Neurological and radiological evaluation of the lumbosacral junction in 33 working German Shepherd dogs : a prospective study over 3 years

Hunold, Katharina

Departement für Kleintiere der Vetsuisse-Fakultät Universität Zürich
Klinik für Kleintierchirurgie
(Direktor: Prof. Dr. P.M. Montavon)

Arbeit unter der Leitung von Dr. F. Steffen und Prof. Dr. M. Flückiger

Neurological and radiological evaluation of the lumbosacral junction in 33 working German Shepherd dogs: a prospective study over 3 years

INAUGURAL–DISSERTATION

Zur Erlangung der Doktorwürde der
Vetsuisse–Fakultät Universität Zürich

Vorgelegt von
Katharina Hunold
Tierärztin aus Glarus

Genehmigt auf Antrag von
Prof. Dr. M. Flückiger (Referent)
PD Dr. M. Hässig (Korreferent)

Zürich 2006

Table of Contents

Neurological and radiological evaluation of the lumbosacral junction in 33 working German Shepherd dogs: a prospective study over 3 years

Neurological and Radiological evaluation of the lumbosacral junction in 33 working German Shepherd Dogs: a prospective study

Katharina Hunold, Frank Steffen, Gernot Scharf, Malgorzata Roos and Mark Flückiger

From the Section of Small Animal Surgery/Neurology (Steffen, Hunold), the Section of Diagnostic Imaging and Radiation Oncology (Scharf, Flückiger) and the Departement of Biostatistics, ISPM (Roos), University of Zürich, Switzerland

Summary

Degenerative lumbosacral stenosis (DLSS) is a common disorder in working German Shepherd Dogs (GSD) and concerned owners increasingly ask for early diagnostic and preventive measures. In this study, a total of 60 randomly selected German Shepherd Dogs (GSD) working as Police dogs were available for a prospective evaluation of their lumbosacral articulation during a 3 to 3.5 year period. Goals of this study included the evaluation of the correlation between clinical and radiological findings at the lumbosacral junction, and to assess progression of clinical and radiological signs of degenerative lumbosacral stenosis. 33 dogs were shown a second time after 3 to 3.5 years and therefore entered the study. All dogs underwent a physical, neurological and orthopedic examination and survey radiographs of the lumbosacral junction were taken at the beginning and the end of the study. On the final examination neurological signs of lumbosacral disease were found in 15 dogs while 18 dogs were normal. 22 dogs were able to perform unrestricted duty and 4 dogs were used for restricted duty including 3 dogs with suspected DLSS. The reason for the restriction was DLSS in 3 dogs. 7 dogs had been excluded from active duty during the period of surveillance, 6 because of DLSS. Of the 15 clinically affected dogs, 13 showed radiological abnormalities in the lumbosacral area. Of the 18 dogs without clinical symptoms, 14 showed radiographic abnormalities of their lumbosacral junction. The progression of abnormalities of clinical and radiological parameters was statistically significant, but statistical correlation analysis showed no association between clinical and radiological findings. In conclusion, plain radiography did not allow to predict development of DLSS. Mildly to moderately affected GSDs may be used as working police dogs for months to even years.

Introduction

Cauda equina syndrome (CES) comprises a complex of neurological signs caused by a lesion of the nerve roots in the lumbosacral area[1,2,3,4]. Degenerative lumbosacral stenosis (DLSS) is the most common aetiology and its most consistent sign is lumbosacral pain [4,5]. DLSS often affects male working dogs of older age and is noted more commonly in German Shepherd Dogs (GSD) than in other breeds [6,7,8] . An early diagnosis is warranted as affected working dogs often have to quit duty untimely after expensive and time-consuming training [9,10] .

To date, radiological signs indicating a future risk for the development of DLSS are sparse and unreliable. They include lumbosacral transitional vertebrae [11,12,13,14] and OCD of the sacral or L7 endplate [15,16]. Asymmetry of the facet joints has been suspected to be associated with DLSS in GSDs, and supporting clinical data have been published [17, 18]. However, the definitive role of the orientation and shape of the articular process joints and their association with DLSS have not been proven so far.

