

COMPUTED TOMOGRAPHY MYELOGRAPHIC FINDINGS IN DOGS WITH CERVICAL SPONDYLOMYELOPATHY

RONALDO C. DA COSTA, RITA L. ECHANDI, DUSTIN BEAUCHAMP

Computed tomography (CT) myelography is used occasionally in the diagnosis of cervical spondylomyelopathy, but the type of lesion found in large- versus giant-breed dogs using this modality has not been characterized. Our purpose was to report the frequency of compressive lesions in large- and giant-breed dogs with cervical spondylomyelopathy and imaged using CT myelography. Fifty-eight dogs were retrospectively studied, 23 large-breed and 35 giant-breed dogs. Multiple sites of compression were found in 12 large-breed dogs (52.2%) compared to 30 (85.8%) giant-breed dogs. The main site of compression was at C5–6 and C6–7 in both large-breed (91.3%) and giant-breed (72.4%) dogs. The main cause and direction of compression was disc-associated and ventral in 19 (82.6%) of the large-breed dogs while osseous changes were the primary cause of compression in 27 (77.2%) of the giant-breed dogs, with most compressions being lateral (51.4%), followed by dorsolateral (14.2%). Osseous compression was observed at C7-T1 in eight giant-breed dogs (22.8%), and at T1-T2 or T2 only in five dogs (14.3%). Four of 23 large-breed dogs (17.4%), and seven (20%) of 35 giant-breed dogs had spinal cord atrophy. Therefore, giant-breed dogs often have multiple compressions, usually caused by osseous changes causing lateralized compressions. In large-breed dogs most compressions are disc-associated and located ventrally. Considering the number of giant-breed dogs with compressions at C7-T1, T1–2, and T2, it is important to include the cranial thoracic region when imaging dogs suspected of having cervical spondylomyelopathy. © 2012 *Veterinary Radiology & Ultrasound*, Vol. 53, No. 1, 2012, pp 64–70.

Key words: canine, dog, myelopathy, spinal cord, wobbler syndrome.

Introduction

CERVICAL SPONDYLOMYELOPATHY IS seen commonly in large- and giant-breed dogs. It is characterized by spinal cord and/or nerve root compression leading to variable degrees of neurologic deficits and neck pain.^{1–4} Neural compression in cervical spondylomyelopathy results from both static and dynamic factors caused typically by disc protrusions or osseous compressions.¹ The pathogenesis is complex and unclear. An example of this complexity is that Doberman Pinschers can have spinal cord compression but no clinical signs of cervical spondylomyelopathy.^{5–7}

The diagnosis of cervical spondylomyelopathy is established via imaging.⁸ Survey radiographs offer limited information and cannot confirm the diagnosis.⁹ Myelography defines the site(s) and direction (ventral, dorsal, lateral) of spinal cord compression and allows stress or dynamic myelographic studies.^{8,9} Magnetic resonance (MR) imaging al-

lows noninvasive assessment of the spinal cord parenchyma in addition to identification of compressive lesions.^{10,11}

Computed tomography (CT) myelography is also used to assess dogs with cervical spondylomyelopathy, but there are few data available regarding the changes to be seen.¹² Our objectives were to describe the CT myelography findings in large- and giant-breed dogs with cervical spondylomyelopathy reporting the frequency of pathologic changes and seizures associated with the procedure.

Materials and Methods

Medical records were searched for dogs diagnosed with cervical spondylomyelopathy between 2003 and 2008. Inclusion criteria were a definitive diagnosis of cervical spondylomyelopathy in a large- or giant-breed dog achieved by CT myelography, with complete medical records and all CT myelography images available for review.

The following information was collected: breed, age at diagnosis, gender, duration of clinical signs (acute ≤ 7 days, chronic ≥ 8 days), contrast medium used for myelography, dose of contrast medium, and prevalence of seizures associated with the procedure.

Fifty-eight dogs met the inclusion criteria. Twenty-three dogs were large-breed and the other 35 belonged to giant breeds. Among the large-breed dogs there were 11 Doberman pinschers, three Weimaraners, three large mixed

From the Department of Veterinary Clinical Sciences, College of Veterinary Medicine, The Ohio State University, Columbus, OH 43210. Dr. Rita L. Echandi's current address is Animal Imaging, Irving, TX 75039. Dr. Dustin Beauchamp's current address is Tufts Veterinary Hospital, North Grafton, MA 01536

Address correspondence and reprint requests to Ronaldo C. da Costa, at the above address. E-mail: dacosta.6@osu.edu.

