# CLINICAL, NEUROLOGICAL AND MAGNETIC RESONANCE ASPECTS IN MYELOMALACIA IN DOGS - 10 CASES

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#### Abstract

The diagnosis and understanding of myelomalacia in dogs rely heavily on clinical, neurological, and magnetic resonance imaging (MRI) aspects. A study conducted at the Faculty of Veterinary Medicine Bucharest reviewed medical records from 10 cases, selected based on their clinical history, neurological evaluation, and MRI findings. The clinical and neurological signs varied depending on the location and severity of the spinal cord injury, with common symptoms including limb weakness or paralysis, coordination issues, walking difficulties, pain, and changes in posture. MRI plays a vital role in diagnosing myelomalacia by providing detailed images of the spinal cord, offering crucial insights into the condition. The cases were classified into three types based on their cause: traumatic (6 cases), degenerative (3 cases), and vascular (1 case), all affecting the thoracolumbar spinal cord. In conclusion, the combination of clinical assessment, neurological examination, and MRI is essential for accurately diagnosing myelomalacia in dogs and determining appropriate treatment options.

Key words: dog, imaging diagnosis, MRI, myelomalacia.

## INTRODUCTION

In veterinary medicine, the term myelomalacia is used in the case of hemorrhagic infarction of the spinal cord that can occur following an acute (traumatic), degenerative (progressive myelomalacia), or vascular injury (Platt and Garosi, 2012).

The leading cause of traumatic myelomalacia is intervertebral disc extrusion, where a significant amount of disc material disperses along the vertebral canal, creating intense pressure on the spinal cord. Over time, this severe compression results in degeneration due to the death of nerve cells (De Risio et al., 2009; Cordle et al., 2023).

In degenerative myelomalacia, the death of the tissue at the level of the spinal cord occurs progressively over time and can be ascending or descending at the spinal cord. The exact cause is not fully understood, but it is thought to be multifactorial. It is often associated with abnormalities of the spine, including vertebral malformations, instability, or compression of the spinal cord. Genetic, nutritional, and biomechanical factors may also play an important role (Schweizer-Gorgas, 2018; Lin et al., 2023).

Vascular myelomalacia can occur due to a fibrocartilaginous embolus through the formation of blood clots that reduce blood flow to the spinal cord. Other vascular accidents or conditions that compromise blood flow to the spinal cord are implicated, such as vasculitis or arterial dissection (Lu et al., 2002).

Clinical signs of myelomalacia can include loss of proprioception in the pelvic limbs, tail, and anus, flaccid paraplegia, absence of deep pain perception, weakened abdominal muscles, and a depressed mental state (Fingeroth et al., 2015). While the clinical signs of myelomalacia are typically seen within the first 24 hours after paraplegia begins, they may sometimes only become apparent during the postoperative period or even several days after paraplegia starts (Platt and Garosi 2012; Schweizer-Gorgas, 2018).

Although myelomalacia itself may not be a common condition that can lead to it, such as intervertebral disc disease (IVDD) or traumatic injuries, are seen relatively frequent in veterinary practice, particularly in certain breeds predisposed to spinal problems such as dogs from the Dachshund or German Shepherd breed (Olby, 2010).

Due to its severity and potential for irreversible damage to the spinal cord, myelomalacia requires prompt diagnosis and appropriate management to minimize neurological deficits and improve the dog's quality of life. The aim of this study was to describe the clinical, neurological, and imaging characteristics, using magnetic resonance, of dogs affected by myelomalacia with different causes.

# MATERIALS AND METHODS

Medical records during the period 2018-2024 from the University Emergency Veterinary Hospital "Prof. Dr. Alin Bîrtoiu", were evaluated from clinical, neurological, and imaging points of view.

The criteria for inclusion in the study were based on the anamnestic data provided by the owners, followed by the clinical and neurological data as well as the imaging data by a specific protocol (Fernoaga et al., 2020; Rebecca and Fernoaga, 2021).

For each case, age, breed, sex, and body weight were documented, along with the time elapsed between the onset of clinical signs reported by the owner and the imaging examination. Additionally, it was noted whether decompressive surgery was performed and the timing of the onset of clinical signs of progressive myelomalacia.