The purpose of this study was to evaluate the correlation between clinical and radiological findings at the lumbosacral junction and to assess progression of clinical and radiological signs of DLSS, if present, over several years in a homogenous population of working GSDs. Radiographic parameters that could potentially predict development of DLSS were analyzed and causes for premature drop out of dogs were analyzed.

Materials and Methods

Of 60 working GSD intially examined 33 were available for reevaluation of their lumbosacral region 3 to 3.5 years after a first anamnestic, neurological and radiographic examination[10]. All dogs had been working at a Swiss police unit during this period.

Prior to the examination, the owners answered a questionnaire for information about the general health status of their dog and any concurring disorders diagnosed by their veterinarian. With respect to potential lumbosacral disease, owners were specifically asked to judge their dogs' ability to perform unrestricted duties. Also owners were expected to mention signs of pain, lameness, weakness in the pelvic limbs and/or tail including the occasion of ocurrence.

Each dog underwent a neurological examination by a board certified neurologist focused on careful assessment of gait, posture, anal and tail tone, quality of conscious proprioception, and spinal reflexes (patellar, cranial tibial and withdrawal reflex). Lumbosacral hyperesthesia was evaluated by spinal deep palpation, lumbosacral hyperextension, and hyperextending the tail. To be considered as suspicious, dogs had to be tested positive on the neurological examination with reproducible lumbosacral pain being the minimal requirement. A presumptive diagnosis of DLSS was made after exclusion of orthopaedic or other neurological disorders that might mimck the disease.

For the radiographic evaluation each dog was sedated with intravenous application of 5-10 µg/kg Medetomidine[a] and Propofol[b] to effect. Medetomidine was antagonised with 5-10 µg/kg Atipamezol[c] intravenously after the examination.

Two non-contrast radiographs[d] centred at the lumbosacral region were taken, one taken with the dog in lateral recumbency with the femora perpendicular to the spine and one with the dog in dorsal recumbency with the stifles abduced and the X-ray beam angled

cranially by 7-10º and thus parallel to the lumbosacral endplates [10]. The radiographs were compared to those taken 3 years ago and reviewed for the same radiological parameters [10]. The dorsal and ventral lumbosacral angle was determined by two methods and any lumbosacral malalignment was registered [19, 20]. Radiographs were assessed for evidence of lumbosacral transitional vertebrae, severity of spondylarthrosis, and ventral and lateral spondylosis deformans, specifically noting any progression. Degree of spondylosis was grouped in mild, moderate, and severe [21]. The intervertebral space was assessed for signs of mineralization of the nucleus and for narrowing. The height of the spinal canal was measured at the level of L7 and S1, the height of the body of L7 at two sites (centre and endplate site), the width of L7 and S1 endplates axially, and dorsal flattening of S1. Shape and contour of S1 were evaluated. Presence of mineralization, bony fragments, and lysis within the spinal canal were noted.

For statistical evaluation, the width of the LS intervertebral disc space was normalized by dividing it by the height of the body of L7.

Data were statistically analysed using StatView Vers 5. Descriptive statistics (mean, SD, median, Q1, Q3) together with boxplots were provided. The Wilcoxon Signed Rank Test was used to estimate progression of radiological and clinical parameters with time within the time period. A multiple logistic regression analysis (SPSS Vers. 10) was performed to assess associations of the various radiographic and neurologic parameters with DLSS. Forward and backward logistic regressions were used in order to identify the most important radiological signs that are associated with a later development of DLSS.

Dorsal and ventral lumbosacral angles were not statistically evaluated because the dogs' positioning was not standardized during radiographic examination resulting in highly variable data.

Results

Of the original 60 dogs investigated three years earlier 15 had died in the meantime. The causes of death were variable but not associated with DLSS. Another 12 dogs were lost or not available to follow-up, leaving 33 dogs for reevaluation. Their mean age was 7.0 years (4 to 11 years), 5 were females (4 intact, 1 neutered) and 28 males (all intact).
In the questionnaire, 13 owners reported problems with a potential association with a lumbosacral disorder (39%). Ten dogs (30%) had difficulties jumping. Seven (21%) showed signs of lower back pain and 11 (33%) had a mild lameness/weakness in the pelvic limbs.
Twenty-two dogs were able to perform unrestricted duty and 4 dogs were used for restricted duty. The reason for the restriction was DLSS in 3 dogs and thoracolumbar disc disease in one dog. Seven dogs had been excluded from active duty during the period of surveillance, 6 because of DLSS and one due to a non-medical reason.

