Received: 18 September 2020 | Revised: 31 January 2021 | Accepted: 9 February 2021

DOI: 10.1111/vru.12974

ORIGINAL INVESTIGATION

WILEY

Magnetic resonance imaging of the caudal portion of the digastric muscle in canine idiopathic facial neuropathy

Ombeline McGregor | **Mark J. Plested** | **Elsa Beltran**

Royal Veterinary College, Hatfield, UK

Correspondence
Elsa Beltran, Department of Veterinary Clinical Science and Services, Royal Veterinary College, University of London, Hawkshead Lane, North Mymms, Hatfield, Hertfordshire AL9 7TA, England.
Email: ebeltran@rvc.ac.uk

Abstract

Idiopathic is the most common etiology for acute onset of facial neuropathy in dogs with limited number of studies describing MRI characteristics. A retrospective, observational study was performed using archived records, aiming to describe the MRI features of the caudal portion of the digastric muscle in dogs diagnosed with idiopathic facial neuropathy and to determine correlation with prognosis. Client-owned dogs presented to a referral hospital between 2009 and 2019, diagnosed with unilateral idiopathic facial neuropathy and having undergone MRI, with images including the caudal portion of the digastric muscle, were included (n = 19). MRI appearance of the affected muscle, including degree of muscle atrophy, signal intensity, enhancement post-contrast, and enhancement characteristics of the affected facial nerve, was described and compared to the contralateral, clinically unaffected caudal portion of the digastric muscle. Correlation between MRI appearance and outcome at 1-month and 3-months following onset of clinical signs was investigated. The majority of patients demonstrated some degree of muscle atrophy (n = 17, 89%), hyperintensity in T2W (n = 17, 89%), and pre-contrast T1W (n = 15, 79%) images, as well as contrast enhancement of the affected muscle (n = 14, 74%) and affected facial nerve (n = 9, 47%). There was no statistically significant correlation between atrophy or enhancement of the affected caudal portion of the digastric muscle nor between enhancement of the affected facial nerve and outcome. Hyperintensity both in T2W images and pre-contrast T1W images was significantly correlated with a worse prognosis. Ensuring inclusion and evaluation of this muscle in MRI may therefore be indicated in canine idiopathic facial neuropathy.

KEYWORDS
contrast enhancement, cranial nerve, denervation, muscle atrophy, signal intensity

1 | INTRODUCTION

The facial nerve is the seventh cranial nerve (CN VII) and provides motor function to the muscles of facial expression, the caudal portion of the digastric muscle and sensory function to the rostral two-thirds of the tongue and palate (providing the sense of taste) and inner part of the pinna. The parasympathetic component innervates the lacrimal glands, the glands of the nasal mucosa, palatine mucosa, and the mandibular and sublingual salivary glands.[1,2]

Previous presentation or publication disclosure: No previous presentation or publication.

EQUATOR network disclosure: An EQUATOR network checklist was not used.

Facial motor neuropathy can result from injuries affecting the nucleus of the facial nerve (located in the rostral part of the medulla oblongata) or its axons at any point in its path. Clinical signs of complete acute paralysis of the muscles of facial expression include ipsilateral drooping of and inability to move the ear and lip with increased exposure of the lip mucosa, saliva falling through the affected side, displacement of the nasal philtrum and upper lip to the unaffected side and inability to blink on the affected side. Dysfunction of the parasympathetic supply of the lacrimal gland and glands of the nasal cavity results in neurogenic keratoconjunctivitis sicca and xeromycteria, respectively. Parasympathetic dysfunction of the facial nerve is mainly seen with lesions at the level of the rostral medulla oblongata, petrosal bone, or pterygopalatine fossa.[1,2]

The most common etiology of facial neuropathy in dogs is idiopathic, accounting for up to 75% of all cases presented with facial neuropathy.[3–6] Diagnosis of idiopathic facial neuropathy is based on neurological signs and exclusion of all other causes. The minimum database includes hematology, biochemistry, and thyroid hormone level. Otoscopy should be performed on all patients as well as a Schirmer tear test and fluorescein staining.[7] Advanced imaging such as MRI is performed to evaluate the path of the facial nerve, including brainstem, inner, and middle ear. Clinical signs associated with idiopathic facial neuropathy may result in ipsilateral paresis or plegia of the muscles of facial expression, with or without associated dysfunction of the parasympathetic innervation, depending on the region of the facial nerve affected. Associated ipsilateral clinical signs of peripheral vestibular dysfunction are common and have been reported in between 42 and 65% of patients diagnosed with idiopathic facial neuropathy.[3–5] This is likely explained by the close proximity of the facial and vestibulocochlear nerves at the level of the inner ear within the proximal part of the facial nerve canal, so that swelling of the facial nerve at that level may result in ipsilateral peripheral vestibular syndrome and vice versa.[4,6]

The majority of the dogs diagnosed with idiopathic facial neuropathy are given a guarded prognosis for functional recovery.[4,6] Previous studies have reported full recovery of facial motor nerve function in <50% of patients, with recovery often taking weeks to months.[1,4,6] The guarded prognosis does not however suggest a significant reduction in quality of life, although some animals may require lifelong eye lubrication to prevent corneal ulceration, and hemifacial contracture is also reported as a long-term complication of idiopathic facial neuropathy.[6]