Received May 23, 2010; accepted for publication July 26, 2011.

doi: 10.1111/j.1740-8261.2011.01869.x

breeds, two Dalmatians, two Labrador retrievers, one Greyhound, and one German shorthaired pointer. The median age of large-breed dogs was 7 years (range 1–13 years, mean 7.1 years), and there were 11 males and 12 females. Among the giant-breed dogs, there were 18 Great Danes, eight Rottweilers, seven Mastiffs, one Great Pyrenees, and one Bernese Mountain. Their median age was 2.5 years (range 0.5 to 11 years, mean 4.1 years), and there were 21 males and 14 females.

Of the large-breed dogs, seven dogs had an acute onset of clinical signs, 13 had chronic signs, and three had chronic neurologic signs with an acute worsening. The mean duration of clinical signs was 95.7 days and the median 36 days. Of the giant-breed dogs, five dogs had acute signs, 25 chronic signs, and five had a chronic history with acute worsening. The mean duration of clinical signs was 179.1 days with a median of 90 days.

Myelography was performed using iohexol* injected at either L4–5, L5–6, or at the cerebellomedullary cistern. The concentration used for all lumbar punctures was 240 mg/ml, while cisternal injections were performed with using contrast medium with a concentration of either 240 or 180 mg/ml. CT was performed using a single-slice fourth-generation helical scanner.† Dogs were in either dorsal or sternal recumbency. Transverse images were acquired at a slice thickness ranging from 1 to 5 mm based on patient size. CT studies were acquired using either a sharp, bone, or standard algorithm.

All CT myelography images were reviewed by a board-certified veterinary neurologist (RdC) and by a board certified veterinary radiologist (RLE). Diagnostic imaging studies dated 2002–2003 were available on film while those dated 2004–2008 were evaluated on a workstation using eFilm.‡ Both investigators reviewed the images individually and then met to discuss the findings. When the findings were dissimilar the images were reviewed and consensus reached. For each dog the following items were evaluated:

- Main site and additional sites of spinal cord compression;
- Severity of spinal cord compression: graded as none, mild (<25% reduction in the diameter of the spinal cord), moderate (25–50%), and severe (>50%). The degree of compression was established by comparing the diameter of the spinal cord at the compressive site and with cranial and caudal sites;
- Direction of the compressive lesion: either dorsal, ventral, lateral, or dorsolateral (Fig. 1A and B);
- Cause of compression: classified as intervertebral disc, ligamentous hypertrophy, or osseous changes such as

articular process joint osteoarthritis or vertebral malformation (pedicle and/or lamina);

- Presence or absence of spinal cord atrophy at the site of most severe spinal cord compression, based on a circumferential widening of the subarachnoid space, compared with the cranial and caudal transverse sections (Fig. 1C);
- Presence or absence of dorsoventral foraminal stenosis and its severity (absent, mild, moderate, or severe) based on transverse images;
- Other pathologic changes such as synovial cysts (Fig. 1D).

Results

In large-breed dogs, 11 (47.8%) had a single site of spinal cord compression and 12 (52.2%) had multiple compressive sites (Fig. 2). The main site of compression was at C6–7 in 14 dogs (60.8%), at C5–6 in seven dogs (30.4%), and C4–5 and C2–3 with one dog each (4.4%) (Fig. 3). The compression was severe in two dogs (8.7%), moderate in 14 dogs (60.8%), and mild in seven dogs (30.5%). The cause of compression was disc-associated in 19 dogs (82.6%) (Fig. 4). The compression was caused by osseous changes in four dogs (17.4%). These osseous changes were characterized by osteoarthritis of the articular processes in two dogs and malformation of the pedicles and lamina in two dogs. Three of the four dogs with osseous compressions also had synovial cysts and one also had thickening of the ligamentum flavum. The direction of spinal cord compression was ventral in 19 dogs (82.6%), dorsolateral in two dogs (8.6%), dorsal in one dog (4.4%), and lateral in one dog (4.4%). Four of the 23 large breed dogs (17.4%) had evidence of spinal cord atrophy.

Six of 23 large dogs (26%) had intervertebral foraminal stenosis. Four had stenosis at a single site, whereas two dogs had stenosis at two intervertebral regions. The stenosis was mild in three dogs, moderate in two dogs, and severe in one dog. The site of foraminal stenosis was at C5–6 in two dogs, C6–7 in two dogs, and at C5–6 and C6–7 in two dogs.