As part of the inclusion criteria, all dogs underwent general anesthesia to be evaluated by magnetic resonance imaging (MRI). This study utilized a specialized veterinary MRI device, the VET MR GRADE with a 0.3 Tesla power (ESAOTE, Italy), featuring a permanent magnet and region-specific coils. The imaging protocols included T1 Spin Echo (SE) and T2 Fast Spin Echo (FSE) sequences in two planes (sagittal and transverse), with a slice thickness of 2-3 mm. Post-contrast images were obtained in the T1 sequence following intravenous administration of a contrast agent (Clariscan, 0.2 ml/kg). During the MRI exam, the animals were positioned in left lateral recumbency (Neagu et al., 2018).

The following protocol was used: premedication was administered with Butorphanol at 0.2 mg/kg IV, followed by induction with Propofol at 3-5 mg/kg IV. After intubation, anesthesia was maintained using Isoflurane and 100% oxygen. Spontaneous or intermittent positive-pressure ventilation (IPPV) was provided using a volume-cycled ventilator delivering 12-15 breaths per minute, targeting an end-tidal CO<sub>2</sub> of 35-45 mmHg. Oxygen flow initially started at 2 L/min, with the vaporizer adjusted to reach an end-tidal concentration of 2.0% isoflurane within 10 minutes of induction. Once the target concentration was achieved, oxygen flow was reduced to (500 + 10/kg) L/min, and the isoflurane concentration was consistently maintained at 1.5 vol.% for all cases (Tudor et al. 2019; Pavel et al., 2021).

# **RESULTS AND DISCUSSIONS**

After analyzing the medical files, 10 cases with signs compatible with myelomalacia were identified, represented by: French Bulldog (3 cases), Pug (2 cases), Pomeranian (1 case), Chow-chow (1 case), Bichon (1 case), English Bulldog (1 case) and Golden Retriever (1 case). Of whom 4 females and 6 males, aged between 4 and 9 years. They were divided into three categories: post-traumatic mvelomalacia following intervertebral disc extrusion (6 cases), degenerative myelomalacia caused by changes in vertebral alignment (3 cases), and vascular mvelomalacia caused bv а fibrocartilaginous embolism (1 case). The neurological examination established the localization of the disease at the level of the thoracolumbar segment of the spinal cord.

In the case of myelomalacia produced by disc extrusion, the neurological signs were correlated with the degree of compression of the spinal cord and nerve roots, adjacent to the affected area. The evolution of the injuries was superacute, with the immediate onset of nerve deficits, respectively in the first 24-48 hours after the incident.

Thus, in the 6 cases, the pain was observed, both when palpating the spine on the thoracolumbar region, and when initiating movements (different degrees of pain). The neurological evaluation revealed the following abnormalities: kyphosis and scoliosis (as changes in posture) (4/6); proprioceptive deficits of varying degrees on the hind limbs (6/6); proprioceptive ataxia and walking paraparesis (6/6); normal spinal reflexes, but in 2 cases flexion was delayed; panniculus was reduced (6/6), caudal to the lesion. Neurological signs were symmetrical or lateralized.

For degenerative myelomalacia, the neurological signs were present or blurred. The evaluated patients presented: kyphosis and scoliosis (3/3); proprioception deficits of varying degrees on the hind limbs (3/3); proprioceptive ataxia (3/3), paresis (2/3), plegia (1/3); normal spinal reflexes, but with incomplete (2/3) or absent (1/3) flexion. In these patients, no pain was detected when palpating the spine or while walking.

The patient with fibrocartilaginous embolism (vascular ischemia) presented intense pain at the time of injury, triggered by play activity/jumping in the yard. The neurological deficits appeared acutely and evolved progressively in the first 24 hours. Afterwards, the pain was no longer present. Neurological signs were symmetrical or lateralized to one side. Proprioceptive deficits of various degrees, proprioceptive ataxia and walking paraparesis, normal spinal reflexes, and reduced panniculus, behind the lesions, were observed; deep pain perception was reduced. This patient did not present pain when palpating the spine and while walking.