MRI findings in idiopathic facial neuropathy such as enhancement of the affected facial nerve following contrast administration have been described in animals.[7–9] MRI features of the caudal portion of the digastric muscle have however not yet been described in veterinary patients. Studies in humans have demonstrated that the degree of asymmetry between facial muscles prior to facial nerve grafting can be correlated with prognosis to return normal facial nerve function.[10] The facial musculature is not easily assessed in dogs due to the small volume of these muscles resulting in difficulty describing any changes accurately. The caudal portion of the left and right digastric muscles however is clearly visible on MR images, allowing for adequate assessment of changes in muscle mass, signal intensity, and enhancement. This muscle could therefore be used as an early indicator of the severity of injury to the facial nerve. It is hypothesized that MRI features of muscle atrophy, edema, fatty degeneration, and contrast enhancement of the affected caudal portion of the digastric muscle are correlated with a worse prognosis to return to normal facial nerve function by 1 month and 3 months following the onset of clinical signs.

The digastric muscle runs from the paracondylar process of the occiput to the ventral border of the mandible, lying medial to the parotid, and mandibular salivary glands. It is separated into a rostral and caudal portion by a tendinous intersection. The rostral portion is innervated by the mandibular branch of the trigeminal nerve, while the caudal portion is innervated by the facial nerve.[2]

The aims of this study are to describe the MRI features of the caudal portion of the digastric muscle in dogs diagnosed with idiopathic facial neuropathy and to determine whether these features are correlated with prognosis to return to normal facial nerve function by 1 month and 3 months following onset of clinical signs.

2 | MATERIALS AND METHODS

The study was a retrospective, observational design. Medical records of dogs referred to the Queen Mother Hospital for Animals for unilateral facial neuropathy from January 2009 to November 2019 were reviewed. Inclusion criteria included all patients with idiopathic unilateral facial neuropathy with or without associated ipsilateral idiopathic vestibular syndrome, complete neurological examination, head MRI with inclusion of the caudal portion of the left and right digastric muscles, and minimum follow-up at 1 month and 3 months after the onset of clinical signs. Exclusion criteria included significant abnormalities in hematology or biochemistry suggestive of systemic disease, low total T4 measurement, abnormalities in cerebrospinal fluid analysis, MRI suggestive of structural disease including the presence of concurrent otitis media/interna, lack of follow-up information and absence or incomplete inclusion of the caudal portion of the digastric muscles in the MRI field of view. Decisions for subject inclusion or exclusion were made by the consensus of a registered veterinarian (O.M.), a board-certified veterinary neurologist (E.B., European College of Veterinary Neurology [ECVN]), and a senior veterinary radiology resident (M.J.P.).

Medical records for dogs meeting inclusion criteria were retrieved, and clinical data were recorded by the registered veterinarian (O.M.) and the veterinary neurologist (E.B.), both of whom were blinded to the MRI findings at the time of data recording. Information collected included the following: age at onset, sex, breed, clinical signs at presentation, duration of clinical signs prior to MRI, and outcome.

All MRI studies were retrieved and re-reviewed by the registered veterinarian (O.M.), the senior veterinary radiology resident (M.J.P.), and the veterinary neurologist (E.B.). Readers were unaware of the side of the clinical signs at the time of image interpretations. Disagreement was resolved by a consensus reading by the three reviewers. The following MR parameters were recorded: degree of atrophy of the caudal portion of the digastric muscle of the affected side compared to the contralateral side, difference in signal intensity between the affected side and the contralateral side in both T2W images and pre-contrast T1W, enhancement of the affected facial nerve compared

