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MINI HEMILAMINECTOMY FOR REMOVAL OF DISC EXTRUSION IN A FRENCH BULLDOG: CASE REPORT

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Abstract: Intervertebral Disc Disease (IVDD) is the main degenerative disease of the spine and one of the leading causes of neurological problems in small animals. Historically, Hansen and Olsson classified it into Type I (chondroid degeneration, disc mineralization, and acute spinal cord compression by extrusion) and Type II (fibroid degeneration, slow and progressive compression by protrusion) (Hansen, 1951; 1952; Olsson, 1951; 1952).
Diagnostic and genetic advances have allowed the identification of variants such as acute non-compressive extrusion (ANNPE), hydrated nucleus pulposus extrusion (HNPE), and intradural/intramedullary extrusions (IIVDE) (De Risio et al., 2009; Brisson, 2010). In addition, the identification of the FGF4 retrogene on chromosome 12 has broadened our understanding of predisposition in chondrodystrophic breeds (Parker et al., 2009; Brown et al., 2017). Clinically, Type I IVDD mainly affects young animals, with signs varying according to the location, extent, and duration of compression. In the reported case, a 3-year-old male dog presented with acute hind limb paralysis and pain on palpation; magnetic resonance imaging showed compression at L2-L3, confirming lumbar Type I IVDD. Surgical treatment with mini-hemilaminectomy was performed, allowing immediate decompression of the spinal cord. Surgery is considered the treatment of choice in severe cases, with a favorable prognosis, even when postoperative physical therapy is not fully followed.

INTRODUCTION

Intervertebral Disc Disease (IVDD) is the most frequently described neurological condition in dogs. It is a condition caused by degeneration of the intervertebral disc, which can lead to disc extrusion or herniation, causing compression of the spinal cord or nerve roots, and even spinal cord concussion. The clinical presentation is variable and depends on the location of the lesion, the volume of material inside the canal, and the speed at which this material is ejected.

Degenerative changes can affect any disc, but the effects are likely to be more severe when they involve the discs in the most mobile regions; the discs in the neck and lumbosacral junction are especially susceptible.

Treatment methods in dogs with IDD include decompressive surgery or conservative clinical treatment consisting of confinement and the use of anti-inflammatory drugs. The goals of surgical treatment in IDD are: decompression of the spinal cord, removal of material from inside the spinal canal, reduction of edema, pain relief, and prevention of future extrusions.

This case report aims to describe a case of intervertebral disc extrusion with moderate left ventro-uterine extradural spinal cord compression at L2/L2-L3, consistent with extruded disc material with extensive perilesional edema, without a history of trauma, performing a comparative analysis of the patient with pain and after the mini hemilaminectomy surgical procedure.

LITERATURE REVIEW

ANATOMY

Lumbar Vertebra Anatomy

A typical vertebra is divided into the body, vertebral arch (formed by the pedicles and right and left laminae), and its processes (articular, spinous, transverse, accessory, and mammillary). The body of the lumbar vertebrae is flattened dorsoventrally and increases in width from the first to the last. The vertebral arch and the body of the vertebra form the vertebral foramen, and the union of these foramina form the vertebral canal, whose diameter is widest in the cervical vertebrae and decreases towards

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the sacral vertebrae, with the exception of the cervical and lumbar bulges of the spinal cord (EVANS; de LAHUNTA, 2012). As can be seen in Figure 1, the anatomical structures of the lumbar vertebrae are more easily understood and visualized when divided into their more dorsal structures and those located more ventrally. Dorsally, its most evident structure is the spinous process. Cranially, it is possible to observe the cranial articular process and other structures that are laterally positioned on both sides, cranial to the spinous process and caudal to the cranial articular process, which are the mammillary processes. Caudally to the spinous process is the caudal articular process. Cranially to these processes, positioned ventrally respectively, are the accessory processes and the transverse processes, also on both sides (FOSSUM, T. W, 2019).

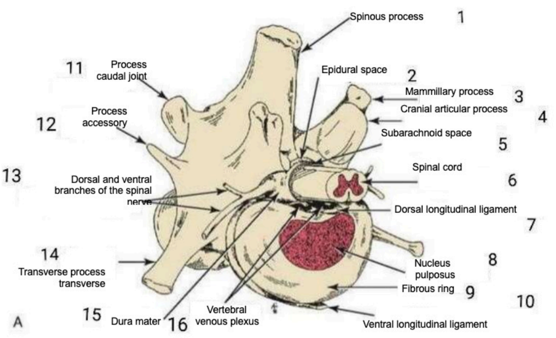

Figure 1: Lumbar vertebra anatomy

Source: Modified from Small Animal Surgery, Theresa Welch Fossum, 5th ed., 2019

Anatomy of the Intervertebral Disc

Ventrally, the structures present are the fibrous ring composed of fibrocartilaginous material arranged in concentric chambers and an ovoid center of gelatinous material, the nucleus pulposus (NP), forming the intervertebral disc (IVD) itself. The AF has a ventral and lateral spacing that is dorsal, which may explain the propensity of NP herniation into the vertebral canal (TOOMBS; BAUER, 1998). At each junction of two vertebrae, there is an intervertebral disc between them, and along the body of the vertebra is the ventral longitudinal ligament, connecting the vertebrae. The space between the vertebrae in front of the cranial articular process ( ) is called the epidural space (DYCE, 2010; FOSSUM, T. W, 2019).

Between the cranial and caudal parts described above runs the spinal cord, covered by the dura mater, forming a space between these structures called the subarachnoid space. Ventral to the described set and dorsal to the vertebral body are the vertebral venous plexuses and the dorsal longitudinal ligament (Figure 1) (FOSSUM, T. W, 2019).

Spinal Cord Anatomy

In cross section, it is possible to observe the spinal cord. The spinal cord is divided into two parts: the gray matter of the spinal cord, formed by the nerve cell bodies, is located centrally in the spinal cord and is responsible for the reflex arc. The white matter, which surrounds the gray matter, is formed by nerve fibers, where the ascending tracts (sensory fibers) and descending tracts (motor fibers) are located (PARENT, 2010). It can be observed that the spinal cord is not rounded throughout its entire length, but rather flattened in its anteroposterior direction. In the anterior part, the anterior median fissure (deepened groove) can be seen, while in the posterior part, the posterior median groove and the intermediate median grooves are found (Figure 2) (LECOUTEUR; CHILD, 1992).