Several musculoskeletal pathologies not related to the lumbosacral joint were diagnosed: Fibrosis of the semitendinosus muscle (2 dogs), thoracolumbar disc disease (1 dog), rupture of the cranial cruciate ligament (2 dogs), forelimb lameness due to elbow arthrosis (4 dogs) and pelvic limb lameness due to hip dysplasia (7 dogs; mild in 3 dogs, moderate in 2, and severe in 2).
Dogs with clinical signs of DLSS during the last 3 years had received various forms of medical or surgical treatment by their veterinarians. NSAIDs had been given repeatedly to 3 dogs during painful episodes; in 2 of them with good temporary relief, in one dog without effect. Another dog had received NSAIDs on a daily basis with a good clinical

effect. One dog had been treated with a long acting steroid every third month with satisfactory clinical results.

Surgical decompression of the cauda equina and nerve roots had been performed in two dogs using standard dorsal laminectomy. The outcome in one was excellent initially, but at presentation, the dog's performance was restricted. The other dog was euthanatized 6 months after surgery because of poorly specified reasons but not related to DLSS.

At the time of the final examination 18 dogs were free of clinical signs of lumbosacral disease, while 15 dogs showed clinical signs of DLSS . All affected dogs exhibited lower back pain elicited by digital palpation of the lumbosacral area and hyperextension of the lumbosacral junction. Additional signs in 8 dogs included abnormalities in gait, posture and mild neurological deficits including proprioceptive deficits in 5 dogs.

Of the 15 clinically affected dogs, 13 showed radiological abnormalities in the lumbosacral area. Of the 18 dogs without clinical symptoms, 14 showed radiological LS-abnormalities. Radiographic abnormalities included mineralization of the intervertbral disc (n=12), sclerosis of the lumbosacral endplates (n=4), spondylosis deformans (n=25) and mineralisation within the spinal canal (n=7). Also, lumbosacral transitional vertebrae were noted in 2 dogs and mild lumbosacral malalignment in 16 dogs.

Compression of the cauda equina was confirmed by epidurography, computed tomography, myelography, magnetic resonance imaging or surgery in 9 dogs.

No associations between clinical and radiological findings were found ($p > 0.05$).

The progression of various clinical and radiological parameters within the time period was analysed. The following clinical signs showed significant progression: gait

abnormalities, conscious proprioceptive deficits in the pelvic limbs, and pain on palpation of the lumbosacral area. Spinal reflex quality did not change significantly.

Statistically significant changes in radiological parameters included progression of mineralization of the lumbosacral nucleus, narrowing of the intervertebral space, spondylosis deformans, mineralization within the spinal canal, opacification within the canal of S1 and height of the spinal canal at the level of L7 and S1. No significant changes were found for the following radiological parameters: dorsal flattening of S1, contour of the cranial endplate of S1, width of L7 and S1 endplates, lumbosacral malalignment, spondylarthrosis, and height of the body of L7 at the endplate site (Table 2).

No radiological signs that would predict a later development of DLSS could be identified by multiple logistic regression analysis (Table 3).

Discussion

The most common and earliest finding in dogs with DLSS is pain elicited on palpation and hyperextension of the lumbosacral junction. Although not specific for DLSS this test is easy, sensitive and the positive predictive value is 91-100%[3,4]. The pain arises from compression of the cauda equina and, eventually, the seventh lumbar nerve roots. Other potential sources of pain include the lumbosacral disc or the articular facets. Although DLSS was not proven by evidence via advanced diagnostic imaging in all dogs in this study, the presumptive diagnosis “DLSS” was supported by the results of the repeated neurological examinations and by exclusion of other diseases that may mimick the disease. The diagnosis was confirmed in 9 of 15 clinically affected GSDs with contrast radiography, computed or magnetic resonance tomography.

