Constitutional thoracic vertebral body malformations are common in brachycephalic dogs with a screw tail (defined by the American Kennel Club as “a naturally short tail twisted in more or less spiral formation”). Although some controversy exists surrounding the appropriate terminology for these anomalies, hemivertebrae are considered the most prevalent type of malformation. The reported prevalence of hemivertebrae in screw-tail brachycephalic dogs ranges from 78% to 94%, depending on the breed and source population. 

Hemivertebrae result from an abnormal, uneven growth between the 2 halves of a vertebral body during embryological development. This can result in incomplete fusion between the 2 halves, resulting in a wedge-shaped vertebral body. The base of wedge-shaped vertebral bodies can be orientated dorsally, ventrally, or laterally with resultant angular deformity of the vertebral column. Kyphosis refers to an abnormal ventrodorsal angulation of the vertebral column, whereas scoliosis refers to a laterolateral angulation.

Most thoracic vertebral body malformations are associated with no clinical signs and should therefore be considered an incidental finding on imaging of the vertebral column.

However, in a small number of dogs, these malformations can result in neurologic abnormalities and spinal cord dysfunction. Most dogs with clinically relevant constitutional vertebral body malformations have progressive pelvic limb ataxia and paralysis. Although age at the time of initial evaluation for clinical signs varies widely, most dogs are < 1 year of age at that point. Because clinical signs associated with thoracic vertebral body malformations have the potential for progression, several surgical techniques have been developed with the aim of providing spinal stabilization with or without decompression. Surgical intervention is indicated when neurologic deficits are present or worsening. 

### OBJECTIVE
To characterize outcomes following nonsurgical treatment of constitutional thoracic vertebral body malformations causing neurologic deficits in dogs.

### DESIGN
Retrospective case series.

### ANIMALS
13 client-owned dogs treated nonsurgically for constitutional thoracic vertebral body malformations at 3 veterinary referral hospitals from June 2009 through May 2016.

### PROCEDURES
Data were extracted from the medical records regarding dog signalment, duration and type of clinical signs before referral, general physical and neurologic examination findings, radiographic and MRI findings, and treatments provided after diagnosis. Follow-up data were obtained from records of recheck examinations and via a standardized owner questionnaire.

### RESULTS
All included dogs were screw-tail brachycephalic breeds with a median age of 6 months. All dogs had ambulatory paraparesis and ataxia, and in 1 dog, signs of spinal hyperesthesia could be elicited. Nonsurgical treatments consisted of restricted exercise without (n = 5) or with (3) physiotherapy, physiotherapy without restricted exercise (3), and no exercise modification (2). Seven dogs received additional nonsurgical treatment consisting of prednisolone (n = 5) or gabapentin (2). Four dogs were eventually euthanized because of progressive neurologic deterioration, 2 underwent surgery for the same reason, and the remaining 7 dogs survived for ≥ 170 days after diagnosis, despite progressive neurologic deterioration.

### CONCLUSIONS AND CLINICAL RELEVANCE
Nonsurgical treatment of constitutional thoracic vertebral body malformations was associated with an unfavorable outcome in this group of dogs. Despite this treatment, all dogs had progression of clinical signs.
is not without risk, however, and these procedures can be invasive and technically demanding.\textsuperscript{8,13}

In contrast, anecdotal reports\textsuperscript{8,13} suggest that mild clinical signs associated with congenital thoracic congenital vertebral body malformations may stabilize or even improve when dogs become skeletally mature. Consequently, some authors recommend delaying surgical intervention until vertebral growth is complete (approx 9 months of age).\textsuperscript{8,9,13} Although such recommendations may support consideration of medical management for certain patients, no studies have been conducted to specifically evaluate the results of nonsurgical treatment of congenital thoracic vertebral body malformations in dogs.

The purpose of the study reported here was to determine outcomes of nonsurgical treatment in dogs with clinically relevant, congenital thoracic vertebral body malformations. We anticipated that nonsurgical treatment would provide a fair prognosis and that no progression of clinical signs would be observed in dogs with only mild clinical signs.