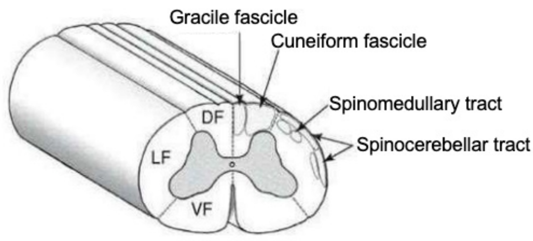

Figure 2: Spinal cord anatomy. DF: dorsal fascicle, LF: lateral fascicle, and VF: ventral fascicle.

Source: Modified from Dewey CW: A practical guide to canine and feline neurology, ed 2, Hoboken, N.J., 2008, Wiley Blackwell.

The spinal cord (SC) is part of the central nervous system (CNS) and is located within the vertebral canal, extending from the caudal limit of the brainstem (at the foramen magnum) to, on average, the sixth lumbar vertebra (this may vary depending on breed and species), where it forms the conus medullaris. The spinal cord is also divided into segments, as many as the segments of the spine, but their location does not correspond to the vertebral segments. Segments C1-C5 are within vertebrae C1-C4, C6-T1 are in C5C7, T2-L3 are between vertebrae T1-L2, segments L3-L4 are between T1-L2, L4-L7 are within L3-L4, and segments S1-S3 are in L5 (De LAHUNTA; GLASS, 2009). Each spinal segment gives rise to a pair of spinal nerves (right and left), which are directed caudally to their segment, each with a dorsal root (sensory) and a ventral root (motor), which form the peripheral nervous system (PNS). The spinal cord has two swellings, the thoracic and lumbar, which are where the cell bodies that provide movement and sensitivity to the thoracic and pelvic limbs are located, respectively (Figure 3) (LECOUTEUR; CHILD, 1992).

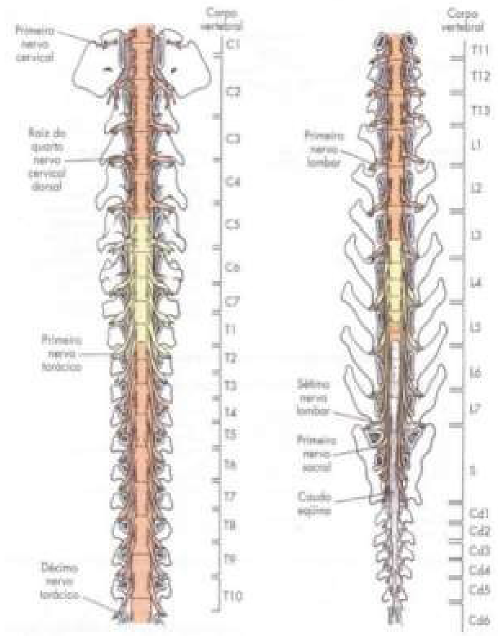

Figure 3: Schematic illustration of the spine after removal of the dorsal lamina, showing the relationship between the spinal segments and the vertebral bodies.

Source: Fossum, T. W, 2007

INTERVERTEBRAL DISC DISEASE

Etiology

IDD is a degenerative lesion in the intervertebral disc, with chondroid or fibrous characteristics. The etiology and pathogenesis of Intervertebral Disc Disease (IDD) have not yet been fully clarified. However, it is believed that biochemical and structural changes that can occur in the IDD are strong predisposing factors for the occurrence of the disease (BRAUND, 1996). Disc degeneration is an expected process that occurs with aging. This process usually occurs in dogs over 6 years of age (BACH et al., 2014). It is characterized by biochemical and structural changes, which can be fibrous or chondroid. Chondroid metaplasia with weakening of the fibers of the annulus fibrosus in chondrodystrophic breeds and fibrous metaplasia in non-chondrodystrophic breeds (Figure 4) (BRISSON, 2010).

In chondroid metaplasia, disc dehydration occurs, where there is a reduction in water and proteoglycans, leading to NP invasion by hyaline cartilage , reducing its ability to absorb shocks (BRISSON, 2010). Dehydration leads to disc mineralization, decreasing its hydrostatic properties and weakening the fibers of the annulus fibrosus (SHARP; WHEELEY, 2005). This process can begin before the age of two in chondrodystrophic animals. Around 75 to 90% of these animals have most of their NP transformed into hyaline cartilage by their first year of age, while non-chondrodystrophic animals remain with high levels of proteoglycans into old age, suggesting a genetic predisposition. (BRISSON, 2010).

In fibrous metaplasia, dehydration also occurs, but there is invasion of the NP by fibrocartilaginous tissue and simultaneous degeneration of the AF, and this process can occur anywhere in the spine, leading to future disc protrusion. This degeneration is not characteristic of any particular breed, but it is more commonly seen in non-chondrodystrophic animals. It is a later process and does not usually lead to disc calcification. Around 40 to 60% of seven-year-old animals already show evidence of this degeneration, but 10 to 30% exhibit macroscopic protrusion (BRISSON, 2010).

Another point to consider regarding DDIV is the anatomy of the annulus fibrosus, which can weaken and become unstable, resulting in disc displacement (BRISSON, 2010).

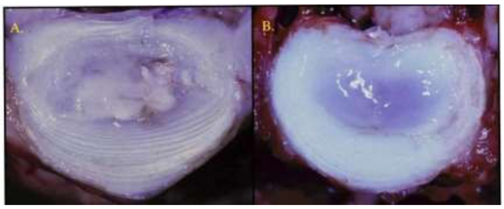

Figure 4: A: Degenerated DIV of a chondrodystrophic dog, NP tissue replaced by mineralized and chondroid material. B: Normal DIV.

Source: Brisson, B. A, 2010

In addition to the animal's genetics, mechanical and anatomical factors are important, noting that trauma can lead to acute extrusion of this degenerated disc. Other contributing factors include hypothyroidism and autoimmune diseases (BRISSON, 2010). The risk of disc extrusion is unrelated to body weight parameters, body condition score, or the animal's activity level. (LECOUTEUR; CHILD, 1992) Structural changes in the annulus fibrosus (AF) could be the primary cause. An increase in the enzymes responsible for degrading both collagen and elastin could weaken its structure, causing instability and consequently disc displacement (BRAUND, 1996).