In giant-breed dogs, five (14.2%) had a single site of compression, whereas 30 (85.8%) had multiple sites of compression (Figs. 2 and 5). The main compressive site was at C6–7 in 13 dogs (44.8%), C5–6 in eight dogs (27.6%), C4–5 in five dogs (17.3%), C2–3 in two dogs (6.8%), and C3–4 in one dog (3.5%) (Fig. 3). Six giant-breed dogs had two or three sites of compression of similar severity and a main lesion could not be established. Osseous compression was also observed at C7-T1 in eight dogs (22.8%), and T1-T2 or T2 only in five dogs (14.3%) (Fig. 5). The primary cause of compression was osteoarthritic proliferations or osseous malformations in 27 dogs (77.2%) with two dogs having concurrent synovial cysts, disc-associated in four dogs (11.4%), and a combination of disc protrusion and osseous changes in four

*Omnipaque, GE Healthcare Inc., Princeton NJ.

†Picker PQS Philips Medical Systems, Inc., N.A., Bothell, WA.

‡eFilm Merge Healthcare 2006, Milwaukee, WI 53214.

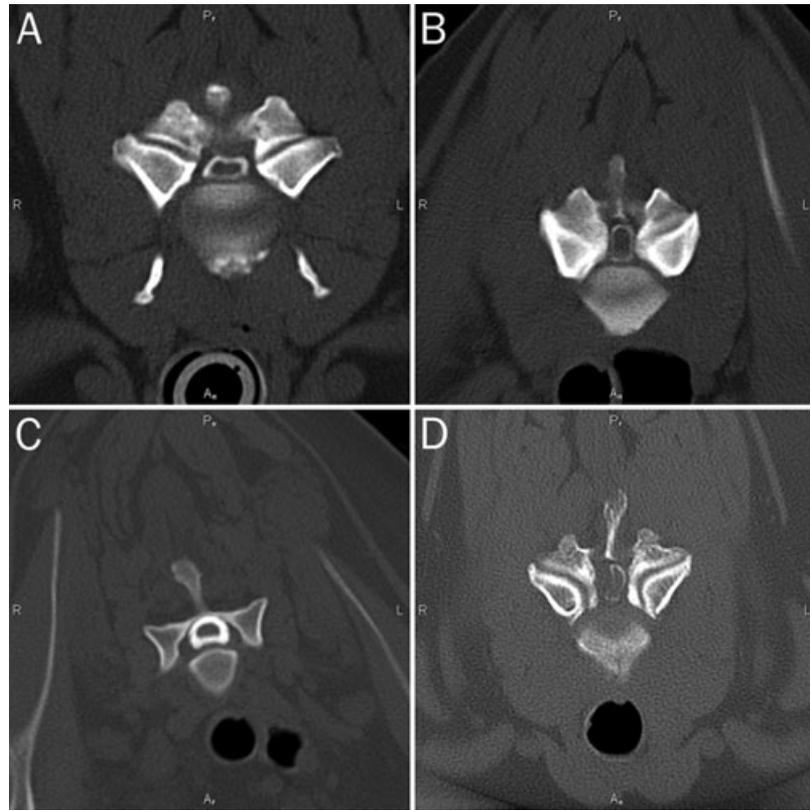


FIG. 1. Directions and types of spinal lesions observed. (A) Dorsal compression at C4–5; (B) bilateral compression at C6–7; (C) Spinal cord atrophy at C6–7; note the circumferential widening of the subarachnoid space; (D) osseous lesion with lateralized compression from extradural synovial cyst at C5–6.

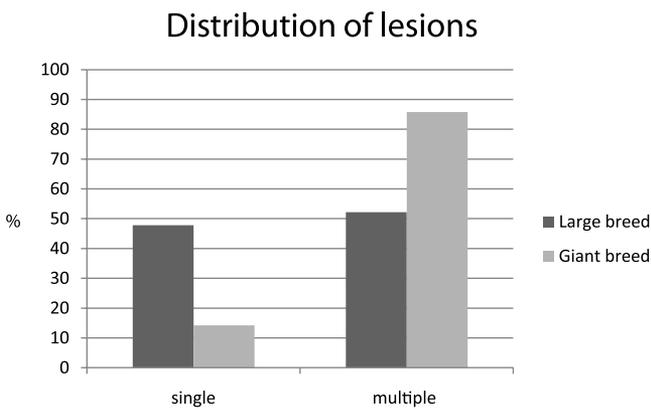


FIG. 2. Distribution of single versus multiple compressive lesions in large-versus giant-breed dogs.