\section*{Materials and Methods}

\subsection*{Case selection criteria}

The electronic medical records of 3 large veterinary referral hospitals were searched from June 1, 2009, through May 31, 2016, to identify dogs that received nonsurgical treatment for congenital thoracic vertebral body malformations. Dogs were included in the study when they had at least 1 thoracic vertebral body malformation with associated neurologic dysfunction. Dogs were excluded when medical records or MRI scans were incomplete or unavailable for review or dogs had concurrent orthopedic or neurologic disorders that could interfere with their clinical assessment or outcome. All medical records and MRI scans were reviewed by 2 investigators (SW and SD [a board-certified veterinary neurologist]) to determine study eligibility. The study protocol was approved by the Royal Veterinary College Clinical Research Ethical Review Board (protocol No. URN 20161463).

\subsection*{Medical records review}

Information was retrieved from the medical records regarding dog signalment at the time of initial referral evaluation, duration and type of clinical signs before referral, general physical and neurologic examination findings, diagnostic test results (including those of diagnostic imaging), and treatments provided after diagnosis. Neurologic deficits were scored by a study investigator (SW) in accordance with the MFS system\textsuperscript{16} as follows: 0, paraplegia with no deep nociception; 1, paraplegia with no superficial nociception; 2, paraplegia with nociception; 3, nonambulatory paraparesis; 4, ambulatory paraparesis and ataxia; 5, spinal hyperesthesia only; and 6, no dysfunction.

For each dog, a diagnosis of a thoracic vertebral body malformation had been made by a high-field MRI of the vertebral column, with the dog anesthetized. Although protocols and sequences varied among hospitals, all MRI examinations included a minimum of T1- and T2-weighted sagittal and transverse images. For selected dogs, MRI examination was combined with spinal radiography. Radiography was also performed with the dog anesthetized, and orthogonal views and images of the complete thoracic and lumbar vertebral column were included. All acquired images were evaluated for study purposes by use of a DICOM viewer.\textsuperscript{9} One investigator (RGQ) evaluated the degree of spinal kyphosis on MRI images by measurement of the modified Cobb angle with the aid of commercially available DICOM software\textsuperscript{17} as described elsewhere.\textsuperscript{17} When multiple hemivertebral were present, the vertebral segment with the greatest degree of spinal angulation was chosen for measurement. For dogs that received repeated MRI examinations after initial diagnosis, Cobb angle measurements were also repeated to evaluate any progression of spinal curvature.

\subsection*{Follow-up data collection}

Short-term follow-up information on dog status 4 to 6 weeks after the diagnosis of hemivertebra was obtained from the medical records of the participating referral hospitals. Long-term follow-up information was obtained by a study investigator (SW) via telephone interview with referring veterinarians and through access to patient medical records held at referring veterinary practices. For the dogs that had died or had been euthanized, the cause of death and last recorded neurologic status were recorded. All findings from recheck neurologic examinations were scored in accordance with the MFS system.

In compliance with ethics and welfare committee guidelines, follow-up contact with dog owners was performed only when dogs were believed to be alive at the time of data collection. For this purpose, owners were sent a letter explaining the study and a standardized questionnaire. Although the questionnaire was not formally validated for the study, questions were based on previously published owner-perceived quality of life assessments\textsuperscript{18,19} (Supplementary Appendix S1, available at: avmajournals.avma.org/doi/suppl/10.2460/javma.253.6.768). Questions were designed to collect data on various aspects of the disease in each dog, including signs of pain, activity level, lameness, paresis, urinary and fecal continence, types of nonsurgical treatment provided, and response to treatment. For those owners who did not respond to mailed questionnaires within 4 weeks after sending, telephone interviews were conducted by use of the same questionnaire.

\section*{Results}

\subsection*{Animals}

Twenty dogs with nonsurgically treated, clinically relevant, congenital thoracic vertebral body malformations were identified during the study period. Five of these dogs were excluded because of concurrent neurologic or orthopedic disorders that could have
contributed to clinical signs. These disorders included medial patella luxation, intervertebral disk extrusion, and syringomyelia. Another 2 dogs were excluded owing to a lack of complete follow-up data, leaving 13 dogs for inclusion in the study.

Included dogs were a median of 6 months of age at the initial referral evaluation (range, 4 months to 94 months). Breeds included Pug (n = 9), French Bulldog (3), and English Bulldog (1). Eight dogs were male (7 sexually intact and 1 neutered), and 5 were female (2 sexually intact and 3 spayed).

Clinical findings

Duration of clinical signs prior to referral evaluation ranged from 2 to 987 days (median, 35 days; mean, 120 days) and consisted of progressive, ambulatory paraparesis and pelvic limb ataxia in all dogs. All dogs were scored as having an MFS of 4 (ambulatory paraparesis and ataxia) at the time of diagnosis. Signs of hyperesthesia could be elicited in 1 dog.