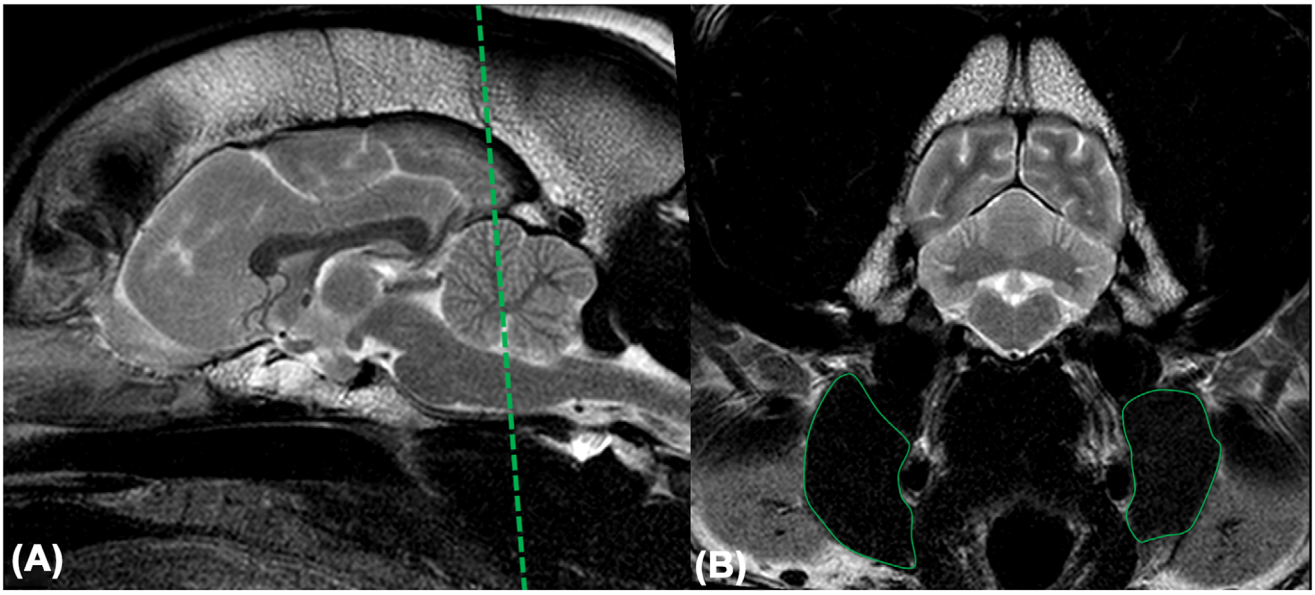

FIGURE 1 Seven-year-old, male neutered Staffordshire Bull Terrier with left sided facial neuropathy. T2-weighted sagittal (A) and transverse (B) magnetic resonance (MR) images demonstrating manually traced regions of interest around the caudal portion of the digastric muscle bilaterally, at the level of the fourth ventricle. Patient positioned in dorsal recumbency; field strength: 1.5T; slice thickness: 3.5 mm; TR: 6000 ms; TE: 110 ms. In the transverse image, the patient's right side is displayed on the left

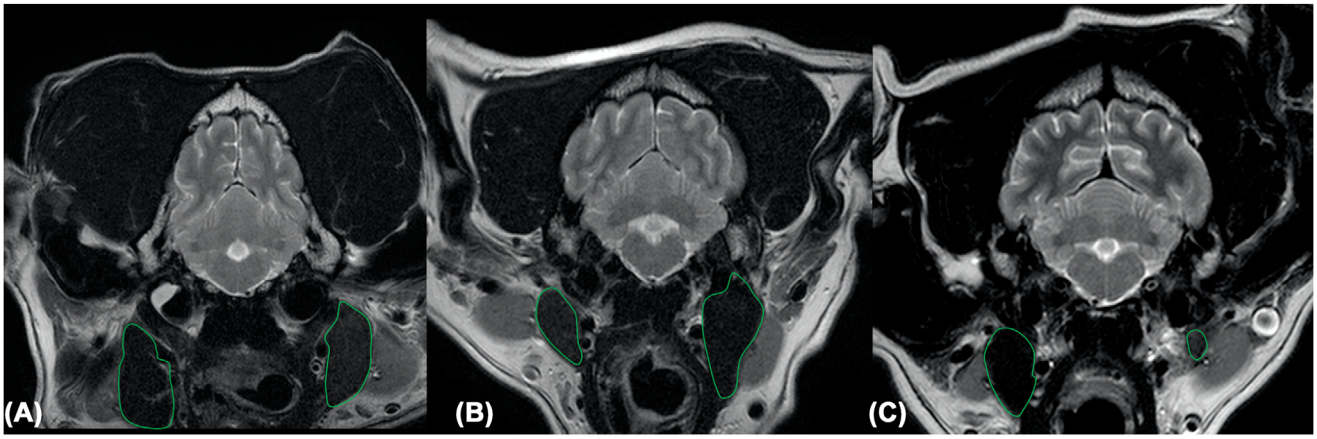

FIGURE 2 T2-weighted transverse MR images demonstrating mild (<25%) (A), moderate (25–50%) (B) and severe (>50%) (C) atrophy of the affected caudal portion of the digastric muscle compared to the contralateral side. (A) Left-sided facial neuropathy in a 6-year-old, female neutered Boxer, (B) right-sided facial neuropathy in a 6-year-old, female neutered Beagle, and (C) left-sided facial neuropathy in a 6-year-old, male neutered Cocker Spaniel. For each patient, positioning was in dorsal recumbency; field strength: 1.5T; slice thickness: 3.5 mm; TR: 6000 ms; TE: 110 ms. In each image, the patient's right side is displayed on the left

to the contralateral side (on T1W images after contrast administration) and enhancement of the affected caudal portion of the digastric muscle compared to the contralateral side (on T1W images after contrast administration).

The transverse sectional area of the caudal portion of the left and right digastric muscles was measured on a transverse T2W image at the level of the fourth ventricle in all patients (Figure 1). Regions of interest were manually traced around the muscle bilaterally. Regions of interest were linked between T2W and T1W sequences using point-based registration. The area of each region of interest was recorded in cm^2. The percentage of atrophy was determined using the following equation:

$$\frac{(\textit{cross sectional area normal side} - \textit{cross sectional area affected side})}{\textit{cross sectional area normal side}} \times 100$$

The severity of the caudal digastricus muscle atrophy was classified for the purpose of this study as mild (<25%) (Figure 2A), moderate (25%–50%) (Figure 2B), and severe (>50%) (Figure 2C). The difference in signal intensity was obtained by comparing the mean