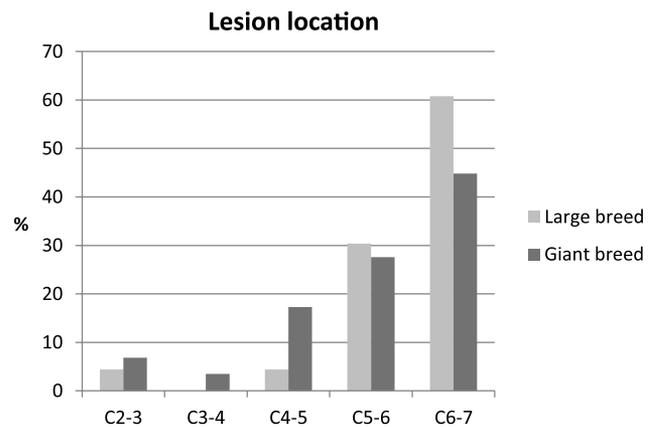


FIG. 3. Distribution of the location of the main compressive lesion in large-versus giant breed dogs.

dogs (11.4%). The direction of compression was bilateral in 18 dogs (51.4%), dorsolateral in five dogs (14.2%), dorsal in three dogs (8%), ventral in five dogs (14.2%), and a combination of two or more directions in four dogs (11.4%). The main spinal cord compression was severe in nine dogs (25.7%), moderate in 19 dogs (54.3%), and mild in seven dogs (20%). Seven dogs (20%) had spinal cord atrophy.

Twenty-three of the 35 giant-breed dogs (65.7%) had intervertebral foraminal stenosis. Nine dogs (39.1%) had

a single site of stenosis (four at C5–6, four at C6–7, and one at C2–3), whereas 14 dogs (60.9%) had multiple sites of foraminal stenosis. Multiple sites of foraminal stenosis affected two sites in nine dogs and three sites in five dogs. Based on the most severe site, the foraminal stenosis was mild in three dogs, moderate in two dogs, and severe in 11 dogs.

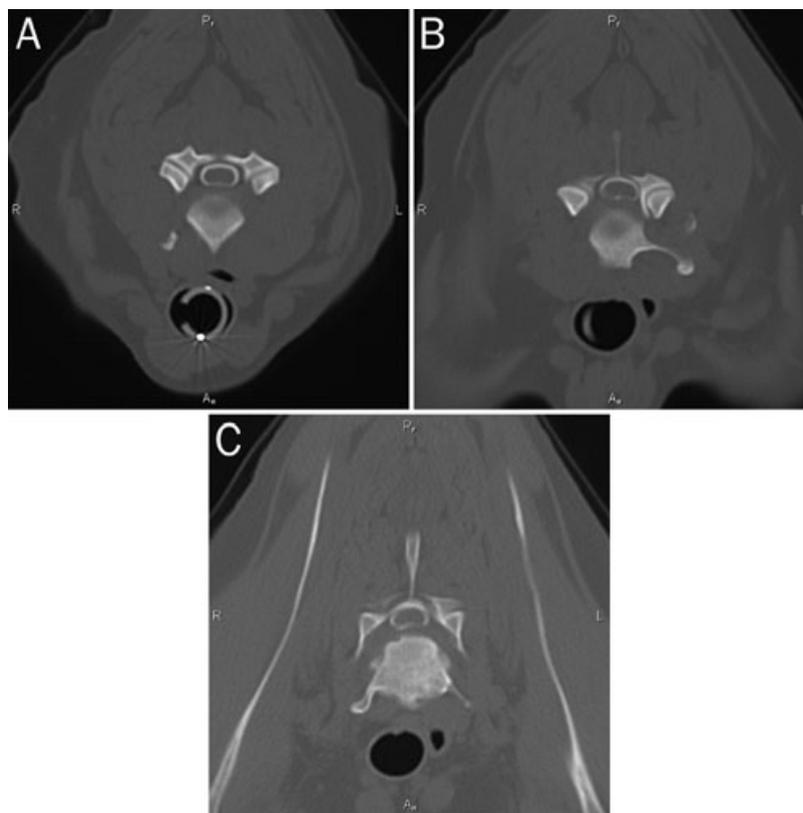


FIG. 4. CT myelogram images of a Weimaraner with ventral, disc-associated spinal cord compression at C4–5 (A), C5–6 (B), and C6–7 (C).