All dogs had ≥1 thoracic vertebral body malformation identified on MRI, and these malformations were associated with kyphosis in 11 dogs. The number of abnormal vertebra ranged from 1 to 8 (median, 2), affecting the vertebral column from T3 through T13. The modified Cobb angle ranged from 3° to 86° (median, 43°; Figure 1). The 2 dogs with no kyphosis had absolute stenosis of the vertebral canal.

Repeated MRI examinations were performed for 2 dogs at 18 and 3 months following initial diagnosis, revealing progression of the Cobb angle by 4° and 11°, respectively. This imaging had been performed because these dogs had progression of clinical signs.

Treatment

Nonsurgical treatment consisted of exercise restriction for 8 dogs, and this approach was combined with physiotherapy or hydrotherapy for 3 of these dogs. Physiotherapy or hydrotherapy without exercise restriction was provided for another 3 dogs. No exercise modification was advised for the remaining 2 dogs. Seven dogs received additional nonsurgical treatment, consisting of a tapering course of prednisolone (initial dose, 0.5 to 1.0 mg/kg [0.23 to 0.45 mg/lb], PO, q 12 h; n = 5) and gabapentin (10 mg/kg [4.5 mg/lb], PO, q 8 h; 2).

Outcome

Two dogs were euthanized 7 or 9 days after discharge from the hospital because of neurologic deterioration. Both dogs had progression to more severe ambulatory paraparesis with ataxia, although the MFS for both dogs remained 4 at the time of euthanasia. One dog underwent surgical intervention 7 days after discharge because of neurologic deterioration characterized by a progression in the severity of ataxia and paresis; the MFS at the time of surgery was 4. Although this dog had postoperative complications related to wound infection, evidence of neurologic improvement was noted for up to 6 months after surgery. Despite this, the dog remained moderately paraparetic and ataxic. All 10 remaining dogs had various degrees of neurologic deterioration detected during their 4 to 6-week recheck examination. This deterioration was largely characterized by loss of fecal or urinary continence or both with progression of ataxia and paresis, although again the MFS remained 4 for all dogs.

Duration of long-term follow-up following initial diagnosis of thoracic vertebral body malformation for the remaining 10 dogs ranged from 55 to 1,730 days (median, 518 days). Two dogs were euthanized because of progressive neurologic deterioration 200 and 559 days after initial diagnosis. The MFS had progressed to 3 (nonambulatory paraparesis) at the time of euthanasia in both dogs, which had complete loss of fecal and urinary continence. Another dog underwent surgical intervention 85 days after initial diagnosis because of an unsatisfactory response to nonsurgical treatment and neurologic deterioration. Although this dog developed postoperative complications related to implant infection, evidence of neurologic improvement was observed for up to 10 months after surgery. Despite this improvement, the dog remained severely paraparetic and ataxic with an MFS of 4 and partial urinary and fecal incontinence. Another dog underwent surgical intervention 85 days after initial diagnosis because of an unsatisfactory response to nonsurgical treatment and neurologic deterioration. Although this dog developed postoperative complications related to implant infection, evidence of neurologic improvement was observed for up to 10 months after surgery. Despite this improvement, the dog remained severely paraparetic and ataxic with an MFS of 4 and partial urinary and fecal incontinence. The remaining 7 dogs had progressive pelvic limb ataxia and paraparesis to various degrees. In 4 of these dogs, the clinical signs appeared to stabilize after an initial period of neurologic deterioration, 14 to 330 days after discharge from the hospital. Median age at stabilization was 1 year and 8 months. The MFS remained 4 in all 7 surviving dogs despite obvious deterioration of clinical signs.

The owners of the 7 dogs that remained alive at the time the study was performed (ie, ≥170 days after diagnosis) consented to respond to the follow-up
questionnaire. All owners indicated that they perceived their dogs as having a reasonable quality of life as suggested by their apparent mentation, freedom from pain, and enthusiasm to interact and play. Despite this perception, 6 dogs reportedly needed assistance with ambulation, 1 had complete fecal incontinence, 1 had partial urinary incontinence, and 1 had partial urinary and fecal incontinence.