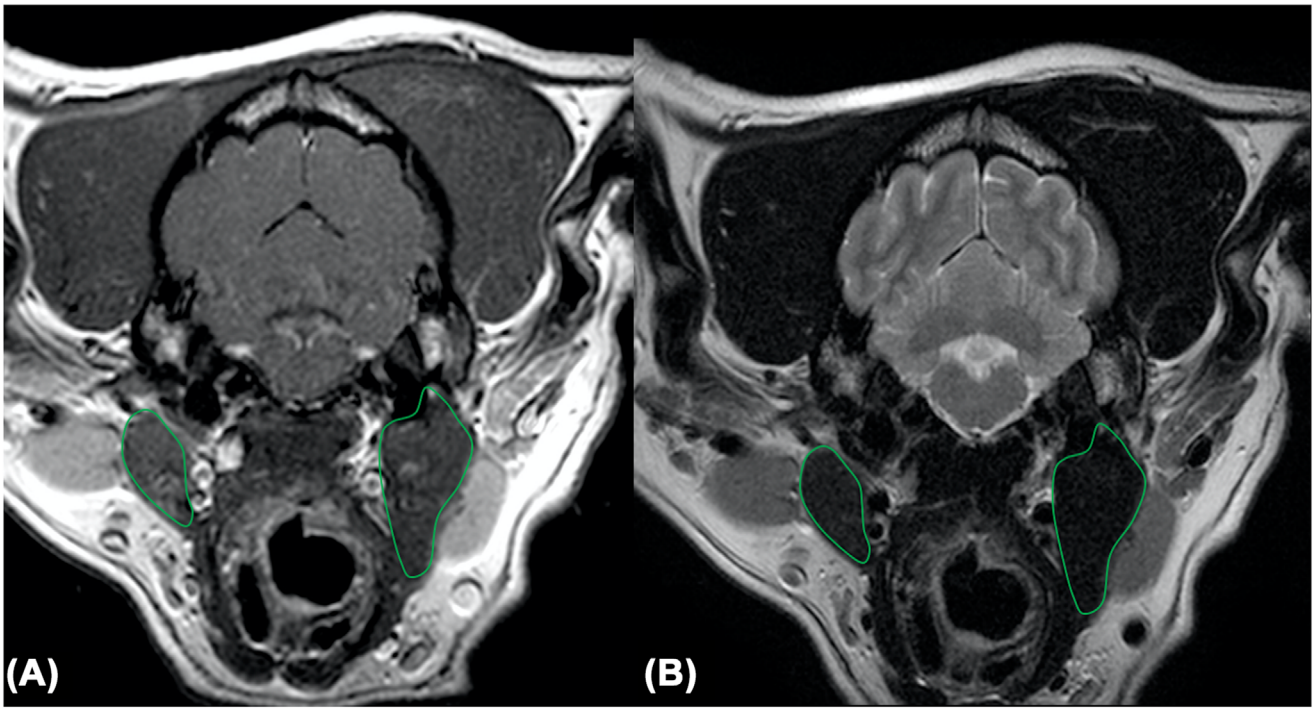

FIGURE 3 Six-year-old, female neutered Beagle with right-sided facial neuropathy. Pre-contrast T1-weighted (A) and T2-weighted (B) transverse MR images demonstrating hyperintensity of the caudal portion of the digastric muscle on the affected side, compared to the contralateral side. Patient positioned in dorsal recumbency; field strength 1.5T; slice thickness: 3.5 mm. For pre-contrast, T1-weighted image (A): TR: 450 ms; TE: 15 ms. For T2-weighted image (B): TR: 6000 ms; TE: 110 ms. In each image, the patient's right side is displayed on the left

signal intensity of the regions of interest of the affected and non-affected muscles, prior to contrast administration both in T1W and T2W images (Figure 3). Enhancement of the facial nerve was classified subjectively as present or absent by comparing signal intensity in the area of the left and right facial nerve in transverse plane T1W images pre- and post-contrast administration. Enhancement of the caudal portion of the digastric muscle was assessed in T1W images by measuring the mean signal intensity of the region of interest pre- and post- contrast administration (Figure 4).

For each patient, follow-up was performed either by clinical re-examinations or evaluation of the clinical records post-diagnosis.

The referring veterinarians were contacted for the clinical records post-diagnosis of the patients. Outcome data were retrieved from the clinical notes provided by the referring practices. Outcome (at 1 month and 3 months post-diagnosis) was classified as "deterioration" in patients where neurological examination revealed clinical deterioration from the clinical signs at presentation, "no improvement" in patients where neurological examination remained unchanged compared to presentation, "improving" in patients demonstrating improvement with some remaining neurological deficits, and "normal" for all patients demonstrating an unremarkable neurological examination at the time of re-examination.

Statistical analyses were performed by a registered veterinarian (Ombeline McGregor) under the guidance of experts in veterinary statistics, using a commercial software (SPSS 26.0, SPSS Inc., Chicago, IL, USA). Repeated Fisher's Exact tests were performed to determine correlation between each of the MRI findings (facial nerve enhancement, hyperintensity of the caudal portion of the digastric muscle in T2W images and pre-contrast T1W, caudal portion of the digastric muscle atrophy, and enhancement post-contrast) and outcome at 1 and 3 months. Statistical significance for all tests was defined as $p < 0.05$.