Radiographic signs suggesting lumbosacral degeneration have been shown to be unreliable for the diagnosis of DLSS in previous reports [1,10]. However, in a recent human study, radiographic parameters were able to distinguish different stages of degeneration, whereas magnetic resonance imaging could only detect advanced stages of degeneration strengthening the role of this modality [23]. Further justifications for the use of plain radiography for screening working dogs for signs of lumbosacral disease included its cost effectiveness, non-invasiveness and its wide availability. In the present study, a significant progression of radiographic signs suggesting lumbosacral degeneration was noted in dogs with clinical signs of DLSS. Overall, radiological signs increased from 60% to 89% within 3 years. However, none of the radiological parameters correlated significantly with clinical signs of DLSS. This finding is supported by the subpopulation of dogs without signs of DLSS that showed a similar proportional increase of radiological LS-degeneration from 50% to 71%. This affirms that survey

radiographs are virtually of no diagnostic value not only in retrospective studies but also on a prospective basis.

Malalignement of the lumbosacral junction, i.e. ventral subluxation of the sacrum relative to the seventh lumbar vertebra deserves special comment. Lumbosacral malalignement has been considered to be a sign of instability by several authors [1,7] ,whereas others found no significant differences between affected and unaffected dogs [20,24]. Schmid and Lang in 1993 found that a ventral subluxation of more than 4 mm was strongly suggestive of an abnormal lumbosacral junction and suggested to follow affected dogs over several years to gain more information about the clinical significance of this finding. In the present study, 16 dogs showed lumbosacral malalignment varying between 1-3 mm. When comparing their current radiographs with those taken 3 years ago, no progression of the malalignment was noted radiologically and this finding was not associated with clinical signs of DLSS.

The association between radiographically apparent spondylosis deformans and clinical manifestation of DLSS is discussed controversially in the veterinary literature. Spondylosis and type II disk disease have been suspected to be associated in a recent study [25] and higher rates of spondylosis at sites of type II disk protrusions have been found [26]. In contrast, our results confirm the finding of Scharf et al. in 2004 that radiographically apparent spondylosis is not consistently associated with clinical signs in the lumbosacral junction. Spondylosis was a common radiographic finding in dogs with DLSS in the present study, but it was neither associated with clinical signs nor did it predict future development of the disease.

The number of dogs with clinical signs of DLSS increased from 37% to 46% within 3 years. Gait abnormalities and conscious proprioceptive deficits progressed statistically

significant in affected dogs reflecting the progressive nature of the disease. Our findings are in accordance with the findings of other reports stating that older dogs are more commonly and more severely affected than younger ones [4,8]. Of the 33 GSDs re-evaluated for the present study, 22 were able to perform unrestricted duty. Four dogs were in restricted duty: 3 dogs due to DLSS and 1 dog due to thoracolumbar disc disease. Six dogs were excluded from duty because of DLSS and one dog because of unrelated motives of the owner. Thus, DLSS was responsible for exclusion or restriction from duty in 9/33 GSDs (27%). In a pathology based study 19.4% of GSDs in military working service were found to be affected by spinal cord and cauda equina disease [9]. The higher incidence in our study may be explained by the fact, that slightly to moderately affected dogs were included. These degrees are unlikely to represent a reason for euthanasia.

Comparing the severity of impairment in DLSS-affected dogs with their ability to perform duty our findings revealed that, interestingly, six dogs with clinical signs of DLSS were able to perform unrestricted duty. Five of them showed only pain during the clinical examination, and only one dog showed advanced neurological deficits. Probably, milder forms of DLSS do not interfere with working capabilities in working GSDs due to their temperament and higher level of pain tolerance.

Our data must be interpreted with some caution as imaging modalities such as epidurography, CT or MRI were not used to demonstrate compression of the cauda equina nerves in all cases. Radiographic diagnosis was based solely on plain radiographs and their role was rather to rule out other diseases than proving presence of DLSS. Despite this presumptive character of the diagnosis it was strengthened by several facts. Non-neurological differential diagnoses of DLSS such as degenerative

joint disease of the hip or stifle could be excluded by repeated physical and radiological examinations. Neurological diseases that mimic DLSS such as the lower-motor neuron form of degenerative myelopathy or vertebral/spinal neoplasia in the lumbosacral area would have become overt during the three years of surveillance and, therefore, could be excluded with great certainity. The only likely differential diagnosis remaining was a disc protrusion outside the lumbosacral junction. As this prospective study spanned only a maximum of 3.5 years it is possible that some dogs with radiographic abnormalities but no current clinical signs of DLSS will develop the disease later on. Therefore, our statement that plain radiography is of very limited diagnostic power for DLSS may prove to be incorrect for surveys over a longer period of time.