Fourteen animals (24.1%) had seizures upon anesthetic recovery. Six were giant-breed dogs and eight large-breed dogs. All dogs recovered without complications. The mean dose per kilogram and total volume of iohexol used in the dogs with seizures was 0.33 ml and 12.9 ml (median 0.32 and 11 ml, range 0.1–0.5, and 8.8–22.4), respectively. Six dogs were injected in the lumbar cistern and six in the cerebellomedullary cistern. The site of injection was not recorded for two dogs. Dogs that did not have seizures had a mean dose per kilogram of 0.24 ml, and a mean total volume of 11.7 ml (median 0.23 and 10 ml, range 0.11–0.47, and 6.5–32 ml), respectively.

Discussion

An important finding of this study was that almost 15% of giant-breed dogs had neural compression in the T1–T2 region. We were not able to find evidence of prior reports of compression at this site. In addition, 22.8% of giant-breed dogs also had neural compression at the C7–T1 region. These compressions were not the main source of neural compression; they were a component of multiple compressions seen in 85.8% of giant-breed dogs. Nevertheless, they were directly impinging upon the spinal cord. The identification of these caudal lesions has two implications. First,

the cranial aspect of the thoracic spine should always be included in the field of view when planning imaging studies of dogs with cervical spondylomyelopathy; we recommend imaging from C2–3 to T2–3. Even though we only observed caudal neural compression in giant-breed dogs, it is reasonable to image this far caudally in any dog suspected of having cervical spondylomyelopathy. The importance of a large field of view for imaging cervical spondylomyelopathy has been emphasized by others.¹³ Second, the identification of cranial thoracic neural compression can have therapeutic and prognostic implications. Giant breed dogs are typically treated with decompression or ventral fusion.^{10,14–17} Dorsal laminectomy extending from C3 to T2 would be very extensive. Ventral fixation would be even more problematic because it would require ventral exposure of T3. It is possible that these caudal lesions would not progress to cause severe neurologic deficits, as spinal lesions in Dobermans with disc-associated cervical spondylomyelopathy tend to progress slowly.¹⁸ The natural progression of cervical spondylomyelopathy in giant-breed dogs is currently unknown. An important difference between large- and giant-breed dogs with cervical spondylomyelopathy is that giant-breed dogs develop osseous compressive lesions at a younger age; median age 2.5 years versus 7 years for large-breed dogs in this study. Therefore, cervical spondylomyelopathy can progress in giant-breed