3 | RESULTS

All the results are detailed in Table S1. Forty-eight dogs were diagnosed with unilateral idiopathic facial neuropathy. Of these, 19 dogs met the inclusion criteria. Ages ranged from 5 years to 13 years old, with a mean of 7.2 years old. Four of eight males and three of 11 females were sexually intact. Affected breeds included Staffordshire Bull Terriers ($n = 3$, 16%), Boxers ($n = 3$, 16%), Crossbreeds ($n = 2$, 11%), Cocker Spaniels ($n = 2$, 11%), Cavalier King Charles Spaniels ($n = 2$, 11%), Beagles, ($n = 2$, 11%), Border Collie ($n = 1$, 5%), Golden Retriever ($n = 1$, 5%), Chihuahua ($n = 1$, 5%), Vizsla ($n = 1$, 5%), and English Springer Spaniel ($n = 1$, 5%).

Both facial neuropathy and peripheral vestibular syndrome were present in 17 cases (89%), and motor facial neuropathy alone was present in two cases (11%). Only cases with unilateral clinical signs were included, being right-sided in seven cases (37%) and left-sided in 12 cases (63%).

At the 1-month follow-up, clinical signs were unchanged in nine dogs (47%), improved in eight dogs (42%), and two dogs were reported to

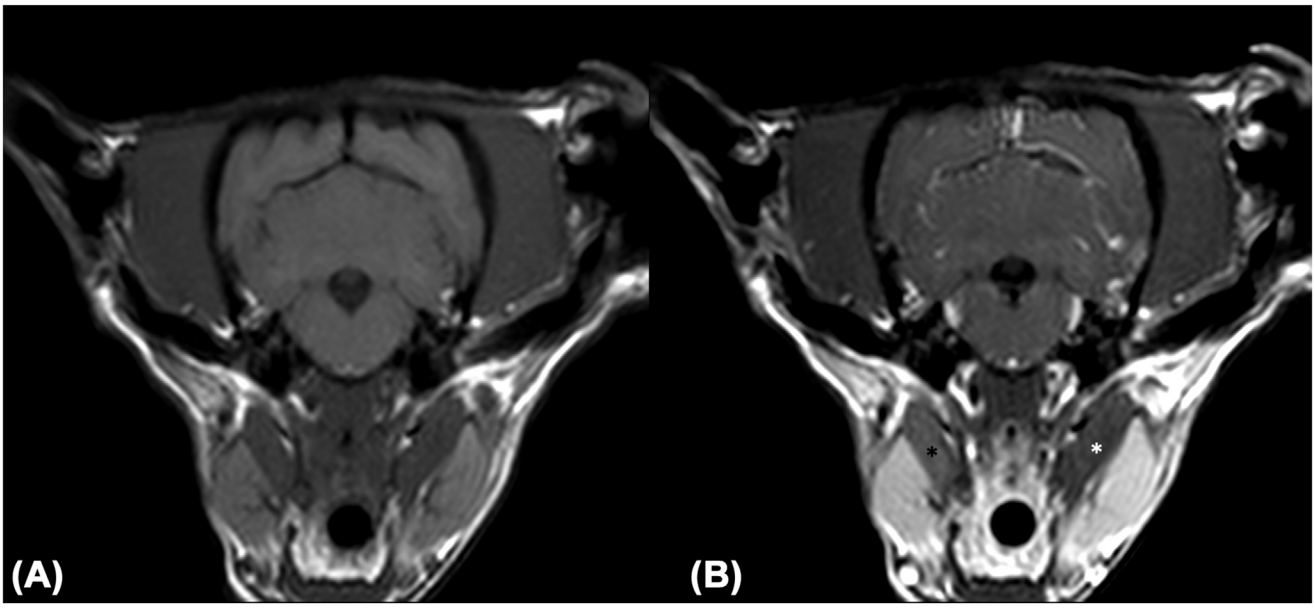

FIGURE 4 Seven-year-old, male neutered Chihuahua with right sided facial neuropathy. Pre-contrast T1-weighted (A) and post-contrast T1-weighted (B) transverse MR images demonstrated increased contrast enhancement of the affected caudal portion of the digastric muscle (black asterisk) compared to the contralateral side (white asterisk). Patient positioned in dorsal recumbency; field strength: 1.5T; slice thickness: 3.5 mm; TR: 450 ms; TE: 15 ms. In each image, the patient's right side is displayed on the left

be normal (11%). Follow-up at 3 months revealed that the clinical signs of facial neuropathy had resolved in seven cases (37%) and partially resolved in five cases (26%). No improvement was noted in seven cases (37%). The duration of the clinical signs prior to MRI ranged from 2 days to 42 days with a mean of 17 days.

MRI of the head was performed using a 1.5 Tesla scanner (Intera CX, Philips Medical Systems, Eindhoven, The Netherlands). Patients were positioned in dorsal recumbency, and images were acquired using a flex coil. Two-dimensional sequences were obtained, including a transverse T2-weighted (T2W) turbo spin echo (TSE) with an echo time (TE) of 110 ms, repetition time (TR) of 6000 ms, slice thickness of 3.0–3.5 mm and an interslice gap of 0—1.0 mm and a transverse T1-weighted (T1W) TSE with a TE of 15 ms, TR of 450 ms, slice thickness of 3.0–3.5 mm and an interslice gap of 0—.0 mm. Transverse T1W sequences were also acquired 1–10 min after intravenous administration by hand of paramagnetic contrast medium, gadobutrol (Gadovist 1.0 mmol/mL, Bayer Inc.) at a dose of 0.1 ml/kg. MR images were analyzed using a DICOM image viewer (OsiriX MD version 11.0.2, OsiriX Imaging Software, OsiriX Foundation, Geneva, Switzerland).

