UNIVERSIDADE DE TRÁS-OS-MONTES E ALTO DOURO

Disorders Affecting the Cauda Equina in Dogs

(A Retrospective Study of 11 Cases and Literature Review)

Dissertação de Mestrado Integrado em Medicina Veterinária

Bárbara Gomes dos Santos

Orientador: Professor Doutor Artur Severo Proença Varejão

Vila Real, 2019

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Declaro que esta dissertação de mestrado é resultado da minha pesquisa e trabalho pessoal e das orientações dos meus supervisores. O seu conteúdo é original e todas as fontes consultadas estão devidamente mencionadas no texto e na bibliografia final. Declaro ainda que este trabalho não foi apresentado em nenhuma outra instituição para obtenção de qualquer grau académico.

Vila Real, de 2019
Bárbara Gomes dos Santos

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Abstract

A compression of the lumbosacral spinal nerve roots may lead to the development of a cauda equina syndrome (CES). Multiple aetiologies can lead to a CES and in this paper, there is a breve description of them, classified as: degenerative, anomalous/developmental, neoplastic, infectious/inflammatory, ischemic/vascular and traumatic. In a fully developed CES, multiple signs of sensory disorders may appear, such as low-back pain, anaesthesia, pelvic limbs' (PL) weakness or chronic paraplegia and, bladder dysfunction.

To evaluate the severity of the lesion, it is necessary to examine the patient and collect all clinical signs and results of the neurological examination and establish a prognosis. The diagnosis is based essentially on the neurological exam and imaging techniques, such as computed tomography and magnetic resonance.

The therapy options usually include the conservative treatment in cases that are considered less severe, and/or the surgical treatment for patients with more severe neurological deficits. The prognosis is mainly dependent upon the neurological deficits, the aetiology of the disease and the chosen therapy.

The practical component of the study includes the analysis of 11 clinical cases of dogs who were submitted to a CT scan, in the presence of clinical signs compatible with CES. The statistical analysis was based on collected data from Hospital Veterinário da UTAD (Vila Real, Portugal), between August 2017 and November 2018.

The obtained results allowed to conclude that large/giant breed dogs are more predisposed to CE disorders, constituting 55% of the sample. Neurological deficits, in CE disorders, are dependent of the nerve root(s) involved and are usually LMN in nature. Degenerative lesions are the most common lesion type regarding CE region, with an expression of 55%. DH II (83% of the degenerative cases) are more common than DH I in the lumbosacral region. The most frequent site for DH, in the CE region, is the L7/L8-S1 IVS, representing 83% of the cases. There is a male predilection for DLS, with an expression of 67%. Large/giant breed middle aged to older dogs have higher predisposition to develop DLS. Medical therapy had a success rate of 50% with 100% recurrence, for DLS, and surgical therapy had a success rate of 100%. The second most frequent type of lesion was the vascular type (FCE), representing 18% of cases. Neoplastic, traumatic and infectious lesions had an expression of 9%.

Keywords: cauda equina syndrome, cauda equina disorders, disc disease, hernia, lumbosacral, fibrocartilaginous embolism, osteosarcoma, sciatic neve injury, lumbosacral discospondylitis, dog.

Resumo

A compressão das raízes nervosas do plexo lombossagrado pode levar ao desenvolvimento de uma síndrome de cauda equina. Múltiplas etiologias podem estar envolvidas neste processo e neste estudo encontra-se uma breve descrição de algumas delas, classificadas como: degenerativas, anómalas/de desenvolvimento, neoplásicas, infeciosas/inflamatórias, isquémicas/vasculares e traumáticas. Numa síndrome da cauda equina totalmente desenvolvida, múltiplos sinais de distúrbios sensoriais podem aparecer, como: dor lombar, anestesia, fraqueza muscular nos membros pélvicos ou paraplegia crónica e disfunção da bexiga.

Para avaliar a gravidade da lesão, é necessário examinar o paciente, correlacionar todos os sinais clínicos com os resultados do exame neurológico e estabelecer um prognóstico. O diagnóstico baseia-se essencialmente nos exames neurológica e imagiológicos, como a tomografia computadorizada e a ressonância magnética.

As opções terapêuticas costumam incluir o tratamento conservativo, nos casos considerados menos graves, e/ou o tratamento cirúrgico, nos casos em que o paciente apresenta défices neurológicos de gravidade superior. O prognóstico depende principalmente dos défices neurológicos presentes, da etiologia e da terapêutica escolhida.

Na componente prática inclui-se a análise de 11 casos clínicos de cães que foram submetidos a uma tomografia computadorizada, na presença de sinais clínicos compatíveis com síndrome de cauda equina. A análise estatística baseou-se nos dados recolhidos no Hospital Veterinário da UTAD (Vila Real, Portugal), entre agosto de 2017 e novembro de 2018.

Os resultados obtidos permitiram concluir que os cães de raças grandes/gigantes são mais predispostos a lesões de cauda equina, constituindo 55% da amostra. Os défices neurológicos são dependentes das raizes nervosas envolvidas e são, geralmente, de neurónio motor inferior. As lesões degenerativas são as mais comuns na região, com expressão de 55%. Hérnias discais do tipo II (83% dos casos degenerativos) são mais comuns do que as do tipo I, na região lombossagrada. O local mais frequente para hérnias discais, nesta região, é o espaço intervertebral L7/L8-S1, representando 83% dos casos. Há uma predisposição do sexo masculino para estenose vertebral lombossagrada degenerativa, com uma expressão de 67%. Cães de raças grandes/gigantes com idade avançada apresentam uma maior predisposição para desenvolver estenose vertebral lombossagrada degenerativa. O tratamento conservativo teve uma taxa de sucesso de 50% com uma taxa de recidiva de 100% e a

terapia cirúrgica teve uma taxa de sucesso de 100%. O segundo tipo de lesão mais frequente foi o vascular, representando 18% dos casos. As lesões neoplásicas, traumáticas e infecciosas apresentaram uma expressão de 9%.

Palavras-chave: Síndrome de cauda equina, lesões da cauda equina, doença do disco intervertebral, hérnia lombossagrada, embolismo fibrocartilagíneo, osteosarcoma, lesão do nervo ciático, discospondilite lombossacral, cão.

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Abbreviations/Symbols/Acronyms List

% – Percentage
AF – Anulus fibrosus disci intervertebralis
ALB – Albumin
ALT - Alanine Aminotransferase
BCHM – Biochemistry
BID – Twice a day
C – Cervical
Ca – Caudal vertebra
CBC – Complete blood count
CE – Cauda equina
CES – Cauda Equina Syndrome
CL – Compressive lesions
CM – Contrast media
CNS – Central nervous system
CSF – Cerebrospinal fluid
CT – Computed Tomography
DH I – Disc herniation(s) Hansen type I
DH II – Disc herniation(s) Hansen type II
DLS – Degenerative lumbossacral stenosis
EOS – Eosinophils
ERI – Erythrocytes
ESC - Extradural synovial cysts
FCE - Fibrocartilaginous embolism
GLOB – Globulins
HCT – Haematocrit
HGB – Haemoglobin
HV-UTAD – Hospital Veterinário da Universidade de Alto Douro e Trás-Os-Montes
IM – Intramuscular
IV – Intravenous
IVD – Intervertebral disc
IVF – Intervertebral foramen
IVS – Intervertebral space
L – Lumbar vertebra
LEU – Leucocytes
LMN – Lower motor neuron
LPL – Left pelvic limb
LS – Lumbosacral
LSTV - Lumbosacral transitional vertebra
m – month(s)
MC – meningocele
Mg – Milligram
MHCT – Microhematocrit
Min - Minutes
MMC – Meningomyelocele
MONO – Monocytes
MR – Magnetic ressonance
MRI – Magnetic ressonance imaging
MVP - Mean plaquette volume
N – Normal
NEU – Neutrophils
NP – Nucleus pulposus
NSAIDs – Non-steroidal anti-inflammatories
PCR - Polymerase chain reaction test
PL – Pelvic limb(s)
PO – Per oral
RETIC – Reticulocytes
RPL – Right pelvic limb
S – Sacral vertebra
SC – Spinal cord
SID – Once a day
spp. – Species
T – Thoracic vertebrae
T1W – T1 weighted image
T2W – T2 weighted image

Tab – Tablet(s)

TCS - Tethered cord syndrome

TID – Three times a day

TL – Thoracolumbar

UMN – Upper motor neuron

UTAD – Universidade de Trás-os-Montes e Alto Douro

VC – Vertebral canal

WBC – White blood cells

wk – Week(s)

y/o – Years old

CHAPTER I – Introduction

The cauda equina syndrome (CES) is a complex entity, with multiple possible aetiologies. Clinical signs may differ in each individual patient and vary according to the location and extent of the lesion. These are highly nonspecific and can be persistent or episodic and heterogenous. Neurological deficits are dependent of the nerve root involved and may differ between right and left sides. When present, they are LMN (lower motor neuron) in nature. CES is usually characterized by low-back pain, bilateral sciatica, saddle hypoesthesia or anaesthesia, motor weakness and neurological deficits of the pelvic limbs (PL), impairment of anal, rectal and bladder sphincter's dysfunction as well as sexual impotence (Orendáčová *et al.* 2001; Platt, 2010).

For the diagnosis of this syndrome, the accuracy of the anamnesis and the physical, orthopaedic, and neurological exams play a very important role. A definitive diagnosis should be supported by imaging results. The therapy options usually include the conservative treatment, in cases considered less severe, and/or the surgical treatment for patients with more severe neurological deficits (Meij & Bergknut, 2010; Lanz & Rossmeisl, 2012). The prognosis should be established considering the neurological deficits, the aetiology of the disease and the chosen therapy (Griffin IV *et al.,* 2009b).

This study includes a literature review about the CES, as well as the clinical approach, diagnosis, treatment and prognosis for the different aetiologies that can be involved in the disease process. The practical component of the study includes the analysis of 11 clinical cases of dogs who were submitted to a CT scan, in the presence of clinical signs compatible with CES. The statistical analysis was based on collected data from Hospital Veterinário da UTAD (Vila Real, Portugal), between August 2017 and November 2018.

This study has the goals of: characterize the sample (breed size, age, gender, weight), characterize the severity and aetiology of the lesion analyse the clinical course of the disease and the recovery with and without decompressive surgery, and analyse and associate the treatment options with the recurrence of clinical signs.

CHAPTER II – Literature review

1. Anatomic description of the vertebral column

1.1. The vertebrae

The number of vertebrae and spinal cord (SC) segments is variable between species. Individual variations can occur, especially in transitional vertebrae. There are approximately 50 vertebrae separated into five distinctive regions: seven cervical (C1-C7), thirteen thoracic (T1-T13), seven lumbar (L1-L7), three fused sacral vertebrae (S1-S3) that constitute the sacrum and twenty caudal (Ca1-Ca20) vertebrae, approximately (Thomson and Hanh, 2012). When analysing the vertebral formula, the first letter of the word designates each group by means of abbreviation, followed by a digit, that designates the number of the vertebra in the specific group. For example, the canine vertebral formula is C7T13L7S3Ca20-23 (Sisson, 2002).

All vertebrae remain separate and articulate with adjacent vertebrae, creating movable joints, except for the sacral vertebrae. The vertebral column has considerable flexibility (Badoux, 2005). Vertebrae contribute to support the head, to provide attachment for the muscles that are responsible for body movements and to protect the SC and roots of the spinal nerves. There are two types of joints between vertebrae: one cartilaginous that involves the direct connection of the bodies of the vertebrae, and a synovial one on the vertebral arches, between articular processes (Sharp & Wheeler, 2005).

Between two adjacent vertebrae there is the interposed cartilaginous disc, the articulations between them and the connecting ligaments, which form a functional unit, complemented by the nerves and blood vessels that leave the vertebral canal (VC) through the intervertebral foramina (IVF), and the muscles, covering different regions (Liebich & König, 2004a).

The typical vertebra will present a cylindrical and dorsally flattened body, which faces into the VC. It may present a median crest ventrally and an arch that encloses the vertebral foramen, with right and left pedicles and laminae. The pedicles extend dorsally, on each side, from the dorsolateral surface of the vertebral bodies, with cranial and caudal vertebral notches. The notches of each side of adjacent vertebrae originate the IVF, through which the spinal nerves, arteries and veins pass. Occasionally, there can be an additional lateral vertebral foramen in the pedicle near to the IVF (Budras *et al.*, 2007; Dyce *et al*., 2010b).

Each vertebra has processes for muscular or articular connections and insertions, classified by transverse, spinous, articular, accessory and mammillary processes, a convex cranial articular surface and a centrally depressed caudal articular surface. Dorsally, the vertebral arch has right and left laminae that unite in the dorsal midline to form a spinous process. The space between adjacent arches is the interarcuate space, where the yellow ligament is dorsally located. The union of the vertebral arch with the vertebral body originates the vertebral foramen, a set of tubes that come together to form the VC, which encloses the SC (De Lahunta *et al.*, 2015).

In the lumbar region, the vertebrae are longer, with more uniform bodies than the thoracic vertebrae (Dyce *et al.,* 2010b). The lumbar vertebrae are usually dorsoventrally flattened, increasing their width (until L7) and length (until L6) caudally. The body of the L7 has approximately the same length L1. The pedicles and laminae of the lumbar vertebrae are longer and larger. The spinous processes are higher and more massive in the mid-lumbar region, with their height decreasing caudally from L4. The accessory processes are well developed on the first three to four lumbar vertebrae and absent on the fifth or sixth (Sisson, 2002; Fletcher, 2013). Other regional features of the lumbar vertebrae are the prominent mammillary and, in some cases, accessory processes (Badoux, 2005).

The sacrum is short, wide and wedge-shaped. It lies between the ilium and articulates with it. The first sacral vertebra has a larger body than the other two combined. There's a median sacral crest that originates from the fusion of the three spinous processes of the sacral vertebrae (Thomson & Hahn, 2002). In the dorsal surface of the sacrum, there are two pairs of dorsal foramina, through which the dorsal divisions of the sacral spinal nerves and spinal vessels will pass. Medially to these foramina, there are the fused mamilloarticular processes of adjacent segments that form the intermediate sacral crest (Budras *et al*., 2007). The pelvic surface bears two pairs of foramina, larger than the corresponding dorsal foramina, situated laterally to the sacral body. Blood vessels and the ventral branches of the first two sacral nerves pass through them. Laterally to the pelvic sacral foramina there are fused transverse processes that form the thin lateral sacral crest (Liebich & König, 2004b; Morales & Montoliu, 2012).

The wing of the sacrum is the enlarged, prismatic-shaped lateral portion, which has a rough auricular surface that articulates with the ilium. The base of the sacrum faces cranially, articulating with the last lumbar vertebra. Above its articular surface, begins the sacral canal, formed by the coalescence of the three vertebral foramina (Fletcher, 2013; De Lahunta *et al.*, 2015). The sacral canal is compressed dorsoventrally (Sisson, 2002). The cranioventral part of the base of the sacrum has a transverse ridge, the promontory. The caudal extremity of the sacrum is the apex, which articulates with the first caudal

vertebra. Occasionally, the first caudal vertebra appears fused to the sacrum (Evans & De Lahunta, 2013).

1.2. The intervertebral discs

The intervertebral discs lie between adjacent vertebra, in the intervertebral spaces, except for the space between C1 and C2 and for the sacrum (Griffin IV *et al*., 2009b; Brisson, 2010). These are thick pads that form fibrocartilaginous joints and allow the movement of the vertebrae, keeping them together and contributing for the flexibility of the vertebral column, while protecting the SC from mechanical forces (Marinho *et al*., 2014; Smolders & Forterre, 2015).

The intervertebral disc (IVD) consists of an outer fibrous ring, the anulus fibrosus disci intervertebralis (AF), which surrounds an inner amorphous and gelatinous centre, the nucleus pulposus (NP), a semifluid remnant of the notochord. The AF is thicker ventrally, constituted by parallel fibres, creating oblique bands, between vertebral bodies. This fibrous ring is responsible for transmitting stresses and strains that are required by lateral and dorsoventral movements of the vertebral column. When approaching the NP, the AF becomes less fibrous and more cartilaginous. When the AF ruptures or degenerates, bulging can occur due to pressure caused by the vertebral body movements (Jeffery *et al*., 2013; Fingeroth & Thomas, 2015).

1.3. The ligaments of the vertebral column

The ligaments of the vertebral column can be organized into short ligaments, that connect successive vertebrae, and long ligaments, that go across several vertebrae (Liebich & König, 2004a).

The supraspinous, the ventral longitudinal and the dorsal longitudinal ligaments extend along considerable portions of the vertebral column. The supraspinous ligament is a thick band, particularly noticeable on the thoracic region, extending from the spinous process of the first thoracic vertebra (T1) to the third caudal vertebra (Ca3). During movements of flexion, the supraspinous ligament prevents abnormal separation of the spine, together with the thin interspinous ligaments (Griffin IV *et al.,* 2009b; Evans & De Lahunta, 2013).

The ventral longitudinal ligament is located ventrally to the surfaces of the vertebral bodies, from the axis to the sacrum, attached to the intervertebral discs (Liebich & König, 2004b; Morales & Montoliu, 2012).

The dorsal longitudinal ligament is thicker than the ventral longitudinal ligament. It runs from the dens of the axis to the sacrum, along the dorsal surfaces of the bodies

of the vertebrae, constituting part of the floor of the VC. The dorsal longitudinal ligament narrows over the middle of each vertebral body and widens when crossing the intervertebral discs (Klopp, 2010a).

The intertransverse ligaments unite the transverse processes of the lumbar vertebrae and are only distinct in this region. These ligaments are tensed during lateral flexion and rotation movements (Liebich & König, 2004b).

The yellow ligaments are located between the arches of adjacent vertebrae, blended with the articular capsules that surround the articular processes. The epidural space is located ventrally to the yellow ligaments and draws apart the ligaments and arches of the vertebrae from the dura covering the SC (Evans & De Lahunta, 2013).

1.4. The spinal cord and the cauda equina

The SC is enclosed by the VC. In medium to large-breed dogs, the SC terminates at the L6–L7 vertebral level and in small-breed dogs, one-half to one vertebral body more caudally. Functionally, the SC can be divided into four regions: cranial-cervical (C1–C5); cervicothoracic (C6–T2); thoracolumbar (T3–L3); lumbosacral (L4–S3) (Dellman & McClure, 1986; Budras *et al.*, 2007).

The innervation of the body is organized in a segmental pattern. Each cutaneous region of the body (dermatome) and group of muscle fibres (myotome) is innervated by one SC segment. A SC segment is defined as a portion of the SC that gives rise to one pair of spinal nerves. In the dog and cat, there are 8 cervical, 13 thoracic, 7 lumbar, 3 sacral and at least 2 caudal SC segments (Platt & Olby, 2012). Some SC segments lie within the vertebra of the same annotation, whilst others lie cranial to the corresponding vertebra. In the last two thoracic and the first two or three lumbar segments, the spinal segments are found within their corresponding vertebrae. All the other ones are located cranially to the vertebra of the same number (Dewey & Da Costa, 2016). Usually, the three sacral segments lie within the fifth lumbar vertebral foramen, and the caudal segments lie within the sixth lumbar vertebral foramen (De Lahunta *et al.*, 2015).

There are two enlargements of the SC, caused by an increase in white matter and cell bodies, associated with the innervation of the thoracic (cervical intumescence) and PL (lumbar intumescence). The cervical intumescence can be found within the fifth to seventh cervical vertebral foramina (caudal cervical region) and the first thoracic segment. The lumbar intumescence is usually located between the fourth and sixth or seventh lumbar segments (Thomson and Hahn, 2012). Caudal to the lumbar intumescence, usually at the level of the L5 vertebra, the SC becomes conical, in what is called the conus medullaris. These segments appear successively smaller,

surrounded by caudally directed spinal roots. The conus medullaris is continued by the filum terminale, and, beyond it, by sacral and caudal spinal roots within the VC. The cauda equina (CE) is constituted by these nerve roots, which normally descend through the VC of vertebrae L6, L7 and sacrum. (Budras *et al*., 2007; Fletcher, 2013).