In conclusion, it is tempting to interpret the radiographic signs of lumbosacral degeneration as a diagnostic criterion for presence of DLSS as the contrary will be difficult to prove in the absence of lifelong survey studies of radiographically suspicious and unsuspicious dogs. However, it can be stated with great certainity that radiographic signs of lumbosacral degeneration may preceed or appear simultaneously or appear even after onset of clinical signs. Thus, they are not helpful as predictors for development of DLSS. Further efforts such as combination of radiographic and magnetic resonance imaging screening or identification of genetic markers suggesting a hereditary predisposition for DLSS may be necessary to identify dogs that are at risk of developing the disease somewhen during their lifespan. DLSS is an important reason for premature termination of the working career in GSDs but this study demonstrates that slight to even moderately affected dogs may be still to be used in police service for months and even years.

Footnotes

[a] Domitor®; Dr. E. Gräub AG Bern, Switzerland

[b] Propofol 1%®; Fresenius AG, Sarnen, Switzerland

[c] Antisedan®; Dr. E. Gräub AG Bern, Switzerland

[d] Fuji AC3-System; Fujifilm AG, Dielsdorf Switzerland

References

1. Morgan JP, Bailey CS. Cauda equina syndrome in the dog: radiographic evaluaton. J Small Anim Pract 1990; 31:69-77.

2. Sisson AF, LeCouteur RA, Ingram JT, et al. Diagnosis of cauda equina abnormalities by using electromyography, discography, and epidurography in dogs. J Vet Intern Med 1992; 6:253-263.

3. Ness MG. Degenerative lumbosacral stenosis in the dog: a review of 30 cases: J Small Anim Pract 1994; 35:185-190.

4. De Risio L, Thomas WB, Sharp NP. Degenerative lumbosacral stenosis Vet.Clin North Am Small Anim Pract 2000; 30:111-132.

5. McKee M. Differential diagnosis of cauda equina syndrome. In Practice 1993; 15:243-244.

6. Oliver JE Jr, Selcer RR, Simpson S. Cauda equina compression from lumbosacral malarticulation and malformation in the dog. J Am Vet Med Assoc 1987; 173:207-214.

7. Jaggy A, Lang J, Schawalder P. Cauda equina-Syndrom beim Hund. Schweiz Arch Tierheilkd 1987; 129:171-192.

8. Linn L, Bartels KE, Rochat MC, et al. Lumbosacral stenosis in 20 military working dogs: Epidemiologic Findings and Outcome after Surgical Intervention (1990–1999). Veterinary Surgery 2003;32:21-29.

9. Moore GE, Burkman KD, Carter MN, et al. Causes of death or reasons for euthanasia in military working dogs: 927 cases (1993-1996). J Am Vet Med Assoc 2001; 219:209-214.

10. Scharf G, Steffen F, Grünenfelder F, et al. The Lumbosacral Junction in Working German Shepherd Dogs – Neurological and Radiological Evaluation. J Vet Med 2003; 51:27-32.

11. Morgan JP. Congenital Anomalies of the Vertebral Column of the Dog: A Study of the Incidence and Significance Based on a Radiographic and Morphologic Study. Vet Radiol and Ultrasound 1968;18:21-29.

12. Morgan JP. Transitional lumbosacral vertebral anomaly in the dog: a radiographic study. J Small Anim Pract 1999; 40:167-172.

13. Steffen F, Berger M, Morgan JP. Asymmetrical, Transitional, Lumbosacral Vertebral Segments in Six Dogs: A characteristic spinal syndrome. J Am Anim Hosp Assoc 2004; 40:338-344.

14. Flückiger M, Damur-Djuric N, Hässig M, et al. A lumbosacral vertebra in the dog predispositions to cauda equina syndrome. Vet Radiol Ultrasound 2006; 47:39-44.

15. Lang J, Häni H, Schwalder P. A sacral lesion resembling osteochondrosis in the German Shepherd dog. Vet Radiol and Ultrasound 1992; 33:69-76.