MRI findings revealed atrophy of the affected caudal portion of the digastric muscle compared to the normal side in 17 cases (89%). The atrophy was classified as mild in 12 cases (70%), moderate in three cases (18%), and severe in two cases (12%). There was no evidence of atrophy in two cases (11%). There was no statistically significant difference in outcome at 1 month or outcome at 3 months between patients with caudal portion of the digastric muscle atrophy and patients without (with *p*-values of 1.0 and 0.713, respectively).

Hyperintensity of the affected caudal portion of the digastric muscle was present for 15 cases (79%) in pre-contrast T1W images and for 17 cases (89%) in T2W images. There was statistically significant correlation between hyperintensity in both T2W and T1W images pre-contrast and outcome at both 1 month and 3 months, within each case a *p*-value of <0.01.

Contrast enhancement of the caudal portion of the digastric muscle was present in 14 cases (74%). There was no statistically significant difference in outcome at 1 month or outcome at 3 months between patients with enhancement of the caudal portion of the digastric muscle and patients without (with *p*-values of 0.480 and 1.0, respectively).

Contrast enhancement of the affected facial nerve was present in nine cases (47%). There was no statistically significant difference in outcome at 1 month or outcome at 3 months between patients with facial nerve enhancement and patients without (with *p*-values of 0.255 and 0.122, respectively).

4 | DISCUSSION

This study describes the MRI features of the affected caudal portion of the digastric muscle in dogs diagnosed with idiopathic facial neuropathy. The majority of dogs demonstrated some degree of atrophy and increased enhancement of the affected muscle post-contrast when compared to the normal side, although no correlation was found between these MRI features and prognosis. Hyperintensity of the affected muscle when compared to the normal side in both pre-contrast T1W and T2W images, resulting from a combination of edema and fatty degeneration, was also demonstrated in a majority of dogs, with these MRI features found to be correlated with a worse prognosis for return to normal facial nerve function.

MRI appearance of muscle following CN motor denervation has been well described in humans and has been correlated with prognosis to return to normal function.[11–14] Denervated muscles undergo a characteristic pattern of changes on MR images as the denervation progresses from the acute phase through a subacute and then chronic phase. The time course of denervation is poorly defined; however it is generally agreed that acute changes are those that occur within a month following denervation, subacute changes occur within 12–20 months, and chronic changes occur after 12–20 months.[11,14,15] In the acute phase, there is evidence of muscle swelling, T2W signal hyperintensity, and increased post-contrast enhancement of the affected muscle.[11,14,16,17] These changes are important to be aware of as the increased muscle mass, signal intensity, and abnormal contrast enhancement may occasionally be mistaken for a mass.[14,17,18] It has been suggested that the muscle swelling and T2W signal hyperintensity in the acute phase may be related to edema, while the increase in post-contrast enhancement may be related to shift of water from the intra- to extra-cellular compartment allowing for greater accumulation of contrast.[11,16,17,19] Furthermore, studies in animals have found that denervated muscle has relatively increased vascularity per volume of muscle, contributing further to contrast enhancement.[20–23] In the subacute and chronic phases, there is progressive fatty replacement of muscle tissue, resulting in hyperintensity, as well as progressive loss of muscle volume from as early as 7 days after injury.[11,14,16,17] It is useful to be aware of normal imaging findings in the chronic phase as atrophied muscle associated with abnormal signal intensity and hypertrophy of surrounding musculature may again occasionally be mistaken for a mass lesion.[14,17,18] The temporal relationship between the onset of the muscle denervation and the onset of clinical and imaging changes varies between individuals and also between different CNs and type of injury.[11,14,17] It is likely that such variation also exists between humans and dogs. The majority of the patients included in this study presented within 1 month following the development of clinical signs assoicated with facial neuropathy and as such would have been classified as presenting in the "acute phase" following denervation. The imaging findings however demonstrated varying degrees of chronicity, with the majority of patients demonstrating some degree of muscle atrophy on the affected side. Further research is necessary to better understand the timeline of denervation injury and associated imaging findings in dogs.

One study in humans has demonstrated that the degree of asymmetry between the muscles of facial expresion prior to facial nerve grafting can be correlated with prognosis, with a worse prognosis for recovery to function in patients with moderate to severe atrophy of the affected muscles compared with those displaying minimal atrophy prior to the procedure. This may be explained by the need for a minimum or "critical" muscle mass to allow for recovery of function.[13] Imaging of the affected nerve and target musculature may therefore be of prognostic benefit. The facial musculature is not easily assessed in dogs due to the small volume of these muscles resulting in difficulty describing any changes accurately.[10] In the current study, the caudal portion of the digastric muscle was clearly visible on MR images allowing for adequate assessment of changes in muscle mass, signal intensity, and enhancement. This muscle could therefore be used as an early indicator of the severity of injury to the facial nerve.

This study did not demonstrate a statistically significant correlation between atrophy of the caudal portion of the digastric muscle nor increased enhancement post-contrast and outcome in this group of dogs. MRI is commonly included as part of the diagnostic work-up for facial neuropathy in small animals and as such is performed often in the acute setting. In this study, the majority of dogs underwent MRI within 1 month following onset of clinical signs. As such, it is possible that atrophy of the caudal portion of the digastric muscle and enhancement post-contrast could not be reliably assessed and correlated with prognosis due to variation in the time between onset of clinical signs and MRI being performed, as well as individual variability between patients in the acute phase. It may be of benefit in a future study to consider a set time between onset of clinical signs and MRI in an attempt to reduce variability.