The CE nerve roots have the structure of a typical peripherical nerve and are partially unsheathed by the meninges. The CE is constituted by L6, L7, S1, S2, S3 and coccygeal 1 to 5 nerve roots. After leaving the foramina, L6, L7 and S1 nerve roots constitute the sciatic nerve. S2 and S3 nerve roots contribute to the formation of the pudendal and pelvic nerves. These nerve roots tolerate deformation better than SC. There is also a large epidural space in the region of the CE (figure 1). Thus, they are usually more resistant to injury than the SC tissue, but if severe damage occurs, recovery is unlikely (Sharp & Wheeler, 2005).

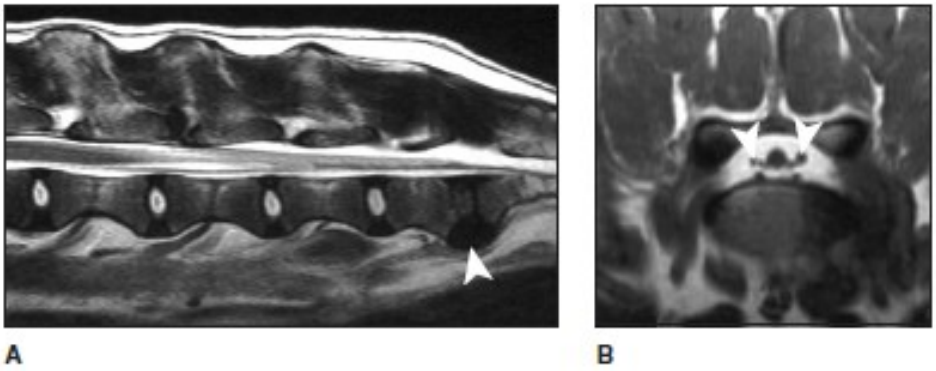

Figure 1 – Normal lumbosacral MRI. A) Sagittal, T2-weighted MRI of the lumbosacral spine in extension. There is loss of signal of the L7/S1 NP (arrowhead) but there is no dorsal displacement of the disc. B) Transverse, T1-weighted MRI through the L7/S1 disc space and foramen of the same dog as image A); the nerve roots (arrowheads) are surrounded by high-signal epidural fat (adapted from: Sharp & Wheeler, 2005).

1.5. Vascularization

A longitudinal ventral spinal artery and usually one or two dorsal spinal arteries course the full length of the SC, together with the spinal nerve (De Lahunta *et al.*, 2015). On the surface of the SC, the longitudinal arteries are connected to a diffuse plexus, sending branches into the parenchyma, through the IVF. The origin of these branches is variable, according to the region of the vertebral column where they are situated. The first thoracic segments are supplied by the thoracic vertebral artery, a branch of the subclavian artery (Uemura, 2015). The remaining thoracic segments are supplied by branches of the intercostal and thoracic arteries. The lumbar area is supplied by lumbar segmentations of the abdominal and thoracic aorta (Morales & Montoliu, 2012).

Each spinal artery has one or two dorsal, dorsal spinal arteries, and a ventral root, the ventral spinal artery, that are responsible for the irrigation of the SC. The ventral spinal artery sends branches into the ventral median fissure, giving rise to the vertical artery, responsible for the irrigation of the grey matter (Budras *et al.,*2007; Uemura, 2015).

The SC veins have similar distribution to the arteries, presenting a ventral spinal vein in the ventral median fissure, a superficial venous plexus on the surface of the SC, with connections to the ventrolateral and dorsolateral veins, and a dorsal spinal vein. In the epidural space, on the floor of the VC, there are ventral internal venous plexuses. The venous sinuses of each side, on the floor of the VC, collect blood from vertebrae, meninges, and nerve roots. Dorsally, in the epidural space, interarcuate veins pass and connect the ventral internal vertebral venous plexus with the dorsal external vertebral venous plexus (Sjöström, 2003; De Lahunta *et al.,* 2015).

2. Clinical assessment of the neurological patient

The first step of the clinical evaluation is to take a proper and complete history. The following table (table 1) exhibits the data that should be obtained from the owner (Lorenz *et al*., 2011). A clear and concise description of the main concern should be obtained from the owner. In the absence of clinical findings, the owner's description can be the basis to institute an anatomical and differential diagnosis. Video footages could offer valuable information and clarify some doubts that the clinician may have. It is very important to get information about the onset, evolution and course of illness (see table 2), as it may provide insight into specific differential diagnoses (Schatzberg, 2017). The evolution of the condition should be characterized as progressive, static, improving or "waxing and waning". It is important to identify factors that can trigger or improve the signs, previous therapy and its effect on disease course (Dewey & da Costa, 2016).

2.1. General physical examination

The neurological examination should be preceded by a general physical examination of all other body systems to detect abnormalities that can affect the nervous system, mimic a primary neurological disorder or influence the prognosis. An orthopaedic examination should be done in all animals with gait disturbances. Vital signs should be examined, and blood and urine collected for further laboratory analysis. Evaluation of the bladder function is very important, since urinary incontinence can be, sometimes, the only clinical sign of some CE disorders (Varejão *et al*., 2004; McKee, 2007).

General information	• Age of the patient • Sibling numbers and health • How long the owner has the pet • Vaccination status • Travel history • Parasite treatments (internal and external) • Access to toxins • History of trauma • Environment • Existence and health of other animals in the same household • Diet, including supplements • Current or recent medication • Medical and surgical history • Allergies (including drug allergies)
Specific complaint	• When did it start? • How did it start? • Alterations since onset • Existence of pain manifestations • Is there any possible association between medication and a change in the condition?
Systemic health	• Appetite and thirst • Vomiting/regurgitation/diarrhoea/coughing/sneezing • Urinary and faecal incontinence • Exercise tolerance • Assessment of vision

Table 1 – Information that should be obtained from the owner during the anamnesis (adapted from: Platt & Olby, 2012; Schatzberg, 2017).

Acute	Onset over minutes to hours
Subacute	Onset over days
Chronic	Onset over several days, weeks or months
Episodic	The animal returns to the normal status between episodes

Table 2 – Characterization of the onset of the patient's illness (adapted from Platt & Olby, 2012).

2.2. Neurological examination

The neurologic examination intends to evaluate the integrity and function of the nervous system. It helps the clinician to elaborate a list of differential diagnosis, to identify the aetiology of the disorder, to determine its prognosis and to help choosing the most adequate treatment (Varejão *et al*., 2004). Having a list of all the steps can be a powerful and helpful tool in order to register the results and remember all parameters. Video recordings are important to ensure that the neurologic examination was fully done and to have a permanent record with the important data about the patient, allowing to evaluate the progression/regression of the clinical signs (LeCouteur & Grandy, 2010). The neurologic examination should aim to answer the following questions:

- Do the clinical signs observed refer to a nervous system lesion?
- Within the nervous system, where is the lesion located?
- What types of disease process can explain the clinical signs?
- What is the severity of the disease? (Platt & Olby, 2012).

It is very important to ensure that the animal is comfortable and cooperating during the examination. Ambulatory patients should be allowed to explore the examination room, in order to evaluate their mental status, behaviour, posture and gait. If the animal is anxious or nervous, the sensory evaluation should not be done right away. The clinician should be aware that sedation, analgesia or neurological conditions, such as epileptic seizures, can influence the results of this neurological evaluation (Platt, 2010; Thomas & De Risio, 2015).

Part I – hands-off examination

The first part of the neurological examination is constituted by evaluation of the mental status and behaviour, evaluation and characterization of posture and body positioning at rest, gait evaluation, and identification of abnormal involuntary movements (e.g. tremors) (Platt, 2010).

On CE disorders, one of the most common neurological presentations is gait disturbances (see table 3). The animal's ability to generate and make coordinated movements should be assessed in a place where the patient can move freely, over a non-slip surface, preferably with the owner walking the animal. If the animal is not trying to walk, body support should be provided, with a sling or harness, so that any subtle voluntary movement can be noticed. Normal gait requires intact function of the brainstem, cerebellum, SC and sensory and motor peripheral nerves, neuromuscular junctions and muscles (Costes, 2012; Thomas & De Risio, 2015).

Ataxia (uncoordinated gait)	Hypometria	Shorter protraction phase of gait.
	Hypermetria	Longer protraction phase of gait.
	Dysmetria	A combination of hypometria and hypermetria.
Paresis/Paralysis	Paraparesis/ Paraplegia	Paresis/paralysis of both PL.
	Monoparesis/ Monoplegia	Paresis/paralysis of one limb, usually caused by a LMN lesion.
Lameness	Usually presents with a short stride of the affected limb and a long stride of the contralateral limb. It can be associated with nerve root signature.	

Table 3 – Important parameters to evaluate on a patient with CES (adapted from Platt & Olby, 2012).

Part II – hands-on examination

The hands-on examination should be constituted by an assessment of the cranial nerves; postural reaction tests; spinal reflexes, muscle tone and size examination and evaluation and sensory evaluation tests (Platt & Olby, 2012). Tables 4 to 6 describe the important tests to diagnose a CES.

The entire nervous system should be able to perform postural reactions (see table 4). The primary aim of postural reaction testing is to detect any subtle deficits that are not obvious on gait evaluation. Lesions affecting the anatomical sensory and/or motor components could result in abnormal postural reactions (Chrisman *et al.*, 2003; Schatzberg, 2017).

The evaluation of spinal reflexes should be considered as a continuation of gait evaluation and postural reaction testing. It helps to classify the neurological disorder as UMN or LMN type, allowing to localize the lesion in specific SC segments or peripheral nerves (Lorenz *et al*., 2011). Spinal reflexes are segmental, which means that they only evaluate the spinal segment(s) within the intumescences corresponding to the stimulated nerve. In dogs, spinal reflexes and muscle tone must be evaluated with the patient in lateral recumbency. The animal can be unconscious. The most reliable spinal reflex tests for the PL' evaluation are the patellar and withdrawal reflexes. Other spinal reflexes are more difficult to perform and interpret (see table 5) (Garosi, 2004; Parent, 2010).

The evaluation of the sensory system depends on tests for pain perception (nociception). It requires a noxious stimulus and evaluation of the animal's response. The purpose of testing pain perception is to detect and map out any areas of sensory loss (see table 6) (Platt, 2010). Pain perception can be classified as:

- Anaesthesia (complete loss of all forms of sensation),
- Hypoesthesia (diminution of sensation),
- Hyperesthesia (increased sensitivity to a normal level of stimulation),
- Analgesia (complete loss of sensitivity to pain),
- Hypoalgesia (decreased sensitivity to pain),
- Hyperalgesia (increased sensitivity to pain) (Chrisman *et al.*, 2003; Coates, 2012).

Proprioceptive placing	Proprioceptive placing is evaluated by placing the paw turned over so that the dorsal surface is in contact with the ground and determining how quickly the animal corrects its position. The animal should be standing squarely on all four limbs and most of its bodyweight supported. The test should be repeated until the examiner is confident with the result.
Sliding paper test	A piece of paper is placed under the weight-bearing foot of the animal and slowly pulled laterally. A normal animal will pick up the limb and replace it in the correct position. This test mainly evaluates proprioception in the proximal part of the limb.
Extensor postural thrusting	The animal is supported by the chest, caudal to the thoracic limbs, and the PL are lowered to the floor, forcing the animal to walk backwards. This test highlights pelvic limb weakness and ataxia.
Hemi-walking	Tests the ability of the animal to walk on the thoracic and PL on one side, whilst holding the limbs of the other side. The animal should be pushed away from the side on which its limbs are supported, and the speed and coordination of the movements assessed. This is best performed on a non-slip surface.

Table 4 – Important postural reaction to evaluate in a patient with CES (adapted from Platt & Olby, 2012; De Thomas & De Risio, 2015).

Spinal Reflex test	Segments evaluated	Execution
Withdrawal (flexor) reflex	L4–S2 and associated nerve roots; femoral and sciatic nerves.	A noxious stimulus is applied to the tested limb by pinching the nail bed or digit with the fingers or a haemostat. A normal reflex would be flexion of the hip (femoral nerve function), stifle and hock (sciatic nerve function). The hock must be extended to evaluate sciatic function. When absent, individual toes should be tested to detect specific nerve deficits.
Patellar reflex	L4–L6 and associated nerve roots; femoral nerve.	The stifle should be slightly flexed. The limb being tested is supported by placing one hand under the thigh. Striking the patellar tendon with a reflex hammer induces extension of the limb due to a reflex contraction of the quadriceps femoris muscle. A weak/absent reflex indicates a L4–L6 segments or the femoral nerve lesion. A similarly weak or absent reflex can be seen with previous stifle disease or as age-related changes. The patellar reflex can be pseudo-hyperreflexic with sciatic nerve or L6–S2 lesion, due to decreased tone.
Cranial tibial reflex	L6–S1 and associated nerve roots; peroneal nerve.	Elicited by striking the proximal part of the cranial tibial muscle with a reflex hammer and observing for flexion of the tarsus.
Gastrocnemius reflex	L7–S1 and associated nerve roots; tibial peripheral nerve.	Elicited by placing a finger over the gastrocnemius muscle and striking it with a hammer. The normal reaction is extension of the hock.
Perineal reflex	S1–Cd5 and associated nerve roots; caudal nerves of the tail and pudendal nerve.	When normal, stimulation of the perineum with a haemostat results in contraction of the anal sphincter and flexion of the tail.

Table 5 – Important spinal reflex tests for the evaluation of a patient with CES (adapted from: Dewey & da Costa, 2016).

Sensory test	Evaluation
Nociception testing	Conscious pain perception must be assessed in the limbs, tail and perineal region. The expected reaction is a behavioural response. The animal is placed on lateral recumbency, ideally with a second person distracting it. A gentle squeeze is applied to the digits to elicit the withdrawal reflex. If the animal doesn't manifest pain, heavier pressure is applied. Withdrawal of the limb is due to the flexor reflex and should not be taken as evidence of pain sensation.
Palpation	Palpation and manipulation should be performed last, to avoid losing cooperation. The degree of pressure applied should increase progressively. Palpation can be useful to evaluate musculoskeletal conditions that could mimic a neurological disorder and to detect focal muscle atrophy, which could be indicative of neurological disease or orthopaedic condition.

Table 6 – Sensory tests for evaluation of the sensory system of the neurologic patient with CES (adapted from: Parent, 2010; Platt & Olby, 2012).

3. Cauda equina syndrome

3.1. Clinical signs

Problems affecting the caudal lumbar region are very common in large-breed dogs. Clinical signs associated with CES vary according to the location and extent of the lesion. These are highly nonspecific and can be persistent or episodic and heterogeneous. Clinical signs may be bilateral or unilateral (Parent, 2010).

Neurological deficits are dependent of the nerve root involved and are usually LMN in nature. Neurological dysfunction of the PL can range from paraplegia, paraparesis, monoplegia or monoparesis to mild proprioceptive deficits which do not affect gait (Danielsson & Sjöström, 1999). A shortened and stiff stride of the PL may be observed. Dragging the claws of the digits and ataxia are manifestations of decreased proprioception. The sciatic, cranial tibial, gastrocnemius, anal and flexor withdrawal reflexes may be normal, decreased or absent. If significant sciatic deficits are present, failure of hock flexion during withdrawal reflex test may be seen. Muscle atrophy may be observable in the sciatic distribution. The patellar reflex test may exhibit a false exaggerated response (patellar pseudo-hyperreflexia), which must be differentiated from the increased reflex that occurs with UMN deficits, observed in lesions cranial to the L4 segment (Ness, 1994; Dewey, 2013).

Patient history frequently includes lower lumbar or PL pain, which can be manifested in various ways. The tail can be flaccid or carried low due to pain, hypotonic, or paralytic. Lower lumbar pain can be triggered by the extension of the lumbosacral

joint, when the animal jumps, climbs stairs, stands from a prone position or crawls. Unwillingness to exercise or jump into a car, stiffness during physically demanding exercise, scuffing of the toenails of the PL, intermittent lameness, limb dysfunction exacerbated by activity, or LMN deficits may be indicative of a CES (Sjöström, 2003) (see table 7). The patient may show stiffness of the musculature surrounding the lumbosacral region (Orendácová *et al.,* 2001; Mckee, 2007).

There are several techniques to isolate lumbosacral painful responses: traction or elevation of the tail; per rectum application of pressure to the L7-S1 disc space; performing the hyperextension or lordosis test, which consists in elevating the pelvic limbs off the ground, guaranteeing extension of the hips, and applying lumbosacral pressure; and rotation of the lumbosacral joint by swinging the PL bilaterally (Platt, 2010; Lanz & Rossmeissl, 2012).

In a general way, when analysing compressive lesions (CL), the first fibres to be compromised are the proprioceptive fibres and the last ones are the nociceptive fibres. Nociceptive dysfunction reflects a severe, extensive lesion (Meij & Bergknut, 2010; Lanz & Rossmeissl, 2012). In cases of CL of increasing severity, the clinical signs will start with spinal hyperpathia/sensitivity, due to meningeal irritation, proprioceptive dysfunction, loss of motor function manifested through paresis, loss of superficial pain, paralysis, and finally loss of all nociception. Loss of urinary and faecal continence can occur when the damage is severe enough to cause marked paresis or paralysis. Faecal incontinence is associated with poor anal tone and may be present even with a normal anal reflex. Urinary incontinence is usually a result of pelvic and pudendal nerve dysfunction (S1-S3), which causes urethral or anal sphincter hypotonia and possibly altered sensation in the perineal region (Platt, 2010; Thomson & Hanh, 2012). Occasionally, urinary incontinence, with dribbling of urine associated with an easy manual bladder emptying, and faecal incontinence may be the only clinical manifestations of lumbosacral disease. (Watt, 1991; Meij & Bergknut, 2010).

Hyperesthesia	Manifested through signs of pain, discomfort and lameness. Can result from compression and/or inflammation of meninges and nerve roots, or from the bones, IVD or articular facet joint capsules.
Paraesthesia and dysesthesia	Can be caused by irritation or trauma to sensory nerves or nerve roots. Patients can manifest it by staring or biting the areas around the rump and PL.
Proprioceptive deficits on the PL	Can be mild, manifested as delayed proprioceptive positioning reactions, or severe, manifested by pelvic limb weakness by dragging the dorsal aspect of the toes. Can be symmetrical or asymmetrical.
Voluntary motor deficits	Damage to the caudal SC segments and/or nerve roots can be noticed by a decreased to absent tail tone and movement. In some cases, the tail can appear in an abnormally low position or with a deviation to one of the sides. A lesion of the sciatic nerve is manifested by pelvic limb weakness.
Abnormal reflex activity	Decreased to absent withdrawal, gastrocnemius and cranial tibial reflexes can be noticed. The patellar reflex in usually normal or hyper-reflexive. Patellar pseudo-hyperreflexia may be observed when the nerve supply to the caudal thigh muscles is disrupted, removing their tonic antagonistic influence over the quadriceps. The perineal reflex can be decreased to absent.
Urinary and/or faecal incontinence	These signs are characteristic of severe presentations and are less reversible.
Nociceptive deficits	Can be observed in the PL, perineum and tail with severe presentations of CE disorders.

Table 7 – Clinical signs of CE disorders (summarized) (adapted from Dewey & da Costa, 2016).

3.2. Differential diagnosis

A differential diagnosis list, according to signalment, history and neurological findings, is essential. Diagnostic tests should aim to confirm or rule out the differential diagnosis in the list, without replacing clinical evaluation. The primary differential diagnosis for the CE region are: degenerative lumbosacral stenosis, discospondylitis, neoplasia, and extradural synovial cysts (Platt & Olby, 2012).

After localizing the lesion, disease processes can be classified according to the cause, using the mnemonic DAMNITV. Each process has a typical signalment, onset, progression, and distribution within the nervous system (see figure 2 and table 8) (Varejão, 2004; da Costa & Moore, 2010). The list may be narrowed down through the neurological examination of the animal, depending on the presence, type, and severity of neurological deficits that arise from the CE region (L4-S3). Geriatric patients frequently

have more than one disease entity, which may interfere with the diagnosis. Orthopaedic conditions or other neurological diseases should be considered, since they may mimic or exaggerate signs of lumbosacral disease. Dogs with orthopaedic disease will have a normal neurological examination (Mckee, 1993; Meij & Bergknut, 2010).