Regardless of the cause that determined the myelomalacia (traumatic, degenerative, and vascular), in patients with neuro-localization on the T3-L3 vertebral segment, the urinary bladder was spastic (9/10), and in those with localization on the L4-S3 segment, the urinary bladder was flaccid (1/10).

After the neurological examination and neurolocalization, the MRI examination was used to confirm the lesion.

In the case of traumatic myelomalacia, the aim was to identify and locate the intervertebral disc extrusion and highlight the imaging characteristics. The MRI examination localized the disc extrusion in the T11-L5 segment (Figure 1). Imaging signs included hyperintensity on T2 sequences and isointensity on T1 sequences, without contrast enhancement at the level of the spinal cord. The extramedullary disc extrusion was visible as T2 hypointensity located dorsally in the vertebral

canal, compressing the spinal cord. The intramedullary change was identified around the extruded disc fragment.



Figure 1. Sagittal planes of the thoracolumbar spine, showing a T2 hyperintensity of the spinal cord ascending and descending from disc extrusion L4-L5 (A), respectively L2-L3 (B)

In 2 of the 6 cases, the extent of the spinal cord injury was both cranial and caudal to the extrusion site. The most likely cause of this effect is the long time elapsed between the onset of the event and the MRI scan. Another cause may be the delay of the surgical intervention (Castel et al., 2017; Castel et al., 2019), mainly due to the hesitation of the owners.

Disc extrusion in the lumbar area can be a risk factor for the occurrence of myelomalacia (Castel et al., 2017; Castel et al., 2019). Previous reports show that disc extrusion, complicated with myelomalacia, is more frequently encountered at T12-T13, T13-L1, and L1-L2 (De Risio et al., 2009). In the present study, disc extrusion was detected on the T11-L5 segment, which led to the extension of the spinal cord injury.

In 3 cases, the presence of degenerative myelomalacia was established, based on the anamnestic signs corroborated with the neurological and imaging ones (Figures 2 and 3).

The evolution of the patient over time was also followed. The patients presented changes in vertebral alignment, as a result of congenital vertebral anomalies, which led to the alteration of the structure of the spinal cord, represented by T2 hyperintensity in the adjacent area. A recent study carried out on French Bulldog dogs in the thoracolumbar segment, showed that changes in the spine alignment, caused by the presence of congenital vertebral anomalies, lead to swelling of the spinal cord (Fernoaga et al., 2021).



Figure 2. Sagittal planes of the thoracolumbar spine (A and B), showing a T2 hyperintensity of the spinal cord with abnormal vertebral alignment



Figure 3. Transversal planes of the thoracolumbar spine (A and B), showing a T2 hyperintensity of the spinal cord

In one case, the presence of changes in the spinal cord, represented by hyperintensity in T2, without the presence of disc extrusion or congenital vertebral anomalies, was found, which led to the suspicion of vascular myelomalacia (Figure 4).

The main cause of vascular myelomalacia is medullary intraparenchymal arterial obstruction, following the embolization of fibrocartilaginous material, which leads to the appearance of local ischemic lesions (Schweizer-Gorgas, 2018). This material is histologically and histochemically similar to the nucleus pulpous, but its origin and appearance mode in the bloodstream are not yet elucidated (De Risio, 2015). Other causes include thrombi. parasites, bacteria, fat embolism, or neoplastic embolism (Schwezer-Gorgas, 2018).



Figure 4. Sagittal plane (A) and transversal planes (B and C) showing reduced T2 hyperintensity on the spinal cord - most probably asses as a vascular ischemia (fibrocartilaginous embolus)

It was noted that the prognosis for dogs in the study with myelomalacia varied significantly based on the underlying cause and the extent of spinal cord damage. In some instances, with timely and proper treatment, dogs were able to recover partial or full mobility within 2 to 6 weeks after the onset of the condition (Olby, 2010; Fingeroth et al., 2015).

## CONCLUSIONS

The MRI findings in collaboration with clinical sign and neurological evaluation are important methods in the diagnosis of progressive myelomalacia. The imaging finding of the appearance as a hyperintense signal in the T2 sequences of the spinal cord was associated with progressive myelomalacia.

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