This study demonstrates a statistically significant correlation between hyperintensity in both pre-contrast T1W and T2W images and outcome, with hyperintensity being correlated with a worse prognosis to return to normal. Based on these results, it is possible that patients that do not demonstrate hyperintensity on these sequences are more likely to regain normal facial nerve function. This may be explained in the acute phase by the absence of significant edema, suggestive of less severe injury. In the more chronic phase, this may be explained by the maintenance of greater functional muscle mass due to minimal replacement of muscle fibers with fatty tissue.

Facial nerve enhancement has been reported in cases of idiopathic facial neuropathy (Bell's palsy) in humans, with conflicting evidence as to its use as a prognostic indicator. Some studies suggest a correlation between facial nerve enhancement and outcome,[24,25] while the majority suggest no such correlation.[26–30] Facial nerve enhancement in cases of idiopathic facial neuropathy has also been described in dogs,[7,8] with one study suggesting a possible correlation with outcome, although the study included few animals.[7] This study did not demonstrate a statistically significant correlation between enhancement of the facial nerve and outcome.

The main limitations of this retrospective study include the low number of cases (n = 19), lack of pathologic confirmation of imaging features, and lack of standardization of MRI protocols. MRI protocols would have been improved by the standardization of patient positioning to reduce the impact of external factors on the shape of the caudal aspect of the digastric muscle, consistent inclusion of the caudal portion of the digastric muscle in all patients being investigated for facial neuropathy, inclusion of fat-suppressing sequences and sequences which highlight edema to better understand the relative roles played by these changes in the imaging features described, and finally by the use of a subtraction technique to highlight subtle findings that may not have been discernible by comparing only pre- and post-contrast T1W images.

Cocker Spaniels have been reported to be over-represented in cases of idiopathic facial neuropathy.[3,5,31] In this study, while Staffordshire Bull Terriers and Boxers were seen slightly more commonly than other breeds, consistent with what has been reported in other studies,[6,7] no

single breed was found to be significantly more at risk, and this breed profile may simply be a representation of the population in the studied area rather than any true predisposition for the disease. Idiopathic facial neuropathy is reported to occur in middle-aged to older dogs.[5] In this study, a median age of onset of 7.2 years was consistent with previous reports.[5,6,7] The present study found more female dogs to be affected than male dogs as well as disease more commonly lateralized to the left-hand side. Predilection of the disease for one side or for a certain sex has not been described previously, and these findings are more likely a reflection of the small number of dogs. This study deliberately included only patients with unilateral clinical signs with the aim to use the contralateral, clinically unaffected side for comparison. Both facial paralysis and vestibular signs were present in majority cases (89%), consistent with previous reports.[4–6,9]

In conclusion, findings from this sample of dogs indicated that MRI characteristics of idiopathic facial neuropathy included ipsilateral atrophy, pre-contrast T1W and T2W hyperintensity, and enhancement post-contrast of the caudal portion of the digastric muscle on MR images. No correlation was found between atrophy or post-contrast enhancement and outcome, although further investigation using a greater number of cases, standardizing time between onset of clinical signs and MRI, and monitoring for a period of 12 months or longer may be of benefit. Significant correlation was found between hyperintensity of the affected caudal portion of the digastric muscle in both pre-contrast T1W and T2W images and outcome, suggesting that MRI of patients diagnosed with idiopathic facial neuropathy may be of benefit not only as a diagnostic tool, but also to better predict prognosis to return to normal facial nerve function.

LIST OF AUTHOR CONTRIBUTIONS

Category 1

(a) Conception and Design: McGregor, Plested, Beltran
(b) Acquisition of Data: McGregor, Plested, Beltran
(c) Analysis and Interpretation of Data: McGregor, Plested, Beltran

Category 2

(a) Drafting the Article: McGregor, Plested, Beltran
(b) Revising Article for Intellectual Content: Plested, Beltran

Category 3

(a) Final Approval of the Completed Article: McGregor, Plested, Beltran

CONFLICT OF INTEREST

Authors do not have conflict of interest.