Pathophysiology

DDIV is characterized by degeneration of the DIV and a neurological syndrome of compression or injury to the ME, which occurs due to herniation of the disc or parts of it into the spinal canal. Disc herniation can occur due to protrusion or extrusion of the disc or parts thereof, and can be an acute or chronic process with varying symptoms (BRISSON, 2010).

Intervertebral disc disease (IVDD) is a comprehensive term for different changes that compromise the intervertebral disc in dogs (Table 1). Since the first descriptions by Dexler in the late 19th century (Dexler, 1896; Dexler, 1897), etiological understanding and nomenclature have evolved. Initially described as *enchondrosis intervertebralis,* it was later recognized as degeneration of the nucleus pulposus (Dexler, 1896; Dexler, 1897; Olsson, 1951).

In the 1940s and 1950s, Hansen and Olsson established a classification based on histopathological changes, distinguishing chondroid metaplasia (chondrodystrophic breeds) and fibrous metaplasia (non-chondrodystrophic breeds), resulting in the well-known Hansen Type I and Type II (Hansen, 1951; 1952; 1959; Olsson, 1951; 1952).

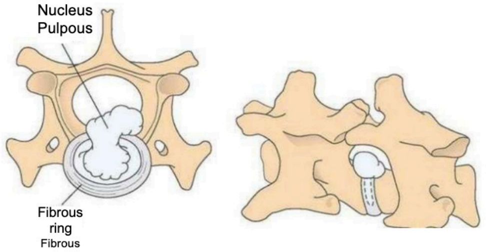

Figure 5: Hansen Type I herniation

Source: Fossum. T.W, 2019

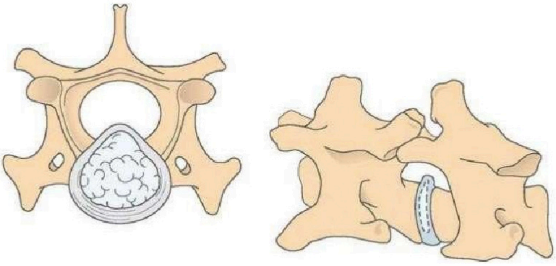

Figure 6: Type II Hansen herniation

Source: Fossum. T.W, 2019.

With advances in diagnosis, descriptions of relatively hydrated nucleus extrusions emerged (Griffiths, 1970; Wright, 1980; McKee, 2002). These atypical forms gave rise to new terminology (Table 2), notably Hansen Type III, now more uniformly referred to as acute non-compressive nucleus pulposus extrusion (ANNPE) (Griffiths, 1970; De Risio et al., 2009; Olby et al., 2004; Kent et al., 2010). In addition, other variants have been described, such as intradural or intramedullary extrusions (IIVDE) and acute compressive extrusion of hydrated nucleus (HNPE) (Rodriguez-Velasquez, 2011; Penning et al., 2006; Brisson, 2010).

The terminology used by Hansen and Olsson varied between "protrusion," "extrusion," "prolapse," and "herniation," often as equivalents (Hansen, 1952; Olsson, 1951). Currently, "extrusion" refers to acute protrusion of degenerated nucleus pulposus, "protrusion" refers to chronic thickening of the annulus, and "disc herniation" is used as a generic term (Adams & Roughley, 2006; Brinjikji et al., 2015).

Recent advances show that both chondrodystrophic and non-chondrodystrophic breeds can develop chondroid metaplasia, although at different ages and frequencies (Olsson, 1952; Bray & Burbidge, 1998; Bergknut et al., 2012). In addition, the discovery of the expression of an FGF4 retrogene on chromosome 12 has provided new genetic insights into predisposition to disc extrusion (Parker et al., 2009; Plassais et al., 2016; Brown et al., 2017).

The use of magnetic resonance imaging and other modern methods has expanded the identification of DDIV variants in dogs and cats, reinforcing the need to update and standardize nomenclature to reduce ambiguities and promote higher quality research.

Current/proposed consensus	**Terms reported in the literature**
(Hansen Type I/Acute) DIV extrusion	Hansen Type I herniation/ extrusion/IVD disease; Acute herniation/extrusion/IVD disease; IVD protrusion; Type I protrusion/prolapse
(Hansen Type II/Chronic) DIV protrusion	Hansen Type II DIV herniation/protrusion/disease; DIV protrusion; Type II protrusion/ prolapse
Acute DIV extrusion with extensive epidural hemorrhage	Acute DIV extrusion with extensive epidural hemorrhage; Disc extrusion with extensive epidural hemorrhage (DEEH); Spinal epidural hematoma + DIV extrusion
Acute non-compressive nucleus pulposus extrusion (ANNPE)	ANNPE; Hansen Type III DDIV; High-velocity/low-volume extrusion; Traumatic DIV extrusion; DIV "explosion"; Traumatic DIV prolapse; "Missile discs"

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Current/proposed consensus	Terms reported in the literature
Hydrated nucleus pulposus extrusion (HNPE)	HNPE; Hydrated nucleus pulposus herniation; Acute compressive extrusion of hydrated nucleus; Partially degenerated disc extrusion; Intraspinal cyst; Canine disc cyst
Intradural/intramedullary extrusion (IIVDE)	IIVDE; Intradural/intramedullary herniation of DIV; Intramedullary extrusion of DIV
Traumatic extrusion of DIV	Traumatic extrusion of DIV; Traumatic prolapse of DIV
Fibrocartilaginous embolism myelopathy (FCEM)	FCEM; Fibrocartilaginous embolism (FCE); Ischemic myelopathy; Spinal cord infarction

Table 1: Current proposed consensus on DDIV

The trauma that occurs with hernias can be concussive or compressive. The injury to the spinal cord will depend on the speed of extrusion or the length of time the spinal cord was compressed. The spinal cord tolerates a certain amount of displacement before neurological signs begin to appear, depending on the type of hernia, the volume of disc within the canal, the clot formed, and the inflammation of the injury. When the ME no longer compensates for its displacement, local hypoxia and demyelination occur, leading to axonal degeneration, which can progress to myelomalacia, which is acute and progressive ischemic or hemorrhagic necrosis (tissue death) that occurs in the spinal cord (TOOMBS; WATERS, 2003; BRAUND, 1996).