Discussion

Overall, of the 13 dogs treated nonsurgically for clinically relevant congenital thoracic vertebral malformations in the present study, 4 were euthanized because of progressive neurologic deterioration, 2 underwent surgical intervention owing to progressive neurologic deterioration, and 7 survived to the time of writing, despite some degree of neurologic deterioration since the initial referral evaluation. Prior to this study, no information was available regarding nonsurgical treatment for such conditions. The signalment and clinical signs of included dogs were similar to those in other studies. Many dogs (8/13) were < 1 year of age, and most (11/13) had a chronic, progressive, nonpainful, ambulatory paraparesis and ataxia of the pelvic limbs. All dogs were screw-tail brachycephalic breeds.

Although we anticipated that nonsurgical treatment would provide a fair prognosis and that no progression of clinical signs would be observed in dogs with only mild clinical signs (once they became skeletally mature [i.e., approx 9 months of age]), all included dogs deteriorated despite treatment. For dogs that had eventual stabilization of clinical signs, this stabilization occurred at > 9 months of age. Although the generalizability of our findings is limited owing to the small number of included dogs, we believe the findings suggest that nonsurgical treatment of congenital thoracic vertebral body malformations does not provide a good prognosis and that stabilization of clinical signs does not often occur at skeletal maturity. Despite inclusion of patients from 3 large referral hospitals over a 7-year period, only 13 dogs met the inclusion criteria for the study. This small number could be considered an important limitation; however, it also reflected the general rarity of thoracic vertebral body malformations as the primary cause of spinal cord dysfunction in dogs with no contributing conditions. Although congenital vertebral malformations are commonly recognized on imaging of screw-tail brachycephalic breeds, they only rarely result in clinical signs, and 1 study performed at one of the referral hospitals that participated in the present study, thoracic hemivertebrae were identified in 93.5% of French Bulldogs, 17.6% of Pugs, and 73.2% of English Bulldogs that underwent thoracic CT for reasons unrelated to spinal disease. Interestingly, compared with the breed distribution in the entire patient population during the period in which the study was conducted, only 0.95% of all French Bulldogs, 4.7% of all Pugs, and not a single English Bulldog received a diagnosis of thoracic vertebral body malformation as the cause of their clinical signs. This information suggests that a diagnosis of clinically relevant vertebral malformations should only be made after more common spinal cord conditions have been excluded.

Clinically relevant vertebral body malformations associated with kyphosis have been reported in the human medical literature. In contrast to suggestions in the veterinary medical literature,9 careful monitoring and early surgical intervention before skeletal maturity is recommended for affected humans because of the potential for clinical progression. Interestingly, the 2 dogs in the present study that underwent surgical intervention because of an insufficient response to nonsurgical treatment had clinical improvement following surgery. Because these dogs continued to have neurologic deficits, we could not exclude the possibility that a greater extent of neurologic improvement would have been obtained if surgery had been performed when the dogs were younger. Although the purpose of the study was not to compare nonsurgical versus surgical treatment of clinically relevant thoracic vertebral malformations, such comparisons would be important to explore and improve treatment options and long-term outcome for affected dogs.

The reason all dogs in the study reported here had progression of clinical signs remains unclear. However, the fact that all dogs had been referred to tertiary care centers could have introduced selection bias toward inclusion of dogs that were more severely affected than those treated in primary care hospitals. Dogs with mild and nonprogressive gait abnormalities may not be referred for specialist diagnostic investigations. Therefore, we are uncertain whether the findings would apply to the general population of dogs receiving nonsurgical treatment for clinically relevant thoracic vertebral malformations. This uncertainty is further complicated by the uncertain pathophysiologic nature of congenital thoracic vertebral body malformations. More specifically, the reason such malformations only rarely result in clinical signs is unknown, as are the mechanisms responsible for development of clinical disease.

The development of clinical signs in dogs with thoracic vertebral body malformations is likely influenced by many variables, with both dynamic and static factors involved. Vertebral instability, vertebral canal stenosis, subluxation, and kyphosis may contribute to repetitive and progressive spinal cord injury. Some investigators have suggested that the degree of vertebral kyphosis should be considered a key factor in the development of clinical signs. More specifically, the degree of vertebral kyphosis has been suggested to exceed a critical threshold value of 35° before clinical signs are likely to occur. Although most dogs included in the present study had spinal kyphosis angles that exceeded this threshold value, 2 dogs had no abnormal vertebral curvature (Figure 1). These findings...
supported the supposition that the development of clinical signs in dogs with thoracic vertebral malformations is multifactorial, and this multifactorial nature further complicates development of treatment guidelines for dogs with congenital thoracic vertebral body malformations.