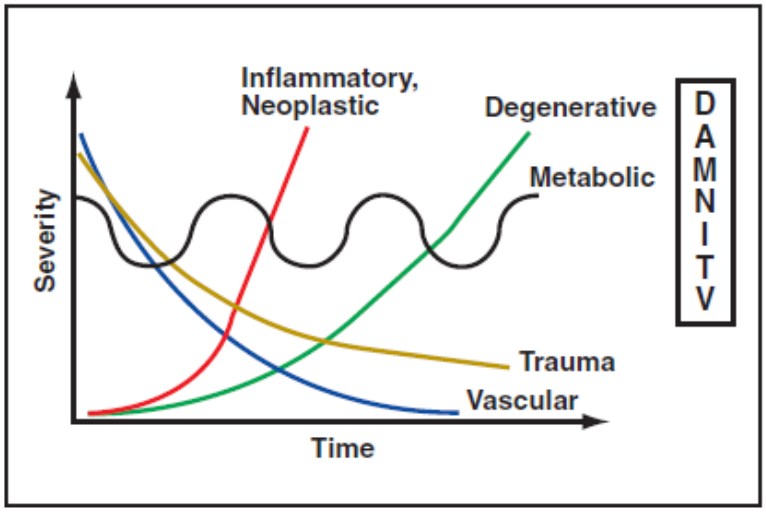

Figure 2 – Onset and progression of neurological diseases of differing causes (adapted from: Platt & Olby, 2012).

Acute progressive with asymmetrical clinical signs	• Degenerative (e.g. IVD disease) • Neoplastic • Inflammatory/infectious disease
Acute, progressive with symmetrical signs	• Metabolic disorders • Nutritional disorders • Neoplastic • Inflammatory/infectious disease • Toxicity
Acute, non-progressive (often asymmetrical)	• Idiopathic • Trauma • Vascular disorders
Chronic, progressive with symmetrical signs	• Degenerative disorders • Anomalous disorders • Metabolic disorders • Neoplastic disorders • Nutritional disorders • Inflammatory/infectious disease • Toxicity
Chronic, progressive with asymmetrical signs	• Degenerative (e.g. IVD disease) • Neoplastic disease • Inflammatory/infectious disease

Table 8 – Disease categories based on onset and progression of signs (adapted from: LeCouteur & Grandy, 2005; Da Costa & Moore, 2010; Dewey & da Costa, 2016).

3.2.1. The mnemonic DAMNITV

a) D – Degenerative

Degenerative diseases involve morphological degeneration of the nervous tissue and can affect any part of the nervous system. Many are hereditary. The onset is typically insidious with slow progression of the clinical signs. The age of onset is variable, but it is more frequent in young animals. In young animals, shortly after birth, the clinical signs are often symmetrical. In adults, clinical signs can have an asymmetrical presentation (LeCouteur & Grandy, 2005; Da Costa & Moore, 2010).

b) A – Anomalous

Neurological signs can result from malformations that directly involve the nervous tissue or the surrounding tissue. These are usually recognized early in life and signs tend to be non-progressive or slowly progressive. In some cases, vertebral malformations only result in neurological signs when the animal reaches adulthood (Denoix, 2005; Coates, 2012).

c) M – Metabolic

Animals of any age can be affected by metabolic disorders. The clinical onset of neurological signs is variable, but usually acute. Clinical signs are often subacute to chronic. Neurological signs are typically bilateral and symmetrical. Most of these conditions tend to wax and wane with time (Lorenz & Kornegay, 2004).

d) N – Neoplastic, Nutritional

Neoplasia is more common in older animals (more than 5 years old) but can occur at any age. Neurological signs are usually chronic and progressive, although acute deterioration can be seen. Determining factors of the clinical expression are: lesion size, histological nature, growth rate, associated inflammatory response and location. Neurological deficits can be asymmetrical or symmetrical, often suggesting a focal lesion (Brehm *et al.,* 1995; Lorenz & Kornegay, 2004).

As with metabolic diseases, neurological signs of nutritional diseases affecting the nervous system are typically bilaterally symmetrical. The onset of clinical signs is variable and often slowly progressive. The distribution of clinical signs can be diffuse or multifocal, as some nutritional diseases can affect selective areas of the CNS (LeCouteur & Grandy, 2005).

e) I – Inflammatory, Infectious, Idiopathic

The onset of sterile inflammatory (immune-mediated) or infectious diseases is cause-dependent and can be acute, subacute or a more insidious. Neurological signs usually progress without treatment, but, in some cases, may wax and wane early after onset. Neurological deficits can refer to a focal or multifocal lesion and can be asymmetrical or symmetrical.

Idiopathic disorders usually result in acute onset of non-progressive or regressive signs. Neurological deficits are variable, according to each syndrome. These disorders frequently affect the PNS (Greene, 1998; Tipold & Stein, 2010).

f) T – Traumatic, Toxic

The onset of traumatic disorders is usually peracute or acute. Signs usually remain static or improve over time. Neurological deficits can be symmetrical or asymmetrical and lesions can be focal or multifocal.

Toxicities often result in acute onset disease. Sings can be diffuse or bilaterally symmetrical and are non-progressive (McKee, 1990; Lorenz *et al*., 2011).

g) V – Vascular

Vascular disorders can result from loss of blood supply (ischaemia) or from haemorrhage into the nervous system. Their onset is typically peracute or acute, with non-progressive or regressive signs. Usually, deficits are initially focal and often asymmetrical. Haemorrhage may be an exception since it can be responsible for a more progressive onset over a very short period. Clinical signs usually regress after 24–72 hours, due to the diminution of the mass effect secondary to haemorrhage and reorganization or oedema resolution (Platt, 2010; Bartholomew *et al.*, 2016).

3.3. Diagnosis

The diagnosis of a CE disorder should be based on a complete patient history and clinical signs and subsequent performance of careful physical, orthopaedic, and neurologic examinations. A definitive diagnosis should be supported by radiological and advanced imaging results (Meij & Bergknut, 2010; Lanz & Rossmeisl, 2012).

3.3.1. Laboratorial tests

Basic laboratorial tests should be done in all animals. These should integrate: an hemogram, a biochemical and type II urine analysis, in order to discharge some diagnosis in the differential diagnosis list and evaluate anaesthesia associated risks (Chrisman *et al.*, 2003).

3.3.2. Neuroimaging

a) Spinal radiography

Spinal radiographs can be used to identify fractures, luxations, discospondylitis, some vertebral tumours, congenital abnormalities and degenerative changes, and provide circumstantial evidence of disc herniation (Brisson, 2010).

Radiographs should be executed with a sedated animal in order to achieve adequate positioning, except if spinal instability is suspected. In this case, only lateral views should be acquired initially, to avoid iatrogenic neural injury. For an accurate interpretation, the spine should not be rotated, and padding ties and troughs are recommended (Varejão *et al*., 2004; Kinns *et al.,* 2006).

The following aspects should be evaluated on plain radiographs: basic anatomy (number of vertebrae, presence of processes and ribs); alignment of the vertebrae in two planes; width of the IVD space, comparing each space with the disc space immediately cranial and caudal; shape and opacity of the IVF; integrity of the vertebral endplates, which should be examined for lysis and sclerosis indicative of infection; evidence of vertebral neoplasia (lysis, sclerosis and distortion of the bone outline); degenerative changes of the vertebrae or articular processes (see figure 3) (Kinns *et al*., 2006; Gavin & Levine, 2015).

Radiographic studies are often the first diagnostic test performed in dogs with clinical signs of lumbosacral disease. These should extend from the L4 to Ca2 or Ca3, to allow examination of the entire lumbosacral region and CE nerve roots. Both lateral and ventrodorsal views should be obtained. Bone lesions can later be delineated more accurately using CT (Meij & Bergknut, 2010).

Dynamic radiographs have limited diagnostic value for CE disorders due to individual and breed variations in normal range of motion, VC diameter, and size of the L7-S1 IVD space. However, these can help to show instability and abnormal movement at the lumbosacral joint (Mattoon & Koblik, 1993; McKee, 1993; Schmid & Lang, 1993). Survey radiographs may help to exclude discospondylitis, neoplasia, fracture, luxation,

idiopathic lumbosacral stenosis or other causes of pelvic limb dysfunction which may mimic a CES (Morgan & Bailey, 1990; Steffen *et al.*, 2007).

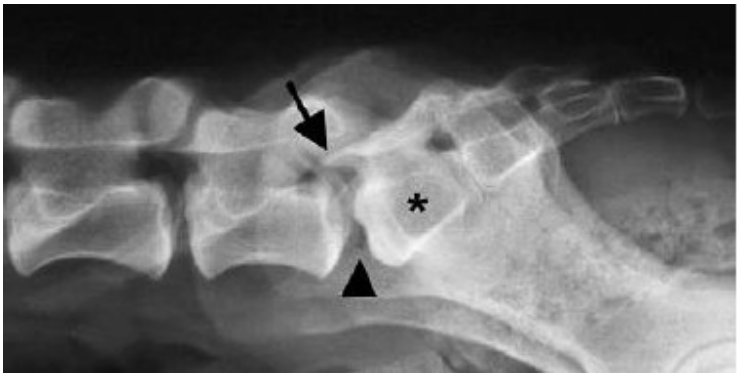

Figure 3 – Lateral radiograph of the lumbosacral region of a dog. Note the presence of a transitional vertebra (asterisk), elongation of the sacral lamina into the caudal aperture of L7 (arrow), and the vacuum phenomenon between L7 and S1 (arrowhead) (adapted from: Meij & Bergknut, 2010).

b) Myelography

Myelography is a satisfactory imaging study, when CT and MRI are not available. This technique is performed by injecting a non-ionic contrast medium (CM) (iohexol or iopamidol) into the atlanto-occipital (cisterna magna) or lumbar (L5-L6) interarcuate space (Hecht *et al.*, 2009). Advantages and disadvantages of this technique are expressed on table 9.

Injection of contrast media (CM) into the lumbar spine has a decreased risk of iatrogenic trauma and seizures, and improves delineation of CL, when compared to the cervical myelography. However, it is easier to introduce the needle into the atlanto-occipital subarachnoid space. Injection of contrast medium into the lumbar subarachnoid space may lead to epidural leakage, interfering with the assessment of the lumbosacral VC (Hecht *et al.*, 2009; Newcomb *et al.*, 2011).

Like survey radiography, myelography may be useful to exclude pathological changes that mimic the clinical signs of a CE disorder, such as neoplasia and IVD disease. Dogs have a variable and an unpredictable termination site of the dural sac. Therefore, myelography can only successfully be used to assess the CE if the patient's dural sac extends caudally into the sacrum, which is an impossible feature to predict (Meij & Bergknut, 2010). If the patient has the dural sac elevated from the ventral vertebral floor or a dorsally located compressive lesion, myelography will not be successful. Laterally CL can be missed in myelographic studies (Ramirez & Thrall, 1998). (Danielsson & Sjöström, 1999).

To interpret a myelogram, three basic pathological patterns must be recognized, according to the location of the lesion: intramedullary, intradural/extramedullary and extradural. For a precise interpretation, radiographs should be taken in ventrodorsal, lateral and right and left oblique views (Hecht *et al*., 2009; Newcomb *et al.*, 2011).

This procedure is not indicated if the general anaesthesia can't be performed safely, if there is an increase in the cerebrospinal fluid (CSF) pressure or an inflammatory lesion of the central nervous system. Since myelographic findings can raise doubts regarding the presence or absence of a lesion, CT should be used following myelography, since it allows a more precise anatomical lesion localization and allows to detect the CM in the subarachnoid space (Sharp & Wheeler, 2005; Gavin & Levine, 2015).

If needed, CSF analysis should be completed prior to performing a myelogram. CM induces a mild meningitis that complicates interpretation of CSF samples for up to one week after the study. CSF analysis is useful to dismiss inflammatory diseases. It can be collected either from the *cisterna magna* or the *cisterna lumbalis*, on the sedated animal, preferably caudally to the lesion (Griffin IV *et al*., 2009a; Brisson, 2010).

Advantages	Disadvantages
Easily available.	Contrast medium toxicity, leading to apnoea, asystole, meningitis, kidney failure, subarachnoid haemorrhage, lesion of the SC, seizures and neurological deterioration (10-20% of patients) or even death.
Higher sensitivity for lesion localization than plain radiographs.	Invasive procedure.
Helps to determine which patients need surgery and to choose the most appropriate surgery technique.	Insensitivity to some intraparenchymal conditions – infarction, oedema, syringomyelia.
	CSF samples are difficult to interpret after this study, as contrast medium induces a mild meningitis.
	Difficulty of the imaging study.
	Artefacts, due to injection of contrast medium into the epidural space.

Table 9 – Advantages and disadvantages of the myelography. (Adapted from: Hecht *et al*., 2009; Newcomb *et al*., 2011; Coates, 2012; Gavin & Levine, 2015).

c) Epidurography

For the evaluation of CE lesions, myelography is often inadequate, since the subarachnoid space ends at the conus medullaris (in the L6 region in most patients). Epidural contrast injection may help to delineate compressive CE lesions, especially at the L7/S1 IVD space. Epidurography has a low level of morbidity (Sisson *et al.*, 1992; Ramirez & Thrall, 1998).

After aseptic preparation of the chosen site (L7-S1, sacrocaudal junction or between one of the caudal intervertebral spaces), the contrast material is injected (iohexol or iopamidol; 0.1–0.2 ml/kg body weight). L7-S1 contrast injection can produce an unsatisfactory epidurogram, in some cases (Garcia-Pereira *et al*., 2010).

The epidural space has an irregular contour, when compared to the subarachnoid space. Consequently, the contrast columns of an epidurogram will appear rough and uneven, in comparison to the contrast columns of a myelogram. Multiple radiographic views are helpful (lateral films taken with the coxofemoral joints in neutral, flexed and hyperextended positions). In a normal epidurogram, contrast fills the epidural space evenly, with the pelvis in any position. Potential epidurogram's abnormalities include complete obstruction of cranial flow of CM past the L7-S1 space or dorsal deviation of the ventral contrast column over this space, which may be exacerbated on extended views and alleviated on flexed views. Occasionally, ventral deviation of the dorsal contrast column can occur (Roberts & Selcer, 1993; Dewey & da Costa, 2016).

d) Discography

Discography is also associated with low level of morbidity, but it is used less often than epidurography to evaluate L7-S1 disc lesions. This technique involves injection of iohexol or iopamidol (0.1-0.3 ml/kg body weight) directly into the NP of the disc. Then radiographs are taken (Kahanovitz *et al.,* 1986; Ramirez & Thrall, 1998).

The injection site must be aseptic prepared. The needle is placed directly into the L7-S1 disc, preferably under fluoroscopic guidance. The injection of contrast is not easy in a normal disc (Weinstein *et al*., 1988).

It is possible to perform a combination discography/epidurography procedure, using a single needle puncture: after the discogram is performed, the needle is withdrawn from the disc into the epidural space and additional contrast is injected and radiographs are taken (Robertson & Thrall, 2011; Dewey & da Costa, 2016).

e) Computed tomography (CT)

CT is a non-invasive and extremely rapid advanced imaging technique, being exposure to radiation its only known side effect. This cross-sectional imaging modality uses x-rays, a detector that produces raw numerical data, and a computer system that converts raw data into images (De Risio *et al*., 2000). CT creates image slices, which overcomes the problems associated with superimposition of structures on plain radiographs and allows the clinician to reformat images in other planes, helping in the spatial localization of lesions, and into 3D volume renderings, which can be helpful to assess complex fractures and other malformations. The possibility of three-dimensional reconstruction may aid surgical planning and be a useful post-surgical tool. The evaluation of a CT study should include an assessment of the normal anatomy and symmetry, identification of areas of contrast enhancement and examination for artefacts (Jones *et al*., 2000; Israel *et al.,* 2009).

CT has better contrast resolution than radiography, enhancing the distinction between different types of tissues and an enhanced conspicuity of small lesions that are not visible on radiographs and myelograms. CT allows visualization of lateral recesses, IVF, articular process joints and the VC. Inherent contrast provided by the epidural fat enables direct visualisation of individual nerve roots. However, it doesn't allow to differentiate the NP and the AF of the IVD (Kärkkäinen *et al.*, 1993; Robertson & Thrall, 2011).

In CE disorders, CT may provide helpful findings, such as loss of epidural fat within the VC or IVF, L7-S1 disc protrusion into the VC, presence of compressive soft tissues within the IVF or VC, thecal sac displacement, vertebral subluxation, osteophytosis, sacral osteochondrosis, hypertrophy of the interarcuate ligament and the vacuum phenomenon (Olby *et al.,* 2000; Suwankong *et al.*, 2006).

CT can be associated with myelography, increasing its sensibility to localize and characterize lesions. This technique helps to differentiate between intramedullary and extradural lesions. It also allows to identify disorders in the subarachnoid space. However, CT-myelography has the disadvantage of being invasive. CM toxicity is also a concern; however, the required dose is inferior to myelography in ¼ (Griffin IV *et al.,* 2009a; Dennison *et al.,* 2010).

f) Magnetic resonance imaging (MRI)

The interaction of atomic nuclei with magnetic properties and electromagnetic radiation and magnetic fields results in absorption and release of detectable levels of energy. This energy, through a computer assisted technique (MRI), may be used to construct a diagnostic anatomic image. MR images are interpreted diametrically. It is the physiochemistry of the tissue that creates the signal and contrast on the image (see table 10) (Thomson *et al.,* 1993; Gavin & Levine, 2015).

It is important to have experienced specialists analysing and interpreting MR images to ensure that the findings are complementary to the clinical findings. Metallic objects should be eliminated from the magnetic field, since they can be responsible for creating artefacts. Ferrous objects carried into the room can become lethal projectiles due to the extremely high field strength of some magnets. Flow and chemical shift artefacts are a complex series of artefacts, associated with MRI (Mayhew *et al.*, 2002; Cooper *et al*., 2014).

MRI findings in dogs with CE disorders are similar to those observed on CT. MRI provides excellent soft tissue contrast resolution (higher than CT), allows imaging in any plane without degradation of the image due to reformatting, allows for earlier detection of disc degeneration than CT and doesn't require the use of ionizing radiation. Longer duration and limited access constitute the main disadvantages of MRI. CT allows for a better distinction of osteophytes, articular process joint disease, soft tissue calcification, and soft tissue gas opacities, but is less sensitive for discriminating soft tissues within the spinal canal (De Risio *et al*., 2000; Jones *et al*., 2000).

Tissue	T1 ± contrast	T2 (fast spin-echo)	T2 FLAIR	STIR	Gradient-echo (T2)
Fat	Hyperintense	Hyperintense	Hyperintense	Hypointense	Hyperintense
CSF	Hypointense	Hyperintense	Hypointense	Hyperintense	Hyperintense
Tissue edema	Hypointense	Hyperintense	Hyperintense	Hyperintense	Hyperintense

Table 10 – The appearance of fat, CSF and tissue oedema on commonly used imaging sequences. Intensity is described relative to normal CNS parenchyma (source: Platt & Olby, 2012).

4. Causes of cauda equina disorders of dogs

4.1. Degenerative

a) Degenerative lumbosacral stenosis (DLS)

This is a common syndrome that usually affects middle-aged to older large-breed dogs. Stenosis of the lumbosacral canal occurs due to a combination of anatomic factors causing compression of the CE. Some authors refer a male predilection for this disease. German shepherds, Labrador retrievers, Border collies, or crossbreeds are frequently affected by this disorder and predisposed (Lang *et al.,* 1992; Janssens *et al.*, 2009).

Stresses on the AF can lead to proliferative changes and IVD protrusion, a Hansen type II IVD degeneration, with additional changes in the articulations and vertebral end plates. Narrowing of the IVD space has the consequence of a smaller intervertebral foramen. When there are osteophytes around the foramen, the L7 spinal nerve is entrapped (see figure 5). In some cases, ventral displacement of the sacrum occurs relative to L7, further narrowing the VC. Proliferation of the soft tissues of the joints, AF, and interarcuate ligament contributes to further compression. Extension of the joint causes additional folding of these tissues, thereby increasing pressure on the nerve roots (see figure 6) (Lorenz *et al.,* 2011; Suwankong *et al.,* 2008). Congenital vertebral anomalies, like lumbosacral transitional vertebrae, can be a predisposing factor for this disorder (Mattoon & Koblik, 1993).