Acute trauma is divided into two parts, where mechanical damage to the spinal cord occurs first, followed by inflammatory processes. In this initial phase, there may be bleeding due to laceration of the spinal cord, concussion, and cell death. The next ph s characterized by edema and a local inflammatory process that alters blood flow, disrupting electrolyte hemostasis, causing the release of free radicals and cell death. This process causes compression at the site of the injury, causing further damage to the spinal cord, lasting for months and possibly leading to scarring or worsening of the case (LAHUNTA, 2009). Chronic trauma acts predominantly on the white matter, with greater damage to the lateral and ventral regions, affecting the motor tracts because they are thicker, and loss of sensitivity ends up being rarer in chronic cases.

Clinical signs of lumbosacral syndrome.

The clinical signs of LDD are very broad, ranging from pain, claudication, monoparesis, paralysis, and quadriplegia, and can lead to death. The variation is due to the location of the herniation and the amount of material in the spinal canal (TOOBS; BAUER, 1998).

Lumbosacral DIV affects the DIVs in the L4-S3 region, with evident clinical signs in the pelvic limbs, urinary bladder, and tail, and with no obvious changes in the thoracic limbs, animals may present paresis or paralysis of the pelvic limbs, urinary bladder dysfunction, and paralysis or paresis of the anal sphincter, resulting in fecal incontinence (LECOUTEUR; CHILD, 1992).

Disc extrusion located between L4-L6 presents more specific signs of unilateral or bilateral loss of proprioception, paresis or plegia of the pelvic limbs, with absence or decrease of spinal reflexes. Neurogenic atrophy of the pelvic limbs also occurs, which may or may not be accompanied by deep pain, depending on the intensity of the extrusion and the amount of material in the spinal canal (LAHUNTA, 2009). When the extrusion is located between L7-S3, the cauda equina is affected, causing cauda equina syndrome, a sign present in other diseases besides DDIV.

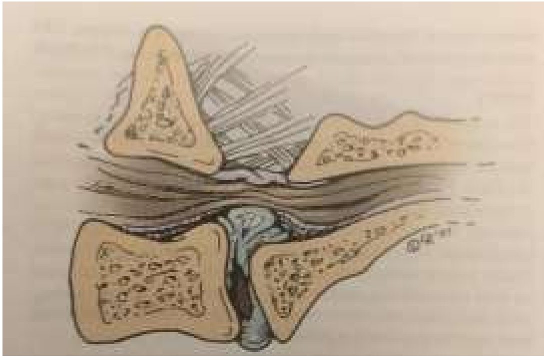

Figure 7: Schematic illustration of a disc protrusion between the L7-S1 DIVs

Source: Fossum. T.W, 2019.

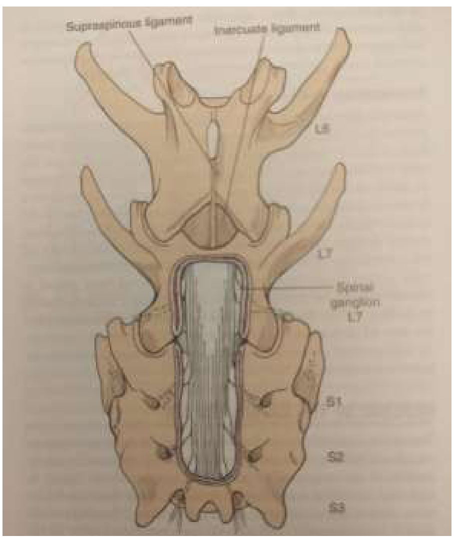

Figure 8: L7-S1, S2 laminectomy with exposure of the cauda equina.

Source: Fossum. T.W, 2019.

Diagnosis

A presumptive diagnosis can be obtained based solely on age, breed, history, and clinical signs, but a neurological examination is necessary to locate the lesion and thus confirm the diagnosis with complementary tests (plain and contrast imaging, cerebrospinal fluid examination, complete blood count, and biochemical tests), which will also rule out other diseases that affect the spinal cord (LECOUNTER; CHILD, 1992; LORENZ; KORNEGAY, 2006; De LAHUNTA; GLASS, 2009).

Clinical Examination

In the clinical examination, it is necessary to first obtain the clinical history, perform a physical and neurological examination, and possibly perform additional tests to accurately determine the affected region and subsequently define the appropriate treatment (de LAHUNTA; GLASS, 2009). In cases of DDIV, it is common to observe painful reflexes during palpation of the spine. It is important to begin the examination with less pressure, gradually increasing it throughout the examination. Palpation should be performed from caudal to cranial, on both sides of the spinous processes of the vertebrae (COATES, 2000).

Palpation of the limbs will indicate whether there is atrophy. When it is very intense and acute, we call it neurogenic, and it occurs when there is damage to the vertebral plexuses. When atrophy occurs slowly, it is due to disuse and happens when the injury is at the front of the limb that is not being used. The evaluation of postural reactions is done through conscious proprioception tests (figure 9), tactical positioning reaction, which is used in cats, and the hopping test (figure 10), which evaluates limb strength. The most important reflexes to be evaluated are the withdrawal of the extensor cruciatum, the extensor carpi radialis, the patellar (Figure 11), perineal, and cutaneous reflexes of the body (should be seen on both sides of the spine, making it possible to assess whether the extrusion is lateralized or not) (LORENZ; KORNEGAY, 2006; COSTA, 2001; PARENT, 2010). To assess conscious pain, also called deep pain, the fingers are pinched (figure 12) and the animal must try to prevent the pain stimulus from persisting, either by vocaliz r pulling the paw. It only occurs in severe cases of ME compression and may be present at the first moment of injury and disappear if it is not treated. (From LAHUNTA; GLASS, 2009; LORENZ; COATES; KENT, 2011)

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Figure 9: Conscious proprioception test.

Source: Fossum. T.W, 2019.

