.[27,28] None of the dogs tested (19/26, 73%) with ITN had abnormal thyroid test results. These results failed to demonstrate any association between hypothyroidism and ITN. Therefore, our results did not confirm the association between hypothyroidism and ITN.

Categorization of the disease process and diagnosis based upon MRI findings, and further supported by clinical follow-up, appeared to be accurate in 95.9% (47/49) of cases; however, confirmatory histopathologic examination of tissues was not performed in any patient. Diagnosis based on clinical and MRI was uncertain in only two cases. Of these cases, one was finally classified as neoplasia and the other as neuritis based on clinical follow-up. Based on MRI characteristics previously described in the literature, MRI diagnosis of trigeminal neuropathies on a low-field MRI is feasible and accurate.[2–5,7,13–16]

Our study had several limitations, mainly owing to its retrospective aspect. A histopathological diagnosis was not established in any animal because no dog was euthanized at time of diagnosis and no owners agreed for necropsy at time of death or euthanasia. Histopathologic analysis is the diagnostic gold standard for trigeminal neuropathies, especially for dogs with contrast enhancement of the trigeminal nerve but lacking a mass effect. This may be especially true to differentiate early neoplasia from neuritis. However, in our study, initial diagnosis was confirmed by information obtained during follow-up telephone calls with owners and included survival time and information about the neurologic evolution of the condition over time.[3,5] The use of a low-field MRI was also a limitation. Therefore, very small or subtle lesions, and especially mild neuritis versus ITN, may have been misdiagnosed.

Conclusion

Clinical bilateral trigeminal dysfunction without other cranial nerve deficits, except for facial or vestibulocochlear nerve deficits and Horner's syndrome, and without other central nervous system deficits, is very suggestive of ITN (95.7%). Unilateral lesions are more likely to be caused by an inflammatory or neoplastic process. Even with marked brainstem neoplastic involvement, central neurological deficits may be absent. A tumor should be the main differential diagnostic consideration in unilaterally affected dogs, especially if >10 yr old. Sensory impairment may indicate trigeminal neuritis or neoplasia. Ipsilateral MEE strongly suggests neoplasia. Horner's syndrome does not seem useful in narrowing the differential diagnosis.

FOOTNOTES

[a] Vet-MR; Esaote, Köln, Germany
[b] Vet-MR Grande; Esaote, Köln, Germany
[c] 3D SST1; Esaote, Köln, Germany
[d] STATA, version 14.0; StataCorp LP, College Station, Texas

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