Ventral spondylosis is commonly seen at the lumbosacral junction. By itself, it is a radiographic finding with little clinical significance. Some anatomical characteristics of the German Shepherd dog make them eight times more likely to develop DLS, such as having different orientation of vertebral articular processes (facets) in the caudal lumbar and lumbosacral regions of the spinal column, and a higher degree of facet joint tropism in this region, that translates in an asymmetry of the angle of orientation between left and right facets. These dogs have a smaller VC than other large breed dogs and a higher step between L7 and S1 (Denoix, 2005; Dewey & da Costa, 2016).

Lumbosacral pain is the most consistent and earlier finding. Pelvic limb lameness can be unilateral or bilateral. These clinical signs can be acute or chronic and episodic or persistent, which can mislead the clinician to diagnose the process as an orthopaedic condition. However, if not treated properly, clinical signs may progress to loss of proprioception in the PL, voluntary motor weakness in the distribution area of the sciatic nerve, and urinary/faecal incontinence (Tarvin & Prata, 1980; Janssens *et al.*, 2009).

The treatment may be surgical or conservative, being this decision based upon the severity of clinical signs, age of the patient and concurrent diseases. For patients experiencing their first episode of lumbosacral pain or whose pain is mild and intermittent, medical therapy is often recommended (Gödde & Steffen, 2007; Platt, 2010). Medical therapy consists of enforced rest up to 14 weeks, followed by a period of regular short walks to maintain muscle mass that often lasts several months. Anti-inflammatory medication and body weight reduction are recommended. Conservative treatment is recommended in older patients with multiple orthopaedic or systemic conditions (Meij & Bergknut, 2010; De Decker *et al.*, 2014).

An approximate success rate of 50% has been reported in dogs managed conservatively, which included animals showing improvement or resolution of clinical signs. Improvement is often transient, once resumption of normal activity frequently elicits recurrence of clinical signs, especially in working dogs. Long periods of rest may be associated with a worse prognosis if neurological deficits develop during that time (Ness, 1994; Dewey, 2013).

Indications for surgical management include failure of conservative management, severe pain, and severe neurologic deficits. Choices of surgical procedures include dorsal decompression, discectomy, facetectomy, foraminotomy, and fixation-fusion (De Risio *et al.,* 2001). The dorsal laminectomy over the L7-S1 interspace, usually combined with the removal of hypertrophied tissue, is the most used surgery technique. Lateral foraminotomy may be needed in cases of stenosis. Facetectomy may be needed in cases which the L7 nerve root is compressed. When a bilateral facetectomy is performed, surgical stabilization of the lumbosacral joint is recommended. In cases of lateralized foraminal stenosis, foraminotomy may need to be performed (Wood *et al.*, 2004; Suwankong *et al*., 2007).

After surgery, confinement is recommended for 3 to 4 weeks. Pain management and local application of cold packs may be used initially. Following confinement period, a period of exercise restriction with a gradual return to normal activity is recommended, for 6 to 8 weeks. Inadequate rest after surgery increases the risk of a poor recovery. Physical therapy, leash walks, swimming and underwater treadmill walks for 2 to 3 months may improve long-term outcome (Bagley, 2003; De Decker *et al.*, 2014).

The prognosis usually ranges from good to excellent with surgical intervention. Successful outcomes range from 66.7-95% of cases. Reported recurrence rates for DLS vary between 3 and 18%. In working dogs, the prognosis is less favourable due to higher performance demands. Resolution of incontinence is not likely to occur with either treatment type (Dewey & da Costa, 2016). Adequate bladder care and prevention of

faecal soiling on the perineal region may be necessary. In such cases, the patients should be kept on clean with soft bedding. (De Risio *et al.*, 2000).

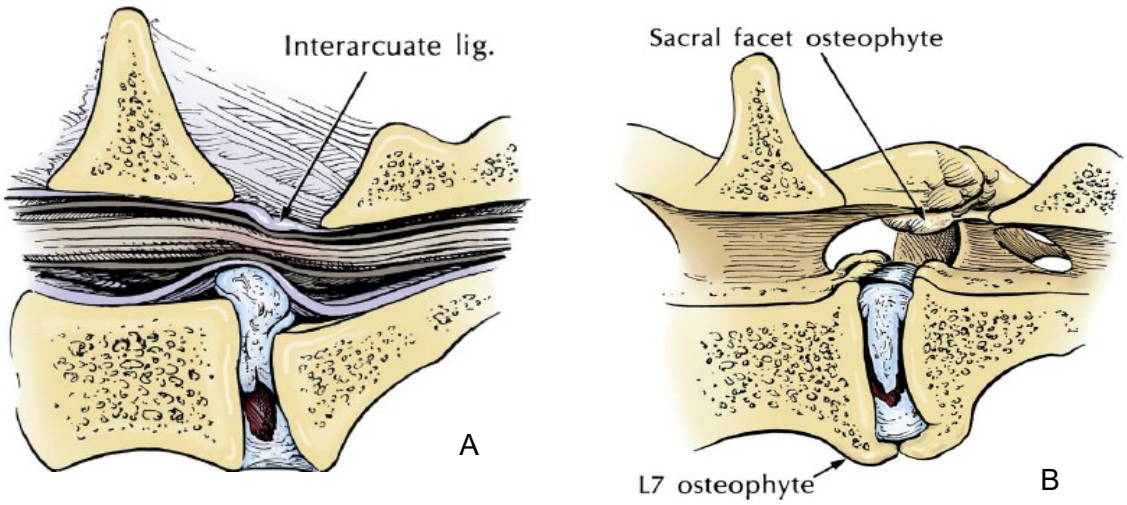

Figure 4 – (A) DLS. Compromise of intervertebral foramen by articular osteophytes that have formed on facet and vertebral body (adapted from: Lorenz *et al.* 2011). **(B)** DLS. Compression of the CE in a sagittal plane by the combined effects of disk herniation and ventral folding of the interarcuate ligament (adapted from: Lorenz *et al.* 2011).

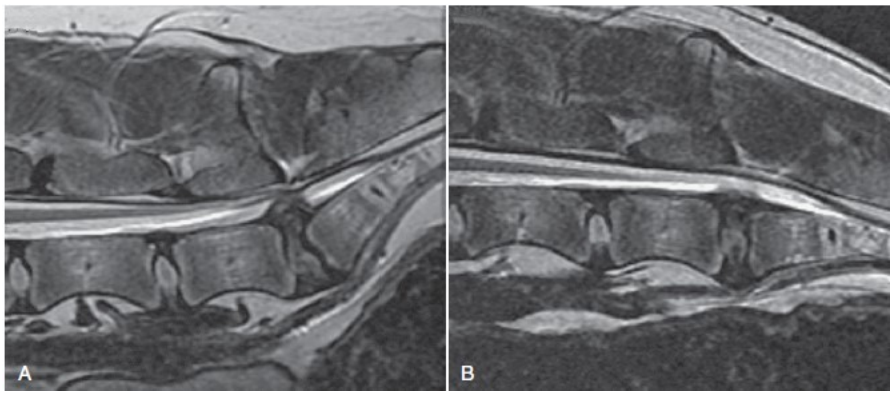

Figure 5 – Sagittal T2W MR images of the caudal lumbar spine in extension and flexion of a German shepherd with IVD degeneration of the L6-L7 and L7-S1 disk. A) Sagittal T2W MR image of the caudal lumbar spine in extension of a German shepherd dog with hyperesthesia over the lumbosacral spine demonstrating dorsal and ventral compression of the CE and loss of hyperintensity of CSF in the thecal sac. Note the IVD degeneration of the L6-L7 and L7-S1 disk. B) Sagittal T2W MR image from the same dog with the spine in flexion. This thereby demonstrates a dynamic lesion of the lumbosacral disk space. (adapted from: Lorenz *et al.* 2011; copyright: 2010 University of Georgia Research Foundation, Inc.).

b) Herniated lumbosacral discs

IVD herniation can result from extrusion of the NP to the VC (Hansen type I) or from the protrusion of the AF (Hansen type II) (Cooper *et al.,* 2014). Hansen type I disc extrusion (DH I) usually causes rapidly developing clinical signs (minutes/days), whereas Hansen type II disc protrusions (DH II) typically cause chronically developing clinical signs (weeks to months, sometimes years). There is a higher prevalence in young, small breed dogs, particularly in the chondrodystrophic breeds (Olby *et al.,* 2000).

Partial or total paralysis in both legs, with impaired sensitivity, urodynamics and micturition and sphincter dysfunction may develop acutely or subacutely, when the CE is compressed by a massive protrusion (DH II) or sequestration of a disc. Approximately 90% of disc herniations occur at the L4-S1 level (Cricun & Cricun, 1988; Gleave & MacFarlane, 1990). Dogs with coccygeal disc extrusion have severe pain reactions in the tail, sacrococcygeal and lumbosacral regions, without neurological deficits. When elevating their tails to defecate, the pain is usually exacerbated (Coates, 2004). Pelvic limb “root signature” may occur with lateralized disc extrusions in the caudo-lumbar region. The abnormal limb will be held in a flexed position and caudal extension of that limb elicits a painful response, due to stretching of irritated nerve roots. This is more commonly seen in Cocker Spaniel dogs (Fingeroth & Thomas, 2015).

The diagnosis is based upon signalment, history, clinical signs, and results of complementary diagnostic tests (especially CT and/or MRI) (Lamb *et al.*, 2002).

Treatment can be medical or surgical. Medical treatment consists of strict cage confinement, for 3-4 weeks. The cage should have enough space for the patient to change positions, but not enough space to walk or jump. Daily activity should be reduced to short walks to urinate and defecate. If the patient stops improving or gets worst, surgical options should be considered (Wood *et al.,* 2004). If necessary, anti-inflammatory drugs can be administered. Anti-inflammatory drugs should not be administered without cage confinement, since pain alleviation will result in increase of activity, which can lead to herniation of more disc material (De Risio *et al*., 2000). Success rates for medical treatment is approximately 50%, but initial success with subsequent disease relapse can be experienced by about 30%-50% of the dogs (Dewey & da Costa, 2016).

The prognosis depends upon the severity of the lesion, the animal’s neurological deficits and the type of treatment (Salger *et al*., 2014). Surgical treatment usually has positive outcomes, with the prognosis ranging from good to excellent (Downes *et al*., 2009). Usually, loss of nociception for more than 48 hours bears a worst prognosis

(Coates, 2000). The same occurs when there is no recovery of nociception for 2 to 4 weeks after the surgical intervention (Olby *et al.*, 2003).

4.2. Anomalous/Developmental

a) Congenital malformations of the cauda equina

Many vertebral malformations are incidental discovers and don't cause clinical problems. Some may be manifested by clinical signs of dysfunction, due to static or progressive spinal stenosis, as the patient grows into adulthood. Vertebral instability can be present. There are congenital malformations that are more relevant to the lumbosacral area (Bailey, 1975; Scott *et al.*, 2001), such as:

Lumbosacral transitional vertebra (LSTV)

A significant feature of transitional anomalies is the increased incidence of lumbosacral disc disease and nerve root compression that occurs in dogs with a lumbosacral transitional malformation (Damur-Djuric *et al.*, 2006).

The clinical signs are the ones typical for CES. It is a relatively common diagnosis for large breed dogs, especially German shepherd dogs, exhibiting back pain and neurologic dysfunction (Jones *et al.*, 1996; Widmer & Thrall, 2013).

LSTV is classified according to the radiological appearance of the transverse processes:

- Type-1, or lumbar type – indicates no sacroiliac fusion;
- Type-2, or intermediate – indicates partial fusion but with the tip of the process free;
- Type-3, or sacral – indicates complete fusion (Johnson *et al.*, 2005; Fluckinger *et al.*, 2006).

Flückiger and his collaborators investigated the association between the occurrence of LSTV and the CES in dogs. In their study, they had: 4000 control dogs without signs of CES, in which 3.5% had LSTV, and 92 dogs with CES, 16.3% had LSTV. They reported that, according to their results, the lesion causing CES always occurred between the last lumbar vertebra and the LSTV. Dogs with a LSTV were eight times more likely to develop CES than dogs without a LSTV (Flückiger *et al.*, 2006).

Idiopathic lumbosacral stenosis

Lumbosacral stenosis represents one of many causes of chronic CES. It occurs primarily in small to medium-sized dogs, more likely due to an abnormal embryologic formation of the neural arch. Abnormally thick laminae and articular facets of the vertebrae can be present since birth. Over time, due to degenerative changes of the soft tissues (e.g. thickened ligamentum flavum and facet joint capsule), the bony canal is narrowed, applying compressive forces to the CE. Clinical signs are variable and depend upon which nerve roots involved and the nature of the compromise. The prognosis is usually favourable with surgical decompression (Davidson, 1986; Kroll, 1992).

Spina bifida

Spina bifida occurs due to a failure of the neural arches to close dorsally to the SC, during embryological development. Alone, it does not result in clinical signs (Arias *et al.*, 2008; Kiviranta *et al.*, 2011).

Meningocele/myelomeningocele

A meningocele (MC) is a tube-like extension of the meninges, containing CSF, through a spina bifida defect of a vertebra that attaches to the overlying skin. The protruding meninges may extend dorsally to connect to the skin but remain as a closed, blind ending sac or remain “open” at the skin and leak CSF. Meningomyelitis due to contamination with environmental microorganisms (ostensibly bacteria) may occur. A meningomyelocele (MMC) is a less-common variant of a meningocele which contains cord segments. If a meningocele contains a large piece of skin-associated fat, it is called as lipomeningocele. These abnormalities are more common in the sacrococcygeal area of the spine and are frequently associated with spina bifida. English bulldogs and Manx cats appear to have predisposition to these disorders (Clark & Carlisle, 1975; Song *et al.,* 2016).

In animals with malformations without involvement of neural structures, the animal will likely have normal neurologic function. In some cases, changes in the hair coat and skin associated with a defect on the dorsal midline can be seen, such as: abnormal streaming of the hair (see figure 8), externally visible depressions and crusting cutaneous lesions associated with drainage of the CSF to the skin (Plummer *et al.,* 1993). With lesions involving the sacral and caudal nerves, gait may be normal, and predominant clinical signs consist of urinary and/or faecal incontinence, hyperalgesia, protrusion of the penis and decreased or absent tail tone. If the subarachnoid space is

continuous with the environment, chronic loss of CSF may lead to hyponatremia and hypochloraemia (Song *et al.*, 2016).

Definitive diagnosis of inflammatory CNS disease requires histopathology. Since biopsy is not usually performed, a presumptive diagnosis is obtained through a combination of CSF analysis, advanced imaging and serology for infectious aetiologies (Ricci *et al,* 2011; Ployart *et al.,* 2013).

In cases of MC or MMC, with a patent meningocutaneous tract that is allowing CSF to drain, medical management can be considered, by keeping the cutaneous lesion clean. Anti-inflammatory medication and antibiotics may be considered. Surgical exploration should be considered in cases with higher severity (Wilson *et al.*, 1979; Adzick *et al.*, 2011).

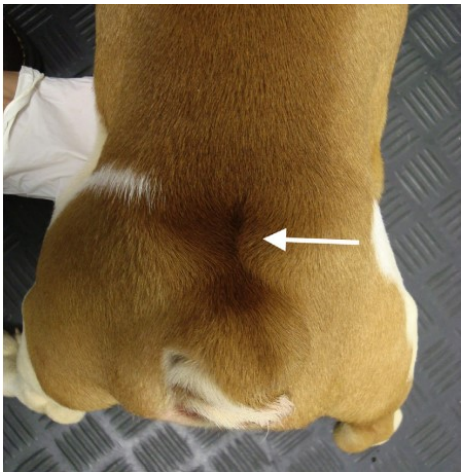

Figure 6 – A dorsal view of the haircoat anomalies (arrowed) of a 6-month-old English bulldog with meningomyelocele (adapted from: Song *et al.,* 2016).

Tethered cord syndrome

Tethered cord syndrome (TCS) is a rare syndrome in which excessive stretching and tension on neural tissues occur, due to abnormal attachments to the vertebrae or skin. May occur in conjunction with MMC and spina bifida or isolated, due to failure of the neuroectoderm to separate from the ectoderm. In the lumbosacral region, due to abnormal fixation of the filum terminale during the embryonic development, skeletal growth places caudal traction on the CE, causing neurological deficits (Fingeroth *et al.,* 1989, Shamir *et al.*, 2001).

Making a diagnosis of TCS in dogs is challenging due to the variations in the termination of the conus medullaris and filum terminale. MRI is the recommended imaging method to diagnose this malformation. Sometimes, clinical signs can be alleviated by surgical intervention (Plummer, 1993; De Decker *et al.*, 2015).

b) Developmental vertebral conditions of the cauda equina

Stenotic vertebral canal

In contrast to the congenital condition, this condition progresses until skeletal growth is completed. The stenosis can be relative or absolute. Lesions of the CE may result in a highly characteristic motor disturbance, known as intermittent neurogenic claudication (Tarvin & Prata, 1980; Suwankong *et al.,* 2008).

c) Dermoid sinus (pilonidal sinus)

Dermoid sinus is a congenital defect caused by incomplete separation of the skin and SC after closure of the embryonic neural tube where a sinus tract with a small cutaneous opening, on the dorsal midline, extends ventrally to various depths, sometimes reaching the subarachnoid space. This is commonly encountered in Rhodesian Ridgeback Dogs but has been reported in many other breeds. Common locations for the sinus are the cervical, cranial thoracic and, more rarely, sacro-caudal regions, although it can occur anywhere along the spine. It is thought to be a heritable condition. According to the extent of penetration of tissues under the subcutaneous layer, dermoid sinuses can be classified as types I-IV or I-V, as following (Motta *et al.*, 2011):

- Types I and II – extend to the supraspinous ligament. Deeply type II is a closed fibrous band (Dewey & da Costa, 2016).
- Type III – is more superficial than types I and II (Bowens *et al*., 2005)
- Type IV – extends to the VC, with or without an obvious laminal defect, and attaches to the duramater. Dermoid sinus type IV has rarely been reported in veterinary medicine (Kiviranta *et al.,* 2011).
- Type V – Is a true cyst, with no tract or skin opening and should be referred to as a dermoid cyst (Dewey & da Costa, 2016).

Rarely reported cases of spina bifida accompanied by dermoid sinus types IV and V can be found in the literature (Pratt *et al.,* 2000; Tong & Simpson, 2009).

Clinical signs are variable, from irritation due to bacterial infection of the sinus (most commonly), to evidence of meningitis and myelitis, if communication of the sinus with the meninges and neurological tissues occurs. Clinical signs usually start at a young age, although they can occur at any age. The sinus can often be palpated subcutaneously on the dorsal midline, as a thick fibrous cord (Cornegliani *et al.*, 2001; Cólon *et al.*, 2007).

Diagnosis is made upon signalment, history, clinical signs and imaging techniques. MRI proved to be a useful tool in further characterization of the lesion and planning an optimal surgical approach (Bowens *et al.,* 2005). However, MRI findings may not clearly indicate the full extent of the tract and can be misleading in cases of a dermoid cyst without a tract (Rahal *et al.,* 2008, Kiviranta *et al.,* 2011).

For treatment, broad-spectrum antibiotics based upon culture and sensitivity results and complete surgical excision of the sinus tract are recommended. Surgical excision without disruption of the sinus is the treatment of choice in neurologically affected cases, and 87.5% of the cases may show either partial or complete recovery. However, potential for recurring episodes exists (Lambrechts, 1996; White, 2003).

Prognosis is variable, according to the existence and extent of neurologic dysfunction and whether the entire sinus tract is successfully removed by surgical intervention. Usually, it goes from guarded to good (Miller & Tobias, 2003).

d) Sacral osteochondrosis

Sacral osteochondrosis is a developmental disease characterized by failure of the endochondral ossification of the articular epiphyseal and physeal cartilages. It is occasionally seen in young adult German Shepherds. It has also been associated with DLS. A 5:1 gender predisposition towards males has been shown, which may be related to the male's larger size and weight, resulting in greater biomechanical forces. A familial predisposition is possible, since this condition is rare in other breeds. The average age at presentation is 4 years old (Lang *et al.,* 1992; Mathis *et al.,* 2009).