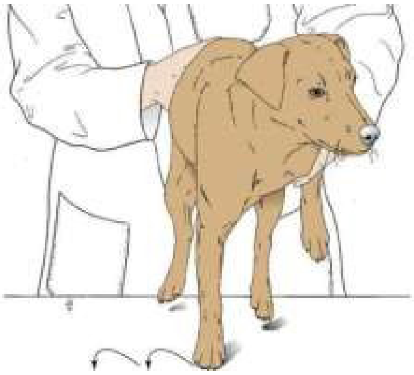

Figure 10: Hopping test

Source: Fossum, 2019.

Figure 11: Patellar reflex technique

Source: Fossum, 2019.

Figure 12: Patellar reflex technique

Source: Fossum, 2019.

Diagnostic Imaging

An accurate diagnosis is only possible with the use of diagnostic imaging tests, such as X-rays, myelography, computed tomography, and magnetic resonance imaging.

A simple radiograph is the best way to rule out other changes such as fractures, dislocations, and congenital abnormalities (LECOUTEUR; CHILD, 1992). Two positions should be taken, lateral and ventrodorsal. Preferably, the animal should be anesthetized to avoid unwanted movements and prioritize successful positioning, often using supports and wedges to avoid sudden movements that could aggravate the injury due to the risk of increased extruded material (LECOUTEUR; CHILD, 1997).

The signs of DDIV found are: decreased intervertebral space (most useful sign on plain radiography), narrowing of the facet joints, opacity of the intervertebral foramen, mineralization of material within the vertebral canal, and the existence of the vacuum phenomenon (very rare). (BRISSON, 2010; COATES, 2000; LECOUTEUR; CHILD, 1997).

Myelography (contrast radiography) can confirm spinal cord compression, assisting in the strategic decision to perform surgery. The contrast is injected into the subarachnoid space, exposing two radiopaque lines within the spinal cord (Figure 13) (TOOMBS; WATERS, 2003; WHELLER; SHARP, 1999). The contrast, which may be iopamidol or iohexol, should be injected into the region called the cerebellospinal cistern or cistern magna, or into the lumbar region between the L4-L5 or L5-L6 vertebrae, depending on the location of the lesion, as determined by clinical examination.

Figure 13: Myelography of a disc extrusion at C2-C3

Source: Fossum, 2019.

Before injecting the contrast medium, cerebrospinal fluid must be removed to prevent an increase in pressure (BRISSON, 2010; TOOMBS; BAUER, 1998; WHELLER; SHARP, 1999). X-rays should be taken in the ventrodorsal, lateral, and oblique positions at the site of the suspected hernia. If the contrast column appears attenuated, diminished, or deviated, it is considered a diagnosis of hernia or edema at the site (BRISSON, 2010). In rare cases, myelography may appear without obvious changes when there is extrusion of disc laterally or into the foramen, but attention should be paid to increased density within this foramen (LECOUTEUR; GRANDY, 2004).

Of all imaging methods, magnetic resonance imaging is the best method for early detection of disc degeneration. It is also the best method for observing and evaluating the ME, DI, and adjacent structures, but it has poor bone resolution. MRI enhances the contrast of the soft tissues of the ME, allowing for direct visualization (FOSSUM, 2019; BRISSON, 2010). In a study comparing pyelography with MRI, conducted with 24 small breeds suffering from DDIV in the thoracolumbar region, MRI proved to be more accurate in identifying the location of the lesion, making MRI a good tool for surgical guidance and decision-making, providing a good idea of the size and location of the laminectomy. however, a slightly larger surgical window is recommended to ensure that all extruded material is removed from the canal (BRISSON, 2010).

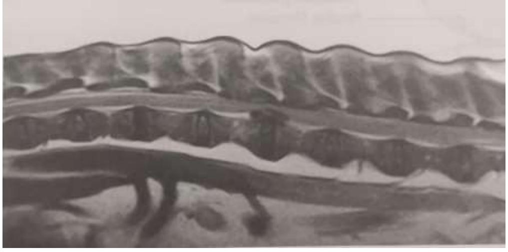

Figure 14: Magnetic resonance image showing a disc rupture at L2L3

Source: Fossum, 2019.

An increase in the density of the IM image is associated with a poor prognosis for recovery in paraplegic dogs, since of the animals that lost deep pain, 31% achieved satisfactory recovery. Conversely, a low-intensity IM image in paraplegics is associated with successful recovery (BRISSON, 2010). MRI can differentiate between hemorrhoids in the vertebral sinus, extensive extradural compression, or ME edema, identifying variations in image intensity and differentiating between disc material and hemorrhage, something that is not possible with myelography. Despite all the benefits of MRI, it is still considered an expensive exam, making it less accessible in veterinary medicine (TOOMBS; BAUER, 1998).

Figure 14: Magnetic resonance image showing an extrusion of the ID in the cervical region

Source: Fossum, 2019.

Treatment

IVDD can be treated surgically or clinically. The choice of treatment will be made by assessing the patient's neurological status and will depend on the veterinarian's experience and the owner's financial condition and interest (LECOUTER; CHILD, 1997; BRAUD, 1996).

Clinical treatment

For clinical (conservative) treatment to be recommended, the animal must show clinical signs indicating DDIV, but the symptoms should be limited to pain or mild neurological impairment, and the animal should still be able to walk, albeit unsteadily (LECOUTEUR; GRANDY, 2004). This conservative treatment is also suggested for paralyzed animals with no conscious pain for more than 48 hours, even though it is rare for these animals to respond to this treatment after that time (BRAUD, 1996).

Clinical treatment consists of physical restriction (confinement) for two to six weeks, combined with physical therapy and the use of anti-inflammatory muscle relaxants to control pain and hyperesthesia (hypersensitivity). The most important part of this treatment is to confine the animal to a small environment, facilitating recovery of the injury site, reducing inflammation, and stabilizing the disc through fibrosis. Rest will prevent more material from entering the spinal canal and worsening the clinical condition. If the animal does not improve, a reassessment should be made to consider surgical treatment. (TOOMBS; WATERS, 2003; LECOUTEUR; GRANDY, 2004).

In clinical treatment, certain medications are used to control edema, such as corticosteroids in animals with a history of acute symptoms. Corticosteroids are used to reduce the pain caused by compression of the nerve roots, which causes inflammation and ischemia at the site. (LECOUTEUR; GRANDY, 2004). Muscle relaxants are used to treat muscle spasms (BRAUND, 1996).