ORCID

Elsa Beltran https://orcid.org/0000-0002-0807-7817

REFERENCES

1. de Lahunta A, Glass E, Kent M. Lower Motor Neuron: General Somatic Efferent, Cranial Nerve. *Veterinary Neuroanatomy and Clinical Neurology*. 4th ed. Missouri: Elsevier Saunders; 2015:162–196.
2. Evans HE. Cranial Nerves. *Miller's Anatomy of the Dog*. 4th ed. Philadelphia: W.B. Saunders; 2013.708–730.
3. Braund KG, Luttgen PJ, Sorjonen DC, Redding RW. Idiopathic facial paralysis in the dog. *Vet Rec*. 1979;105(13):297–299.
4. Chan MK, Toribio J-A, Podadera JM, Child G. Incidence, cause, outcome and possible risk factors associated with facial nerve paralysis in dogs in a Sydney population (2001-2016): a retrospective study. *Aust Vet J*. 2020;98:140–147.
5. Kern TJ, Erb HN. Facial neuropathy in dogs and cats: 95 cases (1975-1985). *J Am Vet Med Assoc*. 1987;191(12):1604–1609.
6. Jeandel A, Thibaud JL, Blot S. Facial and vestibular neuropathy in 16 dogs. *J Small Anim Pract*. 2016;57:74–78.
7. Varejao ASP, Munoz A, Lorenzo V. Magnetic resonance imaging of the intratemporal nerve in idiopathic facial paralysis in the dog. *Vet Radiol Ultrasound*. 2006;4:328–333.
8. Schultz RM, Tucker RL, Gavin PR, Bagley R, Saveraid TC, Berry CR. Magnetic resonance imaging of acquired trigeminal nerve disorders in six dogs. *Vet Radiol Ultrasound*. 2007;48(2):101–104.
9. Smith PM, Goncalves R, McConnell JF. Sensitivity and specificity of MRI for detecting facial nerve abnormalities in dogs with facial neuropathy. *Vet Rec*. 2012;171(14):349.
10. Borges A. Imaging of denervation in the head and neck. *Eur J Radiol*. 2010;74:378–390.
11. Connor SE, Chaudhary N, Fareedi S, Woo EK. Imaging of muscular denervation secondary to motor cranial nerve dysfunction. *Clin Radiol*. 2006;61(8):659–669.
12. Fischbein NJ, Kaplan MJ, Jackler RK, Dillon WP. MR imaging in two cases of subacute denervation change in the muscles of facial expression. *Am J Neuroradiol*. 2001;22:880–884.
13. Kaylie DM, Wax MK, Weissman JL. Preoperative facial muscle imaging predicts final facial function after facial nerve grafting. *Am J Neuroradiol*. 2003;24:326–330.
14. Russo CP, Smoker WRK, Weissman JL. MR appearance of trigeminal and hypoglossal motor denervation. *Am J Neuroradiol*. 1997;18:1375–1383.
15. Fleckenstein JL, Watumull D, Conner KE, et al. Denervated human skeletal muscle: MR imaging evaluation. *Radiology*. 1993;187:213–218.
16. Bendszus M, Koltzenburg M, Wessig C, Solymosi L. MR imaging of denervated muscle: experimental study. *Am J Neuroradiol*. 2002;23(8):1427–1431.
17. Kato K, Tomura N, Takahashi S, Watarai J. Motor denervation of tumors of the head and neck: changes in MR appearance. *Magn Reson Med Sci*. 2002;1(3):157–164.
18. Harnsberger HR, Dillon WP. Major motor atrophic patterns in the face and neck: cT evaluation. *Radiology*. 1985;155(3):665–670.
19. Davis SB, Mathews VP, Williams DW. Masticator muscle enhancement in subacute denervation atrophy. *Am J Neuroradiol*. 1995;16:1292–1294.
20. Bondy SC, Purdy JL, Carroll JE, Kaiser KK. The rate of nutrient supply to normal and denervated slow and fast muscle, and its relation to muscle blood flow. *Exp Neurol*. 1976;551:678–683.
21. Carpenter S, Karpati G. Necrosis of capillaries in denervation atrophy of human skeletal muscle. *Muscle Nerve*. 1982;5:250–254.
22. Hassler O. The angioarchitecture of normal and denervated skeletal muscle. *Neurology*. 1970;20:1161–1164.
23. Hudlicka O. Blood flow and oxygen consumption in muscles after section of ventral roots. *Circ Res*. 1967;20:570–577.
24. Girard N, Raybaud C, Poncet M. 3D-FT MRI of the facial nerve. *Neuroradiology*. 1994;36:462–468.
25. Kress B, Griesbeck F, Stippich C, Bähren W, Sartor K. Bell palsy: quantitative analysis of MR imaging data as a method of predicting outcome. *Radiology*. 2004;230:504–509.
26. Burmeister HP, Baltzer PA, Volk GF, et al. Evaluation of the early phase of Bell's palsy using 3 T MRI. *Eur Arch Otorhinolaryngol*. 2011;268:1493–1500.

27. Engstrom M, Thuomas KA, Naeser P, Stalberg E, Jonsson L. Facial nerve enhancement in Bell's palsy demonstrated by different gadolinium-enhanced magnetic resonance imaging techniques. *Arch Otolaryngol Head Neck Surgery*. 1993;119:221–225.
28. Kohsyu H, Aoyagi M, Tojima H, et al. Facial nerve enhancement in Gd-MRI in patients with Bell's palsy. *Acta Otolaryngol Suppl*. 1994;511:165–169.
29. Murphy TP, Teller DC. Magnetic resonance imaging of the facial nerve during Bell's palsy. *Otolaryng Head Neck*. 1991;105:667–674.
30. Song MH, Kim J, Jeon JH, et al. Clinical significance of quantitative analysis of facial nerve enhancement on MRI in Bell's palsy. *Acta Otolaryngol*. 2008;128:1259–1265.
31. Garosi LS, Lowrie ML, Swinbourne NF. Neurological manifestations of ear disease in dogs and cats. *Vet Clin North Am Small Anim Pract*. 2012;42(6):1143–1160.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

How to cite this article: McGregor O, Plested MJ, Beltran E. Magnetic resonance imaging of the caudal portion of the digastric muscle in canine idiopathic facial neuropathy. *Vet Radiol Ultrasound*. 2021;62:455–462. https://doi.org/10.1111/vru.12974