16. Kriegleder H. Cauda Equina Compression through Osteochondrosis-like lesion in the Sacrum: Clinical Signs, Treatment and Outcome in 10 German Shepherd Dogs. 2000; 10th ESVOT Congress, Munich, 23-26th March.

17. Seiler GS, Häni H, Busato AR, et al. Facet joint geometry and intervertebral disk degeneration in the L5-S1 region of the vertebral column in German Shepherd Dogs. Am J Vet Res 2002; 63:86-90.

18. Rossi F, Seiler G, Busato AR, et al. Magnetic Resonance Imaging of articular process joint geometry and intervertebral disc degeneration in the caudal lumbar spine

(L5-S1) of dogs with clinical signs of cauda equina compression. Vet Radiol Ultrasound 2004; 45:381-387.

19. Feeney DA, Wise M. Epidurography in the normal dog: technique and radiographic findings. Vet Radiol 1981; 22:35-39.

20. Wright JA. Spondylosis deformans of the lumbo-sacral joint in dogs. J Small Anim Pract 1980; 21:45-58.

21. Eichelberger H, Wurster H. Untersuchungen zur Spondylosis deformans bei Boxern. Kleintierpraxis 1982; 27:59-72.

22. Danielsson F, Sjöström L. Surgical Treatment of degenerative Lumbosacral Stenosis in Dogs. Veterinary Surgery 1999; 28:91-98.

23. Benneker LM, Heini PF, Anderson SE, et al. Correlation of radiographic and MRI parameters to morphological and biochemical assessement of intervertebral disc degeneration. Eur Spine J 2005; 14:27-35.

24. Schmid V, Lang J. Measurement on the lumbosacral junction in normal dogs and those with cauda equina compression. J Small Anim Pract 1993; 34: 437-442.

25. Levine GJ, Levine JM, Walker MA, et al. Evaluation of the association between spondylosis deformans and clinical signs of intervertebral disk disease in dogs: 172 cases (1999-2000). J Am Vet Med Assoc 2006; 288:96-100.

26. Morgan JP. Spondylosis deformans in the dog. Ad acta Orthop Scand Suppl 1967; 96:7-88.

Acknowledgement

Many thanks to Dr. Frank Steffen and Professor Mark Flückiger for their support and motivation and to Mark Flückiger for being my referent. To PD Michael Hässig for being my correferent, to Dr.Gernot Scharf helping me with the evaluation of the radiographs and to Dr. Ulrike Michal for her great help with the modification of the text. Thanks to Dr. Malgorzata Roos for her patient help with the statistical analysis and to the owners for giving us the opportunity to investigate their dogs.

Table 1

Descriptive statistics (n = 33) of the neurological and radiological examination of 33 German Shepherd Dogs at beginning of the study (1) and at the end (2).

	Mean	SD	Median	Q1	Q3
Age 1 (years)	3.63	1.69	3.50	2.10	4.54
Age 2 (years)	7.02	1.73	6.69	6.14	8.46
Time intervall (months)	41.29	8.04	37.37	35.23	49.58
Gait abnormalities 1 (score)	0.03	0.17	0.00	0.00	0.00
Gait abnormalities 2 (score)	0.48	0.97	0.00	0.00	0.25
Deficits of conscious proprioception 1 (score)	0.00	0.00	0.00	0.00	0.00
Deficits of conscious proprioception 2 (score)	0.46	1.09	0.00	0.00	0.00
Painful LS area 1 (score)	0.46	1.09	0.00	0.00	0.00
Painful LS area 2 (score)	1.36	1.52	0.00	0.00	3.00
Spinal reflexes 1 (score)	0.00	0.00	0.00	0.00	0.00
Spinal reflexes 2 (score)	0.00	0.00	0.00	0.00	0.00
Mineralisation of the nucleus 1 (mm)	0.36	1.19	0.00	0.00	0.00
Mineralisation of the nucleus 2 (mm)	3.12	6.1	0.00	0.00	6.00