For cases of lumbar DDIV, clinical treatment must always initially include the use of nonsteroidal anti-inflammatory drugs, analgesics, and changes or reductions in exercise and weight loss, similar to the treatment of osteoarthritis (MEIJ; BERGKNUT, 2010; WHEELER; SHARP, 2005). Systemic corticosteroids have caused controversy because NSAIDs promote analgesia similar to corticosteroids but produce fewer side effects. Epidural corticosteroid injections into the affected area of the waist have recently been reported as a treatment that improved 79% of patients clinically. The injection site may have a minor adverse effect. The exercise pattern for these patients is to take regular short walks in order to maintain muscle tone and quality. Clinical treatment cannot cure the hernia, but it can control the pain (MEIJ; BERGKNUT, 2010).

Surgical treatment

Surgery is always indicated for animals with chronic episodes of DDIV, those that do not respond to clinical treatment, or when they have progressive, severe, or acute lesions (; ALVARENGA, 1999). Most cases that do not respond to conservative clinical treatment are due to large amounts of extruded material compressing the ME. These animals definitely require surgical treatment for spinal cord decompression. Contrast radiographic or

MRI examinations should be performed and interpreted correctly, accurately identifying the location of the affected disc to avoid surgical error. Most patients undergoing surgery show neurological improvement, unless there has been extensive damage to the spinal cord (SHARP; WHEELER, 2005).

Patients undergoing spinal cord decompression surgery have a better chance of faster and more complete recovery of motor and sensory function than animals treated clinically (COSTA, 2006 apud SANTANA, 2009). Braund (1998) does not recommend surgery for animals that have lost deep pain sensation for more than 48 hours. However, Tudury (2006) apud Santana (2009) disagrees with this statement, suggesting that according to recent studies, conscious analgesia for more than 48 hours should not be indicative of not performing decompressive surgery.

The most commonly used surgical decompression techniques in cases of DDIV are hemilaminectomy, minihemilaminectomy (pediculectomy), and laminectomy, which aim to decompress the spinal cord through less invasive approaches and removing less vertebral bone. These procedures are supposedly faster, provide lateral and ventral access to the spinal canal to remove the extruded material, cause less tissue trauma and less vertebral instability, and lead to faster postoperative recovery (BRISSON, 2010).

Hemilaminectomy

This technique can be used very well to decompress the ME, providing a good ventral and lateral view of the spinal canal, assisting in the removal of extruded material. Hemilaminectomy is suitable for animals with lateral lesions or material in the intervertebral foramen. The technique is based on the removal of the lateral and dorsolateral laminae of the pedicles and facet joints (Figure 15) (FOSSUM, 2019). When performing the surgery, you must carefully dissect and fully understand the nerves and blood vessels of the foramen to avoid bleeding and nerve damage ( ). When the ME is exposed, to avoid manipulation of the spinal cord, the extruded material is removed precisely and delicately (TOOMBS; WATERS, 2003).

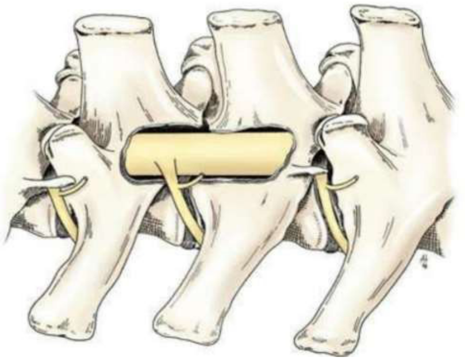

Figure 15: Representation of a hemilaminectomy

Source: Fossum, 2019.

The dorsal approach to the spine to perform a hemilaminectomy allows contralateral access without moving the patient when it is necessary to perform the technique on both sides. As this procedure requires removal of the facet joints, it can cause some vertebral instability (BRISSON, 2010).

It is stated that unilateral hemilaminectomy can be performed along up to three consecutive vertebrae without producing clinically significant vertebral instability. Delayed recovery and deterioration of clinical signs noted one to ten days after surgery was reported in 5.8% of cases of patients who underwent hemilaminectomy, but it was associated with residual compression caused by an incorrect surgical approach leaving material in the canal or by recurrence of extrusion (BRISSON, 2010).

Minihemilaminectomy or pediculectomy. The technique consists of lateral removal of the spinal bone between the vertebral body and its articular process, preserving the articular processes of the two vertebrae adjacent to the injured intervertebral disc (Figure 16). It is also known as minihemilaminectomy (BRISSON, 2010). The window opened in minihemilaminectomy is adequate for viewing the vertebral canal ventrolaterally and provides good access for removing lateralized material or material that is ventral to the ME, in addition to limiting its manipulation. This limited manipulation of the spinal cord avoids complications seen in other techniques (BRISSON, 2010; LUBBE; KIRBERGER; VERSTRAETE, 1994). Preservation of the facet joints minimizes the risk of instability compared to hemilaminectomy. The access provided allows for disc fenestration (BRISSON, 2010). This technique is also simple and quick to perform, as there is not as much bone removal and it provides a direct ventral view of the ME (LUBBE; KIRBERGER; VERSTRAETE, 1994).

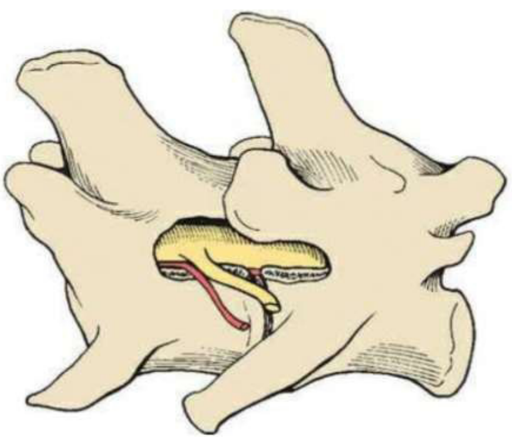

Figure 15: Representation of a minihemilaminectomy or pedicurectomy

Source: Fossum, 2019.