Clinical signs usually are: intermittent lameness in one or both PL, a stilted gait, difficulty rising from a prone position and reluctance to jump. Clinical examination often reveals pain when extending the coxofemoral joints due to indirect lumbosacral extension, pain on palpation of the lumbosacral area and various neurological deficits (Hanna, 2001). It is diagnosed by clinical examination and imaging methods. Radiography, CT or MRI are useful to assist surgical planning (Glyde *et al.*, 2004).

Medical treatment has a variable degree of success. One study reported that conservative therapy was unsuccessful in the long-term, because removal of the compressive lesion could not be achieved with medical therapy (Hanna, 2001). Surgical treatment consists of dorsal laminectomy and removal of the osteochondrosis fragment, with or without dorsal annulectomy, and it is usually successful. CE compression may be caused by static or dynamic components or a combination of the two. Defining the relative importance of each component may help decide the most appropriate surgical intervention (Mathis *et al.,* 2009).

e) Synovial cysts

Extradural synovial cysts (ESC) originate from the zygapophyseal joint of the vertebral column. Histologically, these can be divided into synovial cysts (with synovium-like lining of epithelial cells) and ganglion cysts, which are thought to result from mucinous degeneration of the articular cartilage, presenting a collagenous capsule surrounding myxoid material. These cysts can appear in any region of the vertebral column; however, they are commonly observed in the lumbosacral region, associated with DLS. Most reported dogs with lumbosacral/caudo-lumbar synovial cysts are large-breed, middle-aged or older dogs. The presence of transitional vertebrae may be a risk factor (Lowrie *et al.,* 2014; Schmökel & Rapp, 2015).

ESC produce slowly progressive clinical signs compatible with a myelopathy or radiculopathy but can occasionally be clinically silent. Pelvic limb lameness or weakness with caudal lumbar/lumbosacral pain on palpation are the predominant clinical signs. Confirmation of the diagnosis is best done with MRI, revealing the cysts as well-circumscribed extradural masses, uni- or bi-laterally to the VC. They are hyperintense in T2-weighted images, with variable characteristics on T1-weighted images (Dickinson *et al.,* 2001; Sale & Smith, 2007).

Treatment is typically surgical, usually done at the same time as the decompressive surgery for lumbosacral stenosis, but medical management with activity restriction and anti-inflammatories is recommended initially. The outcome with surgical management is usually excellent (da Costa & Cook, 2016).

f) Arachnoid diverticulum (cyst)

These are CSF filled diverticula of the subarachnoid space. They are not true cysts as they do not have an epithelial lining. The natural pulsation of CSF is enough to produce a bulbous or tear-drop enlargement of the subarachnoid space, which can cause marked SC compression. An underlying aetiology is rarely found, but proposed causes include congenital malformation, trauma, inflammation and neoplasia. Usually, spinal arachnoid cysts are solitary, located dorsally or dorsolaterally, but ventrally located or circumferential arachnoid diverticula have also been reported. Dysraphism or syringomyelia may accompany this condition. Rottweilers may be over-represented, however spinal arachnoid diverticula have been described in other dog breeds and cats (Dyce *et al*., 1991; Skeen et al., 2003).

The onset of clinical signs of neurologic dysfunction is variable, from months to 13 years of age, but most reported dogs have developed signs of myelopathy in young

adulthood. In pugs, the average age of clinical manifestation is at 5-6 years of age. Male predisposition has been found (da Costa & Cook, 2016). Typically, clinical signs of this disorder are slowly progressive ataxia and paresis, depending on the location of the lesion. Spinal hyperpathia can occur, although it is not a prominent clinical feature. Urinary and faecal incontinence can also be present. Arachnoid cysts can also be acquired secondary to trauma or disc lesions (Rylander *et al*., 2002).

The diagnosis is based on spinal imaging. In some cases, the MRI is not clear and CT-myelography facilitates visualization. The typical image is a bulbous, contrast filled diverticulum, continuous with the contrast column in the subarachnoid space, with a characteristic teardrop shape. CSF analysis is typically normal (Jones *et al.*, 1994).

Glucocorticoid therapy can be attempted initially, providing, in some cases, stabilization and management of the disease for long periods of time. Surgical management involves resecting a portion of the meninges comprising the “cyst” wall, to relieve the pressure (surgical decompression) (Mauler *et* al., 2014; Galban & Perkins, 2018).

Prognosis with surgical treatment is usually good, but recurrence of signs can occur. Marsupialization of the “cyst” wall to surrounding tissues at surgery may help to prevent recurrence (Da Costa & Cook, 2016).

4.3. Neoplastic

Histogenetically different spinal neoplasms, classified according to their location into epidural, intramedullary and extramedullary-intradural, form an important category of the CES (Orendáčová *et al.*, 2001). Usually, spinal neoplasia affects patients with more than 5 years old, but some tumours, like lymphosarcoma, are commonly seen in young animals. Larger dogs are more commonly affected by spinal tumours. Boxers and Golden Retrievers have a higher prevalence of meningiomas (Dyce *et al*., 1991; LeCouteur & Grandy, 2010).

Epidural compression is usually accompanied by an early and severe pain, affecting approximately 96% of patients (Gilbert et al., 1978). Weakness of the lower extremities appearing early is reported by approximately two-thirds of patients. Sensory loss and sphincter dysfunction also occur in approximately two-thirds of patients (Orendáčová *et al.*, 2001).

Extradural tumours are the most prevalent type in dogs and intramedullary tumours the less prevalent. Extradural tumours typically cause pain with rapid neurological deterioration. These include vertebral body tumours and those that occupy the epidural space. Discovery of an extradural tumour indicates the possibility of a

primary mass elsewhere, particularly the mammary or thyroid glands, kidney or urinary bladder (LeCouteur, 2001; Chrisman *et al.*, 2003).

Intradural tumours lie within the dura mater but outside the SC parenchyma. The most common tumours in this category are meningiomas and nerve sheath tumours (neurofibroma, neurofibrosarcoma, schwannoma, lymphoma). Meningiomas usually cause pain or chronic discomfort with slowly progressive neurological deficits (Fingeroth *et al.*, 1987). These tumours can invade the SC in some cases. Nerve sheath tumours usually cause a chronic, progressive lameness. Invasion into the SC can occur, but local or distant metastasis is unusual. Both tumours can metastasize occasionally along the subarachnoid space (Brehm *et al*., 1995; Summers *et al.,* 1994).

Signalment, history, clinical signs and spinal imaging can be the base of a tentative diagnosis of spinal neoplasia. Bloodwork abnormalities are not common; however, hyperglobulinemia and proteinuria can be evident in the presence of a myeloma. In cases of vertebral neoplasia, imaging often reveals bony lysis with loss of cortical outlines, with or without evidence of bony proliferation. The vertebral structures should be carefully evaluated, because multiple myeloma tends to cause marked bone lysis with minimal proliferation. For soft-tissue spinal neoplasms, myelography, CT or MRI are helpful in the diagnosis and therapeutic planning, once plain radiographs are usually normal. However, a definitive diagnosis requires a histopathologic evaluation of the affected tissue(s). This is facilitated by surgical intervention, but fluoroscopic, or CT-guided needle biopsy may provide a diagnosis (LeCouteur, 2001; Niles *et al*., 2001; Rao *et al*., 2010).

Therapy can be divided into supportive and definitive treatment. Supportive treatment is directed against secondary sequelae of the tumour, while definitive therapies aim at the elimination of neoplastic tissue. Supportive therapy consists of anti-inflammatory doses of glucocorticoids (e.g. prednisone, 0.5mg/kg PO q 12hrs) and can be increased or decreased as needed, with or without the addition of pain-relieving drugs (e.g. narcotic) (Dewey & da Costa, 2016). Definitive treatment consists primarily on surgery and megavoltage radiation therapy. For lymphosarcoma and myeloma, chemotherapy is indicated. For vertebral neoplasms, surgical decompression can provide temporary relief of clinical signs, but usually there's an extensive bone destruction by the time of diagnosis. Radiation and chemotherapy alone are usually unsuccessful, however, radiation therapy after surgical intervention can increase the survival time (Orendácová *et al.*, 2001; Tyrrel & Davis, 2001).

The prognosis is usually poor, but since most patients are euthanized due to progressive dysfunction within several weeks to months, there is lack of meaningful prognostic information (Orendácová *et al.*, 2001; Platt, 2010).

4.4. Infectious/Inflammatory

a) Discospondylitis

Refers to an infection of the IVD and its contiguous vertebrae, usually by coagulase-positive *Staphylococcus* bacteria. Other bacteria, like *Streptococcus* and *Brucella* have been reported. Due to its zoonotic potential, Brucella needs to be ruled out by rapid slide agglutination test or card test. Fungal organisms, like *Aspergillus* spp., were reported, but these are uncommon. Defective immunocompetence can be a predisposing factor (Butterworth *et al*., 1995; Greene, 1998).

There is a predisposition for this infection in medium- to giant-breed male dogs of any age, however it has been encountered also in small-breed dogs and cats. Discospondylitis can affect any area of the spine, but L7-S1 disc space is the most common site affected (Ruoff *et al*., 2018).

The bacteria usually gain access to the disc by hematogenous spread from other foci in the body, of which the bladder is most common. Occasional cases associated with foreign body migration may occur, usually grass seeds. Iatrogenic cases following surgery are also recognized, especially when an implant has been used. Lesions can be multiple, with progressive nonspecific clinical signs over several weeks, but some dogs can develop signs acutely. Usually, dogs with lumbosacral discospondylitis, tend to walk with a stilted pelvic limb gait, manifesting hyperesthesia associated with the spinal lesion(s). Other nonspecific clinical features such as decreased appetite, weight loss, depression, fever, and reluctance to move may be present. Some patients may be severely paretic or plegic, others have no neurologic deficits or mild evidence of neurologic dysfunction, like proprioceptive deficits with or without mild paresis (Corrente *et al.*, 2010; Tipold & Stein, 2010)

The diagnosis is based on characteristic radiographic findings of the disease, with supportive historical and clinical feature. Radiographs reveal collapse of the affected disc space(s), bony lyses in the endplate regions of the affected disc space(s) and variable component of bone proliferation and endplate sclerosis, usually in chronic cases. Occasionally, the radiographs will be normal even though infection is present (occult discospondylitis). Clinical signs may appear 2-4 weeks before radiographic changes are evident. In some cases, bone scintigraphy, CT or MRI of suspected lesions are valuable (Stefanacci & Wheeler, 1991; Tipold & Stein, 2010). Bloodwork results are often normal, but occasionally there's evidence of leucocytosis on a complete blood count (CBC). Urinalysis may reveal urinary tract infection in some dogs. Bacteria can be cultured from blood, urine or affected disc spaces, by needle aspiration or surgical aspiration. On

nonambulatory patients with severe neurologic deficits, CSF examination and advanced imaging, preferably MRI, should be considered. Contrast radiographs can be useful in emergency situations, to evaluate potentially surgically correctable CL (Kornegay, 1986; Renwick *et al.*, 2010).

Medical treatment should be guided by culture and antibiotic sensitivity tests of the offending organism. Staphylococcus spp. is the most commonly found organism, so first-generation cephalosporins' or beta-lactamase-resistant penicillin drugs are often effective. For severely affected and paralyzed patients, intravenous antibiotics for the first 5-7 days should be administered, followed by the institution of oral antibiotics. If the response to therapy is minimal in the first weeks of treatment, concurrent treatment with antibiotics active against anaerobic bacteria should be considered. The treatment with antibiotics should last around 54 weeks. Analgesic drugs, such as codeine (1-2mg/kg every 6-8 hours), may be needed in some cases. Surgical intervention is recommended in patients with vertebral instability or CL (Moore, 1992; Schwartz *et al.,* 2009). Treatment for *B. canis* is with minocycline (25 mg/kg PO q24h for 2 weeks) and streptomycin (5 mg/kg IM or SQ q12h for 1 week) or gentamycin (2 mg/kg IM or SQ q12 h for 1 week). There is potential for zoonotic spread, and recurrence is common. It may be wise to castrate male dogs with *B. canis* infection as the testes can act as a reservoir of infection (Greene, 1998; Corrente *et al.*, 2010). For infections by Aspergillus spp., itraconazole is recommended (Butterworth *et al.,* 1995; Watt *et al*., 1995).

For bacterial discospondylitis, prognosis is usually favourable, especially in cases with no or mild neurologic deficits. In dogs with resistant bacteria or with severe neurologic deficits, prognosis is more guarded. Generally, it is expected to see clinical improvement in the first week of antibiotic therapy. It is recommended to monitor the progress of the disease with follow-up radiographs of the affected disc spaces, every 1-2 months. For fungal discospondylitis, the prognosis is poor, once the infection is usually disseminated at the time of diagnosis (Renwick *et al.*, 2010).

b) Myelitis/Meningomyelitis

Myelitis refers to an inflammatory process compromising only the SC, but not the meninges. Meningomyelitis refers to an inflammatory process involving both the SC and the meninges (Griffin *et al.*, 2008). Clinically, the two diseases can be distinguished by the presence or absence of spinal pain. Patients with myelitis usually don't have spinal pain, since the SC doesn't have nociceptors. Neurologic deficits, in cases of myelitis, are asymmetric, close to the asymmetry seen in vascular diseases, such as fibrocartilaginous embolic myelopathy. Depending on the lesion localization, spinal

reflexes may or may not be reduced. Clinical signs associated with meningomyelitis can include paresis or paralysis, pain and proprioceptive deficits. The disease can have subacute (3-7 days) and chronic presentations. It is important to keep these diseases on the list of differentials, because a CSF analysis is required to diagnose them (Orendácová *et al.*, 2001; Parry *et al.*, 2009).

Non-infectious meningomyelitis or meningomyelitis of unknown aetiology is diagnosed by ruling out infectious agents with serology, PCR and CSF culture investigations. Treatment consists on the use of corticosteroids and other immunosuppressive agents (Murata *et al.,* 2012).

Several organisms can cause infectious diseases that may result in clinical signs of myelopathy, such as viruses (e.g. canine distemper virus), bacteria (e.g. *Staphylococcus* spp., *Streptococcus* spp., coliforms.), fungi (e.g. *Cryptococcus, coccidioidomycosis*), rickettsia (e.g. *Ehrlichia, Rickettsia,* Rocky Mountain spotted fever); protozoa (*Toxoplasma, Neospora*), parasites (*Dirofilaria, Cuterebra*), algae (*Prototheca*) (Tipold & Stein, 2010; Armstrong *et al*., 2012). The route of infection can be hematogenous or direct (e.g. bite wound, foreign body). Myelography usually shows evidence of extensive purulent epidural fluid accumulation, typically over several vertebral lengths. These can be focal or multifocal. CT can be used, but MRI is preferable to image this condition. On CSF analysis, a moderate neutrophilic pleocytosis with elevated protein concentration is a common finding. CSF culture is typically negative; blood and epidural fluid cultures are often positive. Vertebral osteomyelitis or discospondylitis may or may not be evident of radiographic studies (Parry *et al.*, 2009; Olby, 2010).

Rapid diagnosis and aggressive medical and surgical treatment are important for successful management of this disorder. Patients with mild neurologic deficits and spinal pain can be successfully managed with appropriate antibiotics (Platt, 2010). The presence of spinal epidural empyema is considered by most sources as a surgical emergency, since delayed diagnosis and treatment often leads to poor outcomes (Chrisman, 2003; Platt & Olby, 2012).

4.5. Ischemic/Vascular

a) Intermittent neurogenic claudication

This physiological phenomenon has been proposed to explain exercise-associated pain and lameness, observed in some patients with CE lesions (Markwalder, 1993; Porter, 1996). During exercise, radicular blood vessels dilate to meet metabolic demands. If the IVF are stenotic, blood vessels' dilation can lead to secondary compression and ischemia of the associated nerve roots. Hyperesthesia is usually the predominant clinical manifestation (Kobayashi *et al.*, 2008).

b) Fibrocartilaginous embolism

Fibrocartilaginous embolism (FCE) is an acute ischemic myelopathy, primarily of large or giant breed dogs, which results from occlusion of blood vessels within the SC parenchyma or the adjacent leptomeninges by masses of fibrocartilage. It is frequently associated with trauma or vigorous exercises (Cook, 1988; Neer, 1992).

Neurological deficits are usually asymmetric with acute clinical signs and frequently indicate a very lateralized or focal lesion. LMN deficits are common, but hyperesthesia is unusual. Attention must be given to other systemic diseases that might also give rise to emboli in the absence of FCE (Cook, 1988; Bartholomew *et al.*, 2016).

The diagnosis is based upon history, clinical signs and elimination of other causes of acute compressive myelopathy, such as trauma and IVD herniation. MRI can be useful to evaluate the parenchyma with detail and confirm the diagnosis. Treatment is mainly supportive: rest in cage for 2-3 weeks (Cauzinille, 2000; Urfer, 2007).

Patients with LMN deficits secondary to FCE have a more guarded prognosis than those with UMN deficits. According to Bartholomew and his collaborators, prognosis for recovery of ambulation can be good to excellent (85% of cases), most within 3 weeks, however neurological deficits may persist (49.1% of cases). If nociception is absent, the rate of recovery is lower (10% of cases); however, this data is likely biased by limited follow-up in more severe cases (Neer, 1992; Bartholomew *et al.*, 2016).

4.6. Traumatic

This category includes injuries associated with spinal trauma include SC contusion, vertebral fracture or luxation and traumatic IVD herniation. Patients with spinal trauma usually have concurrent injuries to other major organ systems, requiring rapid and thorough assessment and survey for evidence of other life-threatening injuries (Bagley, 2000).

In case of trauma, the nerve roots of the CE may experience considerable stretching and remain structurally and functionally intact. However, the sparse amount of bone available caudal to the injury for placing implants is often a challenge for surgical repair of fractures/luxations. Sacral fractures often accompany multiple pelvic fractures. Lateralized sacral fractures in dogs usually have a better prognosis for neurologic recovery than the more central ones (Sharp & Wheeler, 2005; Coates, 2012).

Traumatic injuries to the lumbosacral plexus, resulting in pelvic limb monoparesis, most often affect the sciatic truck or nerve. The affected animal should be able to bear weight on the affected limb, but will walk with the paw knuckled over, the stifle joint will not flex, and the tarsus and digits will not flex or extend. In addition, the hock is usually ‘dropped’. Sensation is affected in the entire limb except for the medial aspect (and medial digit) which is supplied by the saphenous branch of the femoral nerve. The flexor reflex is severely affected due to the inability of the patient to flex its stifle, hock or digits. If the medial digit is stimulated, a mild flexion of the hip (femoral nerve) can be observed. Nociception should be checked in all digits to assess the severity of the lesion. A lack of nociception in the digits innervated by the sciatic nerve indicates severe injury (neurotmesis) and a poor prognosis for recovery (McKee, 1990; Shores, 1992).

The peroneal nerve is exposed to traumatic injury as it crosses the lateral aspect of the stifle. The more common causes of injury at this site include intramuscular injections, pressure from orthopaedic casts and surgery of the stifle joint (cruciate ligament repair). Isolated tibial nerve lesions are rare but may occur following intramuscular injection into the thigh muscles. In most animals, tibial nerve lesions occur in association with peroneal or higher (main trunk) sciatic nerve injuries. Pure peroneal or tibial nerve signs are not common (Sharp & Wheeler, 2005; Platt & Olby, 2012).

Spinal cord segments L4–L6 and femoral nerve roots within the vertebral canal can be affected. In these cases, bilateral disease is more common; true femoral monoparesis is a rare occurrence. Peripheral femoral nerve injuries are uncommon because the nerve and its roots are well protected by the sublumbar musculature. However, extreme extension of the hip causing iliopsoas muscle tears can damage the femoral nerve, resulting in femoral neuropathy (Stepnik et al., 2006). Dysfunction of the

femoral nerve causes monoparesis with severe gait abnormalities. The patient cannot bear weight on the affected limb and carries it flexed at the stifle. There is little hip flexion and the patellar reflex is severely decreased or lost. Neurogenic atrophy of the quadriceps muscle usually develops. With severe lesions there is anaesthesia over the medial aspect of the pelvic limb and medial digit. Nociception should be checked in the medial digit to assess the severity of the injury (Shaw *et al.*, 2017). Preservation of deep pain sensation (nociception) in the areas of innervation of the CE nerve roots is a favourable prognostic indicator (Chrisman *et al.,* 2003).