Several surgical techniques have been proposed for the treatment of dogs with thoracic vertebral malformations, and these techniques typically consist of vertebral stabilization with or without additional spinal cord decompression. Although vertebral stabilization without spinal cord decompression has resulted in good outcomes for dogs with severe spinal kyphosis, this technique may not be indicated for dogs with only moderate or minimal spinal kyphosis.

Several dogs in the study developed clinical signs when > 1 year of age, and 2 dogs had progression of vertebral angulation detected when MRI examination of the vertebral column was repeated following neurologic deterioration. Late-onset spinal kyphosis in a Pug has been reported. Screening radiography of the vertebral column was initially performed when the Pug was 2 months old, revealing no vertebral anomalies; however, when 6 months old, the Pug developed pelvic limb ataxia and paresis. Repeated imaging of that dog revealed hemivertebra and severe kyphosis of the thoracic portion of the vertebral column. Growth plate abnormalities, which only became apparent during a period of rapid growth, were suggested as a potential cause of this late development. The clinical relevance and frequency of progression of spinal kyphosis remains unknown for dogs with clinically relevant thoracic vertebral body malformations.

Despite all dogs in the present study having had progression of neurologic signs, owners who completed the follow-up questionnaire perceived their dogs as having a reasonable quality of life. However, all but 1 surviving dog needed assistance with ambulation, had various degrees of urinary or fecal incontinence, or needed assistance and had incontinence. This discrepancy between owner perception and clinical outcome was interesting and emphasized the challenges associated with evaluation of veterinary patients with spinal cord disease. Veterinary professionals often focus on recognizable features, such as motor function, ambulatory status, sensory function, and spinal hyperesthesia to predict outcome. However, human patients with chronic and severe spinal dysfunction can have a good quality of life. Poor functional outcome scores are therefore not necessarily correlated with poor quality of life scores and may not always reflect the complete spectrum of outcome measures. Furthermore, owner-based quality of life assessments are largely subjective and vulnerable to bias.

Bias could have existed in questionnaire responses if owners caring for dogs with severe neurologic dysfunction did not perceive their dogs as having a poor quality of life because such perception would mean they had not made the right decision for their beloved pet or that allowing the dog to continue with a poor quality of life would be considered ethically and socially unacceptable. Social desirability bias is the unconscious enhancement of the tendency to overestimate a socially favorable situation, while underreporting undesirable behavior. Objective evaluation of outcome in the present study was further complicated by the difficulties inherent to grading the severity of neurologic deficits in dogs with chronic spinal disorders. Although an accepted and validated neurologic grading system was used, all dogs were ambulatory at the initial referral evaluation and were therefore assigned the same neurologic grade. Similarly, although all dogs had obvious progression of clinical signs, this was not always reflected by progression to a more severe neurologic grade. It is important to realize that most validated grading systems for dogs with spinal disease were designed for dogs with acute-onset thoracolumbar spinal disorders, and only a few were designed for ambulatory dogs or dogs with chronic thoracolumbar spinal disease.

In addition to the aforementioned limitations to the present study, the reasons that owners chose nonsurgical versus surgical treatment were unknown. Consequently, the possibility remained that factors such as severity of neurologic signs, lack of owner commitment to surgical intervention, and severity of vertebral malformations that were nonamenable to surgery influenced this choice. Furthermore, the retrospective nature of the study precluded standardized assessment and treatment of included dogs; although exercise restriction was recommended for some dogs, other dogs received physical therapy without exercise restriction. The small number of included dogs precluded comparison of responses to various exercise protocols.

Despite these limitations, we believe the results of the study reported here provide new, potentially important information about the effectiveness of nonsurgical treatment of thoracic congenital vertebral body malformations in dogs. Nonsurgical treatment was associated with an unfavorable outcome: all 13 dogs had progression of clinical signs. Additional research is necessary to provide insight into the pathophysiologic nature of thoracic vertebral malformations and to support development of appropriate treatment recommendations. Given the multifactorial nature of thoracic vertebral malformations in dogs, a case-by-case approach may be ultimately found to provide the optimal treatment option.

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Footnotes

a. OsiriX imaging software, version 3.9.2, Pimexo SARL, Berne, Switzerland.

References