As in hemilaminectomy, the window made for pedicurectomy is close to the venous sinus and structures adjacent to the foramen, requiring greater care to avoid bleeding and damage to the nerve root. Pedicurectomy is easily converted to hemilaminectomy when necessary and can even be extended to adjacent vertebrae, as partial pedicurectomy offers a very small window for decompression of extensive lesions due to the difficulty in removing all the material and has the disadvantage of requiring blind probing to remove the material, which can cause damage to the spinal cord or bleeding (BRISSON, 2010).

Prognosis

The prognosis is directly related to neurological signs, anatomical location, and symptoms to treatment, but conscious pain is always the main factor in the animal's recovery. If this sign is present, the prognosis is likely favorable. In animals that had acute IVDD and there was a delay in surgical treatment, recovery is usually slower and neurological sequelae may remain (TOOMBS; WATERS, 2003; LECOUTEUR; GRANDI, 2004; PELLEGRINO; SURANTI; GARIBALDI, 2003).

CLINICAL SIGNS	*PROGNOSIS*
Pain only.	Very good.
Pain, mild ataxia, loss of conscious proprioception.	Good.
Paresis without loss of superficial pain sensitivity.	Reserved to favorable.
Paralysis, bladder control, superficial pain sensitivity present.	Reserved.
Paralysis, absence of bladder control, absence of superficial pain sensitivity.	Reserved to severe.
Paralysis, absence of deep pain sensitivity.	Severe

Table 2: Prognoses associated with clinical signs

Source: Pellegrino, F.; Suraniti, A.; Garibaldi, L., 2003.

The neurological condition improves more quickly in dogs treated surgically compared to dogs that are treated clinically. In one study, small breed dogs recovered more quickly than large breed dogs (4.5 versus seven days), while another study showed that small dogs recovered on average five times faster than large dogs (BRISSON, 2010).

Studies show that patients undergoing dorsal laminectomy with varying degrees of neurological symptoms had a recovery rate of 67% in the first two weeks after surgery and achieved 100% recovery in the period from five to 44 months after surgery (BRISSON, 2010).

Patients who resume walking within 96 hours after surgery are six times more likely to achieve full recovery from neurological dysfunction than those who are not yet walking during this postoperative period. The return of clinical signs has been reported in 0 to 17% of cases after surgical decompression (BRISSON, 2010).

In Thoracolumbar Syndrome, the reported recovery rate has been between 86 and 96% in chondrodystrophic or small breed dogs that still have deep pain before decompressive surgery. The recovery rate for non-chondrodystrophic and large breed animals with Hansen type I thoracolumbar hernia is between 78 and 85%, while those with type II are between 22 and 52% (BRISSON, 2010).

CASE REPORT

A 3-year-old, 11 kg, unneutered male French Bulldog was treated at a private clinic located in the Vargem Grande neighborhood of Rio de Janeiro. During the consultation, the owner reported that the animal had abruptly stopped walking on its hind limbs, denying the possibility of trauma or previous intense physical exercise, and began to show severe pain in the lumbosacral region, unable to support itself on its hind limbs. The patient presented normuria, normochesia, normophagia, and normodipsia.

On clinical examination, the animal was alert, hydrated, with pink mucous membranes, body condition score 3, heart rate 160 bpm, respiratory rate 60 bpm, capillary perfusion time 2, rectal temperature 38.1°C, no pain on abdominal palpation, and no obvious changes in lymph nodes. Palpation revealed severe and acute muscle atrophy of the hind limbs, characteristic of neurogenic muscle atrophy.

During the neurological examination, the patient was seated, supported by the forelimbs. In the conscious proprioception test, the result was absent in the pelvic limbs, and there was no superficial or deep pain in both pelvic limbs. An MRI was then requested for better localization and possible diagnosis of disc extrusion.

The MRI revealed the presence of hypotensive material in relation to the spinal cord observed in T2 and hypotensive in T1 on the floor of the vertebral canal, lateralized to the left, above the L2-3 disc space and displaced cranially over L2, promoting moderate ventrolateral compression-lateral compression on the spinal cord, accompanied by medullary hypersignal in T2 between T12 and L4 with erasure of the liquor column in myelographic sequence between T9 and L5, in addition to extensive perilesional edema (figure 16). Spinal decompression was then indicated using the mini hemilaminectomy technique with removal of the extruded material.

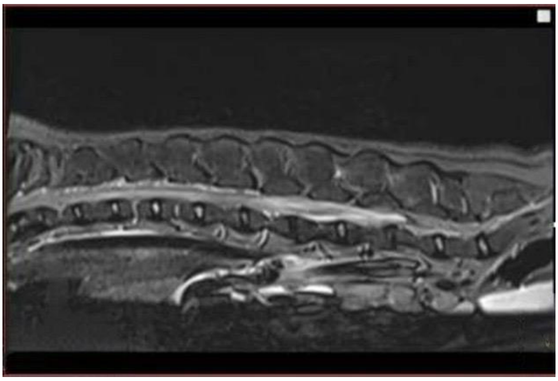

Figure 16: Magnetic resonance image showing disc extrusion at L4-L5

Source: Personal archive 2025

After preparation and antisepsis of the animal and administration of morphine 0.3/kg as a pre-anesthetic medication, anesthetic induction was performed with propofol 3.0mg/kg and inhalation anesthesia maintenance with isoflurane.

For the surgery, the animal was placed in the lateral decubitus position, slightly tilted to the right, with the neck and head positioned cranially, and the surgical table rails positioned at an angle of approximately 45 degrees (Figure 17).

Figure 17: Animal positioned for surgery.

Source: Personal archive 2025

An incision was made slightly lateralized to the left in the region between L2 and L3, and manual blunt dissection of the thoracolumbar fascia was performed until the vertebrae were located. The musculature was retracted and spread apart with the aid of two *Weitlaner* retractors on both sides of the incision, keeping it open for better visualization.

With a good lateral view of the L2 and L3 vertebrae, an electric drill was used to make a slit approximately 1/3 the size of the vertebra, and enlarged with Kerrison forceps, laterally removing the lamina from the lateral wall of the spinal bone between the vertebral body and its articular process, preserving the articular processes of the two vertebrae adjacent to the injured intervertebral disc. During the creation of the slit, simultaneous irrigation with cooled saline solution was performed to prevent heating of the vertebral bone and to remove debris until the inner cortex was visible. Finally, the extruded material was removed from the spinal canal with a dental curette.