The treatment is chosen according to the severity and nature of the underlying primary injury and adapted to the individual patient (see table 12). Neurologic recovery is dependent on the type and severity of primary injury and the effectiveness and timeliness of the treatment of secondary injury (see table 13). Some patients with loss of nociception can recover neurologic function with treatment. Vertebral trauma and luxation have a poorer prognosis, when compared to traumatic disc herniation. Prognosis is also dependent upon other concurrent injuries to other organ systems, so cumulative effect of each individual injury must be considered (Shores, 1992; Shaw *et al.*, 2017).

Primary Injury	**Treatment**	
	Conservative Therapy	**Surgical treatment**
Vertebral fractures and luxations	External coaptation and strict cage rest for 6-8 weeks. Many materials can be used for splint construction, but the splint will not aid proper healing unless the entire vertebral region is immobilized.	Includes surgical decompression, reduction and/or fixation**.** Patients with minimal voluntary motor function or complete paralysis, evidence of unstable fractures and with progression of neurologic signs despite appropriate medical treatment, are indicated for surgery.
Traumatic IVD herniation	Indicated for stable patients with intact voluntary motor function: strict cage rest (3-4 weeks) for resolution of SC edema and inflammation. NSAIDs can be used for initial pain relief.	Most traumatic disc herniations (“type III” discs) are noncompressive and don’t benefit from surgical decompression. If compression is seen, depending on the site and severity of herniation, surgical treatment may be indicated: dorsal laminectomy, hemilaminectomy or ventral slot procedure.
Nerve root injury	Strict cage rest (3-4 weeks). NSAIDs can be used for initial pain relief.	Not indicated.

Table 11 – Therapy options according to the type of primary injuries that can (adapted from: Dewey & da Costa, 2016).

Maintenance of perfusion	Aggressive fluid resuscitation should be implemented; nonresponsive patients to fluid therapy may benefit from vasopressors. Oxygen and ventilation should be closely monitored, and deficits addressed.
Corticosteroids	Corticosteroids have minimal antioxidant effects and are unlikely to have significant neuroprotective effects, however they may reduce discomfort in some cases.
Physical rehabilitation	This type of therapy is important in the recovery of patients with SC injury, by minimizing muscle atrophy, maintaining proper range of motion and reducing discomfort and can possibly hasten the recovery when the injury is reversible. Locomotor training can promote plastic changes in the sensorimotor circuits below the level of injury, leading to improvement of the gait pattern, even in animals with SC transection.

Table 12 – Therapy for secondary injury due to SC trauma (adapted from: Sharp & Wheeler, 2005).

CHAPTER III – Retrospective study

1. Goals

In this chapter, the study sample will be characterized. The progress of clinical signs, the cause of the CE lesion, the diagnostic methods, the type of treatment, the recovery post-treatment and the recurrence of clinical signs will also be analysed.

2. Materials and methods

2.1. Selection and acquisition of clinical cases

The descriptive statistical analysis was based on the data of 11 animals that were examined by the neurological service of Hospital Veterinário da UTAD (Vila Real, Portugal), with clinical signs compatible with CES, between August 2017 and November 2018. All the exposed data was acquired from the individual clinical processes of each patient, the QVET program and the neurological service of HV-UTAD.

All patients were first submitted to a general physical examination and complementary laboratory exams. Upon suspicion of a lumbosacral lesion, plain radiographs were obtained. Then, the clinical cases were referred to and evaluated by the neurological service of HV-UTAD and submitted to a neurological examination to determine the severity and localization of the lesion. All the animals of this study were submitted to a CT scan, to confirm the diagnosis and establish the cause of the CES. It was used a multi-slide helicoidal CT system – *GE Brivo CT325* (Beijing, China).

As for the causes, in this sample, 6 of 11 (55%) patients were degenerative, 1 of 11 (9%) was traumatic, with possible sciatic nerve injury, 1 of 11 (9%) was infectious and 1 of 11 (9%) was neoplastic, with a primary tumour compatible with osteosarcoma. MRI was recommended in 2 of the 11 (18%) cases, to confirm the suspicion of fibrocartilaginous embolism, but the owner's decided they wouldn't do it.

Preanesthetic laboratory exams were requested to evaluate and determine the patients' general health and aesthetic risks during the CT scan. In the anaesthetic plan, it was used a pre-medication with morphine (IM; 0,2 mg/kg), isolated or combined with acepromazine (IM; 0,01 mg/kg). For induction, it was used a combination of butorphanol (IV; 0,1-0,4 mg/kg), diazepam (IV; 0,2 mg/kg) and propofol (IV; 2-4 mg/kg); Isoflurane was used for the maintenance of the anaesthesia.

All patients were submitted to conservative treatment: rest in cage for 2-3 weeks, physiotherapy 3 times a day and supplementation with neurobion™ (100 mg of vitamin B-1, 100 mg of vitamin B-6, and 200 mcg of vitamin B-12 per tablet) and everfit plus™

(200 mg of vitamin C, 30 mg of vitamin E, 6mg of beta-carotene, 300 mg of magnesium, 15 mg of zinc, 200 µg Selenium, 100 µg Chromium and 10mg of Co-enzyme Q-10 per tablet). Only 5 animals (45%) showed up for the revaluation. 2 of 11 (18%) were euthanized.

2 of 11 (18%) were submitted to surgery, which was successfully performed. After surgery, these animals were evaluated again by the neurology service of HV-UTAD. For recovery, rest in cage for 2 weeks with short walks 2-3 times a day was recommended, as well as supplementation with neurobion™ and everfit plus™ and physiotherapy 3 times a day, for 4 weeks.

3. Statistical analysis

Statistical analysis and graphics contained in this practical work were performed using Microsoft Office Excel 2016 program. The statistical tests used were the average and standard deviation.

3.1. Results

3.1.1. Signalment

Case No.	Breed	Gender and neutering status		Age	Weight
1	Giant (Great Dane)	Female	Neutered	6 y/o	38,00 kg
2	Medium (mix breed)	Male	Intact	6 y/o	13,80 kg
3	Medium (Epagneul Breton)	Female	Neutered	6 y/o	16,30 kg
4	Medium (mix breed)	Male	Intact	10 y/o	13,30 kg
5	Large (Estrela Mountain Dog)	Female	Neutered	5 y/o	25,60 kg
6	Small (French Bulldog)	Female	Intact	5 y/o	14,90 kg
7	Large (Labrador Retriever)	Male	Intact	12 y/o	41,40 kg
8	Large (Labrador Retriever mix breed)	Male	Intact	3 y/o	45,00 kg
9	Small (French Bulldog)	Female	Neutered	5 y/o	12,10 kg
10	Large (Labrador Retriever)	Male	Intact	7 y/o	34,40 kg
11	Large (Labrador Retriever)	Male	Intact	11 y/o	34,60 kg

Table 13 – Identification of the studied patients by breed, gender and neutering status, age and weight.

Breed

Comparing breed sizes, there is a higher prevalence for CES in large and giant breeds, representing 55% (6/11) of the cases (see diagram 1).

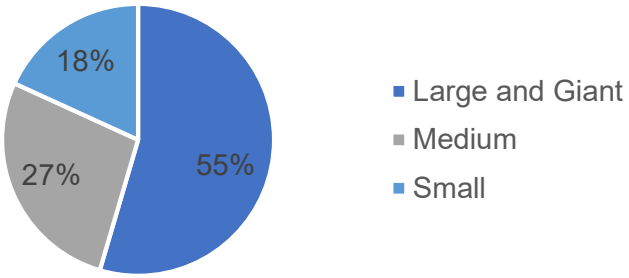

Diagram 1 – Graphic representation, in percentage, of large/giant, medium and small breeds.

Gender

In this sample, males 55% of the patients were male and 45% were female.

Age

The age of the animals in this study ranged from 3 to 12 years old, in average, 6,9±2,8 years old.

Weight

In this sample, the weight of the animals ranged from 12,10 kg to 45,00 kg.

3.1.2. Neurological examination

Case No.	Observation			
	Mental status	Posture	Gait/movement (PL)	Tail position
1	N	N	Reluctance to move, ataxia	N
2	N	N	Reluctance to move, ataxia	N
3	N	N	Reluctance to move	N
4	N	N	Reluctance to move	N
5	N	N	Reluctance to move	Tail held low
6	N	N	Paraparesis	N
7	N	N	Paraparesis	N
8	N	N	Acute LMN paraplegia	N
9	N	N	Paraparesis	N
10	N	LPL flexed	Reluctance to move, monoparesis (LPL)	N
11	N	LPL flexed	Paraparesis	N

Table 14 – Mental status, posture, gait/movement and tail position evaluation of the patients in study. Key: N – normal.

Observation

All patients showed gait disturbances: 54% (6/11) manifested some degree of paresis/plegia; reluctance to move was present in 45% (5/11); and ataxia in 18% (2/11) (see diagram 3). 18% (2/11) of the patients showed abnormal posture – pelvic limb root signature. Abnormal tail position (lowered) was verified in 9% (1/11) of patients.

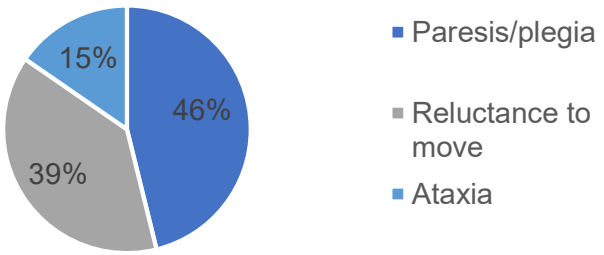

Diagram 3 – Gait disturbances, expressed in percentage, observed in the animals in study.

Case No.	Muscle tone (PL)	Urinary/ faecal function	Sensory function		
			Hyperesthesia	Nociception	Tail elevation
1	RPL (+2) LPL (+2)	N	TL + LS	RPL (+2) LPL (+2)	Pain
2	RPL (+1) LPL (+1)	N	LS	RPL (+2) LPL (+2)	Pain
3	RPL (+2) LPL (+2)	N	-	RPL (+2) LPL (+2)	-
4	RPL (+2) LPL (+2)	N	LS	RPL (+2) LPL (+2)	-
5	RPL (+1) LPL (+1)	N	LS	RPL (+2) LPL (+2)	Pain
6	RPL (+2) LPL (+2)	N	-	RPL (+2) LPL (+2)	-
7	RPL (+1) LPL (+1)	N	LS	RPL (+2) LPL (+2)	Pain
8	RPL (+2) LPL (0)	LMN	LS	RPL (+2) LPL (0)	-
9	RPL (+1) LPL (+1)	N	-	RPL (0) LPL (0)	-
10	RPL (+2) LPL (+1)	N	At coxofemoral extension	RPL (+2) LPL (+2)	-
11	RPL (+2) LPL (+1)	N	LS	RPL (+2) LPL (+2)	-

Table 15 – Evaluation of PL muscle tone, urinary/faecal function and sensory function of the patients in study. Key: (0) absent; (+1) decreased; (+2) normal; (+3) exacerbated; (+4) Clonus; (-) unpresented; (+) present; (X) unexamined.

Case No.	Proprioceptive placing	Flexor reflex (PL) (L6-S2)	Patellar reflex (L4-L6)
1	RPL (0/+1) LPL (0/+1)	RPL (+1) LPL (+1)	RPL (+2) LPL (+2)
2	RPL (+1) LPL (+2)	RPL (+1) LPL (+2)	RPL (+2) LPL (+2)
3	RPL (0) LPL (+2)	RPL (+1) LPL (+2)	RPL (+3) LPL (+3)
4	RPL (+2) LPL (+2)	RPL (+2) LPL (+2)	RPL (+2) LPL (+2)
5	RPL (+2) LPL (+2)	RPL (+2) LPL (+2)	RPL (+3) LPL (+3)
6	RPL (+1) LPL (0)	RPL (+1) LPL (+1)	RPL (+2) LPL (+2)
7	RPL (0) LPL (0)	RPL (+1) LPL (+1)	RPL (+3) LPL (+3)
8	RPL (+2) LPL (0)	RPL (+2) LPL (0)	RPL (+2) LPL (0)
9	RPL (0) LPL (0)	RPL (+1) LPL (+1)	RPL (+1) LPL (+1)
10	RPL (+2) LPL (0)	RPL (+1) LPL (+1)	RPL (+2) LPL (+3)
11	RPL (+2) LPL (0)	RPL (+2) LPL (0)	RPL (+2) LPL (0)

Table 16 – Results of proprioceptive placing and flexor (PL) and patellar reflexes of the patients in study. Key: (0) absent; (+1) decreased; (+2) normal; (+3) exacerbated; (+4) Clonus; (-) unpresented; (+) present; (X) unexamined.

Muscle Tone

In this sample, 6/11 (55%) animals had a decreased muscle tone when presented for evaluation.

Sensory function

73% (8/11) of patients manifested hyperesthesia upon palpation of the lumbosacral area or coxofemoral extension. Within these, 50% manifested pain upon tail elevation (Cases 1, 2, 5 and 7).

Nociception was lost in 2 out of the 11 cases (18%) – cases number 8 and 9. These patients were euthanized.

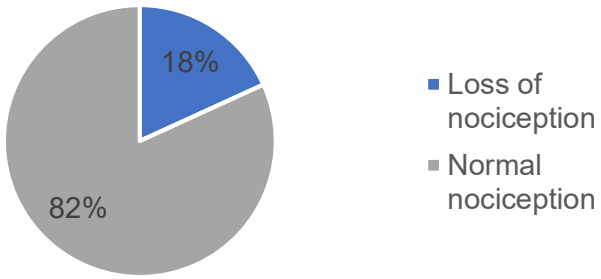

Diagram 4 – Evaluation of nociception, in percentage, of the patients in study.

Evaluation of proprioception, flexor (PL) and patellar reflexes

Proprioception was evaluated by the proprioceptive placing test. It was decreased/absent in 9 out of the 11 cases (82%). Flexor reflex was decreased/absent in 9 of 11 patients (82%). Patellar reflex was hyper-reflexive in 4 of 11 patients. It was decreased/absent in 2 of the 11 patients.

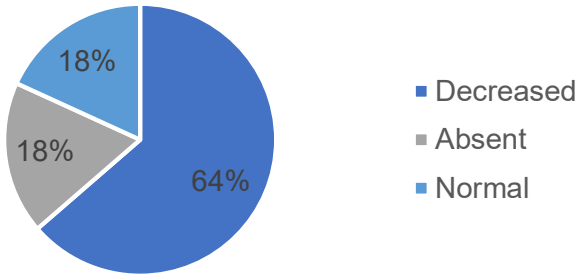

Diagram 5 – Results of flexor reflex evaluation, in percentage.

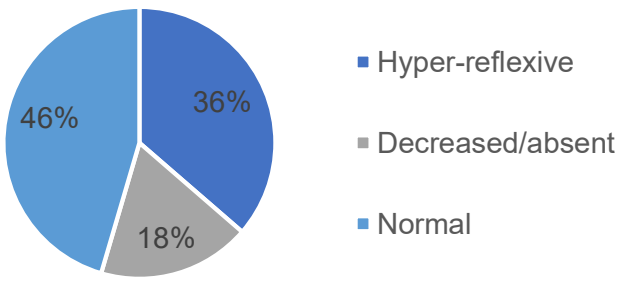

Diagram 6 – Results of patellar reflex evaluation, in percentage.

3.1.3. Complementary diagnostic tests

All patients were submitted to routine complementary laboratory tests (CBC, MHCT, BCHM, total proteins and urinalysis type II). Cases number 5, 8 and 11 showed clinically relevant alterations in one or more complementary tests and further testing was requested (see table 18). All patients were submitted to radiography and CT scan (see table 19).

Case No.	CBC and MHCT	Total proteins	Urinalysis (type II)	Urine culture	Others
5	↑WBC, NEU, MONO, GLOB ↓ERI, HCT, HGB, MPV, ALB	↑α-2 and β-GLOB ↓ALB and α-1 GLOB ↓ALB/GLOB ratio	**Macro**: yellow, turvid. **Micro**: Proteins (+1) Blood (+4) Hemoglobin (+4) **Sediment**: LEU (+3) Bacteriae (+4) ERI (+2)	(+) *E. coli, streptococcus aureus*	**Articular synovial fluid:** (+) *streptococcus aureus*
8	↑MONO, NEU, UREA, ALT ↓EOS, RETIC	N	N		**Blood smear:** (+) Babesia spp.
11	N	↑β-1 and β-2 GLOB ↓α-1 and α-2 GLOB Normal ALB/GLOB ratio	N		**Histopathology:** Eco-guided cytology of the L7 spinal apophysis: highly compatible with osteosarcoma.

Table 17 – Results of complementary laboratory tests requested for each case. Key: ↓ - decreased; ↓ - increased; N – normal; (+) positive for.

Case No.	Radiography	CT	Type of Lesion
1	**L6-S1:** Spondylosis deformans; IVS reduction.	**L6-L7 and L7-S1:** DH II associated with lumbosacral stenosis (see image 7).	Degenerative
2	**L4-S1:** Spondylosis deformans **L7-S1:** IVS reduction.	**L7-S1:** DH II with partially mineralized material. Stenosis of the right sided foramina.	Degenerative
3	**L6-L7; L7-S1:** IVS reduction.	**L6-L7; L7-S1:** DH II without clinical relevance.	Vascular. Suspicion for FCE.
4	**L6-S1:** IVS reduction.	**L6-L7:** DH II with partially mineralized material.	Degenerative
5	**L7-S1:** discospondylitis; increased IVS.	**L7-S1:** increased IVS. Extensive and irregular bone lysis of the endplates and vertebral bodies (see images 8 and 9).	Infectious
6	**L8-S1:** IVS reduction.	**L8-S1:** DH I with highly mineralized material. Severe CE compression. (see image 10).	Degenerative
7	**L7-S1:** IVS reduction.	**L7-S1:** DH II with associated lumbosacral stenosis. Loss of definition of the CE nerve roots due to reduced epidural fat. (see image 11).	Degenerative
8		**L7-S1:** DH II without clinical relevance.	Vascular. Suspicion for FCE.
9	**L7-S1:** IVS reduction.	**L7-S1:** DH II with associated lumbosacral stenosis.	Degenerative
10		**L7-S1:** DH II without clinical relevance.	Traumatic. Suspicion for sciatic nerve lesion.
11	**L7-S1:** IVS reduction; osteophytes. **L7:** lysis.	**L4-L5; L5-L6; L7-S1:** DH II without clinical relevance. **L7**: osteolysis, high risk of fracture. (see images 12 and 13).	Neoplastic. Compatible with osteosarcoma.

Table 18 – Neuroimaging results and lesion characterization for the studied cases.

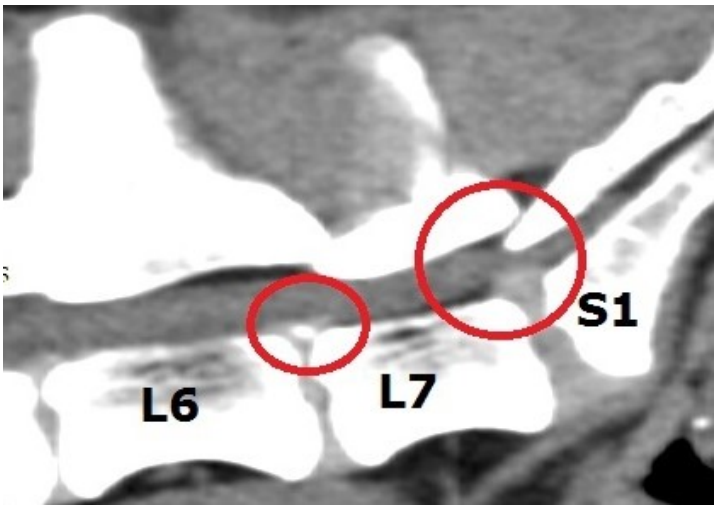

Figure 7 – Sagittal CT image, in soft tissue window, of the lumbosacral area (L6-S2) of patient from case number 1. Note the AF protrusion, with partially mineralized material, for the vertebral canal between L6-L7 and L7-S1. It is possible to observe the elongation of the lamina of the sacrum into L7 (Image gently provided by the neurological service of HV-UTAD).