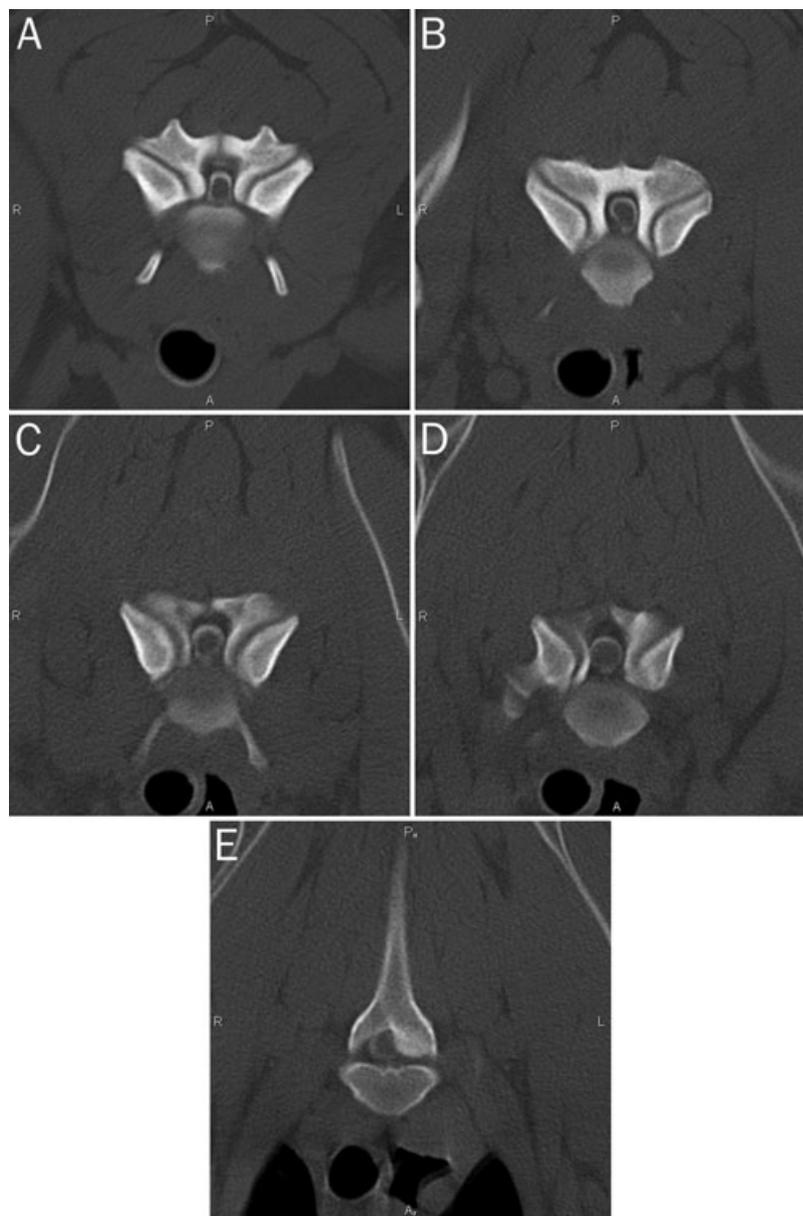


FIG. 5. CT myelogram images of a Mastiff with multiple osseous lesions. (A) bilateral compression at C4–5; (B) bilateral compression at C5–6, worse on the right-side; (C) bilateral compression at C6–7, worse on the left side; (D) bilateral vertebral canal stenosis at C7–T1; (E) Dorsolateral compression at T2.

dogs for many years, allowing sufficient time for these cranial thoracic lesions to become symptomatic.

Interestingly, cranial thoracic neural compression secondary to pedicle malformations similar to those seen in this report was observed in two unrelated 5-month-old Dogues de Bordeaux.¹⁹ These Dogues de Bordeaux, however, had no evidence of cervical myelopathy, and it was stated that myelography alone was of limited value, recommending CT myelography or MR imaging.¹⁹

The CT myelography findings in giant-breed dogs were different from those in large-breed dogs. Most giant-breed dogs (85.5%) had multiple compressive lesions, whereas this

was seen in only approximately 50% of large-breed dogs; this has also been observed by others.^{10,20} On the other hand, some have suggested a more even distribution of single versus multifocal compressions,²¹ while others indicate that most giant-breed dogs have a single compressive lesion.²²

We found that the major site of neural compression in giant-breed dogs was in the caudal aspect of the cervical spine. This is more typical of the site of compression reported in large-breed dogs with disc-associated cervical spondylomyelopathy.^{23–25} It is giant-breed dogs, primarily Great Danes that have been reported to have the most

compressive lesions in the mid-cervical spine.^{20,21} However, most compressive sites in prior work were identified based on survey radiographs or myelography, and it is known that survey radiographs cannot conclusively identify compressive sites and that the most severe site of compression may not be identified based on myelography.¹¹

There was a difference in the source and direction of spinal cord compression in giant-breed versus large-breed dogs. Giant-breed dogs had compression caused by osseous lesions in 77.2% of dogs, whereas only 17.4% of large-breed dogs had compression due to an osseous lesion. Conversely, disc-associated compression was seen in 82.6% of large-breed dogs, but in only 11.4% of giant-breed dogs. There was also an overlap of lesions with 17.4% of large-breed dogs having osseous lesions, and 11.4% of giant-breeds having disc-related compressions. Therefore, even though cervical spondylomyelopathy is generally different in large-versus giant-breed dogs, significant overlap of the pathologic changes occurs. Also, the direction of compression followed the primary source of compression, with disc-associated lesions causing ventral compression and the direction of compression being more variable with osseous lesions. Most giant-breed dogs (51.4%) had bilateral (lateralized) spinal cord compression, followed by dorsolateral (14.2%) and dorsal compression (8%). This is in contrast with other work, based on MR imaging, where dorsal compression occurred in almost 48% of giant-breed dogs with osseous lesions.¹⁰

We identified spinal cord atrophy in 17.4% of large-breed dogs and 20% of giant-breed dogs. We acknowledge that CT myelography may have limited sensitivity to detect this change. Others have reported a prevalence of spinal cord atrophy in Dobermans of 31%, based on CT myelography.¹² In people, spinal cord atrophy is associated with a poor prognosis,²⁶ but it is not known what role spinal cord atrophy plays in dogs.

Five dogs (8.6%) had extradural synovial cysts (two giant-breed, three large-breed dogs). In comparison, synovial cysts were found in 24% of dogs based on MR imaging.¹⁰ This difference is not surprising given the much greater tissue contrast resolution possible with MR imaging compared to CT myelography. All reported synovial cysts

occurring with cervical spondylomyelopathy have been in giant-breed dogs^{10,27,28} whereas we observed three large-breed dogs with a synovial cyst. Consistent with previous reports,^{27,28} all dogs in this study with a synovial cyst of the articular process joint had osseous lesions.