Narrowing of the intervertebral space 1 (mm)	0.44	0.06	0.44	0.41	0.47
Narrowing of the intervertebral space 2 (mm)	0.41	0.06	0.41	0.38	0.44
Spondylosis deformans 1 (score)	0.33	0.48	0.00	0.00	1.00
Spondylosis deformans 2 (score)	0.76	0.44	1.00	0.75	1.00
Mineralisation within the spinal canal S1 1 (mm)	0.06	0.24	0.00	0.00	0.00
Mineralisation within the spinal canal S1 2 (mm)	0.21	0.42	0.00	0.00	0.00
Height of the spinal canal L7 1 (mm)	10.67	1.29	10.00	10.00	11.00
Height of the spinal canal L7 2 (mm)	11.14	1.23	11.00	10.00	12.00
Heigth of the spinal canal S1 1 (mm)	7.02	0.82	7.00	6.38	7.00
Heigth of the spinal canal S1 2 (mm)	7.56	1.09	7.00	7.00	8.00
Dorsal flattening of S1 1 (mm)	0.30	0.17	0.00	0.00	0.00
Dorsal flattening of S1 2 (mm)	0.30	0.17	0.00	0.00	0.00
Contour of cranial endplate of S1 1 (mm)	0.18	0.39	0.00	0.00	0.00
Contour of cranial endplate of S1 2 (mm)	0.18	0.39	0.00	0.00	0.00
Width of L7 endplate 1 (mm)	0.30	0.17	0.00	0.00	0.00
Width of L7 endplate 2 (mm)	0.30	0.17	0.00	0.00	0.00

Width of S1 endplate 1 (mm)	0.18	0.39	0.00	0.00	0.00
Width of S1 endplate 2 (mm)	0.91	0.29	0.00	0.00	0.00
LS malalignment 1 (mm)	0.18	0.86	0.00	0.00	0.13
LS malalignment 2 (mm)	0.44	1.04	0.00	0.00	1.00
Spondylarthrosis 1 (score)	0.06	0.24	0.00	0.00	0.00
Spondylarthrosis 2 (score)	0.00	0.00	0.00	0.00	0.00
Height of the body of L7 at the endplate site 1 (mm)	22.85	1.23	23.00	22.00	24.00
Height of the body of L7 at the endplate site 2 (mm)	23.27	1.46	23.00	22.00	24.00

Table 2

One Sample Sign Test
Progression of clinical parameters time 2 – time 1 (n = 33)

Rate: Number obs. > 0 / n

	p-Value	Accretion	Rate	95% CI Rate
Gait abnormalities	**0.0108**	**8**	0.24	(0.11,0.42)
Deficits of conscious proprioception	**0.0253**	**5**	0.15	(0.5,0.32)
Painful LS area	**0.0016**	**10**	0.3	(0.16,0.49)
Spinal reflexes quality	1.0	0	0	(0, 0.11)

Significant findings in bold

Progression of radiological parameters time 2 – time 1 (n = 33)

	p-Value	Accretion	Rate	95 % CI Rate
Nucleus mineralization	**0.0005**	12	0.36	(0.2,0.55)
Disk space narrowing (normed)	**0.0009**	25	0.76	(0.57,0.89)
Spondylosis deformans	**<0.0001**	20	0.61	(0.42,0.77)
Clouding within the spinal canal of S1	**0.0253**	5	0.15	(0.51,0.28)
Spinal canal height L7	**0.0037**	15	0.45	(0.28,0.64)
Spinal canal height S1	**0.0013**	17	0.52	(0.33,0.69)
Dorsal flattening of S1	0.5905	11	0.33	(0.15,0.49)
Contour of cranial endplate of S1	0.9999	3	0.09	(0.02,0.24)

With of L7 endplate	0.0907	10	0.3	(0.15,0.49)
Width of S1 endplate	0.6180	10	0.3	(0.15,0.49)
LS Malalignment	0.2239	11	0.33	(0.17,0.52)
Spondylarthrosis	0.1573	0	0	(0,0.11)
Height of the body of L7	0.634	15	0.45	(0.28,0.64)

Significant findings in bold

Table 3

Logistic regression
Inclusion criterion: Group of dogs with clinical signs of DLSS
Major parameters are displayed as examples

Covariables (time 1)	OR	95 % C.I. OR (0.240,32.240)
Width of S1 endplate	2.781	(0.240, 32.270)
Spondylosis	2.900	(0.570,14.762)
Clouding within the canal of S1	1229.208	(0.000,3.247E+38)