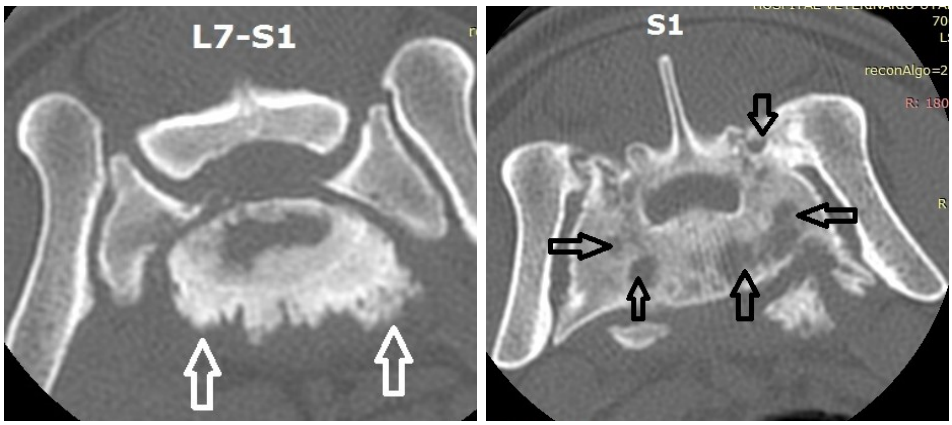

Figura 8 – Transverse CT image, in bone window, of the L7-S1 area of patient number 5. Note an extensive and irregular bone lysis of the terminal plates and vertebral bodies (arrowed), suggesting an acute and active disease process (Image gently provided by the neurological service of HV-UTAD).

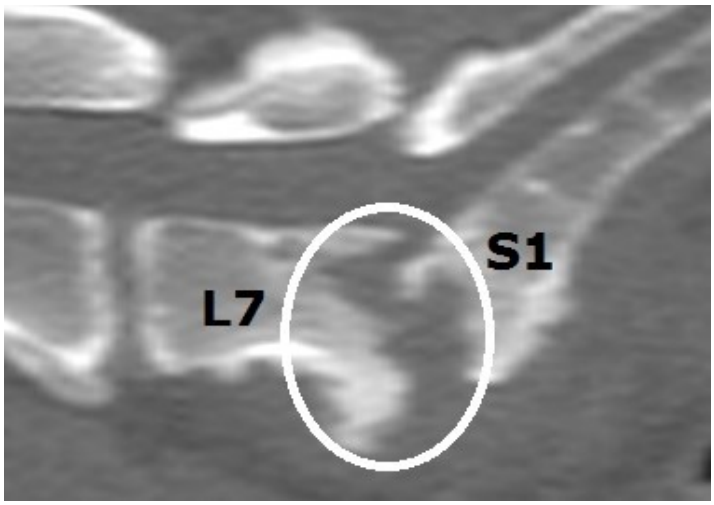

Figure 9 – Sagittal CT image, in bone window, of the L7-S1 area of patient number 5. Note an extensive and irregular bone lysis of the terminal plates and vertebral bodies (circled), suggesting an acute and active disease process (Image gently provided by the neurological service of HV-UTAD).

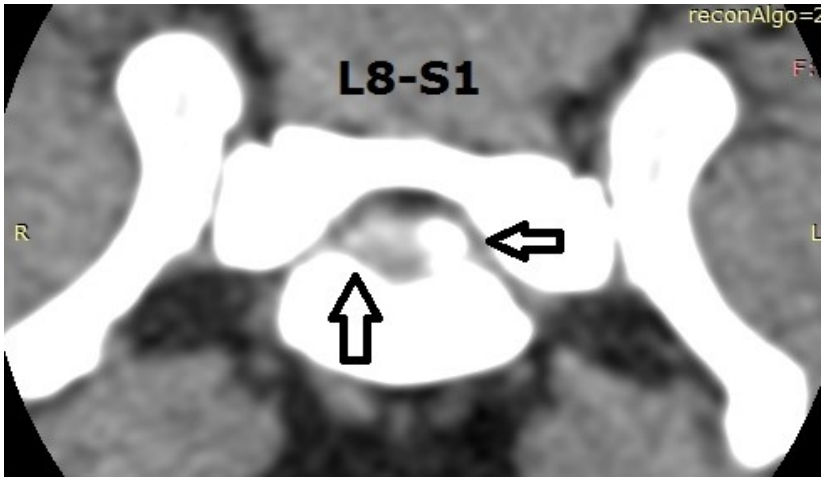

Figure 10 – Transverse CT image, in soft tissue window, of L8-S1 area of patient from case number 6. The arrows indicate the presence highly mineralized material, associated with DH I, severely compressing the nerve roots of the CE (Image gently provided by the neurological service of HV-UTAD).

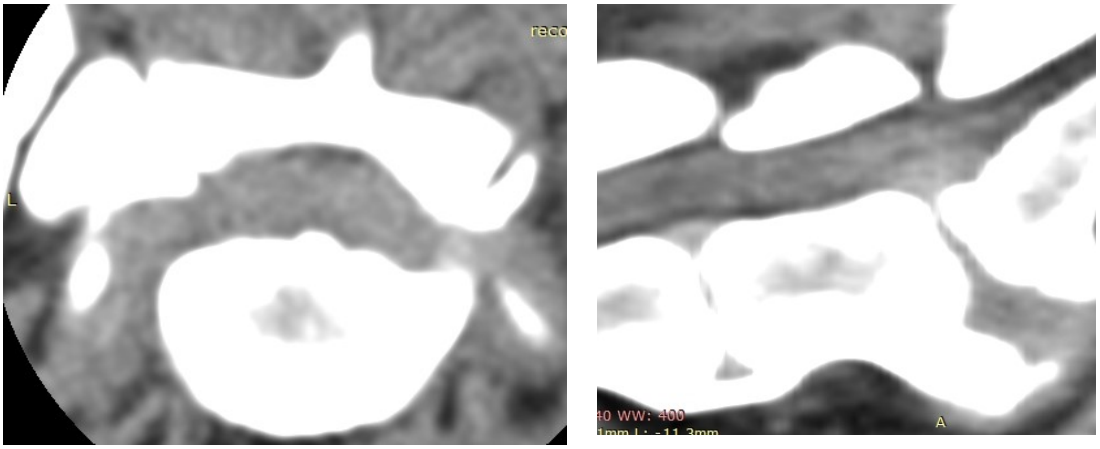

Figure 11 – Sagittal and transverse CT images, in soft tissue window, of the L7-S1 area of patient from case number 7. Note the marked protrusion of the AF into the L-S1 vertebral canal, associated with DH II, and dislocation of the thecal sac. Loss of the nerve roots detail in the lumbosacral region, due to reduced epidural fat. Spondylosis deformans in the same area (Image gently provided by the neurological service of HV-UTAD).

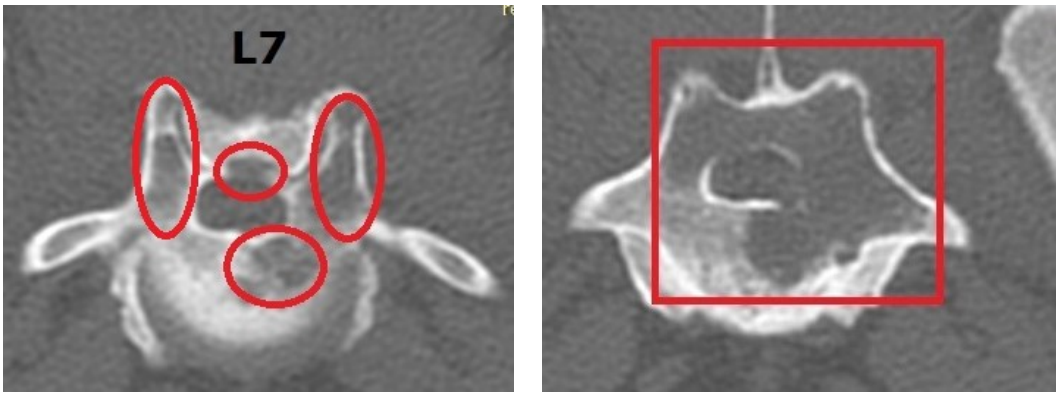

Figure 12 – Sagittal CT images, in bone window, of L7 vertebra from patient from case number 11. Note the marked bone lysis of the spinous process, dorsal lamina, both pedicles and articular processes and right side of the vertebral body

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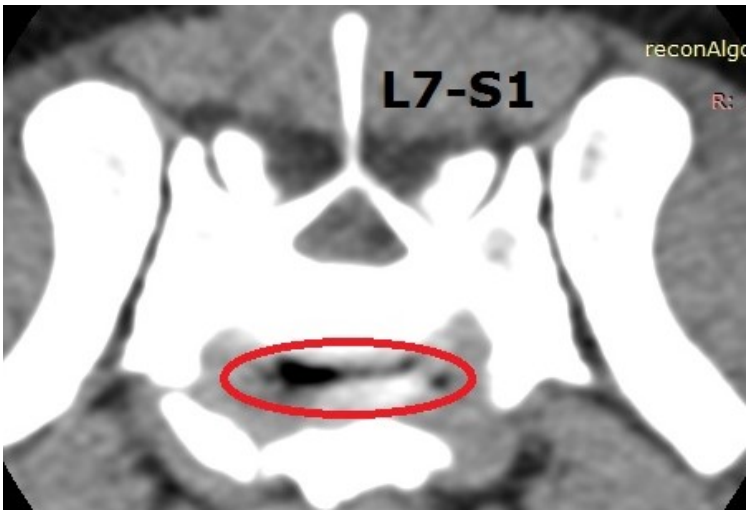

Figure 13 – Sagittal CT images, in soft tissue window, of L7-S1 area from patient from case number 11. Note the presence of vacuum phenomenon and mild protrusion of the AF into the vertebral canal, associated with DH II.

Lesion Localization

The most frequent site of lesion was the L7/L8-S1 IVS, representing 90% (10/11) of the studied cases.

3.1.4. Lesion Characterization

The most frequent type of lesion was the degenerative, representing 55% (6/11) of the studied cases (see diagram 4). Within this group, 83% (5/6) were DH II and only 17% (1/6) were DH I. DH II were associated with lumbosacral stenosis (DLS) in 60% of the cases (3/5). Cases number 3, 8, 10 and 11 had DH II without clinical relevance, so were not accounted. The second most frequent type of lesion was the vascular type, representing 18% (2/11) of the studied cases.

Analysing the breed distribution for degenerative disorders, 33% (2/6) were small breeds, 33% were large/giant breed dogs and 33% medium breed dogs. Regarding gender, 50% of the patients with degenerative lesions were female and 50% were male. Both chondrodystrophic animals in this study were 5 years old. Non-chondrodystrophic animals were between 6 and 12 years old.

The most frequent site for DH, in this study, was the L7/L8-S1 IVS, representing 83% of the studied cases (5/6, cases number 1, 2, 6, 7 and 9). 33% of patients had DH at the L6-L7 IVS (2/6, cases number 1 and 4). Case number 1 had disc herniations in both IVS.

33% (1/3) of the DLS cases were female and 67% (2/3) were male. 67% (2/3) of the animals with DLS were large/giant breed dogs and 33% (1/3) medium breed dogs.

The average age of large breed dogs with DLS was 6 years old and the medium breed 6 years old.

The second most frequent type of lesion was the vascular type (fibrocartilaginous embolism), representing 18% (2/11, cases number 3 and 8) of the studied cases. In this study, large breeds constituted 50% of the sample and medium breeds 50%.

Neoplastic lesion type represented 9% (1/11, case number 11) of the studied cases and the results of the complementary examinations (eco-guided cytology of the L7 spinal apophysis and CT images revealing a severe osteolysis on L7, with high risk of fracture) were compatible with osteosarcoma. This patient was 11 years old.

Infectious causes represented 9% (1/11, case number 5) of the studied cases. This was a large breed dog, with discospondylitis in the L7-S1 IVS, caused by *staphylococcus aureus*.

Lesions of traumatic origin were verified in 9% (1/11, case number 10) of the studied clinical cases. Case number 10 corresponded to a patient who showed up for evaluation 48 hours after an accident. This patient had clinical signs and neurological deficits indicative of nerve root injury (sciatic).

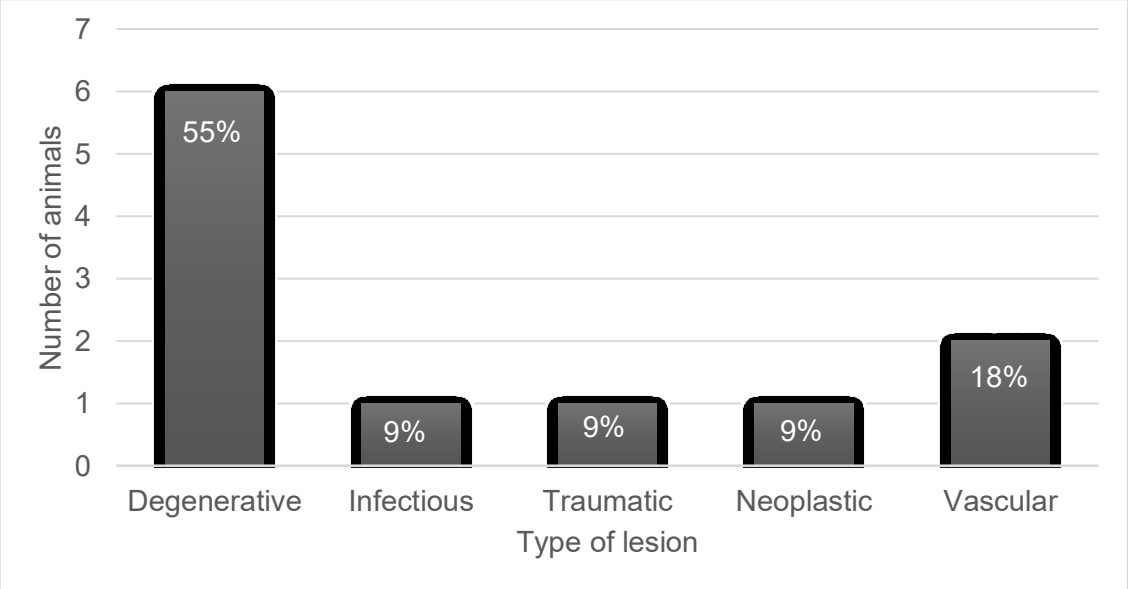

Diagram 7 – Absolute and relative frequencies of the types of lesion found in the cases in study.

3.1.5. Conservative treatment, revaluations and relapses

All cases received conservative treatment with everfit plus™ and neurobion™ (1 tab.; PO; BID) supplementation for 4 weeks. Strict cage rest was recommended for 2-3 weeks and, for patients' number 5, 8 and 10, physiotherapy 3 times a day.

Case number 5, in order to treat the infection caused by *streptococcus aureus*, received treatment with clindamicine (1,9 ml; IV; BID; for 1wk) and enrofloxacine (2,6 ml; IV; BID; for 1wk). IV antibiotics were then replaced by oral tablets of enrofloxacine (5 mg/kg; PO; SID), cefuroxine (10 mg/kg; PO; BID) and metronidazole (20 mg/kg; PO; BID) for 4 weeks.

Pain management

Pain management treatment was given to 64% of the studied cases (7/11; cases number 1, 2, 5, 7, 8, 10 and 11).

NSAIDs: 5/11 cases (45%, cases number 1, 2, 5, 10 and 11) received treatment with NSAIDs. Cases number 1, 2 and 10 (60%, 3/5) were treated with carprofen (2,2 mg/kg; PO; BID, for 5 days). Case number 5 and 11 (40%, 2/5) were treated with meloxicam (0,1-0,2 mg/Kg; PO; SID; for 3wk).

Anticonvulsants: 2/11 animals (18%) were given gabapentin (cases number 2 and 11, 10 mg/Kg; PO; SID and TID, respectively).

Opioids: 4/11 animals (36%) were given opioids. Morphine (0,2 mg/kg; IM; q 4hr for 1wk) was given to case number 8 (25%, 1/4). Methadone (0,1 mg/kg; IM; q 4-6hr, for 2wk) was given to cases number 5 and 7 (50%, 2/4). Case number 11 (25%, 1/4) was treated with tramadol (4 mg/kg; PO; TID; for 4wk).

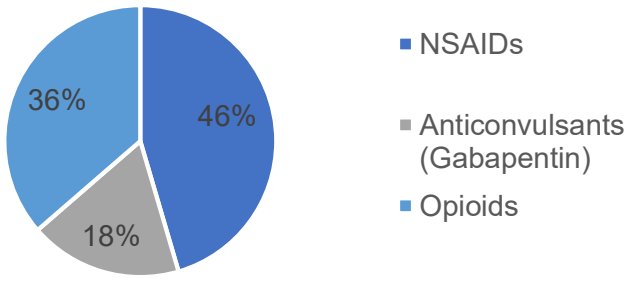

Diagram 8 – Drug groups used for pain management treatment in the studied cases, in percentage.

Case No.	Type of Lesion	Revaluations and Relapses
1	Degenerative (DH II)	**1m later:** slight improvement of clinical signs. Maintenance of evefit plus and neurobion™ for indefinite period. **8m later:** proprioception 0 on both PL. CT images similar to the first evaluation. Maintenance of everfit plus™ and neurobion™. Cage rest for 2wk. Without further information.
2	Degenerative (DH II)	**2wk later:** improvement of LS pain. Without improvement of neurological deficits. Maintenance of treatment for more 2wk. **1m later:** no improvement of neurological deficits. Surgery was proposed and accepted.
5	Infectious (*Streptococcus aureus*)	**2m later:** normal neurological exam. Negative urine and articular synovial fluid cultures.
10	Traumatic. Suspicion for sciatic nerve stretching.	**1wk later:** shows improvement in the ability to support weight, proprioception increased to (+1) on LPL. Maintenance of treatment for more 1wk. Without further information
11	Neoplastic. Compatible with osteosarcoma.	**3m later:** normal neurological exam. CT images similar to the first evaluation. **Relapse 4m later:** manifests pain and reluctance to walk. Maintenance of everfit plus™ and neurobion™. Cage rest for 2wk. **2wk later:** normal neurological exam, improvement of clinical signs. Maintenance of everfit plus™ and neurobion™.

Table 19 – Characterization of the type of lesion of each clinical case and description of the revaluations and relapses.

Revaluations and relapses

Cases number 3, 4 and 6 (27%) didn't show up for revaluation and there is no further information about them. Cases number 8 and 9 (18%) were euthanized. For these reasons, the referred cases were not considered in the following parameters.

Relapses were verified in 60% (3/5) of the patients who showed up for revaluations, from which 67% (2/3) corresponded to degenerative lesions (DLS) and 33% (1/3) to neoplastic lesions (osteosarcoma).

3.1.6. Surgery, post-surgery medical treatment and revaluations

2/11 cases were treated surgically, both with degenerative lesions (DLS). After surgery, medical management consisted of ice therapy (15min; TID; for 2 days), everfit plus™ and neurobion™ (1 tab.; PO; SID; for 3-4wk), physical therapy (TID; for 4wk) and rest in cage with short walks 3-5 times a day for 3 weeks.

Case No.	Surgery	Post-surgery revaluation(s)
2	L5-S3 laminectomy with dorsal fenestration of the disc and L7-S1 distraction.	**1m later:** Normal neurological exam.
7	L7-S1 laminectomy	**2wk later:** Improvement of neurological deficits. Proprioception: RPL (+1), LPL (0) **1m later:** Normal neurological exam.

Table 20 – Surgery technique and post-surgery revaluation(s) for cases 2 and 7.

CHAPTER IV – Discussion

In a general way, when analysing the sample, large/giant breed dogs constituted 55% (6/11) of the patients in study, diagnosed with a CE disorders. This result is in accordance with Dewey & da Costa (2016), who stated that problems affecting the caudal lumbar region are more frequent in large-breed dogs.