It is important to emphasize that CT myelography and MR imaging are complementary.²⁹ In people, the severity of vertebral canal stenosis may be overemphasized with MR imaging and underestimated with CT.²⁹ Discrepancies in the diagnostic accuracy of CT myelography versus MR imaging also exist based on whether the source of the lesion is disc or osseous,³⁰ and for some specific conditions such as articular process osteoarthritis, CT appears to be more reliable than MR imaging.³¹

Compared with MR imaging, CT myelography is invasive, is associated with radiation exposure and seizures, and does not allow assessment of intramedullary lesions. CT myelography requires less time than MR imaging.

Postmyelographic seizures occurred in 24.1% of dogs. Others have reported an incidence of 25% to 42.8% in dogs with cervical spondylomyelopathy.^{11,32,33} Dogs with cervical spondylomyelopathy undergoing CT myelography are predisposed to a higher risk of postmyelographic seizures because of their large body size, injection of contrast medium via the cerebellomedullary cistern, the large volume of contrast medium used, and presence of cervical disease.³²⁻³⁴ It is the total volume of contrast medium, not the dose per kilogram, that is important as a cause of seizures.^{33,34} Dosing contrast medium based on body weight leads to a very large volume in large and giant dogs and this increases the risk of seizures.^{33,34} Fortunately, the volume of contrast medium required for CT myelography is lower than required for conventional myelography.

We conclude that multiple compressive sites caused by osseous lesions are common in giant-breed dogs and that an important number of these dogs have lesions in the cervicothoracic and cranial thoracic regions that need to be considered when planning imaging studies. Spinal cord atrophy was seen in almost 20% of dogs, and more work is needed to determine whether this is associated with a worse outcome.

REFERENCES

1. da Costa RC. Cervical spondylomyelopathy (wobbler syndrome). *Vet Clin North Am Small Anim Pract* 2010;40:881-893.
2. Seim HB. Diagnosis and treatment of cervical vertebral instability-malformation syndromes. In: Bonagura JD (ed): *Current veterinary therapy: small animal practice*, 13th ed. Philadelphia: Saunders, 2000;992-1000.
3. Burbidge HM, Pfeiffer DU, Blair HT. Canine wobbler syndrome: a study of the Doberman pinscher in New Zealand. *N Z Vet J* 1994;42:221-228.
4. VanGundy TE. Disc-associated wobbler syndrome in the Doberman pinscher. *Vet Clin North Am Small Anim Pract* 1988;18:667-696.
5. da Costa RC, Parent JM, Partlow G, et al. Morphologic and morphometric magnetic resonance imaging features of Doberman pinscher dogs with and without clinical signs of cervical spondylomyelopathy. *Am J Vet Res* 2006;67:1601-1612.
6. da Costa RC. Pathogenesis of cervical spondylomyelopathy: lessons from recent years, in Proceedings of the 25th Annual Meet ACVIM Forum, Seattle, WA. 2007;318-320.
7. De Decker S, Gielen IM, Duchateau L, et al. Intraobserver and interobserver agreement for results of low-field magnetic resonance imaging in dogs with and without clinical signs of disk-associated wobbler syndrome. *J Am Vet Med Assoc* 2011;238:74-80.