Figure 18: Slit being irrigated with saline solution,

Source: Personal archive 2025

Figure 19: *Weitlaner* spreaders, rebounding the muscles.

Source: Personal archive 2025

Figure 20: Removal of extruded material.

Source: Personal archive 2025

After removing the material compressing the spinal cord (Figure 21), the site was thoroughly irrigated, and then the thoracolumbar fascia and musculature were synthesized with a simple continuous suture using polyglactin 910 2-0, subcutaneous simple continuous suture using polyglactin 910 2-0, and skin suture using *Sutan* with nylon 3-0.

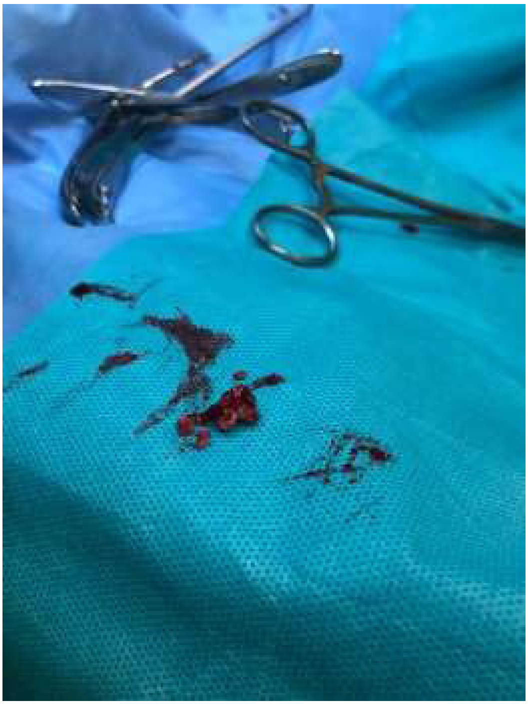

Figure 21: Extruded material removed

Source: Personal archive 2025

There was no significant bleeding from the spinal canal, musculature, and/or subcutaneous tissue. The surgical procedure lasted approximately one hour and thirty minutes. With the surgery, it was possible to confirm the diagnosis of type I DDIV after visualization and removal of the extruded disc material that was compressing the spinal cord.

For the postoperative period, the following were administered as described in Table 2

Medication	**Dosage**	**Route**	**Time**
Gabapentin	5 mg/kg	Oral	TID
Dipyrone	25 mg/kg	subcutaneous	TID
Tramadol	4 mg/kg	subcutaneous	TID
Meloxican	0.05 mg/kg	subcutaneous	SID
Omeprazole	1 mg/kg	Intravenous	SID
Ceftriaxone	30 mg/kg	Intravenous	SID

Table 3: Postoperative medications.

Source: Personal archive

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In the immediate postoperative period, the animal already showed improvement, demonstrating good recovery of sensitivity to superficial pain in the right hind limb, proving to be very responsive to stimuli in the same limb. In the left hind limb, which was the most affected side, the animal was less responsive but showed some superficial pain, indicating almost immediate progress after surgery. The animal was referred for acupuncture therapy, where it only attended the first session.

DISCUSSION

Brisson (2010) stated that the average age of diagnosis of IVD is around six to eight years of age, which is not consistent with the present report since the animal is three years old. This data probably characterizes the case as chondroid metaplasia in a chondrodystrophic animal, in agreement with Brisson (2010), who stated that this process can begin before the first two years of age in chondrodystrophic animals. Around 75 to 90% of these animals have most of their NP transformed into hyaline cartilage by their first year of age, while non-phic chondrodystrophic animals remain with high levels of proteoglycans until old age, thus suggesting a genetic predisposition.

According to Lahunta (2009), disc extrusion located between L2L3 and L3-L4 shows more specific signs of unilateral or bilateral loss of proprioception, paresis or plegia of the pelvic limbs, with absence or decrease in spinal reflexes, which is evident in the case presented. There is also neurogenic atrophy of the pelvic limbs, which was also mentioned in the case, with or without deep pain, depending on the intensity of the extrusion and the amount of material in the spinal canal.

In the reported case, magnetic resonance imaging was used to diagnose DDIV and determine the location of the lesion with a certain degree of accuracy and, consequently, the location of the surgical incision, in agreement with Fossum (2019), who states that magnetic resonance imaging is the best method for early detection of disc degeneration and is the best method for observing and evaluating the ME, DI, and adjacent structures. Even though it has poor bone resolution, MRI enhances the contrast of the soft tissues of the ME, allowing for direct visualization.

In the case reported, palpation of the pelvic limbs revealed severe and acute muscle atrophy, indicative of neurogenic muscle atrophy that occurs when there is damage to the vertebral plexuses, in agreement with the statements of Lorenz and Kornegay (2006). This differs from slow muscle atrophy caused by disuse.

According to Lorenz and Kornegay (2006), in the physical evaluation, the most important reflexes to be assessed are the crossed extensor, radial carpal extensor, patellar, perineal, and cutaneous body reflexes, which were the reflexes assessed in the reported case, proving to be extremely important to be evaluated for the conclusion of the presumptive diagnosis before surgery is performed.

The extruded intervertebral disc in the case report described here was located between the L2 and L3 vertebrae. This information is consistent with Lahunta (2009), who reported that disc extrusion located between L2-L3 presents more specific signs of unilateral or bilateral loss of proprioception, paresis or plegia of the pelvic limbs, with absence or decrease in spinal reflexes. Neurogenic atrophy of the pelvic limbs also occurs, which may or may not be accompanied by deep pain, depending on the intensity of the extrusion and the amount of material in the spinal canal. Several of the pieces of information cited were present in the reported case.

FINAL CONSIDERATIONS

As observed in the reported case, magnetic resonance imaging proved to be the best form of diagnostic imaging for the best formulation of a presumptive diagnosis for DDIV, indicating with some precision the vertebrae and the side that should be focused on by the surgeon at the time of surgery.

It was also possible to conclude that it is indisputable that the best form of diagnosis and treatment for a better prognosis in cases of DDIV is surgical treatment, which has been shown to clearly increase the probability of improvement in the clinical case of animals, even in cases where the prognosis is not so favorable. It also proves that adequate postoperative care, with therapies such as acupuncture, can increase the probability of evident improvement in cases of DDIV.

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