Predominant clinical signs were in accordance with the consulted literature. Coates (2012) stated that on CE disorders, one of the most common neurological presentations is gait disturbances and, in this study, all patients showed gait disturbances.

Neurological deficits, in CE disorders, are dependent of the nerve root(s) involved and are usually LMN in nature. Neurological dysfunction of the PL can range from paraplegia/paraparesis or monoplegia/monoparesis to mild proprioceptive deficits which do not affect gait (Watt,1991; Danielsson & Sjöström, 1999). 54% (6/11) of the studied animals manifested some degree of paresis/plegia. This was the most common gait disturbance observed in the cases in study. Reluctance to move, which can be interpreted as a pain manifestation, was present in 45% (5/11) of animals and ataxia in 18% (2/11). According to Sjöström (2003), patient history frequently includes lower lumbar or PL pain, which can be manifested in various ways. The tail can be flaccid or carried low due to pain, hypotonic, or paralytic. Lumbosacral or pelvic limb hyperesthesia was present in 73% (8/11) of the studied cases. Abnormal tail position (lowered) was verified in 9% (1/11, case number 5) of patients. Within these 73%, 50% manifested pain upon tail elevation (cases 1, 2, 5 and 7).

Abnormal posture was observed in 18% (2/11) of the patients in study, both with the left pelvic limb (LPL) flexed – pelvic limb root signature. According to Plat & Olby (2012) and Fingeroth & Thomas (2015), pelvic limb root signature, with consequent lameness, although not very common in CE disorders, can be observed due to highly lateralized lesions. This was verified in case number 10 and 11, which corresponded to a highly lateralized sciatic nerve lesion and to a lateralized neoplastic lesion, respectively.

Mckee (2007), stated that patients with CE disorders may have a decreased muscle tone of the musculature surrounding the pelvic limbs. In this sample, 6/11 (55%) animals had a decreased muscle tone when presented for evaluation.

Proprioception was evaluated by the proprioceptive placing test. It was decreased/absent in 9 out of the 11 cases (82%). According to Garosi (2004) and Parent (2010), in animals with CE disorders, the most reliable spinal reflex tests for the PL evaluation are the patellar and withdrawal (flexor) reflexes. Other spinal reflexes are

more difficult to perform and interpret. Dewey (2013) states that, due to CE disorders, the patellar reflex test may exhibit a false exaggerated response (patellar pseudo-hyperreflexia). Flexor reflex was decreased/absent in 9 of 11 patients (82%). Patellar reflex was pseudo-hyper-reflexive in 4 of 11 patients (36%). It was decreased/absent in 2 of the 11 patients (18%). Nociception was lost in 2 out of the 11 patients (18%).

All patients were submitted to conservative treatment: rest in cage for 2-3 weeks, supplementation with neurobion™ and everfit plus™ and a request for revaluation 1-2 weeks later. Only 5 animals showed up for the revaluation. 2 of 11 (18%) were euthanized (cases number 8 and 9). 2 of 11 (18%, cases number 2 and 7) were submitted to surgery, which was successfully performed. After surgery, these animals were evaluated again by the neurology service of HV-UTAD. For post-surgery recovery, rest in cage for 2 weeks with short walks 2-3 times a day was recommended, as well as supplementation with neurobion™ and everfit plus™ and physiotherapy 3 times a day, for 4 weeks.

Pain management medication was given to 64% of the studied cases (7/11; cases number 1, 2, 5, 7, 8, 10 and 11). 5/11 cases (45%, cases number 1, 2, 5, 10 and 11) received treatment with NSAIDs (carprofen or meloxicam). 2/11 animals (18%) were given gabapentin (cases number 2 and 11), for pain management. 4/11 animals (36% cases number 5, 7 and 8) were given opioids (morphine or methadone). 1/11 patients (9%, case number 11) was treated with tramadol.

Relapses were verified in 60% (3/5) of the patients who showed up for revaluations, from which 67% (2/3) corresponded to degenerative lesions (DLS) and 33% (1/3) to neoplastic lesions (osteosarcoma).

The most frequent type of lesion was the degenerative, representing 55% (6/11, cases number 1, 2, 4, 6, 7 and 9) of the studied cases. Cases number 3, 8, 10 and 11 had DH II without clinical relevance, so were not accounted. According to some authors, DH II are commonly found in the L7-S1 IVS. DH I are more frequently found in the cervical and thoraco-lumbar regions (Brisson, 2010; Marinho *et al.,* 2014). Within this group, 83% (5/6, cases number 1, 2, 4, 7, and 9) were disc protrusions (DH II) and only 17% (1/6, case number 6) disc extrusions (DH I).

Analysing the breed distribution for degenerative disorders, 33% (2/6) were small breeds, 33% were large/giant breed dogs and 33% medium breed dogs. The consulted literature describes a higher prevalence for DH of the lumbosacral region in large breed dogs (Lorenz *et al.*, 2011; Platt & Olby, 2012). However, a bigger sample with greater breed diversity and distribution would be required to take relevant conclusions on this parameter.

Both chondrodystrophic animals in this study were 5 years old, meaning that these results are within the age range, in which, according to Priester (1976), the peak of higher incidence of DH in chondrodystrophic breed dogs is verified (2 to 6 y/o). Non-chondrodystrophic animals were between 6 and 12 years old, which is in accordance with the age range (older than 5 y/o) described by some authors for non-chondrodystrophic breeds (Meij & Bergknut, 2010; LeCouteur & Grandy, 2010).

Regarding gender, 50% of the patients with degenerative lesions were female and 50% were male. These values are in accordance with Brisson (2010), who supports that there is not a gender predisposition for DH, however, some studies claim the existence of a higher predisposition for females (Priester, 1976; da Costa & Platt, 2017). A bigger sample would be required to take relevant conclusions on this parameter.

Regarding weight, there was a disparity in the obtained values, however, given the different characteristics inherent to each breed, it is not possible to take relevant conclusions about this parameter.

The most frequent site for DH, in this study, was the L7/L8-S1 IVS, representing 83% of the studied cases (5/6, cases number 1, 2, 6, 7 and 9). 33% of patients had DH at the L6-L7 IVS (2/6, cases number 1 and 4). Case number 1 had disc herniations in both IVS. The obtained data are in conformity with the consulted literature, which states that approximately 90% of disc herniations occur at either the L4-L5 or L5-S1 levels (Cricun & Cricun, 1988; Gleave & MacFarlane, 1990).

Regarding clinical signs, treatment options and relapses, cases number 1, 2 and 7 will be discussed separately, since these patients had DH II associated with lumbosacral stenosis, which constitutes a specific degenerative lesion type: degenerative lumbosacral stenosis. Gait disturbances were present in all clinical cases of DH (3/11). Case number 6 and 9 (2/3, 67%) were presented with paraparesis. Case number 4 (1/3, 33%) showed reluctance to move. Lumbosacral pain was present in 33% (1/3) of the cases (cases number 4). Proprioception and flexor reflex were decreased/absent in 67% (2/3) of cases (cases number 6 and 9). Nociception was lost in case number 9 (33%), the remaining patients didn't have any nociception deficits. Patellar reflex was decreased in case number 9 (1/3, 33%) and normal in cases number 4 and 6 (2/3, 67%).

Case number 9 was immediately euthanized, since loss of nociception for more than 48 hours bears a poor prognosis (Coates, 2000) and the owner didn't have the funds necessary for surgical treatment. The remaining patients (cases number 4 and 6) were treated medically: rest in cage for 2 weeks, with everfit plus™ and neurobion™ supplementation for 4 weeks. According to Dewey & da Costa (2016), success rate for medical treatment is approximately 50%, but initial success with subsequent disease

relapse can be experienced by about 30%-50% of the dogs. It wasn't possible to take conclusions about these parameters, since none of the patients showed up for revaluation.

Lumbosacral stenosis was present in 60% (3/5, cases number 1, 2 and 7) of the animals with DH II (degenerative lumbosacral stenosis). Some authors refer a male predilection for this disease, which was verified by this study: 33% (1/3) of the affected animals were female and 67% (2/3) were male. The consulted literature refers a predisposition of middle-aged to older large-breed dogs, which was also verified: 67% (2/3) of the animals with DLS were large/giant breed dogs and 33% (1/3) medium breed dogs. The average age of large breed dogs with DLS was 6 years old (Lang *et al.,* 1992; Janssens *et al.*, 2009).

Muscle tone was normal in case number 1 (33%) and decreased in cases number 2 and 7 (33%), at the time of examination. All patients (100%) manifested lumbosacral pain upon palpation of the area and upon tail elevation. Urinary/faecal function and nociception were considered normal in all patients with DLS. Decreased proprioception and flexor reflex were observed in all patients. 67% (2/3) of the patients had patellar hyperreflexia and 33% (1/3) a normal patellar reflex.

Consulted literature claims an approximate success rate of 50% in dogs managed conservatively, including animals showing improvement or resolution of clinical signs. It is also stated that, when this type of treatment is chosen, improvement is often transient, once resumption of normal activity frequently elicits recurrence of clinical signs, which was verified in this study (Meij & Bergknut, 2010; De Decker *et al.*, 2014). Surgery was recommended for the 3 patients diagnosed with DLS. Case number 1 and 2 (2/3, 67%) decided for the medical therapy, since it was more affordable. Medical therapy consisted of everfit plus™ and neurobion™ supplementation for 4 weeks, rest in cage for 4 weeks and administration of carprofen (NSAID) for 5 days, as recommended by literature (Platt & Olby, 2012; Dewey & da Costa, 2016). For case number 1 (1/2, 50%), an improvement of clinical signs and neurological deficits occurred initially, but recurrence was verified 8 months later. Medical treatment was considered unsuccessful for case number 2 (1/2, 50%), due to lack of improvement of clinical signs. After revaluation, this owner opted for the surgical therapy. Medical therapy had a success rate of 50% (1/2) with 100% recurrence, in this study.

Cases number 2 and 7 were submitted to surgery (laminectomy with dorsal fenestration of the disc and L7-S1 distraction and L7-S1 laminectomy, respectively). After surgery, these animals were evaluated again by the neurology service of HV-UTAD. Both patients had normal neurological exams when they showed up for revaluation (1 month later). Surgical therapy had a success rate of 100% in this study, for DLS. Consulted

literature claims successful outcomes range from 66.7 to 95% of cases, with surgical intervention. Reported recurrence rates vary between 3 and 18% (De Risio *et al.*, 2000; Meij & Bergknut, 2010; De Decker *et al.*, 2014). However, there was no further information regarding these patients, so conclusions about recurrence cannot be established.

The second most frequent type of lesion was the vascular type (FCE), representing 18% (2/11, cases number 3 and 8) of the studied cases. These diagnoses were presumptive, since the owners did not authorize the clinicians to perform MRI, which would be necessary to establish a definitive diagnosis.

According to Cook (1988) and Neer (1992), fibrocartilaginous embolism (FCE) occurs primarily on large or giant breed dogs. In this study, large breeds constituted 50% of the sample and medium breeds 50%. A bigger sample would be required to take conclusions on this parameter. As recommended by the consulted literature, these diagnoses were established by the history, neurological dysfunction signs and by the absence of other signs of acute myelopathy. MRI was recommended by the clinicians of the neurological service of HV-UTAD, to confirm the diagnosis, but the owners decided not to do it (Cauzinille, 2000).

By the time of neurological evaluation, the patient from case number 8 presented acute LMN paraplegia, LMN bladder, decreased muscle tone and absent nociception for the LPL. Case number 3 had normal muscle tone and nociception, with unilateral neurological deficits (RPL): decreased proprioception and decreased patellar and flexor reflexes. None of the patients manifested hyperesthesia. These data are in accordance with Cook (1988) and Bartholomew and his collaborators (2016) who stated that neurological deficits for FCE are usually asymmetric with acute clinical signs, frequently indicating a very lateralized or focal lesion, and that LMN deficits are common, but hyperesthesia is unusual.

Treatment for patients' number 3 and 8 was supportive: rest in cage for 3 weeks with neurobion™ and everfit plus™ supplementation, as recommended by literature (Cauzinille, 2000; Urfer, 2007). Patient number 8 received also IM injections of morphine for pain management. The owner of the patient from case number 8 decided for euthanasia, because nociception was still absent after 1 week of conservative treatment, which bears a poor prognosis. As stated from Bartholomew and his collaborators (2016), if nociception is absent, the rate of recovery is approximately 10%. There is no further information about case number 3, since the patient didn't show up for revaluation.

Neoplastic lesion type represented 9% (1/11, case number 11) of the studied cases and the results of the complementary examinations were compatible with osteosarcoma (eco-guided cytology of the L7 spinal apophysis and CT images revealing

a severe osteolysis on L7, with high risk of fracture). This patient was 11 years old, which is in accordance with consulted literature, that refers that spinal neoplasia usually affects patients with more than 5 years old (Dyce *et al*., 1991).

Consulted literature states that extradural tumours typically cause pain with rapid neurological deterioration (LeCouteur, 2001; Chrisman *et al.*, 2003). The patient manifested a high level of pain in the lumbosacral area and severe neurological deficits on the LPL, such as paresis, pelvic limb root signature, decreased muscle tone and loss of proprioception, flexor and patellar reflexes. Masses in other locations were not found, meaning that this was a primary tumour, which goes against what is stated by LeCouteur (2001), who claims that discovering an extradural tumour indicates the possibility of a primary mass elsewhere.

Since there was an extensive bone destruction at the time of diagnosis and given the age and health status of the patient (chronic cardiac disease), surgery was not considered, and supportive therapy was given: pain management medication and supplementation with everfit plus™ and neurobion™. There is no further information regarding this patient.

Infectious causes represented 9% (1/11, case number 5) of the studied cases. This was a large breed dog, with discospondylitis in the L7-S1 IVS, caused by a *staphylococcus aureus* that according to laboratory tests results, gained access to the disc by hematogenous spread from the bladder (the infectious agent was found in urine and articular synovial fluid cultures). This data is in accordance with the consulted literature that refers a predisposition in medium- to giant-breed dogs for this disorder (Dewey & da Costa, 2016) and a higher prevalence for the IVD between L7-S1, regarding the site of infection (Ruoff *et al.*, 2018). *Staphylococcus aureus* is the most reported infectious agent for this disorder (Butterworth *et al*., 1995; Greene, 1998).

Clinical signs were nonspecific and progressive, as describe by consulted literature: lumbosacral pain, stilted pelvic limb gait, decreased appetite, weight loss, fever and reluctance to move. Proprioceptive deficits were not present and, regarding spinal reflexes, only the patellar hyperreflexia was verified, which is also in accordance with the consulted literature (Corrente *et al.*, 2010; Tipold & Stein, 2010).

The patient received treatment with clindamicine (1,9 mL; IVL; BID; for 1wk) and enrofloxacine (2,6 mL; IVL; BID; for 1wk). IV antibiotics were then replaced by oral tablets of enrofloxacine (5 mg/kg; PO; SID), cefuroxine (10 mg/kg; PO; BID) and metronidazole (20 mg/kg; PO; BID) for 4 weeks, supplementation with everfit plus™ and neurobion™ and physiotherapy TID, for 4 weeks and methadone and meloxicam for pain management, as recommended by Moore (1992) and Schwartz *(*2009).

Renwick and his collaborators (2010) demonstrated that the prognosis for bacterial discospondylitis is usually favourable, especially in cases with no or mild neurologic deficits, which was verified by this study: 2 months later the patient had a normal neurological exam and negative urine and articular synovial fluid cultures.

Lesions of traumatic origin were verified in 9% (1/11, case number 10) of the studied clinical cases. Case number 10 corresponded to a patient who showed up for evaluation 48 hours after an accident.

This patient had clinical signs and neurological deficits indicative of nerve root injury (sciatic): monoparesis and pelvic limb root signature (LPL flexed), decreased muscle tone, absent proprioception, decreased flexor and patellar reflexes and pain upon coxofemoral extension. According to Platt & Olby (2012), these are typical clinical signs of sciatic nerve injury. Nociception was normal, which, according to Mckee (1990) and Shores (1992), is indicative of good prognosis for recovery.

Treatment was conservative: strict cage rest for 3 weeks, supplementation with everfit plus™ and neurobion™ for 4 weeks, carprofen for 5 days and physiotherapy TID, for 4 weeks, as recommended by Dewey & da Costa (2016). 1 week later, there was improvement in the proprioceptive deficits. The animal didn’t show up for any further revaluation.

As previously mentioned, this study presents some limitations. To carry out a study with greater accuracy and higher scientific value, the sample size should be more representative and with a higher distribution of the types of lesion, which had negative influence in the analysis and conclusion obtained for certain parameters, as well as some information which were not registered.

CHAPTER V – Conclusions

Based on the results presented in the study and the information discussed in this dissertation, it is possible to take some conclusions, although influenced by the small size of the sample.

- The general and neurologic examinations intend to evaluate the integrity and function of the nervous system.
- Neurological examination helps the clinician to elaborate a list of differential diagnosis, to identify the aetiology of the disorder, to determine its prognosis and to help choosing the most adequate treatment.
- A differential diagnosis list, according to signalment, history and neurological findings, is essential.
- Complementary tests should aim to confirm or rule out the differential diagnosis in the list, without replacing clinical evaluation.
- CT is a very fast imaging method and may provide helpful findings in CE disorders.
- Longer duration and limited access constitute the main disadvantages of MRI.
- Problems affecting the caudal lumbar region are more common in large-breed dogs.
- Clinical signs associated with CES vary according to the location and extent of the lesion. These are highly nonspecific and can be persistent or episodic and heterogeneous.
- Clinical signs may be bilateral or unilateral.
- Pelvic limb root signature, with consequent lameness, although not very common in CE disorders, can be observed due to highly lateralized lesions.
- On CE disorders, one of the most common neurological presentations are gait disturbances.
- Patient history frequently includes lower lumbar or PL pain, which can be manifested in various ways.
- In animals with CE disorders, the most reliable spinal reflex tests for the PL evaluation are the patellar and withdrawal (flexor) reflexes.
- Neurological dysfunction of the PL can range from paraplegia/paraparesis or monoplegia/monoparesis to mild proprioceptive deficits which do not affect gait.
- The most frequent type of CE lesion is the degenerative.
- The most frequent site for DH, in the CE region, is the L7/L8-S1 IVS.
- Disc protrusions are more common than disc extrusions in the lumbosacral region.
- Degenerative lumbosacral stenosis is a very common CE disorder in middle-aged to older large/giant breed male dogs.

- For degenerative lesions, surgery is a more reliable long-term treatment than the conservative treatment, however it isn't always possible, due to individual limitations of the patient (e.g. advanced age, systemic diseases) and due to its higher costs.
- Fibrocartilaginous embolism (FCE) occurs primarily on large or giant breed dogs.
- The diagnosis of FCE should be established by the history, neurological disfunction signs and by the absence of other signs of acute myelopathy. MRI is recommended to confirm it.
- Neurological deficits for FCE are usually asymmetric with acute clinical signs, frequently indicating a very lateralized or focal lesion. LMN deficits are common, but hyperesthesia is unusual.
- Spinal neoplasia usually affects patients with more than 5 years old.
- Extradural tumours typically cause pain with rapid neurological deterioration.
- There is a predisposition in medium- to giant-breed dogs for lumbosacral discospondylitis and a higher prevalence for the IVD between L7-S1, regarding the site of infection.
- *Staphylococcus aureus* is the most common infectious agent for lumbosacral discospondylitis.
- In cases of lumbosacral discospondylitis, the infectious agent can be found in urine and articular synovial fluid cultures.
- Clinical signs of lumbosacral discospondylitis are usually nonspecific and progressive: lumbosacral pain, stilted pelvic limb gait, decreased appetite, weight loss, fever and reluctance to move.
- The prognosis for bacterial discospondylitis is usually favourable, especially in cases with no or mild neurologic deficits.
- Lateralized clinical signs, such as monoparesis, pelvic limb root signature, decreased muscle tone, absent proprioception, decreased flexor and patellar reflexes and pain upon coxofemoral extension are indicative of sciatic nerve injury.
- For sciatic nerve injury, if nociception is still present, conservative treatment is likely to be effective in improving the neurological deficits and clinical signs.
- Loss of nociception bears a poor prognosis regarding CE disorders.

CHAPTER VI – References

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