8. Seim HB, Withrow SJ. Pathophysiology and diagnosis of caudal cervical spondylo-myelopathy with emphasis on the Doberman pinscher. *J Am Anim Hosp Assoc* 1982;18:241–251.
9. Sharp NJH, Wheeler SJ, Cofone M. Radiological evaluation of ‘wobbler’ syndrome – caudal cervical spondylomyelopathy. *J Small Anim Pract* 1992;33:491–499.
10. Lipsitz D, Levitski RE, Chauvet AE, et al. Magnetic resonance imaging features of cervical stenotic myelopathy in 21 dogs. *Vet Radiol Ultrasound* 2001;42:20–27.
11. da Costa RC, Parent JP, Dobson H, et al. Comparison of magnetic resonance imaging and myelography in 18 Doberman pinscher dogs with cervical spondylomyelopathy. *Vet Radiol Ultrasound* 2006;47:523–531.
12. Sharp NJH, Cofone M, Robertson ID, et al. Computed tomography in the evaluation of caudal cervical spondylomyelopathy of the Doberman pinscher. *Vet Radiol Ultrasound* 1995;36:100–108.
13. Massicotte C, Jones JC, Newman S, et al. Wobbler syndrome due to cervical stenosis in a Great Dane puppy. *Can Pract* 1999;24:18–21.
14. De Risio L, Munana K, Murray M, et al. Dorsal laminectomy for caudal cervical spondylomyelopathy: postoperative recovery and long-term follow-up in 20 dogs. *Vet Surg* 2002;31:418–427.
15. Bergman RL, Levine JM, Coates JR, et al. Cervical spinal locking plate in combination with cortical ring allograft for a one level fusion in dogs with cervical spondylotic myelopathy. *Vet Surg* 2008;37:530–536.
16. Sharp NJH, Wheeler SJ. Cervical spondylomyelopathy. In: Sharp NJH, Wheeler SJ (eds): *Small animal spinal disorders diagnosis and surgery*, 2nd ed. Philadelphia: Elsevier Mosby, 2005;211–246.
17. Galano H, Olby NJ, Sharp NJ, et al. Long-term effect of cervical fusion on neurological status and vertebral canal diameter in giant breed dogs with cervical stenotic myelopathy. *J Vet Int Med* 2005;19:419.
18. da Costa RC, Parent JM. One-year clinical and magnetic resonance imaging follow-up of Doberman pinschers with cervical spondylomyelopathy treated medically or surgically. *J Am Vet Med Assoc* 2007;231:243–250.
19. Stalin CE, Pratt JN, Smith PM, et al. Thoracic stenosis causing lateral compression of the spinal cord in two immature Dogues de Bordeaux. *Vet Comp Orthop Traumatol* 2009;22:59–62.
20. Lewis DG. Cervical spondylomyelopathy (‘wobbler’ syndrome) in the dog: a study based on 224 cases. *J Small Anim Pract* 1989;30:657–665.
21. Denny HR, Gibbs C, Gaskell CJ. Cervical spondylopathy in the dog—a review of thirty-five cases. *J Small Anim Pract* 1977;18:117–132.
22. Trotter EJ, de Lahunta A, Geary JC, et al. Caudal cervical vertebral malformation-malarticulation in Great Danes and Doberman pinschers. *J Am Vet Med Assoc* 1976;168:917–930.
23. McKee WM, Butterworth SJ, Scott HW. Management of cervical spondylopathy-associated intervertebral disc protrusions using metal washers in 78 dogs. *J Small Anim Pract* 1999;40:465–472.
24. da Costa RC, Parent JM, Holmberg DL, et al. Outcome of medical and surgical treatment in dogs with cervical spondylomyelopathy: 104 cases (1988–2004). *J Am Vet Med Assoc* 2008;233:1284–1290.
25. Chambers JN, Betts CW. Caudal cervical spondylopathy in the dog: a review of 20 cases and the literature. *J Am Anim Hosp Assoc* 1977;13:571–576.
26. Badami JP, Norman D, Barbaro NM, et al. Metrizamide CT myelography in cervical myelopathy and radiculopathy: correlation with conventional myelography and surgical findings. *AJR Am J Roentgenol* 1985;144:675–680.
27. Levitski RE, Chauvet AE, Lipsitz D. Cervical myelopathy associated with extradural synovial cysts in 4 dogs. *J Vet Intern Med* 1999;13:181–186.
28. Dickinson PJ, Sturges BK, Berry WL, et al. Extradural spinal synovial cysts in nine dogs. *J Small Anim Pract* 2001;42:502–509.
29. Naganawa T, Miyamoto K, Ogura H, et al. Comparison of magnetic resonance imaging and computed tomogram-myelography for evaluation of cross sections of cervical spinal morphology. *Spine* 2011;36:50–56.
30. Shafaie FF, Wippold FJ 2, Gado M, et al. Comparison of computed tomography myelography and magnetic resonance imaging in the evaluation of cervical spondylotic myelopathy and radiculopathy. *Spine* 1999;24:1781–1785.
31. Lehman RA Jr, Helgeson MD, Keeler KA, et al. Comparison of magnetic resonance imaging and computed tomography in predicting facet arthrosis in the cervical spine. *Spine* 2009;34:65–68.
32. Lewis DD, Hosgood G. Complications associated with the use of iohexol for myelography of the cervical vertebral column in dogs: 66 cases (1988–1990). *J Am Vet Med Assoc* 1992;200:1381–1384.
33. da Costa RC, Parent JM, Dobson H. Incidence of and risk factors for seizures after myelography performed with iohexol in dogs: 503 cases. *J Am Vet Med Assoc* 2011;238:1296–1300.
34. Barone G, Ziemer LS, Shofer FS, et al. Risk factors associated with development of seizures after use of iohexol for myelography in dogs: 182 cases (1998). *J Am Vet Med Assoc* 2002;220:1499–1502.